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**Aline Bütikofer**
*Norwegian School of Economics*

**Kjell G. Salvanes**
*Norwegian School of Economics and IZA*

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**IZA – Institute of Labor Economics**

Schaumburg-Lippe-Straße 5–9
53113 Bonn, Germany

Phone: +49-228-3894-0
Email: publications@iza.org

www.iza.org

# ABSTRACT

## Disease Control and Inequality Reduction: Evidence from a Tuberculosis Testing and Vaccination Campaign*

This paper examines the economic impact of a tuberculosis control program launched in Norway in 1948. In the 1940s, Norway had one of the highest tuberculosis infection rates in Europe, affecting about 85 percent of the inhabitants. To lower the disease burden, the Norwegian government launched a large-scale tuberculosis testing and vaccination campaign that substantially reduced tuberculosis infection rates among children. We find that cohorts in school during and after the campaign in municipalities with high tuberculosis prevalence gained more in terms of education, earnings, longevity, and height following this public health intervention. Furthermore, the gains from the disease control program are not limited to the initially treated cohorts but also affect their children. The results also suggest that individuals from a low socioeconomic background benefited more from the intervention and we present new evidence that a narrowing of the gap in childhood health can lead to a reduction in socioeconomic inequalities in adulthood.

**JEL Classification:** I14, I18, I24
**Keywords:** health programs, tuberculosis, education, inequality

**Corresponding author:**
Kjell G. Salvanes
Department of Economics
Norwegian School of Economics
Helleveien 30,
N-5035 Bergen-Sandviken
Norway
E-mail: kjell.salvanes@nhh.no

* The authors thank the Norwegian Research Council for its financial support. The authors gratefully acknowledge comments by Jérôme Adda, Prashant Bharadwaj, Kai Liu, Janet Currie, Jonathan Guryan, Douglas Miller, and seminar participants at RAND Corporation, the Norwegian School of Economics, Dresden University of Technology, the Annual Congress of the European Economic Association in Mannheim, the Nordic Labor Institute, the Bergen-Stavanger Workshop, and the Austin-Bergen-London Workshop in Labor Economics. Arn-Tore Haugsdal and Erling Risa provided excellent research assistance.

# 1 Introduction

The importance of infectious disease for public health and economic development has been the object of increasing attention among both policy makers and researchers over the last several decades. After World War II, a number of large-scale disease eradication programs and mass childhood vaccination campaigns took place in order to reduce the disease burden. The long-term gains of disease eradication in health and human capital outcomes are potentially very high, as childhood health conditions explain a substantial amount of the variation in the economic capabilities of adults (see Currie and Almond, 2011; Case and Paxson, 2009; Cunha and Heckman, 2007, for an overview). In addition, childhood vaccination programs are shown to significantly increase cognitive test scores for immunized children (Bloom, Canning, and Shenoy, 2012). To date, the literature has largely focused on the long-term consequences of in utero and neonatal exposure to infectious diseases such as influenza or malaria during pregnancy (Almond, 2006; Kelly, 2011; Barreca, 2010), or how policy-induced variations in maternal and early-life health affect individuals in the long run (see, e.g., Bharadwaj, Løken, and Neilson, 2011; Bhalotra and Venkataramani, 2012). In contrast, rather less work has been done to analyze how late childhood or adolescent health affects long-term economic outcomes. A likely reason for this research gap is the difficulty in identifying exogenous changes in the state of public health that specifically targets the health of children after the postneonatal period. An exception is Bleakley (2007), who analyzes the eradication of hookworm in the southern United States. He finds a substantial effect of early-life hookworm exposure on school enrollment and attendance, as well as on literacy and long-term outcomes such as earnings and completed years of schooling.[1]

In this paper, we asses the short- and long-term consequences of a disease-free environment by examining the impact of a tuberculosis testing and vaccination program that commenced in Norway in the late 1940s.[2] At the time, schoolchildren were the most vulnerable to tuberculosis. Children were often infected around the age they started school and suffering from the active disease as teenagers (WHO, 2013c). Infants and toddlers were hardly affected (Statens-Skjermbildefotografering, 1949). Hence, this tuberculosis control campaign affects mostly the health of children after the postneonatal period. We therefore use the tuberculosis control campaign as a source of exogenous variation in adolescent disease exposure and examine the campaign's contemporaneous and long-term consequences using panel data, which allows us to link an individual's

[1]Similarly, Baird, Hicks, Kremer, and Miguel (2015) exploit experimental variation in a deworming program in Kenyan primary schools and show that ten years after the health investment young women who were eligible as girls are more likely to attend secondary school and men are working more hours in the labor market. Moreover, Lee (2012) shows that mandatory school vaccination laws introduced in the US in the 1970s also positively affected adult outcomes, including educational attainment and overall labor force participation.

[2]The campaign began soon after the World Health Organization (WHO) and the United Nations Children's Fund (UNICEF) endorsed the Bacille Calmette-Guérin (BCG) vaccine as a mass childhood vaccine. Similar tuberculosis testing and vaccination campaigns were conducted as part of the so-called International Tuberculosis Campaign in postwar Europe, where nearly 30 million persons underwent tuberculin testing, and almost 14 million were given the Bacille Calmette-Guérin (BCG) vaccine.

disease and program exposure as a child to adult human capital, labor market, and health outcomes. Tuberculosis is not simply an infectious disease; it is also a social disease, with infections exacerbated by inadequate nutrition and spread through cramped and overcrowded living conditions, thereby making tuberculosis a greater health threat for the poor. We therefore also analyze whether individuals from a lower socioeconomic background experienced a larger gain from the public health intervention and whether, through this the campaign, economic inequality in adulthood was lowered. We also test directly whether intergenerational mobility in education increased more in municipalities that experienced a strong reduction in tuberculosis. Furthermore, we study whether the gains from the disease control program are limited to the initially treated cohorts or whether the program also affects their children.

Our identification strategy combines the treatment—the control program—with cross-area differences in pretreatment infection rates as in Acemoglu and Johnson (2007), Bleakley (2007), and Card (1992). In particular, we exploit the cohort variation generated by the plausibly exogenous launch of the tuberculosis control program and the large regional variation in disease spread across municipalities. We therefore expect the gain from the vaccination program to be larger for individuals in municipalities with high infection rates prior to treatment. Individuals in municipalities with very low tuberculosis infection rates, however, may have derived much smaller benefits from the campaign. This heterogeneity in disease exposure permits a treatment and control strategy. That is, this identification strategy allows us to evaluate whether individuals who grew up in a municipality with a high tuberculosis infection rate benefited more after the intervention than those in municipalities with a low infection rate.

Norway is an interesting case in the context of tuberculosis, as the country had one of the highest tuberculosis infection rates in Europe in 1940 (Blom, 1998)—at a time when tuberculosis remained a major health threat across Europe.[3] By 1945, tuberculosis accounted for about 5 percent of all deaths in Norway (Backer, 1963). The introduction of the antibiotic streptomycin in 1946 allowed the effective treatment of tuberculosis, and the disease became less fatal.[4] However, even treated tuberculosis can cause permanent damage to the lungs, making breathing and physical activity difficult. Moreover, tuberculosis may also damage other organs, such as the liver and the heart (WHO, 2013a). Tuberculosis prevention was therefore an important health policy goal.

The infectious nature of tuberculosis denotes a clear externality, which led Norwegian policy makers to enact several laws that valued public health higher than individual freedom, and allowed authorities to control the spread of the disease. The laws included mandatory reporting of new tuberculosis cases by doctors, occupational bans for infected wet-nurses or grocers, and the isolation of infected individuals. Of particular importance in combatting tuberculosis was a nationwide tuberculosis testing and vaccination program launched in 1948. The program included tuberculosis

[3]The tuberculosis mortality rate and infection rates in Norway in the 1940s were comparable to those of sub-Saharan Africa today (WHO, 2013a).

[4]If untreated, the disease is fatal in about 60 percent of active tuberculosis cases (Link, 2005).

testing and made vaccination with BCG mandatory for the entire uninfected population (Bjartveit and Waaler, 1965). Over several years, teams of doctors and nurses from the National Mass Radiology Service (NMRS) went by bus and boat to screen and vaccinate all adolescents and adults aged over 14 years. School children (aged 7 to 14 years) were vaccinated at school (Nøkleby, 2006). The examinations included chest X-rays, tuberculin tests, and BCG vaccination for uninfected individuals (Bjartveit, 1972). Follow-up studies indicated that the examinations were very effective, with about 80 percent of all eligible individuals in Norway tested between 1949 and 1957 (Liestøl, Tretli, Tverdal, and Mæhlen, 2009). In 1948, 124,000 individuals were vaccinated, with the number of vaccine doses increasing until the mid-1950s (Galtung, 1961).

We find that individuals growing up in municipalities with higher levels of tuberculosis infections prior to the control program experienced larger gains in education and earnings following the intervention. Moreover, we find a significant increase in longevity and adult height suggesting that the effect the policy has on earnings might not entirely be driven through an increase in education, but also through better health. Men had a higher tuberculosis morbidity rate than women prior to the intervention, and our results suggest that they also benefited more from the tuberculosis control program in terms of earnings. Women, on the other hand, had an indirect financial benefit: the disease control campaign increased women's marriage market success. In particular, we find that affected women marry men with higher earnings and higher education.

Although children had the most to gain from the campaign, adults also benefited from newly available treatments against tuberculosis such as new antibiotics. Hence, the campaign may have affected adult health and labor market outcomes, and thereby household resources. This might make it difficult to disentangle the direct effect of the campaign on children from the indirect effect via their parents. However, we find no empirical evidence that adults of childbearing age did benefit in terms of human capital accumulation and earnings. Moreover, we analyze whether these healthier children are getting more attention from their parents. We find that the disease control campaign did not alter the number of siblings of the exposed cohorts and that the years of education and the earnings of older siblings who had left school before the start of the control campaign are not significantly affected by the tuberculosis control campaign.

In addition, our findings indicate that the effect was larger for individuals from a low socioeconomic background and that the campaign increased intergenerational mobility in education. Furthermore, we estimate the intergenerational effects of exposure to the tuberculosis control program and find statistically significant positive effects of campaign exposure on birth weight, years of education, log earnings, and height at age 19 (for boys only) of the exposed cohort's children.

The remainder of the paper is structured as follows. Section 2 reviews the disease and the history of treatment for tuberculosis and describes the Norwegian tuberculosis control program. Section 3 outlines the empirical strategy. We discuss the data and provide descriptive statistics in Section 4. We discuss our results and analyze whether there were heterogeneous effects by individual

characteristics in Section 5. Section 6 presents different sensitivity tests. In Section 7, we provide a simple back-of-the-envelope calculation quantifying the costs and benefits of the program. Section 8 provides a brief conclusion.

# 2 Tuberculosis and the Disease Control Campaign

We first provide a brief description of tuberculosis and its long-term health consequences as well as an overview of the quest for treatment and disease prevention. We then provide details on tuberculosis in Norway and the tuberculosis screening and vaccination program launched in 1948.

## 2.1 Tuberculosis

The bacteria *Mycobacterium tuberculosis* causes tuberculosis. In most cases, the disease occurs in the lungs (pulmonary tuberculosis), but tuberculosis can affect all the body's organs (Link, 2005). Tuberculosis was earlier known as 'consumption' because of the way the disease would consume infected individuals from within (Bynum, 2012). Tuberculosis can be either latent or active. Latent tuberculosis describes the situation where the bacteria are present in the body but inactive; an individual with active tuberculosis, however, has bacteria in the lungs that multiply and may cause pneumonia.[5] In addition, the infected individual experiences chest pain and has a persistent cough, which often brings up blood. Further symptoms are enlarged lymph nodes and lungs, weight loss, loss of appetite, and fatigue (Link, 2005). Given these symptoms, it is plausible that active tuberculosis would make schoolwork more difficult and would depress the returns on human capital investment.

As the bacteria spread to other parts of the body, the immune system forms scar tissue or fibrosis around the bacteria. This scar tissue prevents the bacteria from spreading. However, if the bacteria manage to break through the scar tissue, the disease returns to an active state (Bynum, 2012). Only active tuberculosis can be transmitted from person to person through the air. That is, when a person with pulmonary tuberculosis coughs, sneezes, speaks, laughs, or sings, aerosols are released into the air and the bacteria can pass to other individuals. Active tuberculosis is closely linked to overcrowding and malnutrition. Hence, the risk of having active tuberculosis is much higher for poor or other vulnerable people. In addition, infected people with compromised immune systems, such as HIV patients, those suffering from diabetes, or tobacco smokers, have a much higher risk of developing tuberculosis (WHO, 2013a). Tuberculosis may harm the human body permanently, even if the disease is treated. Besides permanent lung damage, there may be long-term damage ranging from defects to the central nervous system and the brain and damage to the circulatory system, skin, lymph nodes, joints, bones, and the heart (WHO, 2013a).

[5]Note that both individuals with either active or latent tuberculosis are so-called tuberculin positive. That is, an individual has the bacteria bacteria *Mycobacterium tuberculosis* in her body and a tuberculin skin tests would reveal a positive result.

Archeological evidence suggests that tuberculosis has existed for at least five thousand years, with evidence of tuberculosis found in the bones of prehistoric man and Egyptian mummies. In Europe, tuberculosis and its contagious nature emerged in the sixteenth and seventeenth centuries, and mortality statistics from 1850 in Europe show that 70 out of every 100,000 people died of tuberculosis (Link, 2005). The history of tuberculosis, and in particular, the history of the treatment for tuberculosis, changed dramatically in 1882, when Robert Koch showed that a single infectious agent caused the disease. Combined with Wilhelm Roentgen's discovery of the X-ray in 1895, physicians were now able to diagnose and track the progression of the disease.[6] The ability to diagnose tuberculosis and knowledge about disease transmission led to a decrease in the number of tuberculosis deaths long before antibiotics or vaccines were available, attributable to improvements in socioeconomic and hygienic conditions in Europe and the US during the postindustrial period (Link, 2005). In addition, tuberculosis treatment underwent a major change in the nineteenth and twentieth centuries with early interventions in many countries, including public health campaigns aimed at preventing spitting in public places and laws preventing infected individuals from taking jobs involving food or small children. In addition, laws were enacted that forced infected individuals who could not be isolated in their own homes (mostly the poor) to enter sanatoria (Bynum, 2012).[7] However, sanatoria, which offered only basic care and medical attention, were no guarantee of a cure, with about 50 percent of those entering a sanatorium in 1916 in Great Britain dying within five years (Bynum, 2012).

By the mid-twentieth century, medical innovation allowed better and easier detection of tuberculosis infection: a skin test measuring the reaction to a small amount of tuberculin introduced into the skin enabled doctors to make fast diagnoses and to detect latent cases. In 1946, the introduction of the antibiotic streptomycin allowed doctors to treat tuberculosis more effectively, and the mortality rate decreased substantially. In addition, Calmette and Guérin developed a vaccine for tuberculosis—the so-called BCG vaccine—in 1921 (Link, 2005). Although the effectiveness of the BCG vaccine was, and still is, questioned (see, e.g., Colditz, Brewer, and Berkey, 1994; Fine, 1995), it was endorsed by WHO and UNICEF as a mass childhood vaccine in 1948. Since then, some four billion BCG doses have been administered (WHO, 2013a).[8]

As noted above, public interest in tuberculosis is largely due to the contagious nature of the

[6] If the immune system forms scar tissue around the tuberculosis bacteria, this tissue may harden like stone in a calcification process. This results in granulomas (rounded marble-like scars) that appear on X-rays.

[7] Sanatoria's benefit was mostly provided through isolating the affected individuals and thereby reducing the likelihood of disease transmission (see Hollingsworth, 2014). Focusing on the spread of viral diseases which cannot be treated by drugs such as antibiotics, Adda (2016) shows for example that policies reducing inter-personal contacts reduce disease prevalence.

[8] Although the BCG vaccine is the world's most widely used vaccine, its use remains controversial. The reason is that different clinical trials have found variable efficacy, with protection granted by the BCG vaccine against pulmonary tuberculosis ranging from zero to 80 percent (Fine, 1995). This variability in efficacy appears to depend on geography and has been attributed to genetic or nutritional differences between populations and environmental influences (sunlight exposure of the vaccine or poor cold-chain maintenance). Clinical trials conducted in Northern Europe have consistently shown higher efficacy, whereas efficacy tends to fall in warmer climates.

disease. The prevention of transmission of the bacteria is crucial. A combination of two strategies can lower the risk of new infections: first, active tuberculosis cases have to be detected and treated; second, vaccination programs can protect those vaccinated from the consequences of later infections and the public from subsequent disease transmission. Medical innovations in the 1940s permitted the coordination of these two disease control strategies. Hence, tuberculosis became a curable infection that could be controlled by large-scale screening and vaccination campaigns in the late 1940s. This combined strategy is generally considered the beginning of the modern era of tuberculosis treatment (Link, 2005).

### 2.1.1 The Situation Today

Today, tuberculosis is curable in most cases and infection rates in Western countries since 1990 have remained very low (WHO, 2013a). The eradication of tuberculosis, however, is 'a job half done,' as the disease still ranks as the second leading cause of death from an infectious disease worldwide after the human immunodeficiency virus (HIV). In 2012, 8.6 million people suffered from active tuberculosis and 1.3 million died from tuberculosis, more than 95 percent of them in low- and middle-income countries (WHO, 2013a). However, the full scope of tuberculosis among children is not fully known because diagnosing childhood tuberculosis is challenging, and infected children often live in poor areas with limited access to health services. One important reason why tuberculosis remains a major global health problem is the HIV/Aids epidemic in low- and middle income countries. HIV/Aids weakens the immune system and makes it more likely that individuals with latent tuberculosis will develop active tuberculosis and transmit the disease (see Bynum, 2012). About 0.3 million of the 1.3 million tuberculosis-related deaths are associated with HIV (WHO, 2013a). In addition, the eradication of tuberculosis is complicated by the emergence of new drug-resistant strains of the disease that are resistant to the two most effective anti-tuberculosis drugs, isoniazid and rifampicin (WHO, 2013b).

## 2.2 Tuberculosis in Norway and the Disease Control Campaign

In the early twentieth century, Norway had one of Europe's highest tuberculosis rates, and the disease was very widespread (Blom, 1998). 20 percent of all deaths in Norway in the early 1900s caused by tuberculosis (Backer, 1963). The seriousness of the disease led policy makers to take some drastic measures, with Norway being the first Nordic country to pass a law to promote the fight against tuberculosis in 1900. This law aimed to protect the healthy and to control the spread of disease from the infected to the rest of the population. Registration of new tuberculosis cases became mandatory.[9] In addition, medical treatment became mandatory and it was prohibited for infected people to take work that involved producing or selling food or working with small children

[9]One result was that official statistics on the regional distribution of tuberculosis were made publicly available from 1900 onwards and published in Statistics Norway's yearly health statistics.

(e.g., nannies and wet-nurses). Authorities were empowered to inspect and disinfect private homes where a tuberculosis case was detected, and if it was not possible to separate the infected from the healthy in smaller homes, patients had to move to a sanatorium. The expenses for treatment were covered by the local municipality, but people tried hard to avoid being sent to a sanatorium in order to avoid the social stigma (Blom, 1998).

Despite a continuous decrease in the number of new infections, the disease still accounted for 5 percent of all deaths in 1945. As discussed, tuberculosis detection and treatment in combination with the vaccination program lowered the risk of new infections. Medical innovations in the 1940s permitted this combined control strategy and led to the passage of the Act of Tuberculin Testing and Vaccination against Tuberculosis (lov om tuberkulinprøving og vaksinasjon mot tuberkulose) in 1947 in Norway (Ot.prp., 1947). The new law made it mandatory for every individual aged over 14 years to participate in tuberculin testing and vaccination against tuberculosis and motivated the launch of a large-scale mandatory screening and vaccination campaign in 1948 (Bjartveit and Waaler, 1965).[10] In addition, the law mandated that all the BCG vaccine was included into the vaccination program for children and that all school children (aged 7 to 14 years) were vaccinated at school (Nøkleby, 2006). However, the act was very controversial and there was significant opposition in the parliament to compulsory vaccination. The enactment of the law was crucially influenced by the testimony of an expert group of doctors in parliament, who confirmed the BCG vaccine was nonhazardous, that it protected people from tuberculosis, and thus justified a mandatory mass vaccination campaign.[11]

The National Mass Radiology Service (NMRS), with its seven mobile fluoroscopes (two mounted on boats to reach remote coastal areas), performed the tuberculosis testing. The NMRS tested about 400,000 individuals annually, and within five years, all municipalities in all counties had been tested. Examinations included chest X-rays which were independently analyzed by two experts,

[10]Besides Norway, Sweden and Denmark also launched vigorous campaigns to vaccinate the tuberculosis-negative population. Following World War II, Sweden legislated an offer to vaccinate schoolteachers and their students given that it made sense to target those who could potentially spread the disease through their workplace. The take-up rate was rather large. In addition, staff at state mental hospitals and dental services as well as trainee nurses and medical students needed to be vaccinated. Sweden, Denmark, and Norway also founded the International Tuberculosis Campaign (ITC). Together with the newly founded UNICEF, this initiative offered help in the form of mass vaccination programs in the war-torn areas of Europe and implemented programs in Austria, the Czech Republic, Finland, Greece, Yugoslavia, Hungary, Poland, and Italy. Almost 30 million persons underwent tuberculin testing, and almost 14 million were vaccinated with the BCG vaccine. Furthermore, the ITC initiated a postgraduate school for physicians, established new laboratories, and introduced hundreds of young doctors and nurses to international public health. Nevertheless, Norway was the only European country with a testing and vaccination mandate that covered the entire adult population.

[11]The so-called Lübeck Disaster in 1929—the most serious vaccination disaster of the twentieth century—raised serious concerns about the BCG vaccine, where 72 babies out of 242 vaccinated died from tuberculosis shortly after their vaccination. Many other infants became ill because of vaccination and many suffered from complications including cirrhosis of the liver or brain damage from tuberculous meningitis. The vaccine used was later found to have been contaminated with a human tuberculosis strain that was being studied in the same laboratory where the vaccine was prepared (Link, 2005).

tuberculin tests (adrenaline Pirquet test[12]), and from 1949 onwards, BCG vaccination for uninfected individuals (so-called tuberculin-negative individuals). The NMRS decided where and when to test inhabitants and informed the local authorities about the arrival of the mobile fluoroscopes. Local doctors' offices were then responsible for calling in all individuals older than 14 years. Depending on the type of fluoroscope, the team of doctors and nurses needed about a week to control and vaccinate all eligible individuals of a municipality of about 11,000 inhabitants in total. The mass testing and vaccination commenced in southeastern Norway, particularly in the Oslo region and central Norway, and then expanded to the south, west, and north of Norway. School children aged 7 to 14 years were vaccinated by the school medical service.

Figure 1 depicts the municipalities in which inhabitants were tested from 1948 until 1952. The examinations were very effective with about 80 to 91 percent of all eligible women and 72 to 84 percent of all men tested from 1949 to 1957 by the NMRS at their place of residence.[13] About 5 percent of the population could not be screened by the NMRS because of illness or temporary absence.[14] The rate of people not tested for unspecified reasons was small (Liestøl, Tretli, Tverdal, and Mæhlen, 2009). The number of new tuberculosis infections substantially decreased during the disease control program. Figure 2, which plots the number of new active tuberculosis infections per 10,000 inhabitants (hereafter infection rate), illustrates the program's success. In early 1940, the infection rate was relatively steady, but by the end of the 1940s, the infection rate began falling. In 1960, the number of new tuberculosis infections per 10,0000 individuals was very low and only at about an eighth of its 1940 level.

Schoolchildren were most vulnerable to new tuberculosis infections: in the mid-1940s, about 18 percent of 15–19 year olds, about 15 percent of school-age children, and less than 1 percent of children under four were found to be tuberculin positive (Statens-Skjermbildefotografering, 1949). The largest increase in infection rates occurred during the teenage years, such that children and young adults benefited most from the control program.[15] The nationwide testing and vaccination

[12]The method applied in Norway required a drop of concentrated tuberculin to be placed on a site on the volar aspect of the underarm. Two 5 mm long scratches were then made in the superficial layer of the skin and the sites left to dry. The reactions were read after two to three days by a public health nurse based on the width of the largest induration across the scratches.

[13]Besides the test campaign directed by the NMRS, the military examined soldiers (about 5 percent of the eligible male population) and the health personnel were vaccinated at hospitals.

[14]Examples in Norway during this time are mostly fishermen and seamen.

[15]There are several reasons why infants and toddlers are less affected by tuberculosis and thereby by a disease control program. First, newborns are protected from viral and bacterial illness by immunoglobulin G antibodies which were produced by the mothers immune system and then transported across the placenta to the fetuss blood supply. Second, the risk of acquiring tuberculosis is directly proportional to the number of bacilli to which a child is exposed. Infants and toddlers are mostly exposed to their parents, siblings and other family members. As described in Section 2.2, the Norwegian law required individuals with active tuberculosis to stay in a sanatoria if they could not be isolated within their house. In particular, infants of parents with an active tuberculosis infection were taken from their parents to prevent infections. Hence, infants and toddlers were relatively well protected from contracting tuberculosis. On the other hand, schoolchildren are in contact with many other children and adults at school and on the commute from and to school and are therefore more exposed to the disease threat. Moreover, as the diagnosis of tuberculosis in children was more difficult than in adults (Piccini, Chiappini, Tortoli, de Martino, and Galli, 2014),

campaign led to a decisive decrease in the number of new tuberculosis infections reported among young people. In evidence, Waaler, Galtung, and Mordal (1971) report that the infection rates among seventh grade school children (13 years old) in Oslo decreased from 17.4 percent in 1945, to 10.9 percent in 1951, and just 0.8 percent in 1968. In addition, Figure 3 depicts the age pattern among newly infected individuals in 1946 and 1960. With 32.3 new active tuberculosis infections per 10,000 individuals, teenagers had the highest rates of new active tuberculosis infections in 1946—the year prior to the control program. This rate is about three times as high as for the general population. Comparing 1946 with 1960 reveals that teenagers had the highest rates of new active tuberculosis infections in the year prior to the control program and that the risk for the same age group was much lower (about two new tuberculosis infections per 10,000 individuals) by 1960. In 1960, infection rates were lower for teenagers than for the general population. Hence, teenagers benefitted most from the disease control campaign in terms of the number of new infections per 10,000 individuals.

The low number of new infections in the 1960s raised some discussion as to whether the continuation of the mass vaccination campaign was still justified. After 1963, the mass tuberculosis testing and vaccination campaign with mobile teams of doctors and nurses ceased and the disease control effort was modified into a more selective testing program for high-risk groups. However, it was not until 1976 that we saw the mandate for X-ray testing abolished. The mandatory vaccination law for children remained in effect right up until 1995.

## 3 Empirical Strategy

The goal of this study is to analyze the benefits of growing up in a tuberculosis-free environment. We use an identification strategy similar to that of Acemoglu and Johnson (2007), Bleakley (2007), and Card (1992). In particular, we compare long-term economic outcomes across municipalities with different tuberculosis infection rates by estimating the following reduced-form relationship:

$$Y_{ijc} = \alpha + \gamma(T_j^{pre} \times Post_{ic}) + \beta X_{ijc} + \delta_c + \theta_j + \varepsilon_{ijc}, \tag{1}$$

where $Y_{ijc}$ denotes the outcomes of interest recorded in adulthood for individual $i$ born in municipality $j$ in cohort $c$. $X_{ijc}$ is a vector of individual-level demographic characteristics, $\delta_c$ are cohort dummies, and $\theta_j$ are municipality fixed effects. The central variable of interest, $T_j^{pre} \times Post_{ic}$, is the interaction term of the pretreatment tuberculosis infection rate in municipality $j$ and a dummy

diagnosis of tuberculosis in children and isolation often only occurred after they potentially already infected other class mates. Third, the lower disease prevalence among toddlers might also be a measurement issue as it takes children about two years to develop active tuberculosis after being infected. It is important to note that the situation today is somewhat different as infants are not necessarily separated from their infected mothers when mothers are receiving treatment against tuberculosis and because tuberculosis is often a co-morbidity of AIDS in children (Newton, Brent, Anderson, Whittaker, and Kampmann, 2008).

variable indicating whether cohort $c$ was exposed to the control program at ages 14 years or younger. More precisely, the pretreatment infection rate, $T_j^{pre}$, is given by the average number of individuals registered with active tuberculosis from 1940 to 1946 per 100 inhabitants in each municipality. Thus, this variable captures the treatment intensity. $Post_{ic}$ describes the cohort variation generated by the launch of the tuberculosis control program and is equal to one for all cohorts leaving school in 1949 and after. To account for serial correlation in the outcomes, standard errors are clustered at the municipality of birth level (Bertrand, Duflo, and Mullainathan, 2004). The parameter $\gamma$ captures the effect of the reduction in tuberculosis morbidity on long-term outcomes. This effect includes the potential direct medical effect of disease prevention on cognitive development and the effect of not missing school because of tuberculosis on human capital accumulation, as well as the indirect effect of changed human capital investment incentives. As the tuberculosis prevention efforts also had a potential impact on adults, the estimated effect may also include indirect benefits for children from having healthier parents and thereby larger household resources.[16]

Alternatively, we use a specification where we spilt the sample at the median in municipalities with high versus low tuberculosis infection rates:[17]

$$Y_{ijc} = \alpha + \kappa(H_j^{pre} \times Post_{ic}) + \beta X_{ijc} + \delta_c + \theta_j + \varepsilon_{ijc}. \tag{2}$$

The variable $H_j^{pre}$ is a dummy variable equal to one if individual $i$ was born in a municipality with a high tuberculosis infection rate. Here, $\kappa$ measures the differences in outcome $Y_{ijc}$ across individuals born in high versus low infection municipalities. Standard errors are clustered at the municipality level to allow for arbitrary correlation of the errors for individuals in the same municipality of birth.

To account for the differences in the length of exposure to the tuberculosis testing and vaccination campaign while at school, we also allow the central variable of interest to vary by the number of years an individual is in school during the campaign. We therefore specify $Exp_{ic}$ to account for the number of years a cohort $c$ was exposed to the control program while in school. The variable $Exp_{ic}$ is zero for cohorts leaving school before the campaign started and increases linearly for those in school during the campaign. That is, $Exp_{ic}$ is equal to one for the cohort leaving school in 1949, two for the cohort leaving school in 1950, and taking values up to seven for cohorts leaving school in 1955 and thereafter.[18] We interact $Exp_{ic}$ with the pretreatment infection rate, $T_j^{pre}$, and estimate the following reduced form:

$$Y_{ijc} = \alpha + \lambda(T_j^{pre} \times Exp_{ic}) + \beta X_{ijc} + \delta_c + \theta_j + \varepsilon_{ijc}. \tag{3}$$

[16]The indirect benefits for children from having healthier parents is discussed in Section 5.2.

[17]A similar specification is used by Adhvaryu, Bednar, Molina, Nguyen, and Nyshadham (2014) in a study of the effect of salt iodization on labor force participation.

[18]The results are similar if we analyze the number of years an individual was exposed to the tuberculosis control campaign since birth.

The above specifications permit us to examine the year and municipality-of-birth cohorts retrospectively. Based on the preexisting tuberculosis infection rates and by using the unexposed cohorts as a comparison group, we can then make comparisons across both municipalities and cohorts.

We also analyze the contemporaneous consequences of the tuberculosis control program. Unlike the long-term analysis, the short-term analysis draws on municipality-level data. To consider the short-term effects of the tuberculosis test and vaccination campaign, we analyze whether the control program had an immediate effect on the percentage of missed schooldays. We estimate the following reduced-form specification:

$$Y_{jt} = \alpha + \rho(T_j^{pre} \times Post_t) + \delta_t + \theta_j + \varepsilon_{jt}, \tag{4}$$

where $Y_{jt}$ denotes the percentage of missing schooldays in municipality $j$ in year $t$. $\delta_t$ are year dummies and $\theta_j$ are municipality fixed effects. The central variable of interest, $T_j^{pre} \times Post_t$, is the interaction term of the pretreatment tuberculosis infection rate in municipality $j$ and a dummy variable indicating whether year $t$ was after the launch of the tuberculosis control campaign. As in the long-term analysis, $T_j^{pre}$ captures the treatment intensity. $Post_t$ is equal to one for all years after 1948.

As discussed in Bleakley (2007), the identification of the effect of the tuberculosis campaign relies on (i) regional variation in tuberculosis exposure, (ii) the program's immediate success, and (iii) the exogeneity of the program launch. In the 1940s, there was a large regional variation in tuberculosis infection rates. Figure 4 displays the average tuberculosis infection rate per 100 inhabitants for each municipality from 1940 to 1946 (the seven years before the law was enacted). Although the highest infection rates are evident in the northernmost municipalities and municipalities in the southeastern part of Norway, there is significant variation in disease rates across the country.[19] Environmental factors affect the risk of infection and disease development, with high humidity, poor ventilation, and overcrowding possibly increasing the chances of contracting tuberculosis. That is, residents of the humid coastal regions in the south and west of Norway were more vulnerable to tuberculosis infections than individuals from the dryer regions in the east.[20] In addition, tuberculosis more severely affected poorer municipalities and cities where overcrowding was most common. Our strategy relies on this heterogeneity in the spread of the disease prior to the intervention. The basic assumption is that municipalities where tuberculosis was widely distributed experienced a greater decline in tuberculosis infections and therefore benefited more from the vaccination campaign than those municipalities with low infection rates. This difference in potential benefits from the campaign allows for a treatment and control strategy. The assumption that municipalities with a high disease

[19] There are two measures concerning the spread of tuberculosis: first, the rate of tuberculin-positive individuals as found by testing, and second, the rate of individuals with active tuberculosis. Because only active tuberculosis is contagious, we focus on the latter.

[20] The average annual rainfall is significantly higher in municipalities in the highest quartile of the disease exposure when compared with municipalities in the lowest quartile of disease exposure.

spread benefit more is supported by Figure 5, which plots the municipality-specific reduction in tuberculosis infection between the campaign years and 1960 as a function of the precampaign morbidity rate. The plot clearly displays convergence in the tuberculosis infection rate across municipalities.

In addition, the identification relies on the rapid success of the campaign. As noted, the mass tuberculosis testing and vaccination campaign lowered the infection rates substantially among young people within a decade (see Figure 2). Follow-up studies indicated that the vaccination campaign protected 80 percent of the possible new infections of individuals aged between 15 and 24 (Bjartveit and Waaler, 1965; Tverdal and Funnemark, 1988; Waaler, 1966). From a historical perspective, this decrease in disease exposure following the launch of the tuberculosis testing and vaccination campaign was a major change. By studying outcomes, including completed years of education, earnings and early mortality, we analyze outcomes up to 60 years after the testing and vaccination program—undoubtedly a relatively long time span compared with the actual duration of the campaign.

Finally, the identification relies on the exogeneity of the disease control program with respect to the prevalence of tuberculosis. In the 1940s, except for a few larger cities, public health infrastructure was limited, large areas of Norway were relatively poor, and some areas had even been destroyed by the end of World War II. Without a nationwide mass testing and vaccination program and government funding, it would have been impossible for most municipalities to establish tuberculosis testing and vaccination facilities. Because the NMRS brought mobile infrastructure and crew to the municipalities by bus and boat, the disease control program represented neither a permanent change in health infrastructure nor a lasting positive income shock for the municipalities. In addition, the timing of the intervention crucially depended on the availability of medical innovations such as the BCG vaccine, X-rays, and tuberculin tests. We argue that these innovations are unrelated to expectations about the future development prospects for different Norwegian municipalities. Thus, the timing of the innovations is not endogenous in the context analyzed.

The identification strategy faces further challenges. First, one concern is that the average outcomes in municipalities with high and with low tuberculosis infection rates might have had different time trends prior to the campaign. That is, tuberculosis infection rates were very high in municipalities where, for example, the level of education was steeply rising, while at the same time, education levels were stable in municipalities with low tuberculosis infection rates. We therefore need to show that the average education level in municipalities with high or low disease exposure follows a similar trend prior to the intervention. Panel (a) of Figure 6 uses census data and plots the average years of education by birth cohorts from 1910 to 1950. The different lines denote averages in municipalities in the first, second, third, and fourth quartile of the distribution of tuberculosis infection rate per 100 inhabitants by municipality from 1940 to 1946. Municipalities are allocated

to individuals using the place of residence in 1960.[21] The vertical line marks the 1933 cohort—the first cohort affected by the disease control campaign while in school. The figure shows that individuals in municipalities with low tuberculosis infection rates are on average better educated. For cohorts born before 1933, the trends in education level are increasing in a parallel fashion. For cohorts born after 1933, the trends in average education are converging and indicating that the gap in average education in areas with high and low tuberculosis infection rate is declining. In Panel (b) of Figure 6 we show the average share of individuals between 20 and 30 years of age who completed high school per municipality in 1930, 1940, 1950, and 1960. The different lines denote averages in municipalities in the first, second, third, and fourth quartile of the distribution of tuberculosis infection rate per 100 inhabitants by municipality from 1940 to 1946. Municipalities are allocated to individuals using their place of residency in either 1930, 1940, 1950, and 1960. The vertical line marks the start of the disease control campaign in 1948. Until 1950, the trends in education level are relatively parallel. However, in 1960—twelve years after the program commenced—municipalities with above-average disease exposure experienced a larger increase in the share of inhabitants with secondary education than municipalities with lower disease exposure. This provides some initial evidence that the increase in postmandatory educational attainment was related to the decrease in tuberculosis exposure.

In addition, unobservable characteristics that vary by municipality of birth and birth year and that correlate with both tuberculosis exposure and accomplishment later in life threaten our identification strategy. We therefore need to include specifications controlling for the trends in public infrastructure and the exposure to other diseases in the municipality of birth. First, we control for preexisting local trends by including the number of inhabitants per doctor in each municipality and the student–teacher ratio in each municipality in the year an individual enters school. A further concern is that the doctors and nurses that passed through the municipalities with the control campaign generally improved the health status in the municipality and that the coefficient $\gamma$ picks up health improvements other than tuberculosis treatment. In general, the control campaign staff stayed only for the time needed to test all individuals for tuberculosis and vaccinate all those who were tuberculosis negative. Hence, their impact on local health systems was rather short. Nevertheless, the reports from the NMRS also show that other respiratory diseases such as silicosis (an occupational health-related lung disease from inhaling dust) or cardiovascular diseases were sometimes diagnosed among adults. In addition, tuberculosis prevention may have strengthened individual health and lowered the likelihood of infection with other diseases. Like tuberculosis, other infectious diseases, such as measles, affect school attendance by children and thereby the human capital accumulation. Measles is a highly communicable disease and this gives an indication of the municipality-specific state of public health programs that could have changed alongside the tuberculosis control campaign. We therefore control for precampaign childhood mor-

[21] Note that Panel (a) only includes individuals who survive until 1960.

tality rates from measles for each county by including the interaction with an indicator variable $Post_{ic}$ that is equal to one for those cohorts $c$ in school during or after the campaign years. This specification allows outcomes to vary discontinuously with measles exposure. Another concern is that some of the affected cohorts also benefited from the introduction of the first antibiotics. The first-generation antibiotics were effective against pneumonia, wound infections, and puerperal sepsis (see, e.g., Bhalotra and Venkataramani, 2012). Antibiotics such as penicillin were also used for nonmilitary purposes in Norway after World War II. We therefore add an interaction term between the indicator variable $Post_{ic}$ and the county-specific childhood mortality rate from pneumonia.

A further challenge to our identification strategy is that the estimates could be an artifact of mean reversion. Thus, we might expect an increase in income and years of education for the postcampaign cohorts, even without the direct effect of tuberculosis on productivity, if the pre-campaign cohorts were highly infected with tuberculosis and had low productivity because of some mean-reverting shock. To account for mean reversion, we use data from the 1930 Census on the number of individuals with secondary education in each municipality and the natural logarithm of the average earnings in each municipality and include their interaction with the year of birth.

# 4 Data and Descriptive Statistics

We compile our data from various sources by linking aggregate data on tuberculosis infection with individual administrative data. Our primary data source is the Norwegian Registry Data, a linked administrative dataset that covers the Norwegian population up to 2013. These data are maintained by Statistics Norway and covering a number of different administrative registers, including the central population register, the family register, the education register, and the tax and earnings register. The data provide information about place of birth and residence, educational attainment, labor market status, earnings, a set of demographic variables, as well as information on families. The aggregate data on tuberculosis infections are from the historical data collection of Statistics Norway and the data collected by the Norwegian Public Health Institute on the tuberculosis testing and vaccination campaign. For our analysis, we focus on those cohorts born between 1930 and 1945. The earlier cohorts in our sample completed mandatory schooling before vaccination was mandatory and when the risk of contracting tuberculose while in school was still high; the later cohorts finished school when the tuberculosis infection rates were increasingly smaller. Table 1 provides summary statistics of the outcome and control variables.

### 4.1 Registry Data

The central population register contains the municipality of birth.[22] We allocate a municipality of residence during the vaccination campaign in the late 1940s and early 1950s to each individual by assuming that they were still residing during the first few years of life in their municipality of birth.[23] The central population register includes identifiers for parents. This enables us to identify socioeconomic background and an individual's siblings. The sibling information allows testing whether the family was still living in the same municipality when younger siblings were born, and indicates the geographic mobility of families. More than 90 percent of the individuals with younger siblings in our sample have younger siblings born in the same municipality.[24] We thus argue that the municipality of birth is a relatively good approximation of the municipality of residence during school age. Educational attainment is from the educational database provided by Statistics Norway. Since 1979, educational institutions report educational attainment directly to Statistics Norway annually, thereby minimizing any measurement error from misreporting. For individuals who completed their education before school year 1973/74, we use information from the 1970 Census. Census data are self-reported. However, the information is considered to be very accurate (see, e.g., Black, Devereux, and Salvanes, 2005). We use two measures of educational achievement: high school is an indicator variable equal to one if an individual received a high school degree. We also consider the number of years of education completed by the individual. On average, 60 percent of men and 51 percent of women in our sample have postmandatory schooling. The average number of years of education is 10.2 for men and 9.5 for women.

We consider earnings when individuals are in their late thirties. The earnings measure is not top-coded and includes discounted labor earnings, taxable sick benefits, unemployment benefits, parental leave payments, and pensions. The average earnings at 37 years of age for employed individuals are Norwegian kroner (NOK) 229,866 for men and NOK 67,248 for women.

As an additional outcome, we consider early mortality, i.e., mortality before average life expectancy at birth for cohorts born from 1930 to 1945. Life expectancy at birth for the cohorts of interest is 65.8 years (FHI, 2012). We calculate early mortality from the cause-of-death registry provided by the Norwegian Institute of Public Health. As tuberculosis mostly affects the lungs, we use this information to create an indicator variable for death from respiratory disease. We classify diseases such as pneumonia and chronic lower respiratory diseases such as bronchitis as respiratory disease. Tuberculosis may also infect the tissues that surround the heart, causing inflammation and fluid accumulation that may affect the heart's ability to pump effectively, a condition known as cardiac tamponade, and which may be fatal (WHO, 2013a). We therefore also consider death

[22] Municipality borders have changed substantially during the last few decades. Smaller municipalities merged and the number of municipalities decreased from 747 municipalities in 1930 to the 428 that remain today. We use the historical municipality borders in our analysis.

[23] The central population register provides the municipality of residence in each year only from 1967 onwards.

[24] In addition, we find that only 15% of the male cohorts born in 1932 and 1933 are living in a different municipality in the year of military enlistment (at age 18) than their municipality of birth.

from cardiovascular disease. About 43 percent of the individuals in our sample died before the age of 66, with about 2 percent dying from respiratory disease and about 8 percent from cardiovascular disease.

The effect of the campaign on long-term outcomes could differ by socioeconomic background. As a proxy variable for parental background for those individuals whose parents were alive in 1960 and are therefore included in the 1960 Census, we use the father's education and profession. We classify fathers with a high school diploma or a higher degree as fathers with a high education. Alternatively, we divide professions into high and low socioeconomic status. We classify engineering and academic professions, jobs in the public administration, and office jobs as high status professions. About 33 percent of fathers have such high socioeconomic professions and about 28 percent of fathers have completed secondary or higher education.

### 4.2 Data on Tuberculosis Infection and Vaccination

Tuberculosis has been a notifiable disease in Norway since 1900 and closely monitored since then. That is, district doctors were required to report new tuberculosis infections, the number of tuberculosis survivors, and tuberculosis-related deaths to the authorities. Statistics Norway collected and published yearly information on the number of new active tuberculosis cases, the number of individuals affected with active tuberculosis by the end of the year, the number of people who recovered from active tuberculosis, and mortality by municipality. For our analysis, we use the average number of individuals infected with active tuberculosis by the end of the year from 1940 to 1946—the seven years prior to the vaccination mandate—per 100 inhabitants in each municipality to measure treatment intensity. As discussed, Figure 4 displays the significant variation in infection rates in Norway, ranging from 0.13 in Stor-Elvdal in Hedmark county in the east to 4.6 in Tranøy in Troms county in the north. It is important to note that the measure we use serves as a proxy for the tuberculosis prevalence among schoolchildren in each municipality. The actual number of individuals *ever* infected with tuberculosis is substantially larger than our treatment intensity measure indicates.[25] Moreover, the infection rates among schoolchildren were substantially larger than in the general population (see Figure 3). As the number of ever infected individuals and infection rates per age category are not reported on municipality level, the average number of individuals infected with active tuberculosis by the end of the year from 1940 to 1946 serves as a proxy. Note that when focusing on county-level data, the average number of individuals infected

[25]For example, in 1946 there were 20,824 individuals in Norway suffering from active tuberculosis by the end of the year. During the same year, 4490 new cases of active tuberculosis were registered and 3906 individuals recovered from active tuberculosis. Hence, the number of individuals that actually had active tuberculosis during the year of 1946 is almost 20 percent higher (see Statistics Norways Yearly Historic Health Statistics 1946, p. 182). Moreover, only half of the individuals that are registered as infected with active tuberculosis are registered in multiple years. Hence, the cumulative number of actively infected individuals during the years 1940 to 1946 is about 3 to 4 times higher as the average number of individuals infected with active tuberculosis by the end of the year from 1940 to 1946 displayed in Figure 4.

with active tuberculosis by the end of the year from 1940 to 1946 is a strong predictor of the number of schoolchildren suffering from tuberculosis. For example, the correlation coefficient between the average number of individuals infected with active tuberculosis by the end of the year from 1940 to 1946 and the percent of tuberculin positive individuals under 19 years from 1949 to 1952 is 0.66 (Statens-Skjermbildefotografering, 1949, 1950, 1951, 1952). Moreover, the correlation coefficient between the average number of individuals infected with active tuberculosis by the end of the year from 1940 to 1946 and the percent of individuals aged 10-14 years and 15-19 years dying from tuberculosis between 1940 and 1946 is 0.51 (Statistics Norway, 1940, 1941, 1944, 1945, 1946).

The data on the tuberculosis testing and vaccination campaign are from the NMRS's yearly reports (Statens-Skjermbildefotografering, 1949, 1950, 1951, 1952). Key statistics for the first four campaign years are reported in Table 2. During the first four years of the program, seven teams tested up to 400,000 individuals annually. Among men without active tuberculosis, 60 to 70 percent were tested tuberculin positive; among women, the rate was 50 to 60 percent. Between 20 to 25 percent of all examined individuals were tested tuberculin negative and therefore vaccinated with the BCG vaccine. Although testing and vaccination for tuberculin-negative people was mandatory, not all individuals were tested. In 1951, 76 percent of all eligible men and 84 percent of all eligible women were tested under the campaign (Statens-Skjermbildefotografering, 1951). The difference between men and women is visible in all test years and reflects that men have a higher probability of being absent from the municipality during the test days because of employment or other obligations (including jobs such as sailor, fisherman, or soldier). Individuals who were not present during the testing in their municipality were called in later for tests at hospitals. The data on the rate of tuberculin-positive individuals, however, include only residents.[26] The vaccination rat among school children was almost 100 percent (Nøkleby, 2006).

The NMRS's yearly reports give a precise picture of the operational costs and the number of employees of the tuberculosis testing and vaccination campaign over the period 1948 to 1952. The costs include wages for doctors, nurses, X-ray assistants, drivers, lab assistants and clerks, operation costs, rent of labs, electricity, and other expenses. Table 3 lists the total costs in each year as well as the average costs by tested individual in 1950 NOK. In 2013 values, the total program costs in 1952 would amount to approximately US dollars (USD) 4,500,000. The costs by tested individual rose from 1949 to 1952 partly because of increases in wages and amounted to about USD 19 per tested person when converted to 2013 values. We estimate the overall program costs for the mass testing and vaccination from 1948 to 1963 to be about USD 80 million.

[26] As discussed, the total number of individuals tested and vaccinated is higher, as the military tested and vaccinated its members and the new recruits and health personnel were also vaccinated if they were found to be tuberculin negative.

### 4.3 Municipality-Level Data

As discussed in Section 3, we include the numbers of inhabitants per doctor and the student–teacher ratio in the year an individual enters school to control for local trends in public infrastructure, and mortality rates from measles and pneumonia to control for the local public health situation. The data on mortality rates from measles and pneumonia per county as well as the number of doctors per 100 inhabitants are from Statistics Norway's historical yearly health statistics. The average number of doctors per 100 inhabitants in each year is plotted in Figure 7. There is also a large geographic spread in doctors per 100 inhabitants: while rural and remote places have fewer doctors, most medical personnel are located in urban areas.

We obtain the student–teacher ratio from Statistics Norway's historical school statistics. The student–teacher ratio varies from 11 students per teacher to 54 (there is no significant difference between city and rural municipalities). Statistics Norway's historical school statistics also provide the number of schooldays missed each year from 1940 to 1950. This allows us to calculate the percentage of school days missed in primary school in each municipality. The missing school days are a possible indicator of sickness absence from school because of tuberculosis. Of course, reasons for missing school days could also be students working on the farms during harvest season or school closures from bad weather conditions. On average, about 9 percent of schooldays were missed prior to the campaign.

We collect the number of inhabitants with a high school diploma and the average income per municipality from the 1930 Census. The 1930 Census is the second census in Norway after 1910, collecting data on income, wealth, taxes, and unemployment. In-kind payments complicate the collection of income and wealth data. The average income for men was NOK 2310 and NOK 1316 for women in 1930 NOK. The average rate of individuals with high school education was 10 percent. Income and education also vary significantly by the degree of urbanization, with the income for men and women and the number of individuals with high school education significantly higher on average in cities than in rural municipalities.

## 5 Empirical Results

### 5.1 Contemporaneous Effects on School Children

In this subsection, we analyze the short-term effects of the tuberculosis testing and vaccination campaign on the percentage of missed schooldays per municipality to analyze whether the control program had an immediate effect on school education. The campaign could lead children to reduced absence from school. A child infected with tuberculosis missed school for several weeks, which could add up to months if the disease returned or the tuberculin-positive child more easily contracted other infectious diseases throughout the mandatory schooling years. As noted above, we base this analysis on municipality-level data. The results from estimating Equation 4 for various samples

and specifications are presented in Table 4. We find a substantial decrease in missing schooldays in municipalities that had high levels of tuberculosis infection from 1940 to 1947. That is, the coefficient on $(T_j^{pre} \times Post_t)$ denotes that in a municipality that has a tuberculosis infection rate of 1 person for every 100 inhabitants, the percentage of missing schooldays would decrease by 2.5 percentage points relative to municipalities without any tuberculosis cases. On average, 9 percent of schooldays were missed before the program launch and the standard deviation across municipalities was 0.08. The standard deviation of the precampaign tuberculosis infection rate was 0.33. That is, an increase in the infection rate by one standard deviation lowers the missed school days by a tenth of a standard deviation.

When limiting the analysis to rural municipalities, the effect remains very similar (Table 4, Column 2). The effect also remains significant even when focusing solely on the postwar years to ensure that missed schooldays were not influenced by the German wartime occupation (Column 3). The size of the effect, however, is smaller. In Column (4), we exclude the two northernmost counties of Finnmark and Troms. These two counties were relatively poor and suffered the greatest damage toward the end of World War II,[27] and had a particularly high tuberculosis infection rate. Also, when imposing this sample restriction, the coefficient on $(T_j^{pre} \times Post_t)$ remains significant at the 5 percent significance level.

The specification in Panel B contains controls for heterogeneity across municipalities. In particular, we add the number of doctors per inhabitant and the student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_t$, and with an interaction term between the dummy variable $Post_t$ and the local childhood mortality rate from pneumonia. We do this to control for trends in local public infrastructure and local exposure to other diseases. The estimated coefficients for $(T_j^{pre} \times Post_t)$ remains unchanged. We include additional controls in Panel C to control for mean reversion across municipalities. We include the average income in each municipality in 1930 interacted with $Post_t$ and the average percentage of inhabitants with a high school education in each municipality in 1930 interacted with $Post_t$. Once again, the point estimates of the effect of tuberculosis control do not change substantially and the estimated effects remain significant. These findings suggest that controlling tuberculosis had a positive effect on the number of days children went to school and potentially on the human capital accumulation of the affected cohorts during mandatory schooling. Thus, when examining these cohorts as adults, we expect to see increases in human capital and productivity in the labor market.

[27] With the beginning of the German withdrawal from northern Norway and the advance of Soviet troops at the end of the war, the Germans applied the so-called scorched earth policy and virtually destroyed all urban areas before retreating. Inhabitants were ordered to evacuate the region immediately.

### 5.2 Contemporaneous Effects on Adults in Childbearing Age

First, we examine whether the tuberculosis campaign affected the educational outcomes of adults. Tuberculosis incidents among adults of childbearing age were lower than for children and teenagers (see Figure 2). In addition, adults might benefit much less in terms of human capital because their educational investment is largely already completed. As with the contemporaneous effects on schoolchildren, we estimate Equation 4 for various samples and specifications and use the percentage of individuals of childbearing age with a high school degree in each municipality as an outcome. We use municipality-level data from the censuses in 1940, 1950, and 1960. We consider the adults, who are potential parents of our cohorts of interest, and look at the average education level of the population that was aged between 20 and 40 years in 1940. We then follow these cohorts in the 1950 and 1960 censuses to examine whether the tuberculosis control campaign altered the education of the parents' generation.

The results are presented in Table 5. Panel A provides the estimates using the 1940 and 1950 censuses and Panel B contains census data from 1940 to 1960. For all specifications, we are unable to reject the null hypothesis that the changes in educational attainment across municipalities with different tuberculosis infection rates were the same. That is, the tuberculosis control program does not appear to affect the education of adults of childbearing age prior to the campaign. Alternatively, we study whether the tuberculosis control campaign altered the educational outcomes of fathers of individuals born between 1930 and 1945. The results are presented in Panel C of Table 5. For all specifications, we find that the tuberculosis control program does not appear to affect the fathers' likelihood of completing high school.

However, these findings do not rule out that our estimates of the short- and long-term effects for children still include indirect impacts from the increase in household resources generated by lower tuberculosis morbidity among parents. We therefore analyze whether the tuberculosis campaign affected adults' incomes. As the census data in 1940, 1950 and 1960 do not include earnings and we only observe earnings in the registry date from 1967, we use census data from 1930 and digitalized data from the national tax statistics for 1948, 1951, 1954, 1957, and 1961, which give us average taxable incomes for adults in each municipality. As above, we consider cohorts of adults, who are potential parents of our cohorts of interest. The results are presented in Panels D and E in Table 5. For all specifications, the tuberculosis control program does not significantly affect the contemporaneous log average earnings of adults. The estimated effects are not as precisely estimated as the effects on earnings for the main cohorts of interest, but they are substantially smaller (see Section 5.3). Based on these results and that tuberculosis incidents among adults of childbearing age were much lower than for children and teenagers (see Figure 2), we expect the indirect effects through the household resources to be smaller relative to the direct effect on children's health.

### 5.3 Long-Term Consequences on Labor Market Outcomes

In this section, we analyze the long-term effects of the exposure to the tuberculosis control program. We start with a simple graphical analysis (see Figure 8). We estimate a simple regression model for each year of birth of our outcomes of interest, $Y_{ijc}$, for individual $i$ born in municipality $j$ in cohort $c$:

$$Y_{ijc} = \beta_c T_j + \eta_c + X_j' \Gamma_c + \nu_{ijc}, \tag{5}$$

where $T_j$ is the pre-campaign tuberculosis infection rate in municipality $j$, $\beta_c$ is a cohort-specific coefficient on tuberculosis, $X_j$ is a vector of municipality-level control variables, and $\eta_c$ and $\Gamma_c$ are cohort-specific intercept and slope coefficients. This specification allows us to study how the relationship between long-term outcomes and pre-campaign tuberculosis rate differs across cohorts. The dots in Figure 8 represent the cohort-specific coefficient on tuberculosis, $\beta_c$. The lines indicate the number of years of potential exposure to the tuberculosis control campaign while an individual was enrolled at school. Cohorts that were born in 1933 or earlier were too old to have benefited from the decrease in the risk to be infected with tuberculosis while enrolled at school. Later cohorts experienced decreased tuberculosis infections during their school years. The pre-campaign tuberculosis infection rate generally predicts lower education and earnings. For cohorts born late enough to have potentially been exposed to the control campaign while enrolled at school, the predicted long-term outcomes are higher.

In a second step, we study the long-term effects of the exposure to the tuberculosis control program by estimating Equation 1. Estimates of the variable of interest, $(T_j^{pre} \times Post_{ic})$, are given for various outcomes and specifications in Table 6. Each cell contains estimates of the coefficient on $(T_j^{pre} \times Post_{ic})$ from a separate regression. The rows report different specifications with additional controls, and the columns the four different outcome variables.

Panel A shows the baseline results controlling only for the municipality and cohort fixed effects. We find a significant increase in years of schooling among those cohorts exposed to the tuberculosis campaign in municipalities that had high levels of tuberculosis infection from 1940 to 1947. In addition, the probability of entering postmandatory schooling increased significantly, and our results show the significant positive effect of the relief of the disease burden on earnings at age 37 years. In particular, the coefficient on $(T_j^{pre} \times Post_{ic})$ indicates that, on average, an individual in a municipality that had a tuberculosis infection rate of 1 person per 100 inhabitants experienced a 0.5-year increase in years of education, a 6 percentage point increase in the probability of postmandatory schooling, and a 7 percent increase in earnings at age 37 compared with individuals in municipalities without any tuberculosis cases prior to the campaign.

The estimated effects seem large compared to the average infection rates plotted in Figure 4. As discussed in Section 4.2, the tuberculosis infection rate we use is only a proxy and a lower bound for the rate of ever infected schoolchildren in each municipality. Lowering the tuberculosis infection rate from 1 to 0 person per 100 inhabitants in the general population relates to a decrease from

11 to 0 percent among schoolchildren. If only the children suffering from active tuberculosis were benefitting, the estimated treatment effect on the treated would still be rather large. However, it is very likely that also children who never suffered from active tuberculosis benefit in terms of education from having fewer classmates that miss school a lot or that need extra attention.

Panel B reports the results for the specification including controls for trends in local public infrastructure and local exposure to other diseases.[28] The estimated coefficients of $(T_j^{pre} \times Post_{ic})$ remain almost unchanged. As pointed out by Bleakley (2007), the estimated effect could also be caused by mean reversion across municipalities. That is, if a temporary shock caused high tuberculosis infection rates and lower income before the campaign, we could observe an increase in schooling and income after the campaign, even in the absence of the direct effect of the control program on schooling and income. The specification in Panel C therefore additionally controls for average income in each municipality in 1930 interacted with the year of birth and the average percentage of inhabitants with a high school degree in each municipality in 1930 interacted with the year of birth. There is some evidence for mean reversion in the coefficients of $(T_j^{pre} \times Post_{ic})$, but they are only slightly smaller and remain significant.

Alternatively, we use Equation 2, where we spilt the sample at the median in municipalities with high versus low tuberculosis infection rates. Hence, this specification does not exploit the full variation in infection rates but classifies the municipalities in only two categories. Estimates of the variable of interest, $(H_j^{pre} \times Post_{ic})$, are displayed for various outcomes and specification in Table 7. Each cell denotes estimates of the coefficient on $(H_j^{pre} \times Post_{ic})$ from a separate regression. Panels A to C show results for different sets of control variables as described above. The results indicate that the tuberculosis control program had a significant positive effect on years of schooling. Individuals attending school during or after the tuberculosis control campaign in municipalities with above median infection rates have about 0.07 more years of education than individuals in municipalities with below median infection rates, the probability of having postmandatory schooling is 2.6 percent higher and earnings at age 37 is approximately 0.8 percent higher. As Equation 2 does not exploit the full variation in infection rates, the estimated effects are smaller than above, but still significant.

Some cohorts received exposure during all their school years to the tuberculosis campaign, whereas for other cohorts it was only for a few years. To account for the differences in the length of exposure to the tuberculosis testing and vaccination campaign while in school, we also allow the central variable of interest to vary by the number of years an individual is in school during the campaign and estimate Equation 3. Estimates of the coefficient of $(T_j^{pre} \times Exp_{ic})$ are reported for various outcomes in Table 8. Panels A to C show results for different sets of control variables as described above. Qualitatively, the results are similar to the results in Table 6 and suggest

[28] As discussed, we add the number of doctors per inhabitant and the student–teacher ratio in the year an individual commenced school, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between the local childhood mortality rate from pneumonia and the dummy variable $Post_{ic}$.

a significant increase in years of schooling, the probability of having high school education, and earnings at age 37 among cohorts exposed to the tuberculosis campaign in municipalities that had high levels of tuberculosis infection from 1940 to 1947. The coefficient on ($T_j^{pre} \times Exp_{ic}$) indicates that, on average, an individual in a municipality with a tuberculosis infection rate of 1 person in every 100 inhabitants and who was exposed to the campaign for one year while in school experiences a 0.06-year increase in years of education, a 2 percentage point increase in the probability of having high school education, and a 1 percent increase in earnings relative to individuals in municipalities without any tuberculosis cases prior to the campaign.

In general, the long-term effects are remarkably robust to successive inclusion of controls for municipality infrastructure characteristics, other diseases, and municipality-level income prior to the campaign. The results in Table 6 on years of education are comparable to the estimated effect of exposure to effective pneumonia treatment early in life (introduction of the sulfa drug in 1939) on the years of schooling in the US (see Bhalotra and Venkataramani, 2012, page 34). The long-term follow-up effects Bleakley (2007) found from the hookworm eradication program on log earnings, however, are somewhat larger than our estimated effects in Table 8.

### 5.3.1 Results by Gender

The prevalence of tuberculosis prior to the campaign was slightly higher for boys than for girls. Thus, boys potentially benefited more from the tuberculosis campaign. The existing literature also suggests that boys are more vulnerable to adverse health shocks during childhood and thus benefit more from medical innovations in childhood diseases (see, e.g., Low, 2000; Gluckman and Hanson, 2005). Focusing on both genders separately, we find that men and women benefited in a similar fashion from the tuberculosis campaign in terms of education (see Table 9). The benefits for women are slightly larger, although the difference is not significant. In terms of earnings, we only find significant results for men. A possible reason for the larger difference of the effect on earnings might be that the career opportunities for men born in the 1930s and 1940s were much better for men than for women. Thus, men could take advantage of the complementarities of better health and more accumulated human capital, whereas women in these cohorts would more likely drop out of the labor market after having children.

Better health might increase women's marriage market success. We therefore analyze the impact of the control program on the probability that a woman is married and her husband's earnings. Marriage rates for women born between 1930 and 1945 are very high (87%) and we do not find a significant increase in marriage rates among men or women that were exposed to the tuberculosis campaign in municipalities that had high levels of tuberculosis infection from 1940 to 1947 (see Table 9). However, we find that control campaign affected the match quality (see Table 9, Panel C). That is, a woman in a municipality that had a tuberculosis infection rate of 1 person per 100 inhabitants married on average men with 3 percent higher earnings at age 37 and 0.3 more

years of education compared with women in municipalities without any tuberculosis cases prior to the campaign. Hence, women's financial situation improved indirectly due to the disease control program.

### 5.4 Long-Term Consequences on Adult Height and Mortality

The effect of the tuberculosis control campaign on income is larger than the effect on education would suggest. Hence, individuals might benefit also in terms of better adult health besides the increase in human capital. On the other hand, improving children's health may not only raise the marginal benefit of education, it may also makes a child a more productive worker and thereby raise the marginal cost of education (see Bleakley, 2010, for a discussion). Hence, the greater importance of physical labor for individuals born between 1930 and 1945 in Norway may have depressed the effect on education and the effect on lifetime earnings might be a better indicator for the overall impact of the tuberculosis control campaign (see also Bleakley, Costa, and Lleras-Muney, 2014). Health measures are scarce for individuals born from 1930 to 1945. However, we are able to analyze whether individuals die at an early age and whether men's adult height increased more during the control campaign in areas with higher pre-treatment infection rates.

Decreased inflammation from reduced exposure to tuberculosis during early life may lead to lower morbidity and mortality from chronic conditions in old age. We use mortality before age 66—the life expectancy at birth of the cohorts of interest—as a long-term health outcome. In addition, we study whether the campaign affected deaths related to respiratory disease and cardiovascular disease.[29] We find a small but significant decrease in the probability of dying before age 66 among cohorts that were exposed to the tuberculosis campaign in municipalities that had high levels of tuberculosis infection from 1940 to 1947 (see Table 10). Thus, the coefficient on $(T_j^{pre} \times Post_{ic})$ indicates that, on average, an individual in a municipality that had a tuberculosis infection rate of 1 person per 100 inhabitants experienced a 1 percentage point decrease in the chance of dying before 66 compared with individuals in municipalities without any tuberculosis cases prior to the campaign. When looking at both genders separately, we only find significant effects for women. In line with our overall mortality estimates, we also find significant negative effects for cause-specific mortality from respiratory disease. This result suggests that reductions in the prevalence of respiratory disease as a cause of death drive the mortality effect discussed above. Again, we only find significant effects for women. The probability of dying from cardiovascular diseases, however, was unaffected.

As discussed in Section 2, tuberculosis was also known as 'consumption' as the disease led to weight loss and among children tuberculosis is associated with stunting (Olofin, McDonald, Ezzati,

[29] Respiratory disease could be a direct long-term consequence of lung damage from tuberculosis infections. Some cardiovascular diseases may be the long-term consequences of severe active tuberculosis infections. For example, tuberculosis may cause pericarditis, an inflammation of the outer surface of the heart, or cardiac tamponade, a fluid accumulation that may affect the heart's ability to pump effectively. Both conditions can be fatal.

Flaxman, Black, Fawzi, Caulfield, and Danaei, 2013). We therefore analyze the impact of the control program on adult height for men born in 1932-1933 and 1942-1945.[30] The results are displayed in Panel D in Table 10. We find a significant increase in adult height among men that were exposed to the tuberculosis campaign in municipalities that had high levels of tuberculosis infection from 1940 to 1947. Our finding of a positive impact of the disease campaign on men's health suggests that the effect the policy has on earnings might not entirely be driven through an increase in education, but also through better health. As women's labor market success may have benefitted less from an increase in physical strength, this result might also explain why the effect on earnings for women is not significant.

### 5.5 Parental Behavior and Spillovers to Older Siblings

In addition to the direct effects from better health during childhood, indirect mechanisms may explain the effects of the tuberculosis control program on education and earnings. Parents might change their behavior in response to having healthier children—for example, changes in relation to fertility choices and investments into other family members. These changes may either reinforce or counteract the direct effects of the program. Testing the behavioral responses of parents is difficult (see, e.g., Almond and Mazumder, 2013), as a second source of exogenous variation is needed to identify the exact mechanisms. However, we can provide two tests for parental behavior. First, we test whether a mother's completed fertility changes when her children benefits from the control program while in primary school. The number of children a woman has is an important family choice and a determinant of children's outcomes. Second, we study spillovers to older siblings to analyze whether families reallocate more resources to older children in the family.

The effect of the disease control program on the number of siblings and the number of younger siblings is shown in Table 11. Families did not change their (subsequent) fertility when one of their children was exposed to the control program while in school. Moreover, we find that completed years of education and the earnings of older siblings who had left school before the start of the control campaign are not significantly affected by the tuberculosis control campaign.[31] However, the effects are mostly positive and suggest that families might, to a small extent, compensate the direct effect of the program and allocate less resources to the healthy children.

The concern remains that nonrandom migration might change the composition of people in the municipality over time or that the location choice might be endogenous. Therefore, we estimate specifications including sibling fixed effects. Moreover, specifications with sibling fixed effects allow

[30] Data for adult height comes from various sources. The data for them men born in 1932 and 1933 are from military conscription records that can be linked to the registry data. The height measures for the men born from 1942-1945 are from two health surveys (see, e.g., Black, Devereux, and Salvanes, 2015, for a discussion of the health survey data).

[31] We consider older siblings of children affected by the program who are born between 1920 and 1933. As we observe earnings only in 1967 when the oldest cohorts are 47 years old, we consider earnings at age 47 instead for the older siblings.

us to analyze whether positive health shocks are reinforced by parental investments. We restrict the sample to families in which there are at least two children born between 1925 and 1945. The results are presented in Panel C of Table 11. The estimated effects are smaller than our main estimates, but mostly remain significant. As the estimated coefficients are smaller when family fixed effects are included, this indicates that positive health shocks are not reinforced by parental investment.

### 5.6 Long-Term Consequences on Inequality Reduction

In this section, we attempt to address the question of whether the reduction in the relative disease burden played a role in the subsequent decrease in inequality and the increase in intergenerational mobility in Norway. International comparisons show that Norway and other Nordic countries have a comparatively high intergenerational mobility in income and education today (Black and Devereux, 2011; Björklund and Salvanes, 2011). Family background, however, was a much more important determinant of adult income in the early twentieth century. For example, Björklund, Jäntti, and Lindquist (2009) show that the correlation in brothers' income fell substantially between cohorts born in the 1930s to those born in the 1950s in Sweden. For later cohorts, the correlation between brothers' earnings is rather stable. For Norway, Pekkarinen, Salvanes, and Sarvimäki (2017) find both a strong regional and socioeconomic convergence in the same period. Both studies indicate that changes in the number of school days may be an important factor behind the increasing intergenerational mobility for cohorts born between 1930 and 1950. Improvements in health conditions and the notable reduction in the relative disease burden for the poor might be another factor explaining this change.

Previous literature also shows that there is a socioeconomic gradient in children's health. For instance, Case, Lubotsky, and Paxson (2002) find a positive relation between socioeconomic status and children's health, and that this relationship becomes more pronounced over time. One mechanism could be that poor health leads to lower human capital accumulation, or alternatively, that negative shocks affect the poor more. In particular, chronic health conditions such as tuberculosis in childhood may be an important determinant of the socioeconomic gradient in children's health. Tuberculosis is a disease of poverty given its close link with overcrowding and malnutrition. Poor children in urban areas were highly exposed in the 1940s in Norway (Andresen, 2008), whereas wealthier parents were better able to establish a safer environment for their children and to purchase medical care and nutritious food. Thus, children from poor families may have benefited more from the free and mandatory treatment and the decrease in the prevalence of tuberculosis. These asymmetric benefits from the tuberculosis control program could have lowered health inequalities during childhood and thereby increased socioeconomic mobility later in life. To analyze the differing impacts of the control program on long-term outcomes for individuals from high and low socioeconomic backgrounds, we examine the long-run effects of the tuberculosis control program separately by socioeconomic background.

Table 12 displays the results from estimating Equation 1 for individuals from low and high socioeconomic backgrounds separately. As described in Section 4.1, we use fathers' education or profession as proxies to divide families into high and low socioeconomic status.[32] The estimated effects are higher for individuals from a low socioeconomic background. Most of the effects on the educational outcomes are not significantly different for high and low status individuals. However, our results indicate a significantly larger increase in earnings for individuals from a low socioeconomic background.[33] These findings suggest that the tuberculosis control program may well have lowered health inequalities during childhood and thus reduced income inequalities decades later.

In a further step, we aim to examine the role of the disease control program in shaping intergenerational mobility. We follow Pekkarinen, Uusitalo, and Kerr (2009) and estimate the effect of the disease control program on the persistence of educational attainment across generations. Hence, we test whether the disease control program can break the link in educational attainment between our cohorts of main focus and their fathers. We use a specification relating the completed years of education of the male individuals in our sample cohorts to a dummy variable $HSF_{ijc}$ indicating whether their father had a high school education interacted with $H_j^{pre} \times Post_{ic}$ and a full set of interactions between municipality and cohort dummies and a dummy variable representing whether the father had a high school education:

$$\begin{aligned} Y_{ijc} = \alpha + \mu HSF_{ijc} + \eta HSF_{ijc}(H_j^{pre} \times Post_{ic}) + \beta X_{ijc} + \\ \xi_c HSF_{ijc} + \tau_j HSF_{ijc} + \phi HSF_{ijc} X_{ijc} + \delta_c + \theta_j + \varepsilon_{ijc}. \end{aligned} \tag{6}$$

We identify the effect of the disease control program on the persistence of educational attainment across generations $\eta$ from the second-level interactions, that is, from the changes in the effect of father's education occurring at the time of the control program. Given the cohort and municipality fixed effects, we identify the effect of the tuberculosis control program on the persistence of educational attainment across generations. That is, we study whether the tuberculosis control program tightened or relaxed the intergenerational persistence in education.

Table 13 presents the main regression results from Equation 6. In Column A, we report the results of the effect of the son's completed years of education on the father's high school status, the term $(H_j^{pre} \times Post_{ic})$, along with the interaction between $(H_j^{pre} \times Post_{ic})$ and father's education. The coefficient of the interaction term of interest is -0.286, indicating that the intergenerational persistence of education is lower in municipalities with a high tuberculosis prevalence after the

[32] As there is little variation in mothers' education, we focus on fathers' education or profession.

[33] As tuberculosis is a disease of poverty, the larger effects for individuals from poor families might be a result of the higher baseline tuberculosis infection rates among the poor. If only individuals infected with tuberculosis benefit from the control campaign, the reduced form effect might be the same for individuals from a low or a high socioeconomic background. As we do not know which exact individual was suffering from active tuberculosis, we cannot analyze the differences in the estimated reduced form effects. Moreover, as discussed in Section 5.3 it is very likely that children from a high socioeconomic background who never suffered from active tuberculosis benefit in terms of education from having fewer low socioeconomic classmates that miss school a lot or that need extra attention.

disease control campaign by about 0.3 years. The estimate is statistically significant at the 1 percent significance level and represents a 14.3 percent reduction in the persistence of educational attainment across generations compared with the precampaign level. The set of controls is increased in Column B and includes controls for heterogeneity across municipalities as well as interactions of these variables with the father's education status.[34] In Column C, additional controls are added to control for mean reversion across municipalities as well as interactions of these variables with the father's education status.[35] The estimated effect of the reduction in persistence is slightly smaller when all control variables are included. The effects are, however, still significant at the 1 percent significance level and amount to about a 13.7 percent reduction in the intergenerational persistence of education compared with its precampaign level. Hence, we find that the tuberculosis control campaign reduced the association of fathers' education and the sons' education. These results suggest that policies that improve childhood health may significantly enhance intergenerational education mobility.

Note that not all public campaigns aimed at improving child health necessarily enhance intergenerational education mobility. As discussed earlier, tuberculosis is more prevalent among poor families and might therefore be particularly well suited to affect intergenerational mobility. While the public health campaigns against diseases such as polio, which was more randomly distributed across the socioeconomic spectrum, might affect intergenerational mobility less, there are many other diseases and health threats that are more prevalent among poor children. Examples include lead exposure, exposure to toxins, pneumonia, measles, AIDS, malaria, intestinal parasites, or dental decay. Hence, the result might not be generalizable to all disease eradication campaigns, but to a large variety of ongoing public health programs in both developed and developing countries.

### 5.7 Long-Term Consequences on the Second Generation

Our data allow us to link across generations and provides a rare opportunity to examine the effect of exposure to the tuberculosis control program on the children of those exposed. Only few studies analyze the intergenerational transmission of health shocks and most of these papers focus on the multi-generational impact of *negative* shocks such as famine, influenza, or radioactive fallout (Almond, Edlund, Li, and Zhang., 2010; Painter, Osmond, Gluckman, Hanson, Phillips, and Roseboom, 2008; Black, Bütikofer, Devereux, and Salvanes, 2013; Richter and Robling, 2016).

There are two possible mechanisms through which the benefits from the program exposure could be transmitted across generations. The first and most direct is through biological changes. The medical literature focuses mostly on the health risks of pregnant mothers who are tuberculin

[34] Controls for heterogeneity across municipalities include the number of doctors per inhabitant and the student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term for a dummy variable indicating whether someone was born after the introduction of antibiotics and the local childhood mortality rate from pneumonia

[35] To control for mean reversion we include the average income in each municipality in 1930 and average percentage of inhabitants with a high school degree in each municipality.

positive and on the health risks of children of tuberculosis infected mothers. The immunological changes associated with pregnancy make it more likely for women to contract tuberculosis during pregnancy and increase the risk of re-activation (Zenner, Kruijshaar, Andrews, and Abubakar, 2012). Moreover, infants born to mothers who have active tuberculosis may contract tuberculosis congenitally, which is associated with a range of birth defects (Lowe, 1964). Jana, Vasishta, Jindal, Khunnu, and Ghosh (1999) and Figueroa-Damian and Arredondo-Garcia (2001) show that maternal tuberculosis is associated with increased morbidity, lower birth weight, and an increased risk of infant death. The other channel for intergenerational effects is a more indirect mechanism. As the program improves socioeconomic outcomes of exposed parents, this could lead to better outcomes for their children.

To avoid confusion, we will refer to persons born between 1930 and 1945 as the first generation and refer to their children as the second generation. Of our sample of people born between 1930 and 1945, 73% of men and 76% of women have at least one child for whom we observe adult outcomes. A likely explanation for this disparity is that women have children at a younger age than men do, increasing their likelihood of having children who are old enough to be in our sample.

A key issue that arises in this analysis is whether there is selection into the sample. To address this, Table 14 shows that exposure to the tuberculosis control program has no effect on the probability of having a child for whom we observe adult outcomes and thereby on the probability of having a child in the second-generation sample. Moreover, the results show that neither the number of children nor the age at first birth are affected by the tuberculosis control program. More broadly, we show that the fertility behavior of the first generation is not affected by the exposure to the tuberculosis control program. This suggests that selection into our second generation sample is unlikely to be an issue.

We estimate the intergenerational effects of exposure to the tuberculosis control program using the same control variables that we used when studying outcomes for the first generation that were pre-determined at the time of the control program. The regressions are weighted such that each first generation parent gets equal weight. We run separate regressions for the children of first generation men and women. Table 15 presents the results. We find statistically significant positive effects of exposure of first generation women on birth weight, years of education, log earnings, and height at age 19 (for boys only) of the second generation. For exposed first generation men, the effects are also positive and statistically significant for their children's years of education and log earnings. As mother's health is an important factor for a child's health at birth, the significant effect of mother's exposure on children's birth weight may imply that program-exposed mothers were of better health. Although our findings are weaker than for the first generation, they still imply that exposure to the tuberculosis control program was important for the second generation. Importantly, these results suggests that a substantial proportion of the positive effects of exposure to the control program is passed on from parents to their offspring.

As in Section 5.6, we can also study the role of the disease control program in shaping intergenerational mobility over multiple (male) generations. These mobility trends over multiple generations are not necessarily monotonic (Nybom and Stuhler, 2013). Hence, it is an empirical question whether a shock that reduces intergenerational persistence in the directly affected generation also decrease in intergenerational persistence for the next generation or whether family ties tighten again. Using Equation 6, we identify the effect of the tuberculosis control program on the persistence of educational attainment across generations. Panels B and C in Table 13 present the results. In Panel B, we report the results of the effect of the second generation son's completed years of education on his father's high school status, the term ($H_j^{pre} \times Post_{ic}$), along with the interaction between ($H_j^{pre} \times Post_{ic}$) and his father's education. In Panel C, we report the results of the effect of the second generation son's completed years of education on his paternal grandfather's high school status, the term ($H_j^{pre} \times Post_{ic}$), along with the interaction between ($H_j^{pre} \times Post_{ic}$) and his grandfather's education.

The effect of interest is the coefficient of the interaction term, which indicates whether the intergenerational persistence of education is higher or lower in municipalities with an initially high tuberculosis prevalence after the disease control campaign. In Panel B, the estimate is negative but not statistically significant at the 5 percent significance level. Comparing second generation sons with their paternal grandfathers in Panel C, yields a coefficient of the interaction term of interest is -0.202. The estimate is statistically significant at the 1 percent significance level and indicates that the intergenerational persistence of education between grandfathers and their grandsons is lowered by the control program. However, the change in the persistence parameter is slightly lower than when comparing first generation men with their fathers (see Section 5.6).[36] Hence, we find that the tuberculosis control campaign reduced the association of grandfathers' education on the second generation sons' education. On the other hand, there are no tuberculosis-related differences in the intergenerational mobility between fathers and second generation sons. As argued by Nybom and Stuhler (2013), policies may reduce the transmission of social status in the directly affected generation. However, this decrease in intergenerational persistence is not necessarily still in place for the next generation as family ties might tighten again and society might enter a new steady state with lower intergenerational mobility.

# 6 Sensitivity Analysis

We present a variety of sensitivity tests. First, we perform a subgroup analysis excluding the two northernmost Norwegian counties, which were relatively poor and most affected by tuberculosis.

[36] In all columns, we include a full set of cohort and municipality fixed effects and interact these effects with the fathers' education. The set of controls is increased in Column B and includes controls for heterogeneity across municipalities as well as interactions of these variables with the father's education status. In Column C, additional controls are added to control for mean reversion across municipalities as well as interactions of these variables with the father's education status. Including further controls does not change the results to a large extend.

Second, we discuss whether different types of heating systems, which may affect air quality and thereby the spread of lung diseases, are correlated with both the tuberculosis exposure and accomplishment later in life and thus threaten our identification strategy. Third, we analyze how varying the timing of the treatment assignment affects the results. Fourth, we provide some falsification checks. Fifth, we include municipality specific time trends. Finally, we use detailed information on the exact year a subsample of municipalities were tested for the first time and use a simple difference-in-difference estimation exploiting the program rollout.

In Panel A of Table 16, we exclude the two northernmost counties of Finnmark and Troms.[37] Most point estimators are larger; the differences, however, are not significant.

The medical literature links childhood tuberculosis to exposure to passive smoking and both indoor and outdoor air pollution (Sumpter and Chandramohan, 2013; Hwang, Kang, Lee, Lee, Kim, Han, and Yim, 2014). In particular, the burning of biomass fuels such as wood is an important concern. We collected data on the percentage of households in each municipality using biomass fuels for heating. In contrast to findings in the medical literature, the tuberculosis infection rates in different municipalities negatively correlate with the percentage of households heating their houses with biomass fuels in cross sections of Norway. To ensure that differential time trends in the percentage of households using biomass fuels are not driving our results, we include these data as a further control in Equation 1. The results are reported in Panel B in Table 16. Controlling for the percentage of households heating with biomass fuels does not alter the results presented in Section 5.3.

The variable $Post_{ic}$ in Equation 1 describes the cohort variation generated by the launch of the tuberculosis control program and is equal to one for all cohorts leaving school in 1949 and after. Hence, some cohorts are exposed to the tuberculosis control campaign during all their school years while other cohorts are exposed for only one year. To test how sensitive our results are to this definition, we use alterative assignment cutoffs and define an individual as exposed to the public health program if she was exposed at least seven, six, five, four, three, or two years. The results are displayed in the Table 17 (Panels A to F). Our main findings in Table 6, where we define an individual as exposed if she was exposed at least one year, are mostly lower than for the alternative timing for the treatment assignment. Hence, our main results are conservative estimates of the impact of the tuberculosis control program.

To analyze whether the control campaign is most effective for school children and less for toddlers who were less affected by tuberculosis, we define $Post_{ic}$ in Equation 1 to be equal to one if an individual is three years old or younger when the tuberculosis control program in launched. We include cohorts born from 1930 to 1950 in this falsification test. The results are displayed in Panel G of Table 17 and show that reassigning the treatment to toddlers instead of schoolchildren does not yield significant results.[38]

[37] These two counties suffered most destruction during World War II.

[38] Note that the standard errors are similar to the standard errors in Table 6. Hence, the insignificant effects for

As noted in Section 3, our identification strategy is not valid if average outcomes in municipalities with high and with low tuberculosis infection rates have different time trends prior to the campaign. For this reason, we study whether including municipality-specific time trends in our main specification (Equation 1) alters our results. Panel C in Table 16 shows that including a linear municipality-specific time trends lowers our main results slightly. However, the effects are still significant and substantially large.

From the reports of the NMRS (Statens-Skjermbildefotografering, 1949, 1950, 1951, 1952), we have detailed data about the first time inhabitants in a subsample of municipalities were tested and vaccinated from 1949 to 1952. We use this information and estimate a difference-in-difference model, which exploits the program rollout:

$$y_{ijc} = \alpha + \eta D_{jc} + \beta X_{ijc} + \delta_c + \theta_j + \varepsilon_{ijc},$$

where $Y_{ijc}$ denotes the outcomes of interest recorded in adulthood for individual $i$ born in municipality $j$ in cohort $c$. $X_{ijc}$ is a vector of individual-level demographic characteristics, $\delta_c$ are cohort dummies, and $\theta_j$ are municipality fixed effects. $D_{jc}$ is an indicator variable taking a value of one if an individual leaves school in or after the year the campaign tested individuals in the municipality of birth and zero otherwise. The coefficient of interest is $\eta$, which shows the effect of the campaign on various outcomes. We cluster standard errors at the municipality of birth level. As we have detailed information about the program only for four years and only for a subset of municipalities, we do not expect the effects to be as precisely estimated as in Section 5.3, but they should point in the same direction. The results are reported in Panel D in Table 16. We find that the years of completed education increase by 0.11 if an individual left school after the municipality of birth was tested and vaccinated against tuberculosis for the first time. The average years of education precampaign were 9.2 years. Thus, we find that the campaign increased the number of years of schooling by about 1 percent. On the other outcomes, we do not find significant effects from the rollout during the first four years of the campaign. This identification strategy using the rollout of the vaccination campaign allows us to compare our results with other studies analyzing different vaccination campaigns. Lee (2012) finds that the introduction of mandatory school vaccination laws in the 1970s in the US increased years of schooling by 0.12 years. This effect is very similar to our estimated effect using the difference-in-difference strategy. This empirical strategy uses variations in when and where the public health campaign tested and vaccinated the population to evaluate the effect of the program on long-term economic outcomes. Hence, we assume that the timing of the arrival of the tuberculosis control campaign is uncorrelated with other determinants of changes in long-term economic outcomes. To distinguish the effect of the control program from differential secular trends, we test for the existence of precampaign trends with an event-study framework. Figure 9 plots event-study estimates as well as the 95 percent confidence intervals for the four main

toddlers are not due to imprecise estimates.

outcomes. Note that we only find significant results in Panel D in Table 16 for years of education. The results provide no evidence of a differential trend in education in treated municipalities before the campaign arrived in the municipality. The estimates of the precampaign effects are relatively small in magnitude and not statistically different from zero at the 5 percent significance level. In the year following the arrival of the control campaign to the municipality, education increases and most effects are statistically significant at the 5 percent significance level. For the other three outcomes we do not find significant results in the generalized difference-in-difference model. However, none of the estimates of the precampaign effects are statistically different from zero at the 5 percent significance level and provide no evidence of a differential trend in the outcomes in treated municipalities before the campaign arrived in the municipality.

# 7 Discussion

In this paper, we present evidence that the tuberculosis testing and vaccination campaign increased education and earnings more for individuals in municipalities with high tuberculosis prevalence. To understand the economic magnitude of our results, we study whether the program benefits outweigh the costs of the program.[39]

Table 3 reports the program costs. The costs per tested individual vary between about NOK 4 to NOK 6 (in 1950 NOK). As each individual was tested about three to four times during the campaign, the approximate cost per individual was at most NOK 24. At today's price levels and exchange rates, this corresponds to about USD 55. Our estimated effects translate into differences between municipalities with a tuberculosis infection rate of 1 person per 100 inhabitants compared with municipalities without any tuberculosis cases. About 45 percent of the individuals in our sample live in a municipality with a tuberculosis infection rate of 1 person per 100 inhabitants or higher. For all other individuals, the benefits of tuberculosis control are smaller. Hence, the campaign costs for those who potentially benefit most are about 2.2 times the amount of the per-person costs computed above and therefore USD 122. While the costs incur when the children are 14 years old or younger, the benefits in terms of earnings occur when the children enter the labor market. We therefore assume that people work from age 21 to 65 and that annual earnings at age 37 reflects the average annual life-time earnings. Thus, the present value of the benefits is given by $\sum_{t=21}^{65} \frac{0.071w}{(1+r)^t}$, where 0.071 is the program's effect on earnings and $w$ is average annual earnings. The internal rate of return, that is the discount rate that equalizes the present values of costs and benefits, is then 0.085. However, a large percentage of tested individuals were adults, with only about 10 percent of the tested individuals being teenagers or school-age children. Hence, the calculated internal rate of return is an upper bound as our estimated effects on income are valid only for every tenth tested person. To compute a lower bound, we divide the total program costs

[39] We will build on the assumptions made in Fredriksson, Öckert, and Oosterbeek (2013) where they calculate the cost-benefit of reducing class size.

by the number of tested individual aged 14 years or younger. This exercise yields an internal rate of return of 0.032. We conclude that the tuberculosis testing and vaccination campaign passes a cost-benefit analysis in the context we study.

However, we should interpret our calculations with some caution. The costs are limited to the direct program costs and do not include the costs of the municipality doctors or potential costs municipalities bore in conjunction with calling in all residents for the testing. In addition, the costs for treating tuberculosis with antibiotics or hospitalization costs are not included.

# 8 Conclusion

The objective of this paper is to evaluate the economic consequences of a tuberculosis control program in Norway that successfully lowered the tuberculosis infection rates. This specific intervention is of interest as its timing is well defined, the program was rapidly implemented, the geographical variance in infection rates permits a treatment/control design (see, e.g., Bleakley, 2007), and the intervention took place sufficiently long ago that we can evaluate the long-term consequences.

We find that the affected individuals in municipalities with a high tuberculosis infection rate prior to the campaign experienced a decrease in the percentage of missed school days and a substantial gain in earnings and education relative to individuals in municipalities with a low tuberculosis infection rate. The effect of the tuberculosis control campaign on earnings is larger than the effect on education would suggest. Hence, individuals might benefit also in terms of better adult health besides the increase in education. We show that there is a significant increase in longevity and adult height suggesting that the effect the policy has on earnings might not entirely be driven through an increase in education, but also through better health. Analyzing gender differences, we find that men benefited more in terms of earnings and women more in terms of longevity. In addition, women had an indirect financial benefit from the campaign: our results suggest that affected women marry men with higher earnings and higher education. We show that the positive effect of the disease control campaign on schoolchildren is unlikely mostly driven by positive effects on parental health and household resources. Moreover, our results indicate that children affected by the policy are not benefitting form higher parental investment. In addition, our results indicate that the effect was larger for individuals from a low socioeconomic background. Moreover, we find that the disease control program reduced the effect of fathers' education on sons' education by about 0.3 years. This amounts to a 14 percent decline in the persistence of educational attainment across generations. These results suggest that policies that improve childhood health may significantly enhance the intergenerational mobility of education. Hence, we present new evidence that the narrowing of a gap in childhood health (here, because of a tuberculosis campaign) can lead to a reduction in socioeconomic inequalities in adulthood. Furthermore, we estimate the intergenerational effects of exposure to the tuberculosis control program and find statistically significant positive effects of exposure of the directly affected generation on birth weight, years of education, log earnings, and

height at age 19 (for boys only) of the children of the affected generation. These findings suggest that a substantial proportion of the positive effects of exposure to the control program is passed on from parents to their offspring. Moreover, we show that although the control campaign reduced the transmission of social status in the directly affected generation, this decrease in intergenerational persistence in education is no longer in place for the next generation.

A key strength of our analysis is also a drawback: to study the long-term effects of the tuberculosis testing and vaccination campaign, we need to consider reforms that happened a long time ago. The health situation and the challenges faced by public health systems in the developed world are different. This makes it difficult to generalize our results to current policies (see also Ludwig and Miller, 2007, for a discussion). The present results, however, suggest potentially substantial benefits of testing and vaccination campaigns in countries where tuberculosis is still widely present. Today, more than one million people die of the disease every year and around one-third of the world's population are believed to be infected (Lougheed, 2017). Due to the compounding problems of drug resistance, HIV epidemics, and poverty, tuberculosis remains one of the most serious problems in world medicine. Nevertheless, it remains an open question whether the gains from tuberculosis control estimated for Norway could also be realized in developing countries. The tuberculosis screening and vaccination campaign in Norway relied on a range of institutional infrastructure factors, such as district doctors and roads or seaports, as well as extensive follow-up testing. In addition, schools need to be in place so that children are able to benefit from the health improvements. However, there are clear similarities of the Norwegian tuberculosis control campaign in the 1940s and 1950s and, for example, current tuberculosis awareness campaigns in India. As in pre-1950s Norway, much of India's tuberculosis problem is socioeconomic—poor housing, poor sanitation, overcrowding, and unhealthy populations both at higher risk of developing active tuberculosis and with limited access to adequate health care. Hence, programs where volunteers visit vulnerable populations to provide information on tuberculosis and refer those with who cough for more than two weeks for examination to public health facilities is part of the solution to ensuring that people receive treatment, do not continue to transmit their infection to others, and potentially achieve a long-term increase in economic outcomes (Prasad, Satyanarayana, and Chadha, 2016). Furthermore, our results give some indication that current policies in developed countries targeting health threats that are more prevalent among poor children (e.g., asthma or lead exposure) might have some long-term and even intergenerational effects.

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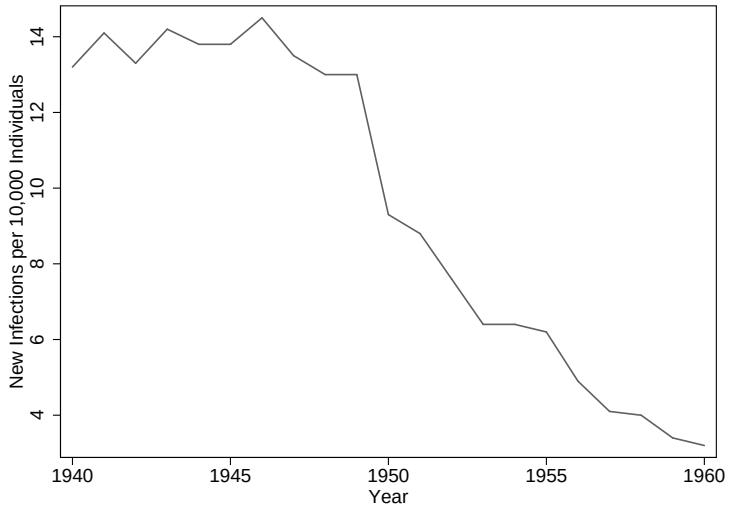
# 9 Tables and Figures

Figure 1: Rollout of the Tuberculosis Control Program

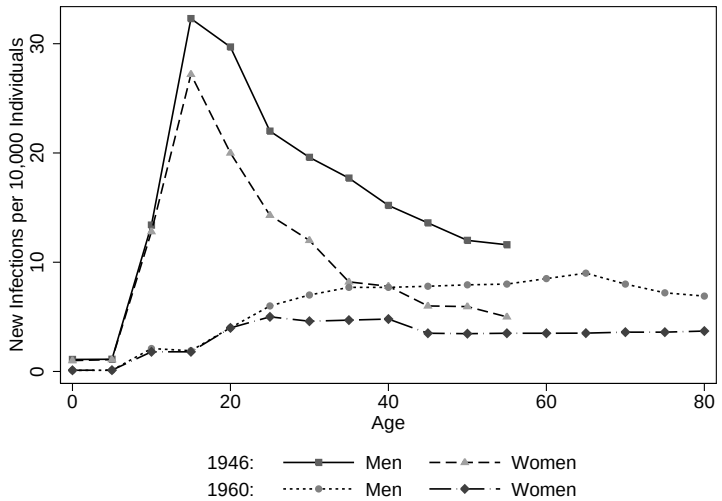

*Notes:* The map displays the municipalities in which inhabitants were tested for the first time from 1948 until 1952. Data source: NMRS's yearly reports.

Figure 2: Number of New Tuberculosis Infections in 10,000s of Individuals



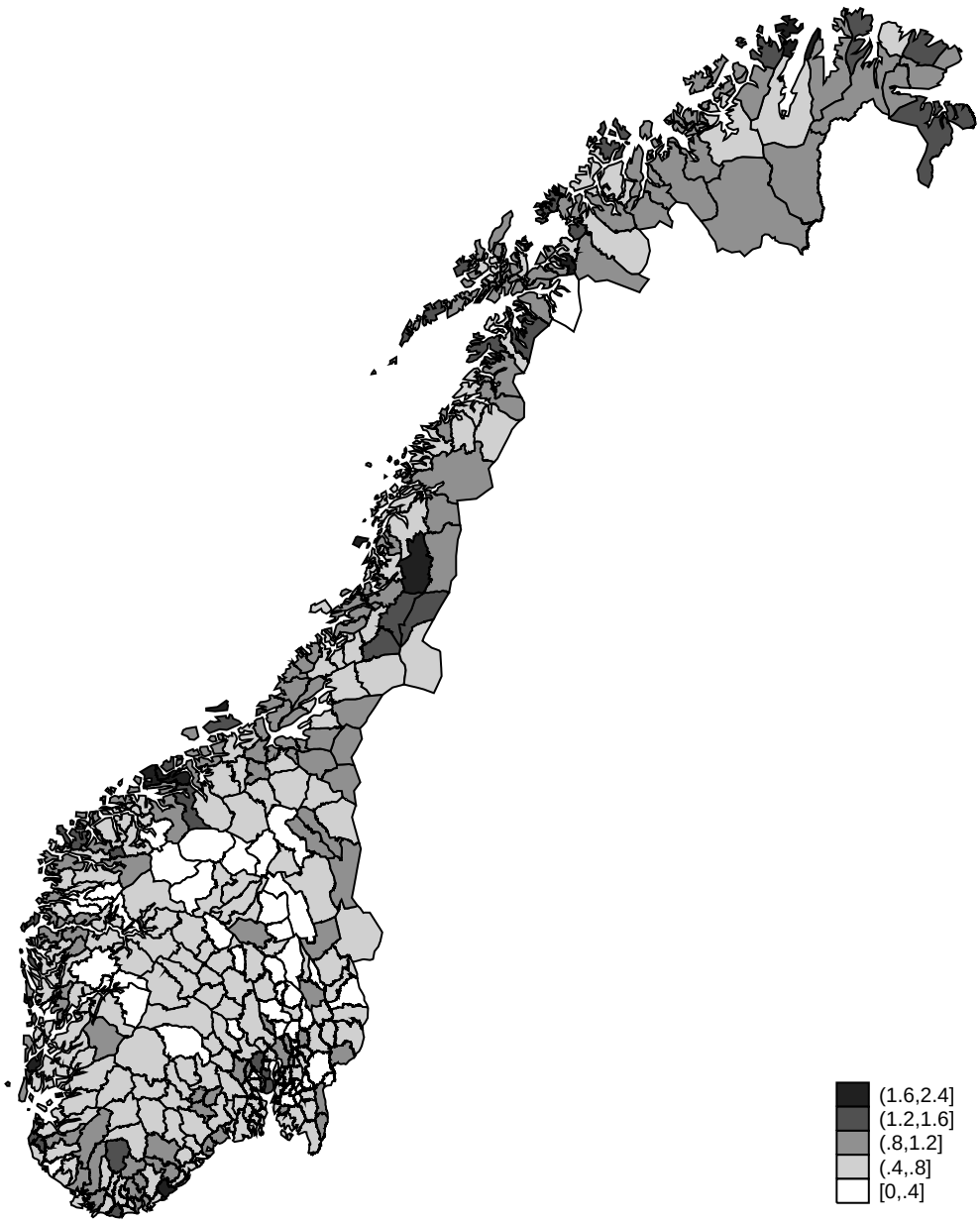

*Notes:* The figure plots the number of new active tuberculosis infections per 10,000 inhabitants from 1940 to 1960. Data source: Statistics Norway's historical yearly health statistics.

Figure 3: Number of New Tuberculosis Infections per 10,000 Individuals by Gender and Age



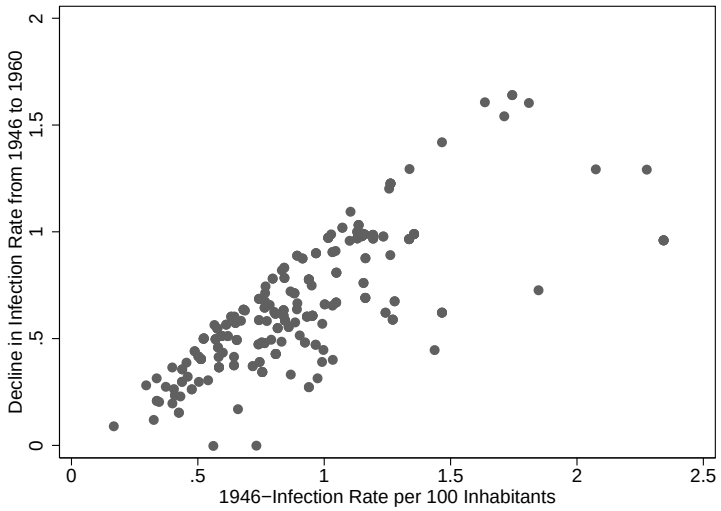

*Notes:* The figure plots the number of new active tuberculosis infections per 10,000 inhabitants in 1946 and 1960 by gender and age. Data source: Statistics Norway's historical yearly health statistics.

Figure 4: Tuberculosis Infection Rate by Municipality



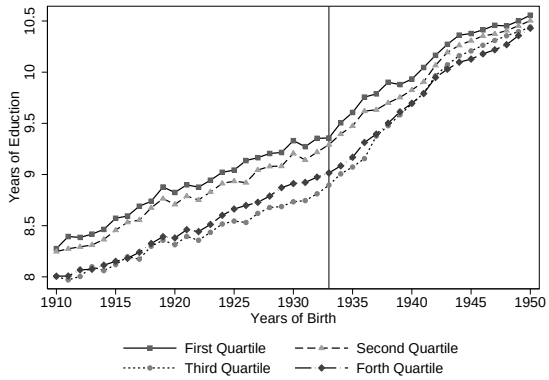

*Notes:* The map displays the average number of individuals infected with active tuberculosis by the end of the year per 100 inhabitants from 1940 to 1946 by municipality. Data source: Statistics Norway's historical yearly health statistics.

Figure 5: Highly Infected Areas Saw Greater Decline in Tuberculosis Infection Rates

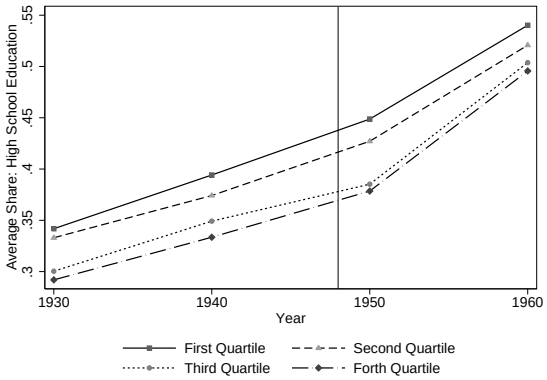



*Notes:* The y-axis displays the decrease in tuberculosis infection rates post-intervention. That is the difference between the precampaign average infection rate (average number of individuals infected with active tuberculosis by the end of the year per 100 inhabitants from 1940 to 1946) and the number of individuals infected with active tuberculosis by the end of the year 1960 per 100 inhabitants. The x-axis is the number of individuals infected with active tuberculosis by the end of the year 1946 per 100 inhabitants. The data are at the municipality level. Data source: Statistics Norway's historical yearly health statistics.

Figure 6: Trends in Education by Tuberculosis Infection Rates



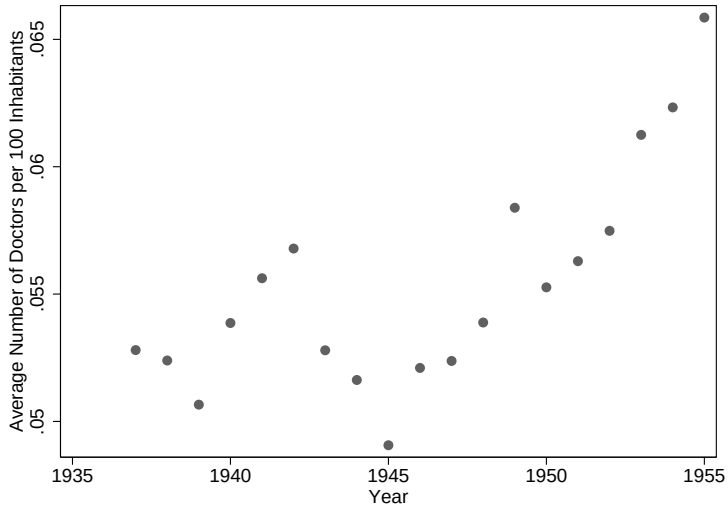

(a) Years of Education by Cohorts




(b) Probability of High School Education by Year

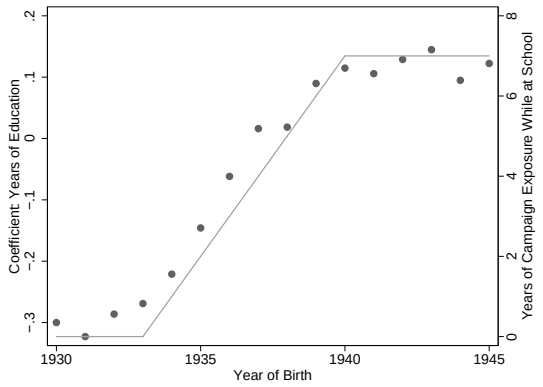
*Notes:* Figure (a) plots the average years of education by birth cohorts from 1910 to 1950. The different lines denote averages in municipalities in the first, second, third, and fourth quartile of the distribution of tuberculosis infection rate per 100 inhabitants by municipality from 1940 to 1946. Municipalities are allocated to individuals using the place of residence in 1960. The vertical line marks the 1933 cohort—the first cohort affected by the disease control campaign while in school. Data source: 1960, 1970, and 1980 census data. Figure (b) plots the average share of individuals between 20 and 30 years of age who completed high school per municipality in 1930, 1940, 1950, and 1960. The different lines denote averages in municipalities in the first, second, third, and fourth quartile of the distribution of tuberculosis infection rate per 100 inhabitants by municipality from 1940 to 1946. Municipalities are allocated to individuals using their place of residency in either 1930, 1940, 1950, and 1960. The vertical line marks the start of the disease control campaign in 1948. Data source: 1930, 1940, 1950, and 1960 census data.

Figure 7: Average Number of Doctors per 100 Inhabitants per Year

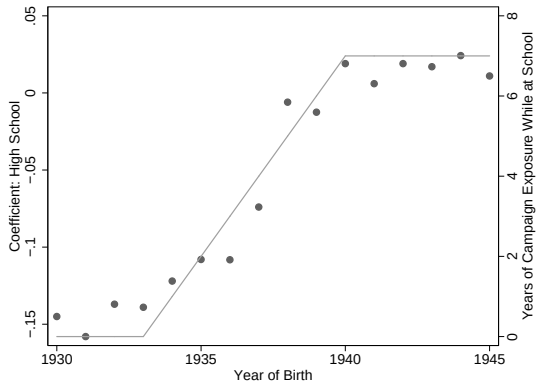



*Notes:* The figure plots the average number of doctors per 100 inhabitants in each year. Data source: Statistics Norway's historical yearly health statistics.

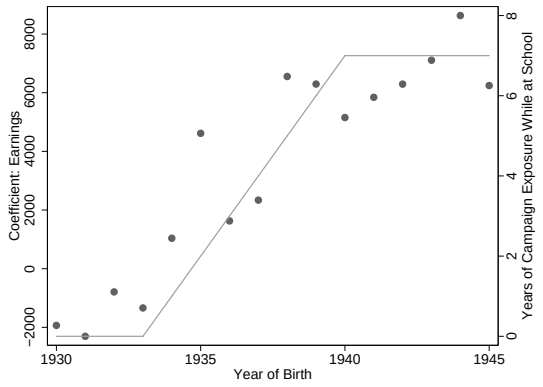
Figure 8: Cohort-Specific Relationships: Long-Term Outcomes and Pre-Campaign Tuberculosis Infection Rates



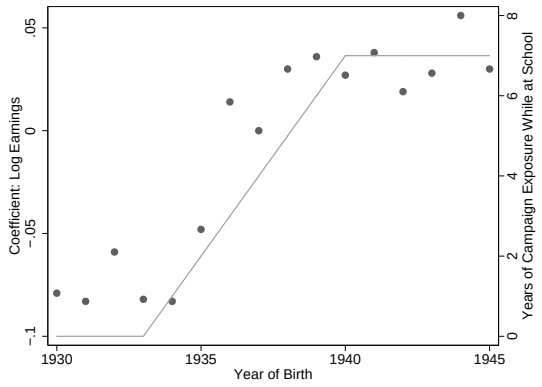

(a) Years of Education




(b) Probability of High School Education



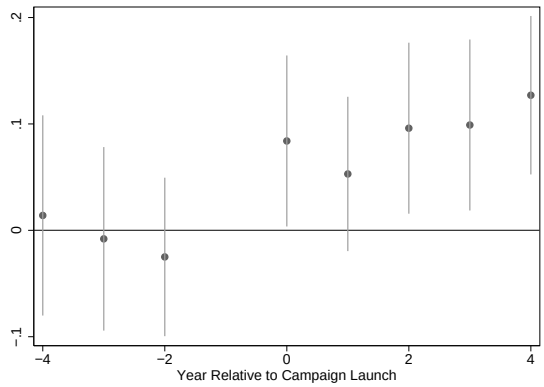

(c) Earnings Age 37



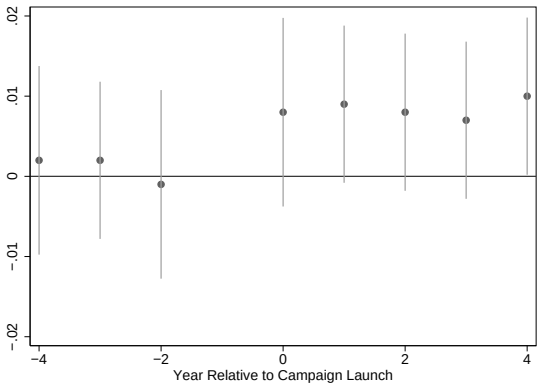

(d) Log Earnings Age 37

*Notes:* The figures plot regressions of education and earnings measures on pre-campaign tuberculosis infection rates by municipality. The left y-axis plots the estimated cohort-specific coefficients on the municipality-level tuberculosis measure. The x-axis is the cohort's year of birth. Each dot represent the cohort-specific coefficient on tuberculosis. The lines indicate the number of years of potential exposure to the tuberculosis control campaign while an individual was enrolled at school. The OLS regression coefficients are estimated for each cohorts separately. The specification contains municipality specific fixed effects, the number of doctors per inhabitant and the student–teacher ratio in each municipality.

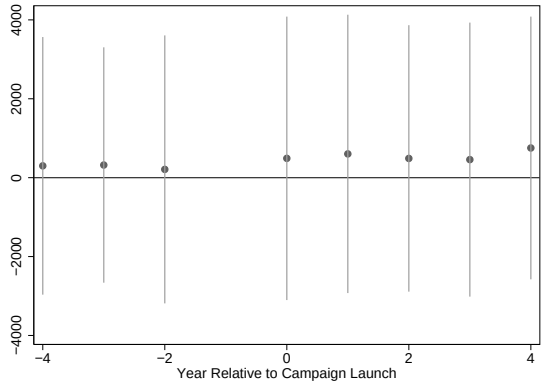
Figure 9: Event-Study Estimates of the Impact of Exposure to the Disease Control Program



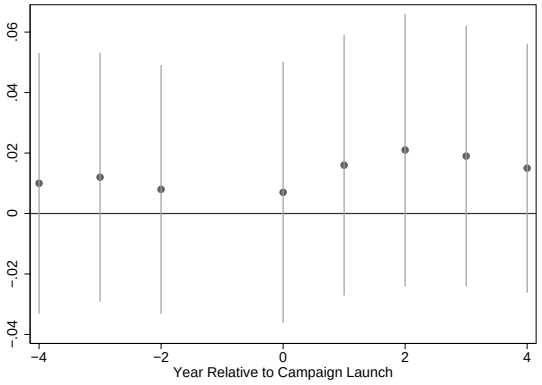

(a) Years of Education




(b) Probability of High School Education




(c) Earnings Age 37




(d) Log Earnings Age 37

*Notes:* The figures plot the post-treatment and anticipatory effects from an event-study specification as well as the 95 percent confidence intervals. Included control variables are municipality dummies and year of birth dummies, the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county, and the local childhood mortality rate from pneumonia. The omitted year is the year before the campaign start.

Table 1: Descriptive Statistics

| | Whole Sample | Men | Women |
|---|---|---|---|
| **Disease spread** | | | |
| Average of tuberculosis infections | 0.861 | 0.861 | 0.861 |
| per 100 inhabitants during 1940–1946 | (0.378) | (0.378) | (0.378) |
| **Outcomes** | | | |
| Years of education | 9.86 | 10.22 | 9.50 |
| | (2.60) | (2.88) | (2.23) |
| High school education | 0.556 | 0.597 | 0.513 |
| | (0.497) | (0.490) | (0.500) |
| Earnings at age 37 years in NOK | 149,053 | 229,963 | 67,427 |
| | (125,191) | (108,676) | (79,356) |
| Early Mortality (before age 66) | 0.432 | 0.441 | 0.418 |
| | (0.495) | (0.496) | (0.493) |
| Early Mortality from | 0.018 | 0.015 | 0.022 |
| respiratory diseases | (0.132) | (0.122) | (0.147) |
| Early Mortality from | 0.080 | 0.104 | 0.043 |
| cardiovascular diseases | (0.272) | (0.305) | (0.203) |
| **Socioeconomic Status** | | | |
| Father has high school education | 0.277 | 0.272 | 0.283 |
| | (0.448) | (0.445) | (0.451) |
| Father has high status profession | 0.334 | 0.327 | 0.341 |
| | (0.472) | (0.469) | (0.474) |
| **Municipality-level controls** | | | |
| Number of doctors per | 0.082 | 0.082 | 0.082 |
| 100 inhabitants | (0.073) | (0.073) | (0.073) |
| Student–teacher ratio | 26.3 | 26.3 | 26.3 |
| | (8.94) | (9.19) | (8.69) |
| Percentage of missed | 0.076 | | |
| school days (1946) | (0.074) | | |
| Percentage of individuals with | 0.027 | 0.027 | 0.027 |
| high school degree in 1930 | (0.017) | (0.017) | (0.017) |
| Average income in 1930 | 1090.2 | 1090.2 | 1090.2 |
| (in 1930 NOK) | (457.7) | (457.7) | (457.7) |
| Number of observations | 444932 | 223447 | 221485 |

*Note*: Means and standard deviations in parentheses.

Table 2: Tuberculosis Testing and Vaccination Program

| Year | No. of X-rayed individuals | No. of test days | Percentage tuberculin-positive men | Percentage tuberculin-positive women | No. of individuals vaccinated |
|---|---|---|---|---|---|
| 1949 | 361,092 | - | 62 | 50 | 60,214 |
| 1950 | 393,133 | 1,201 | 68 | 55 | 62,023 |
| 1951 | 357,659 | 1,204 | 68 | 55 | 60,069 |
| 1952 | 308,153 | 1,220 | 71 | 59 | 48,935 |

*Note*: Data source: NMRS's yearly reports.

Table 3: Program Costs in 1950 NOK

| Year | Total Costs | Average Costs by Tested Individual |
|---|---|---|
| 1948 | 1,391,449 | 4.32 |
| 1949 | 1,371,637 | 3.80 |
| 1950 | 1,450,333 | 3.69 |
| 1951 | 1,681,194 | 4.70 |
| 1952 | 1,969,475 | 6.39 |

*Note*: Data source: NMRS's yearly reports.

Table 4: Contemporaneous Effects on Missed School Days

| | | | | |
|---|---|---|---|---|
| **Panel A: Basic Results** | | | | |
| | Whole sample | Rural municipalities | Postwar years | Without north |
| TB infection rate | -0.024** | -0.024** | -0.018** | -0.010* |
| × Post dummy | (0.005) | (0.005) | (0.004) | (0.005) |
| **Panel B: Heterogeneity Across Municipalities** | | | | |
| | Whole sample | Rural municipalities | Postwar years | Without north |
| TB infection rate | -0.024** | -0.024** | -0.018** | -0.010* |
| × Post dummy | (0.005) | (0.005) | (0.004) | (0.005) |
| **Panel C: Heterogeneity Across Municipalities and Mean Reversion** | | | | |
| | Whole sample | Rural municipalities | Postwar years | Without north |
| TB infection rate | -0.025** | -0.025** | -0.018** | -0.011* |
| × Post dummy | (0.005) | (0.005) | (0.005) | (0.005) |
| Number of observations | 4290 | 4136 | 2340 | 3641 |
| Number of groups | 390 | 376 | 390 | 331 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each column contains a different sample. Each estimate is from a different regression and is the coefficient on ($T_j^{pre} \times Post_t$). Included control variables are municipality dummies and year of birth dummies. The set of controls is enlarged in Panel B and includes controls for heterogeneity across municipalities (the number of doctors per inhabitant and the student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_t$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia). In Panel C, additional controls are added to control for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$).

Table 5: Contemporaneous Effects of Control Program Exposure on Adults' Education and Earnings

| | | | |
|---|---|---|---|
| **Panel A: Percent with High School Degree, 1940 and 1950 Censuses** | | | |
| | Whole sample | Rural municipalities | Without north |
| TB infection rate | 0.003 | 0.004 | 0.003 |
| × Post dummy | (0.005) | (0.005) | (0.006) |
| Number of observations | 738 | 716 | 620 |
| Number of groups | 369 | 358 | 310 |
| **Panel B: Percent with High School Degree, 1940, 1950, and 1960 Censuses** | | | |
| | Whole sample | Rural municipalities | Without north |
| TB infection rate | -0.020 | -0.019 | -0.020 |
| × Post dummy | (0.059) | (0.060) | (0.071) |
| Number of observations | 1114 | 1080 | 937 |
| Number of groups | 386 | 373 | 327 |
| **Panel C: Fathers with High School Degree** | | | |
| | Whole sample | Rural municipalities | Without north |
| TB infection rate | 0.002 | 0.005 | -0.001 |
| × Post dummy | (0.006) | (0.009) | (0.006) |
| Number of observations | 442605 | 151285 | 375684 |
| **Panel D: Average Taxable Income, 1930, 1948, and 1951** | | | |
| | Whole sample | Rural municipalities | Without north |
| TB infection rate | 0.029 | 0.031 | 0.028 |
| × Post dummy | (0.081) | (0.075) | (0.097) |
| Number of observations | 1107 | 1071 | 933 |
| Number of groups | 376 | 364 | 317 |
| **Panel E: Average Taxable Income, 1930, 1948, 1951, 1954, 1957, and 1961** | | | |
| | Whole sample | Rural municipalities | Without north |
| TB infection rate | 0.022 | 0.024 | 0.020 |
| × Post dummy | (0.041) | (0.039) | (0.049) |
| Number of observations | 2215 | 2144 | 1868 |
| Number of groups | 376 | 364 | 327 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each column contains a different sample of adults in childbearing age. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Post_t)$. Included control variables are municipality dummies and in Panel C year of birth dummies. The outcome in Panels A and B is the percent of individuals born between 1900 and 1920 (potential parents of the main cohort of interest) with a high school degree in each municipality (data source: 1940, 1950, and 1960 census); the outcome in Panel C is the likelihood of fathers of individuals born between 1930 to 1945 to have a high school degree (data source: population registry); the outcome in Panels D and E is the average taxable income of the same cohorts in each municipality (data source: 1930 census and national tax statistics for 1948, 1951, 1954, 1957, and 1961).

Table 6: Long-Term Consequences of Control Program Exposure on Labor Market Outcomes

| | Years of schooling | High school | Earnings | Log earnings |
|---|---|---|---|---|
| **Panel A: Basic Results** | | | | |
| TB infection rate | 0.550** | 0.059** | 3928.7** | 0.071** |
| × Post dummy | (0.102) | (0.012) | (987.3) | (0.014) |
| **Panel B: Heterogeneity Across Municipalities** | | | | |
| TB infection rate | 0.546** | 0.058** | 3850.9** | 0.068** |
| × Post dummy | (0.100) | (0.012) | (1054.9) | (0.016) |
| **Panel C: Heterogeneity Across Municipalities and Mean Reversion** | | | | |
| TB infection rate | 0.537** | 0.055** | 3896.1** | 0.068** |
| × Post dummy | (0.100) | (0.012) | (1056.8) | (0.016) |
| Number of observations | 444932 | 444932 | 355848 | 355848 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Every column contains a different sample. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Post_{ic})$. Included control variables are municipality dummies and year of birth dummies. The set of controls is enlarged in Panel B and includes controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia). In Panel C, additional controls are added to control for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$).

Table 7: Alternative Identification Strategy for Long-Term Consequences of Control Program Exposure on Labor Market Outcomes

| | Years of schooling | High school | Earnings | Log earnings |
|---|---|---|---|---|
| **Panel A: Basic Results** | | | | |
| High TB infection rate | 0.075** | 0.026** | 356.5 | 0.008** |
| × Post dummy | (0.064) | (0.001) | (272.6) | (0.002) |
| **Panel B: Heterogeneity Across Municipalities** | | | | |
| High TB infection rate | 0.074** | 0.026** | 350.6 | 0.008** |
| × Post dummy | (0.006) | (0.001) | (279.8) | (0.002) |
| **Panel C: Heterogeneity Across Municipalities and Mean Reversion** | | | | |
| High TB infection rate | 0.074** | 0.026** | 374.3 | 0.008** |
| × Post dummy | (0.006) | (0.001) | (290.5) | (0.002) |
| Number of observations | 444932 | 444932 | 355848 | 355848 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each column contains a different sample. Each estimate is from a different regression and is the coefficient on interaction of a dummy variable $H_j^{pre}$, which is equal to one if the individual $i$ is born in a municipality with above median tuberculosis infection rate, and the dummy $Post_{ic}$). Included control variables are municipality dummies and year of birth dummies. The set of controls is enlarged in Panel B and includes controls for heterogeneity across municipalities (the number of doctors per inhabitant and the student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia). In Panel C, additional controls are added to control for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$).

Table 8: Long-Term Consequences Based on Intensity of Exposure on Labor Market Outcomes

| | Years of schooling | High school | Earnings | Log earnings |
|---|---|---|---|---|
| **Panel A: Basic Results** | | | | |
| TB infection rate | 0.057** | 0.020** | 490.6* | 0.008** |
| × Years of exposure | (0.011) | (0.003) | (216.7) | (0.002) |
| **Panel B: Heterogeneity Across Municipalities** | | | | |
| TB infection rate | 0.056** | 0.020** | 465.8* | 0.008** |
| × Years of exposure | (0.011) | (0.003) | (229.1) | (0.002) |
| **Panel C: Heterogeneity Across Municipalities and Mean Reversion** | | | | |
| TB infection rate | 0.055* | 0.020** | 472.5* | 0.008** |
| × Years of exposure | (0.011) | (0.003) | (230.3) | (0.002) |
| Number of observations | 444932 | 444932 | 355848 | 355848 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each column contains a different sample. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Exp_{ic})$. Included control variables are municipality dummies and year of birth dummies. The set of controls is enlarged in Panel B and includes controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia). In Panel C, additional controls are added to control for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$).

Table 9: Heterogeneous Effects by Gender

| | | | | | |
|---|---|---|---|---|---|
| **Panel A: Men** | | | | | |
| | Years of schooling | High school | Earnings | Log earnings | Ever married |
| TB infection rate | 0.477** | 0.044** | 5186.5** | 0.079** | -0.004 |
| × Post dummy | (0.105) | (0.011) | (1660.6) | (0.015) | (0.005) |
| TB infection rate | 0.048** | 0.017** | 521.4 | 0.009** | -0.001 |
| × Years of exposure | (0.011) | (0.003) | (319.9) | (0.002) | (0.001) |
| Number of observations | 223447 | 223447 | 216671 | 216671 | 223447 |
| **Panel B: Women** | | | | | |
| | Years of schooling | High school | Earnings | Log earnings | Ever married |
| TB infection rate | 0.573** | 0.061** | 440.4 | 0.007 | -0.009 |
| × Post dummy | (0.106) | (0.014) | (942.1) | (0.034) | (0.006) |
| TB infection rate | 0.061** | 0.023** | 144.4 | 0.004 | -0.001 |
| × Years of exposure | (0.012) | (0.004) | (122.3) | (0.003) | (0.001) |
| Number of observations | 221485 | 221485 | 139177 | 139177 | 221485 |
| **Panel C: Women's Husbands** | | | | | |
| | Years of schooling | High school | Earnings | Log earnings | |
| TB infection rate | 0.297** | 0.023 | 3644.5* | 0.031* | |
| × Post dummy | (0.112) | (0.013) | (1803.2) | (0.014) | |
| TB infection rate | 0.030* | 0.010* | 404.2 | 0.006* | |
| × Years of exposure | (0.013) | (0.004) | (298.5) | (0.003) | |
| Number of observations | 186047 | 186047 | 180405 | 180405 | |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each column contains a different sample. Each estimate is from a different regression and is the coefficient on ($T_j^{pre} \times Post_{ic}$) or ($T_j^{pre} \times Exp_{ic}$), respectively. Included control variables are municipality dummies and year of birth dummies, controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia), and controls for mean reversion across municipalities (average income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality). Panel A contains all men born between 1930 and 1945, Panel B all women born between 1930 and 1945, and Panel C contains the first husbands of women born between 1930 and 1945.

Table 10: Long-Term Consequences for Early Mortality and Height

| | | | |
|---|---|---|---|
| **Panel A: Early Mortality** | | | |
| | Whole sample | Men | Women |
| TB infection rate | -0.007* | -0.009 | -0.007** |
| × Post dummy | (0.003) | (0.006) | (0.003) |
| **Panel B: Early Mortality from Respiratory Disease** | | | |
| | Whole sample | Men | Women |
| TB infection rate | -0.005** | -0.003 | -0.006* |
| × Post dummy | (0.002) | (0.002) | (0.003) |
| **Panel C: Early Mortality from Cardiovascular Disease** | | | |
| | Whole sample | Men | Women |
| TB infection rate | -0.001 | 0.003 | -0.006 |
| × Post dummy | (0.100) | (0.006) | (0.006) |
| Number of observations | 444932 | 223447 | 221485 |
| **Panel D: Height** | | | |
| | Whole sample | Men | Women |
| TB infection rate | | 1.53* | |
| × Post dummy | | (0.61) | |
| Number of observations | | 223447 | |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each column contains a different sample. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Post_{ic})$. Included control variables are municipality dummies and year of birth dummies, controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term of a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia), and controls for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$).

Table 11: Effect of Control Program Exposure on Siblings

| | Number of siblings | Number of younger siblings | | |
|---|---|---|---|---|
| **Panel A: Number of Siblings** | | | | |
| TB infection rate | 0.061 | 0.008 | | |
| × Post dummy | (0.036) | (0.031) | | |
| Number of observations | 444932 | 444932 | | |
| **Panel B: Older Siblings** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | -0.017 | 0.010 | 73.412 | 0.004 |
| × Post dummy | (0.029) | (0.005) | (1087.605) | (0.010) |
| Number of observations | 465488 | 465488 | 465488 | 465488 |
| **Panel C: Sibling Fixed Effects** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | 0.476** | 0.014 | 5837.1** | 0.061* |
| × Post dummy | (0.055) | (0.008) | (1585.5) | (0.031) |
| Number of observations | 109637 | 109637 | 109637 | 93020 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Every column contains a different sample. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Post_{ic})$. Included control variables are municipality dummies and year of birth dummies, controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia), and controls for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$). Panel A includes cohorts born between 1930 and 1945, Panel B includes older siblings born in 1920 or later of cohorts born between 1930 and 1945, Panel C contains cohorts born between 1930 and 1945 and their older siblings born in 1925 or later. Earnings in Panel B are measured at age 47, earnings in Panel C are measure at age 42.

Table 12: Heterogeneous Effects by Socioeconomic Background

| | Years of schooling | High school | Earnings | Log earnings |
|---|---|---|---|---|
| **Panel A: Father Has Low Education Level** | | | | |
| TB infection rate | 0.552** | 0.056** | 3898.1** | 0.097** |
| × Post dummy | (0.107) | (0.013) | (1141.9) | (0.019) |
| Number of observations | 318553 | 263407 | 254866 | 254866 |
| **Panel B: Father Has High Education Level** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | 0.406** | 0.034** | -162.9 | 0.033 |
| × Post dummy | (0.126) | (0.012) | (2561.2) | (0.025) |
| Number of observations | 122854 | 122854 | 119674 | 97876 |
| **Panel C: Father Has Low Status Profession** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | 0.547** | 0.070** | 4103.8** | 0.094** |
| × Post dummy | (0.101) | (0.015) | (1303.1) | (0.019) |
| Number of observations | 275433 | 275433 | 219513 | 219513 |
| **Panel D: Father Has High Status Profession** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | 0.516** | 0.024 | 1019.5 | 0.057 |
| × Post dummy | (0.154) | (0.017) | (2988.5) | (0.034) |
| Number of observations | 138142 | 138142 | 110730 | 110730 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each column contains a different sample. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Post_{ic})$ or $(T_j^{pre} \times Exp_{ic})$, respectively. Included control variables are municipality dummies and year of birth dummies, controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia), and controls for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$).

Table 13: Effect of Control Program Exposure on Persistence of Educational Attainment Across Generations

| | (A) | (B) | (C) |
|---|---|---|---|
| **Panel A: Father-Son Comparison** | | | |
| Father has high school education | 2.69** | 2.64** | 2.60** |
| | (0.056) | (0.089) | (0.088) |
| Father has high school education × | -0.286** | -0.286** | -0.273** |
| High TB infection rate × Post dummy | (0.062) | (0.062) | (0.062) |
| Number of observations | 444932 | 444932 | 444932 |
| **Panel B: Father-Son Comparison, Second Generation** | | | |
| Father has high school education | 2.07** | 2.03** | 2.05** |
| | (0.058) | (0.072) | (0.082) |
| Father has high school education × | -0.127 | -0.125 | -0.130 |
| High TB infection rate × Post dummy | (0.087) | (0.087) | (0.87) |
| Number of observations | 196059 | 196059 | 196059 |
| **Panel C: Grandfather-Son Comparison, Second Generation** | | | |
| Grandfather has high school education | 1.73** | 1.75** | 1.77** |
| | (0.040) | (0.045) | (0.045) |
| Grandfather has high school education × | -0.202** | -0.201** | -0.199** |
| High TB infection rate × Post dummy | (0.072) | (0.073) | (0.073) |
| Number of observations | 194628 | 194628 | 194628 |

Significance Levels: ** 1% level, * 5% level

Note: The dependent variable is son's completed years of education. Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each set of estimates is from a different regression and is the coefficient on a dummy variable whether the father has a high school degree and the interaction of a dummy variable whether the father has a high school degree, a dummy variable $H_j^{pre}$, which is equal to one if the individual $i$ is born in a municipality with above median tuberculosis infection rate, and the dummy $Post_{ic}$). Every column contains a different set of control variables. Included control variables are municipality dummies and year of birth dummies as well as interactions of these variables with the father's education status. The set of controls is enlarged in Column B and includes controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term of a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia) as well as interactions of these variables with the father's education status. In Column C, additional controls are added to control for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$) as well as interactions of these variables with the father's education status.

Table 14: Long-Term Consequences of Control Program Exposure on Fertility

| | Men | | | Women | | |
|---|---|---|---|---|---|---|
| **Panel A: Basic Results** | | | | | | |
| | P(child in sample) | Number of children | Age at first birth | P(child in sample) | Number of children | Age at first birth |
| TB infection rate | -0.007 | -0.048 | 0.005 | -0.003 | -0.039 | -0.077 |
| × Post dummy | (0.004) | (0.032) | (0.104) | (0.003) | (0.031) | (0.102) |
| **Panel B: Heterogeneity Across Municipalities** | | | | | | |
| | P(child in sample) | Number of children | Age at first birth | P(child in sample) | Number of children | Age at first birth |
| TB infection rate | -0.007 | -0.048 | 0.009 | -0.003 | -0.037 | -0.073 |
| × Post dummy | (0.004) | (0.033) | (0.104) | (0.003) | (0.031) | (0.101) |
| **Panel C: Heterogeneity Across Municipalities and Mean Reversion** | | | | | | |
| | P(child in sample) | Number of children | Age at first birth | P(child in sample) | Number of children | Age at first birth |
| TB infection rate | -0.007 | -0.045 | 0.011 | -0.002 | -0.035 | -0.071 |
| × Post dummy | (0.004) | (0.033) | (0.105) | (0.003) | (0.031) | (0.102) |
| Number of observations | 228195 | 167475 | 124751 | 226341 | 172957 | 103354 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Every column contains a different sample. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Post_{ic})$. Included control variables are municipality dummies and year of birth dummies. The set of controls is enlarged in Panel B and includes controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia). In Panel C, additional controls are added to control for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$).

Table 15: Long-Term Consequences of Control Program Exposure on Second Generation

| | Exposed Fathers | | | | | Exposed Mothers | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Birth weight | Years of education | Log earnings | IQ (boys) | Height (boys) | Birth weight | Years of education | Log earnings | IQ (boys) | Height (boys) |
| **Panel A: Basic Results** | | | | | | | | | | |
| TB infection rate | 26.6 | 0.166* | 0.030* | 0.066 | 0.113 | 47.7* | 0.171* | 0.024* | 0.099 | 0.410** |
| × Post dummy | (14.5) | (0.067) | (0.012) | (0.041) | (0.114) | (20.9) | (0.070) | (0.012) | (0.053) | (0.143) |
| **Panel B: Heterogeneity Across Municipalities** | | | | | | | | | | |
| TB infection rate | 26.2 | 0.164* | 0.030* | 0.064 | 0.101 | 47.8* | 0.169* | 0.024* | 0.104* | 0.410** |
| × Post dummy | (14.6) | (0.067) | (0.012) | (0.041) | (0.113) | (21.1) | (0.069) | (0.011) | (0.051) | (0.144) |
| **Panel C: Heterogeneity Across Municipalities and Mean Reversion** | | | | | | | | | | |
| TB infection rate | 25.4 | 0.164** | 0.030* | 0.060 | 0.102 | 47.8* | 0.167* | 0.024* | 0.105* | 0.409** |
| × Post dummy | (14.6) | (0.063) | (0.012) | (0.042) | (0.113) | (21.2) | (0.069) | (0.011) | (0.051) | (0.144) |
| Number of observations | 235066 | 392578 | 423576 | 199453 | 210309 | 172908 | 409854 | 443968 | 204736 | 216556 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Every column contains a different sample. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Post_{ic})$. Included control variables are municipality dummies and year of birth dummies. The set of controls is enlarged in Panel B and includes controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia). In Panel C, additional controls are added to control for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$).

Table 16: Sensitivity Analysis

| | | | | |
|---|---|---|---|---|
| **Panel A: Without North (Finnmark and Troms)** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | 0.604*** | 0.061*** | 4135.6** | 0.06** |
| × Post dummy | (0.128) | (0.016) | (1559.2) | (0.024) |
| Number of observations | 420673 | 420673 | 420673 | 336534 |
| **Panel B: Additional Control: Heating with Biomass Fuels** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | 0.535*** | 0.055*** | 3836.2*** | 0.066*** |
| × Post dummy | (0.102) | (0.012) | (1065.6) | (0.017) |
| Number of observations | 454536 | 454536 | 454536 | 363325 |
| **Panel C: Linear Municipality-Specific Time Trends** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | 0.474** | 0.041** | 2417.4* | 0.062** |
| × Post dummy | (0.113) | (0.012) | (1118.3) | (0.017) |
| Number of observations | 454536 | 454536 | 454536 | 363325 |
| **Panel D: Rollout from 1949 to 1952** | | | | |
| | Years of schooling | High school | Earnings | Log earnings |
| TB infection rate | 0.111** | 0.009 | 493.9 | 0.017 |
| × Post dummy | (0.038) | (0.006) | (1723.4) | (0.022) |
| Number of observations | 118435 | 118435 | 118435 | 91815 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Each estimate is from a different regression and is the coefficient on ($T_j^{pre} \times Post_{ic}$). Included control variables are municipality dummies and year of birth dummies, controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term of a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia), and controls for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$). Panel D includes linear municipality-specific time trends.

Table 17: Varying Timing of Treatment Assignment

| | Years of schooling | High school | Earnings | Log earnings |
|---|---|---|---|---|
| **Panel A: 7 Years of Program Exposure** | | | | |
| TB infection rate | 0.681** | 0.122** | 4413.6** | 0.085** |
| × Post dummy | (0.085) | (0.026) | (635.044) | (0.020) |
| **Panel B: 6 Years (or More) of Program Exposure** | | | | |
| TB infection rate | 0.683** | 0.128** | 5026.9** | 0.086** |
| × Post dummy | (0.080) | (0.020) | (1033.4) | (0.015) |
| **Panel C: 5 Years (or More) of Program Exposure** | | | | |
| TB infection rate | 0.640** | 0.105** | 4191.5** | 0.074** |
| × Post dummy | (0.108) | (0.022) | (1005.4) | (0.016) |
| **Panel D: 4 Years (or More) of Program Exposure** | | | | |
| TB infection rate | 0.588** | 0.082** | 3567.1** | 0.075** |
| × Post dummy | (0.109) | (0.026) | (1080.5) | (0.015) |
| **Panel E: 3 Years (or More) of Program Exposure** | | | | |
| TB infection rate | 0.546** | 0.076** | 3618.7** | 0.066** |
| × Post dummy | (0.104) | (0.026) | (1135.6) | (0.020) |
| **Panel F: 2 Years (or More) of Program Exposure** | | | | |
| TB infection rate | 0.549** | 0.058** | 4006.8** | 0.070** |
| × Post dummy | (0.091) | (0.025) | (1115.5) | (0.023) |
| Number of observations | 444932 | 444932 | 355848 | 355848 |
| **Panel G: 3 Years or Younger at Campaign Launch** | | | | |
| TB infection rate | 0.025 | 0.015 | 1870.1 | 0.013 |
| × Post dummy | (0.058) | (0.016) | (1641.7) | (0.009) |
| Number of observations | 678150 | 678150 | 678150 | 564433 |

Significance Levels: ** 1% level, * 5% level

Note: Robust standard errors adjusted for clustering at the municipality of birth level in parentheses. Every column contains a different sample. Each estimate is from a different regression and is the coefficient on $(T_j^{pre} \times Post_{ic})$. Included control variables are municipality dummies and year of birth dummies, controls for heterogeneity across municipalities (the number of doctors per inhabitant and student–teacher ratio in each year and in each municipality, the precampaign childhood mortality rates from measles per county interacted with the indicator variable $Post_{ic}$, and the interaction term between a dummy variable indicating whether born after the introduction of antibiotics and the local childhood mortality rate from pneumonia), and controls for mean reversion across municipalities (average log income in each municipality in 1930 and the average percentage of inhabitants with a high school degree in each municipality interacted with $Post_{ic}$). Panels A to F include cohorts born between 1930 and 1945; Panel G includes cohorts born between 1930 and 1950.