Long-term effects of early life exposure to pneumonia*

Camila Saez†

January 11, 2021

For the latest version click here

Abstract

This paper studies the effect of sulfa drugs, the first widely used antibiotic, on mortality and exploits the introduction of sulfa drugs to identify the causal impact of exposure to pneumonia in infancy on later life outcomes in Chile during the first half of the 20th century. Using new yearly data by province, which includes cause of deaths, I find that sulfa drugs' introduction causes a considerable decline in pneumonia, meningitis, and maternal mortality. Additionally, I exploit the introduction of sulfa drugs to identify the impact of exposure to pneumonia in infancy on later life outcomes, using an intensity of treatment research design. The results show that exposure to sulfa drugs, and thereby less exposure to pneumonia during birth year, led to a statistically significant improvement in education and employment for men of the affected cohorts. For years of schooling, a decrease of one standard deviation in pneumonia exposure resulted in an increase of 0.5 years in schooling for men, an increase of 7 percentage points in the probability of completing primary school and a 3 percentage points in completing secondary school. For employment, men born in an environment with less incidence of pneumonia are 2.8 percentage points more likely to be employed.

*I am grateful to my advisor, Katherine Eriksson, for her guidance and support, and Santiago Pérez and Gregory Clark, for their suggestions and advice. I am also thankful to Rowena Gray, Marianne Bitler, and seminar participants of UC Davis Economic History Coffee Hour for helpful comments and suggestions. Any errors remains my own.

†Department of Economics, University of California, Davis. cfsaez@ucdavis.edu.

Introduction

Pneumonia is the most significant cause of death for children under five globally (figure 1), causing more than 16% of all childhood deaths. Today, pneumonia deaths are found mainly in developing regions; however, historically, this was a global problem. The decline in pneumonia deaths came from the introduction of sulfonamides (also called sulfas), the first antibiotic. In Chile, the pneumonia mortality rate fell from 180 per 100,000 population in 1930 to 14 in 2017 [1].

This paper studies the effect of sulfonamide drugs on mortality and later-life outcomes. In particular, it explores the impact of sulfa drugs on reducing mortality and the long-term effects of being born in a better disease environment during the birth-year. I focus on Chile during the first half of the twentieth century. Chile is an interesting case study because it is a success story. During that period, the US and Chile had very different mortality rates. Chile was a high mortality country (infant mortality in 1940 in Chile was over 200 deaths per 1,000 live births, while the US experienced one-fourth of those mortality levels, while today, the differences in mortality are negligible). Also, while many papers study the effects of being born in a better disease environment during childhood for other diseases like malaria, pneumonia has been under-study. Pneumonia is an important disease because it was, and still is today, the leading cause of death for children under-five.

In this paper, I start by studying the contribution of sulfa drugs to the decline of mortality in Chile. Previous research has shown that the adoption of sulfa medications leads to a significant mortality decline in the United States. This relationship has not been tested empirically for more developing regions. Using new yearly data by province, including the cause of deaths, I estimate the impact of sulfa drugs on mortality, using a difference in difference approach for the period 1930-1950. For several infectious diseases, including pneumonia, sulfa drugs represented the first effective treatment. To see how sulfa drugs helped decrease Chile's mortality, I compare mortality from diseases treatable

[1] In the United States, deaths from pneumonia (and influenza) have steadily declined, from 213 per 100,000 population in 1930 to 15 in 2017.

with sulfa drugs (maternal mortality, pneumonia, and meningitis) versus those unaffected by sulfa drugs (tuberculosis) before and after the adoption of sulfa drugs. Sulfa drugs are an excellent way of testing this because their adoption was fast because of their low cost. I find that the introduction of sulfa drugs caused a considerable decline in pneumonia, meningitis, and maternal mortality. Specifically, sulfa drugs resulted in a drop of 10-28% in maternal mortality. They also led to a 25-50% decline in pneumonia mortality and a 10-40% in meningitis mortality.

In the second part of this paper, I use the introduction of sulfonamides drugs to identify the causal impact of exposure to pneumonia in infancy on later-life outcomes in Chile. There is a consensus that early life shocks can have persistent effects on later life (Barker (1992), Almond (2006), Bhalotra and Venkataramani (2015), Venkataramani (2012), Cutler et al. (2010), Bleakley (2010), Barreca (2010) and Lucas (2010)). My identification strategy exploits the introduction of sulfa drugs to identify the causal impact of exposure to pneumonia during infancy on later life outcomes. The idea is that being exposed to a better disease environment during childhood has short-term and long-term benefits. In the short-term, mortality declines, but in addition, because of this low endemicity there are also long-term benefits associated with a healthier overall population. My paper studies these two effects.

I estimate the effect of introducing sulfa drugs on cohorts born after the introduction on different outcomes (years of schooling, likelihood of completing primary and secondary school, disability, and employment). I causally identify the effects by comparing people born in provinces with high pre-sulfa-drug levels of pneumonia mortality, with those from areas with low prevalence. We should expect that sulfa medications should have a more significant effect on provinces with higher pre-sulfa mortality rates. Provinces with higher pre-sulfa mortality levels should have experienced the largest mortality declines after introducing sulfa drugs; hence they benefit the most from the new treatment versus areas with low mortality levels. This empirical approach is often called an "intensity of treatment" research design, and it is the most widely used approach in the literature of the long-term effects of disease (see Bleakley (2007), Lucas (2010), and Bhalotra and

Venkataramani (2015)).

My results show that exposure to sulfa-drugs, and thereby less exposure to pneumonia in the year of birth, led to a statistically significant improvement in education and employment for men. For years of schooling, a decrease of one standard deviation in pneumonia exposure (mortality) is estimated to have resulted in an increase in 0.5 years of schooling for men (my results are not significant for women). The same effect is observed for my other educational variables. A decrease of one standard deviation in pneumonia exposure (proxy by mortality) is estimated to have resulted in an increase of seven percentage points in the probability of completing primary school and three percentage points in completing secondary school.

For employment, men who were born in an environment with a lower incidence of pneumonia are 2.8 percentage points more likely to be employed. I do not find significant results for the disability variable or mental disability.

As is common in the literature studying the long-term effects of health shocks, my results for women are not significant, and in most cases, they show a negative and insignificant effect for all the variables studied. This is consistent with the idea that males are more vulnerable to shocks in utero (Almond et al. (2007), Almond and Mazumder (2011), Nilsson (2008)); the literature usually finds that the effects for males are always of higher magnitude than those for females. This result is also coherent with Bhalotra and Venkataramani's (2015) results for people exposed to pneumonia in the US. And this is also explained by the fact that pneumonia incidence in childhood was larger for males than for females; therefore, men benefit the most from sulfa drug availability.

These results are likely a lower bound of the positive effects of reducing exposure to an infectious disease in infancy. There are two potential effects: a selection effect and a scarring effect. In the selection effect, the sulfa drug availability will allow babies that otherwise would have died to live because of the drug, and since these children have worse overall health status we should expect a negative impact on future outcomes. In the scarring effect, the availability of sulfa drugs will make the disease nonexistent or shorter for individuals (because of the lower endemicity); thus, the people exposed will

have better adulthood outcomes. My results capture the net of these two effects. The two effects operate in opposite directions, hence my results are a lower bound, where scarring effects dominate selection.

This study relates to a broad literature on the long-term effect of infectious diseases. Literature in epidemiology and economics has studied the impacts of different shocks on early life. The most common studies show that being exposed to infectious diseases in utero or during infancy has negative long-term consequences. Almond (2006) examines the effects of prenatal exposure to the 1918 influenza pandemic in the US and finds that children exposed in utero were less likely to graduate from high school and had lower wages than their counterparts who were not exposed to the pandemic (cohorts born just before and after). Other papers examine the impact of other infectious diseases. For example, Bhalotra and Venkataramani (2015) show that early life exposure to pneumonia affects disability, human capital, and productivity in adulthood. In particular, they found that cohorts exposed to sulfa drugs and thereby less pneumonia in the birth year achieved substantial improvements in schooling, employment and income, and lower risk of disability and poverty in adulthood. For Sweden, Lazuka (2018), also explores the impact of mitigating the pneumonia disease burden in infancy on long-term outcomes, and finds that less pneumonia exposure substantially reduced the probability of receiving disability and increased labor income in late adulthood.

The current study also relates to the literature studying the effects of sulfa drugs on mortality. The only paper exclusively examining the impact of sulfa drugs on mortality is Jayachandran et al. (2010) which find that sulfa drugs led to a 24-36% decline in maternal mortality, 17 to 32% decline in pneumonia mortality, and 52 to 65% decline in scarlet fever mortality between 1937 and 1943. Another related paper is Thomasson and Treber (2008), which studies how being born in a hospital affects maternal mortality. That paper, finds that in the US, giving birth in a hospital did not significantly reduce maternal mortality until after sulfa drugs became widely available in 1937. Also, Smith and Bradshaw (2008) study the impacts of penicillin on life expectancy rather than mortality, and finds that, the annual variation in life expectancy declined after penicillin was introduced in the

United States and England.

My paper contributes to the literature in several ways. First, it contributes to the literature on sulfa drugs impacts on mortality. There is little evidence of sulfa drugs' contribution to the mortality decline, despite being an important medical discovery. All previous research examined the US or similar developed countries. Second, this paper contributes to studying the impact of sulfa drugs on mortality in a developing country with a high mortality rate. This is important because, during that period, the US and Chile had very different mortality rates. Chile was a high mortality country (infant mortality in 1940 in Chile was over 200 deaths every 1,000 live births, while the US experienced one-fourth of those mortality levels).

Also, Chile is an interesting country to study because of its relatively high-quality data. Demographic data has been available since the beginning of the 19th century. The Central Statistics Office has collected vital statistics since 1848. In this paper, I digitize yearly data, including cause of death, mortality, number of hospitals, and maternal mortality. This matters because mortality data for most developing regions usually comes from survey data or census. The lack of yearly data makes it hard to study the causal impact of any policy on mortality. Moreover, Chile has an exceptional performance in reducing mortality, so it is an excellent example for other developing countries about policies that may reduce mortality.

This study also contributes to the literature on the long-term impacts of infectious diseases in early life. While many papers study this for other diseases like malaria, pneumonia has been under-study. This is important because pneumonia was, and is still today, the leading cause of death for children under-five. Therefore, we should expect that getting rid of pneumonia should have larger effects than eliminating other less prevalent diseases. These results are also relevant because they give external validity to the previous results find by Bhalotra and Venkataramani (2015) for the US. My results are similar to those found in that case.

In addition, Chile may be more important as a lesson for the developing world than the examples of more developed countries, given Chile's high mortality levels during that

time. Results using data from developed, high-income countries may not extrapolate to more developing regions. What happened in the US or Sweden, even in the past, tells us relatively little about the impact of childhood health on adult outcomes in contemporary developing countries, where prevalence rates of diseases, educational attainment, and institutions are different from those in developed countries. Also, these countries were already high income and with low mortality levels. This makes Chile more critical as a lesson for the developing world, where contagious and infectious diseases are more prevalent, and infant mortality remains high. As a result of this, we should expect that the benefits of eliminating diseases in the long-term outcomes to be larger in the developing world. For example, my results are of larger magnitude than those find in the US. For instance, for the US, a one standard deviation decrease in pneumonia exposure led to an increase in only 0.1 years of education, while for Chile was an extra 0.5 years of schooling[2]. The differences are due to two reasons. First, mortality was higher in Chile, so declines in mortality should have larger effects, and second, baseline years of education were significantly lower. Becasue these factors also applied to the developing countries today, we should expect that the same more considerable results should be observed for developing regions today.

This issue is also relevant now because, even today, only one-third of children who have pneumonia are able to access antibiotics (World Health Organization, 2019). Moreover, sulfa drugs are still widely used in the developing world, so understanding not only the short-term impacts of reducing mortality but also the long-term impacts is essential. Focusing only on the effects of sulfa drug availability on mortality makes the benefits appear smaller than they are, essentially under-estimating the real benefits of their use.

The rest of the paper is structured as follows. Section 2 gives a brief explanation of the history of sulfa drugs. Section 3 describes the data used. Section 4 provides the results for the impact of sulfa drugs on mortality. Section 5 shows the results for the long-term impacts of being exposed to sulfa drugs, and finally, Section 6 concludes.

[2]In general, the estimated coefficients for Chile are 4 times larger than those find for the US.

History of Sulfa drugs

Sulfonamide drugs were the first drug that effectively treated a series of bacterial diseases. Before the arrival of sulfa medications, pneumonia, and other infectious diseases, were primarily treated with supportive care and immunotherapy. Even though dyes were being tested as antibacterial agents since the 1920s, it was not until 1932 that a German scientist, Gerhard Domagk[3], discovered that a dye, Prontosil (figure B.1), was useful to treated streptococci bacterial infections. The investigation results were not published until 1935.

By 1935, enough clinical trials showed that Prontosil was effective against severe streptococcal sore throat, erysipelas, scarlet fever, puerperal sepsis, and other streptococcal infections. However, it was also proved that it was useless against other bacterial infections. In addition to curing infections, sulfa drugs also brought sharp reductions in patients' recovery time and the amount of nursing time devoted to the care of patients with bacterial infections.

However, Prontosil was not the only medication available; in 1935, a team at the Pasteur Institute showed that a constituent of the Prontosil molecule, a compound later known as sulfanilamide, was at least as effective as Prontosil in animal and clinical trials. This finding was extremely important because Prontosil was patented; however, sulfanilamide was not (it was discovered in 1908, so the patent had already expired), and it was cheaper and easier to manufacture. Sulfanilamide was also better because Prontosil could turn people red from the treatment, while sulfanilamide was free of this side effect.

In the United States, the first major clinical trial of sulfa drugs happened in 1936, when it was used to successfully treat women with puerperal fever. By 1937, sulfa drugs were widely available and used in the US. Sulfa drugs became widely popular fast because chemical manufacturing companies already produced tons of sulfanilamide every year as an intermediate in the dye-making process. Hence, a large supply of the drug was readily available. Also, because the drug was a tablet, it was easy to administer. In Chile, sulfonamides were introduced in 1938 by Dr. Hernan Alessandri, and they were widely

[3]He won the Nobel Prize of Medicine in 1939 for this discovery.

used after that.

The production and use of sulfa drugs grew rapidly after their discovery; their low price partially explains their rapid spread. The next significant medical advance did not occur until the mid-1940s when penicillin and other antibiotics became available.

Data

To determine the impact of sulfa drugs on mortality, I hand-digitize data from the Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, at the national and provincial level from 1930 to 1950. Data include information on deaths, broken down by cause of death by province, year, and gender (See figure B.2). I collect data on causes of deaths[4], and I focus on five causes of death that were shown to be highly responsive to sulfa drugs in clinical trials: maternal mortality, puerperal sepsis[5], pneumonia[6], scarlet fever, and meningitis. I will refer to these diseases as "treated diseases".

In addition, I collect data on comparison diseases that were untreatable with sulfa drugs. According to Jayachandran et al. (2010), the best comparison disease is tuberculosis ("control diseases"), an infectious disease that was not responsive to sulfa drugs in clinical trials. Additionally, I collect data on other chronic diseases like diabetes, circulatory system disease, cancer, as well as diarrhea deaths.

To calculate mortality rates, I use the population linearly interpolated from the census of 1930, 1940, and 1952. For maternal mortality and puerperal sepsis, I use live births data available yearly from the Demographic and Social Assistance Yearbooks.

During the 1940s, Chile had 25 provinces; however, some provinces did not exist during the entire period. For example, in 1934, there were only 18. Hence, to avoid potential issues that may arise, I harmonized the districts, aggregating them when needed to form a unit that does not change over time.

Table 1 reports summary statistics of national mortality rates for 1930 and 1950.

[4]Cause of death by age is not available by province, so the data is not age-standardized.

[5]Literature usually uses maternal mortality as a proxy of puerperal sepsis. In this specific case, using Chilean data, deaths by puerperal sepsis are listed separately, however, given the time period, some deaths by puerperal sepsis maybe not correctly catalogued as puerperal sepsis, but they will be included in maternal mortality. This is because, during this period, most deaths were not certified by a doctor; hence it was easy to determine that the death was related to childbirth, but not necessarily that it was due the infection. Given this, the main results focus only on maternal mortality.

[6]It is worth noting that, since 1939, US data combine influenza and pneumonia mortality in only one category. This is unfortunate because sulfa drugs did not affect influenza mortality. Chilean data, however, separate influenza and pneumonia mortality.

It also reports statistics for the pre-sulfa period and post-sulfa period. In 1930, the maternal mortality ratio was 831.9, i.e., for every 100,000 live births, 831 women died from childbirth consequences. For the post-sulfa period, this number decreased to 596, around a 30% decline. Similarly, deaths from pneumonia declined 54%, from scarlet fever 69%, and from meningitis 57%. Nevertheless, in the same period, mortality from tuberculosis only fell 9%, and most chronic diseases increased their mortality rate.

To determine the long-term effects of exposure to pneumonia, I used individual level data from the census of 1970, 1982 and 1992, available from the Integrated Public Use Microdata Series, International. I pooled this censuses to have a larger sample size and more precise estimates[7]. I do not use previous censuses because the marginal cohort may be too young during that period. Given that sulfa drugs were introduced in 1938, the in-utero cohort (born in 1938) during this time will be around 33 years old for the 1970 census, 43 years old for the 1982 census, and 53 in 1992. Moreover, I don't use the 2002 census because evidence shows that exposure to infectious diseases in early life is also associated with lower life expectancy (Crimmins and Finch (2005), Myrskylä (2010)), hence biasing my results. This is important in my context because life expectancy at birth in Chile in 1940 was less than 50 years.

I also hand-digitized data on the number of hospitals, the number of doctors, and province-specific health expenditures from the Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile. Statistics from these variables are in the table C.1.

[7]However, my results are robust when I use each census.

Effects of sulfa drugs on mortality

Following Jayanchandran et al. (2010), I use two facts about sulfa drugs to test their impact on mortality. First that sulfa drugs discovery can be taken as exogenous because sulfa medications could not be patented (as the patent had already expired). Hence, the diffusion was rapid. Second that, according to the clinical trials, sulfa drugs were only effective against some diseases.

Figure 2 plots total mortality between 1930 and 1950 and figure 3 shows the time series of the four sulfa-treated diseases: maternal mortality, puerperal sepsis, pneumonia, and meningitis. For all the diseases, there is a sharp decline after the 1938-1939 period[8]. Figure 4 shows the trajectory of the control infectious disease (unaffected by sulfa drugs), tuberculosis. It can be seen that the mortality from this disease is relatively flat until 1947 when it started declining (this is consistent with the use of the first antibiotic that effectively treated tuberculosis, streptomycin). The same pattern can be seen from figure B.5 that includes chronic diseases and under-2 year diarrhea; neither of these shows any meaningful change around the time sulfa drugs were introduced. In fact, some of these disease are trending upward. This suggests that the factor explaining the decline of the treated diseases is sulfa drugs and not other relevant medical advances.

To empirically test that sulfa drugs decrease mortality, I use the national mortality level series to estimate the extent to which sulfa drugs contribute to the decline in mortality. I estimate two models. The first model allows for a level change in mortality in 1938, and the second model allows mortality to be a function of a slope change and level change after 1938. I estimate the models with data only until 1943; after that, penicillin was widely used, which can bias my results. The first set of regressions use yearly data

[8]Scarlet fever was also treated by sulfa drugs, however, figure B.3 shows the trajectory, and it can be seen that scarlet fever was endemic in Chile with relatively low mortality, and periodic exacerbations every 4-5 years. Thus, the pre-sulfa drug trajectory is too different from the rest of the diseases under study, so I prefer to omit it in the analysis
I will also omit puerperal sepsis, and just use maternal mortality as a proxy. Figure B.4 shows the trajectory of both series, as it can be seen, they move similarly, so for simplicity I will only focus on maternal mortality.

from 1930 to 1950, for the national mortality series. My first model is given by:

$$log(M)_t = \beta_0 + \beta_1 Year_t + \delta_0 Post\text{-}1938 + e_t \quad (1)$$

where $log(M)_t$ is the log of mortality in year t, $Year_t$ is a continuous year variable centered on 1938, and *Post-1938*, is a dummy variable equal to 1 if the year is 1938 or after. I am interested in whether δ_0 is negative and significant.

The second model, allows both a change in levels and a change in slope and is given by:

$$log(M)_t = \beta_0 + \beta_1 Year_t + \delta_0\, Post\text{-}1938 + \delta_1\, Post\text{-}1938 \times Year_t + e_t \quad (2)$$

In this case, I am interested in if δ_0 and δ_1 are negative and significant.

Table 2 shows the results of the first model. All the diseases treatable with sulfa drugs saw declines in mortality after 1938, the δ_0 are negative and statistically significant. However, the coefficients for tuberculosis and cancer are not significant and the coefficients are much smaller. This suggests a decline in total mortality of about 14.2% between the pre-sulfa and post-sulfa period, a 26% decline in maternal mortality, a 48% decline in pneumonia, and 38% decline in meningitis.

The second model results are in table 3. They also show that δ_0 and δ_1 are negative and significant for our treated diseases. This regression coefficient indicates an effect of a 28.5% decline in maternal mortality, a 50% decline in pneumonia mortality, and a 41% decline in meningitis. If we compare this with the results find by Jayachandran et al. (2010) for the United States, the Chilean coefficients are larger for regressions. This is because mortality levels were much higher in Chile than in the US during that period. As a reference, Chile's infant mortality rate during 1940 was over 200 while in the US, it was 47, so it's expected that mortality declines to be larger.

Differences in differences

Next, I use a difference in difference regression to measure the magnitude of sulfa drugs' effect on mortality. Based on my previous results, I know that sulfa drugs were widely used by 1938. Also, from clinical trials conducted in the 1930s, we know that sulfa drugs were only effective against certain infectious diseases (maternal mortality, scarlet fever, pneumonia, and meningitis), but not against others. For example, we know that sulfa drugs were not efficient in treating tuberculosis. Therefore, I estimate a difference-in-difference model that compares mortality differences between treated and control diseases before and after 1938. Tuberculosis (my control disease) accounts for all the other factors that may affect mortality during 1938. I assume that post-1938 mortality declines for treated diseases, beyond those that occurred for the control disease, are due to sulfa drugs[9].

National level

Using first national mortality data, I estimate:

$$\begin{aligned} log(M)_{dt} = \beta_0 + \beta_1 Treated_d \times Post1938_t \\ + \beta_2 Treated_d \times Year_t + \beta_3 Treated_d + \beta_4 Year_t + \beta_5 Post1938_t + \ e_{dt} \end{aligned} \tag{3}$$

The dependent variable $log(M)_{dt}$ is the log of mortality for disease d in year t. $Treated_d$ is an indicator variable for whether disease d is a treated disease, $Post1938$ is a variable equal to zero before year 1938, and one after, and $Year_t$ is a continuous year variable. Equation (3) estimates changes in the level of mortality after 1938; β_1 measures whether the reduction in mortality was larger for treated diseases after 1938.

[9]Figure B.6 in the appendix show the trajectory of the mortality rates for my selected diseases. It can be seen that previous to the introduction of sulfa drugs, the trend in mortality was similar, but after 1938 started declining only for the treated diseases.

Additionally, I estimate:

$$\begin{aligned} log(M)_{dt} = \beta_0 + \beta_1 Treated_d \times Year_t \times Post1938_t + \beta_2 Treated_d \times Post1938_t \\ + \beta_3 Treated_d \times Year_t + \beta_4 Treated_d + \beta_5 Year_t + \beta_6 Post1938_t + \ e_{dt} \end{aligned} \tag{4}$$

This model allows for changes in both the intercept and the slope after sulfa drugs. In this case,my hypothesis is that β_1 and β_2 are significant and negative. Both models allow for a different linear time trend for control and treated diseases. The results are in table 4.

The first column shows the results from equation (3) and the second column for equation (4). The coefficients of interest are negative and significant for treated diseases (except for maternal mortality). These results suggest that the introduction of sulfa drugs led to considerable mortality declines for diseases that could be treated with the new drugs.

Province level

I repeat the same exercise using province-level data, which allows us to control for province-specific trends. I estimate the following:

$$\begin{aligned} log(M)_{idt} = \beta_0 + \beta_1 Treated_d \times Post1938_t \\ + \beta_2 Treated_d \times Year_t + \beta_3 Treated_d + \beta_4 Year_t + \gamma_{it} + \ e_{idt} \end{aligned} \tag{5}$$

and

$$\begin{aligned} log(M)_{idt} = \beta_0 + \beta_1 Treated_d \times Year_t \times Post1938_t \\ + \beta_2 Treated_d \times Post1938_t + \beta_3 Treated_d \times Year_t \\ + \beta_4 Treated_t + \beta_5 Year_t + \gamma_{it} + \mu_{it} \times Year_t + e_{idt} \end{aligned} \tag{6}$$

The dependent variable is the natural logarithm of the mortality rate in province i for disease d in year t. The rest of the variables are defined as before. I also include

$Province \times Post1938$ fixed effects, which control for the main impact of $Post1938$, an absorb province-level variation in mortality declines. Standards errors are clustered by disease-year.

I estimate the equations separately for each treated disease, focusing only on the period 1930 to 1943. I end my regressions in 1943 to avoid biasing my result because of other medical advances like penicillin. The results for equation 5 are in table 5. The coefficients of interest are negative and significant for all the treated diseases, suggesting that sulfa drugs' introduction led to a mortality decline for these diseases.

But, how much did sulfa drugs reduce mortality?. Using the coefficient from equation (5), we can see that the maternal mortality coefficient of -0.198 implies that sulfa drugs caused a decline of 18% in maternal mortality in the post-sulfa period, equivalent to almost 150 fewer maternal deaths per 100,000 live births between 1933-1938 and 1939-1943. For pneumonia and meningitis, sulfa drugs led to a decline of 25.4% and 9.5% in mortality, respectively

The second model estimate from equation 6, allows for a break in trend and a level change. I find larger effects, that sulfa drugs led to a 20% decrease in maternal mortality, a 43% decrease in pneumonia mortality, and a 22% decrease in meningitis mortality (table5).

Long-term impacts of pneumonia exposure

The second part of this paper, estimates the impacts of being born in an environment with less pneumonia, using individual-level outcome data from the census.

The epidemiological and economic literature suggests that there are long-term impacts of being exposed to infectious diseases in utero and during childhood. The reason for this is because infectious diseases generate an inflammatory immune response that diverts resources away from physical and mental development, and the diversions can have long-term effects in adulthood. Because nutritional demands are high during infancy, exposure during the birth year and childhood will cause the most irreversible damage compared to exposure later in life (Crimmins and Finch (2005), and Eppig et al. (2010)).

For pneumonia in particular, children who survive pneumonia have an increased risk of chronic lung disease. During adulthood, children who survive pneumonia may have worsened exercise ability, cardiovascular disease, and cognitive decline for months or years. Because of this, one should expect benefits from being born in a low endemicity environment.

Furthermore, as previously mentioned, pneumonia is the leading cause of death for children under 5, and it has been one of the leading causes of death for children since 1900. In Chile, in 1935, close to 30% of total pneumonia deaths were children under one-year old. Figure 5 exhibits the incidence of pneumonia mortality by age group in 1935; the deaths are mostly concentrated in children under one-year old.

In this section, I will investigate whether early-life exposure to pneumonia[10] (particularly during the birth year) affects disability and human capital in adulthood. For this, I will use the mortality from pneumonia in their birth year (and birth region) as a proxy for disease morbidity[11]

Because identification is challenging because of selectivity in infection, given that

[10] I focus on lobar pneumonia and non-specified pneumonia only, because this were the ones that react to sulfa drugs. Bronchopneumonia, did not exhibit any response to the drug (Lowenburg and Lowenburg (1939)).

[11] In the economic history literature, mortality is commonly used as a proxy for morbidity. For example, Lleras-Muney and Glied (2008), Almond, et al. (2012) and Bhalotra and Venkataramani (2015).

people who contracted pneumonia may be a selected sample of the population, my identification strategy uses the introduction of sulfa drugs in 1938, to determine the impact of sulfa drugs and pneumonia exposure on long-term outcomes. I cannot use a traditional differences-in-differences estimation strategy because sulfa drugs were introduced across the country at the same time. Hence, I will also use the cross-province difference in mortality. The idea is that provinces with higher pre-sulfa mortality levels should have experienced the largest mortality declines after the introduction of sulfa drugs; hence they benefit more from the new treatment compared to areas with low mortality levels. This empirical approach is often called "an intensity of treatment" research design and it is the most widely used approach in the literature of long term effects impact of diseases (see Bleakley (2007), Lucas (2010), and Bhalotra and Venkataramani (2015)). This can be seen in figure 6, which presents the convergence in pneumonia mortality after the introduction of sulfa drugs. The x-axis has the value of *basepneumonia*, which is the pre-sulfa pneumonia mortality rate in the birth province[12]. The y-axis shows the decline in mortality from pneumonia after sulfa drugs were introduced (hence is *basepneumonia* minus the value of pneumonia mortality in 1943). As the graph shows, there was a strong convergence in mortality levels. Provinces with higher pneumonia mortality levels pre-sulfa drugs experience the largest declines.

To estimate the long-term effects of pneumonia exposure during the birth year, I will compare cohorts born before the introduction of sulfa-drugs to cohorts born after. From my previous analysis, I know that sulfa-drugs became widely used in 1938, so I will compare the cohorts born before 1938 with those born later. The outcomes of interest will be years of schooling, if the person completed primary school, if they completed secondary school, if they are employed, and their disability status in adulthood [13].

I will estimate the following regression:

$$Y_{ibtc} = \alpha + \beta Post_t \times basepneumonia_b + \theta_b + \eta_t + \delta_c + e_{btc} \tag{7}$$

[12] $BasePneumonia$ corresponds to the province-specific average pneumonia mortality rate between 1933 and 1937.

[13] For more details about the variables, see the appendix.

Where Y_{ibtc} is the outcome recorded in adulthood for individual i, born in province b in year t, observed in census year c. $Post_t$ is equal to 1 for cohorts born on or after 1938, the year that sulfonamides were introduced. $basepneumonia_b$ is the pre-sulfa pneumonia mortality rate in the birth province b and is used as the proxy of pneumonia exposure, defined as the average province-specific pneumonia mortality rate from 1933 to 1937. I focus specifically on birth year exposures because it is during the first year of life where it is estimated that is where most nutrients are utilized for brain development, while this declines markedly for subsequent ages (Eppig et al. (2010)).

θ_b, η_b and δ_t are fixed effects for the birth province, birth year, and census year. I also include in my preferred specification province of birth-specific time trends. The province fixed effects capture unobserved differences that are constant across regions, while the birth year and census fixed effects control for changes in national policies, potential life cycle changes across cohorts, and other aggregate factors. Standard errors are clustered at the birth province level.

β compares the change in outcomes between cohorts born before and after sulfa drugs in areas that benefit more from sulfa against the same change for cohorts born in regions with lower mortality. If birth year exposure to sulfa drug led to better adulthood outcomes, we should expect a positive sign for β.

For my regressions I only use the 1970, 1982, and 1992 census, and I only consider the cohorts born between 1933 to 1943. I started with the 1933 cohort to avoid confounding effects from the Great Depression. Chile's GDP fell 13% in 1930 and 18% in 1931[14], and some literature states that being born during a recession led to worse outcomes in adulthood. For example, Thomasson and Fishback (2014) examine how economic conditions at the time of birth influenced various measures of socioeconomic success as adults in the US. They find that individuals born in the Great Depression in low-income states had substantially lower incomes and higher work disability rates during adulthood (see Cutler et al. (2007) for more evidence of this). I stop in the 1943 cohort because

[14]I don't have provincial-level data for economic activity or unemployment, but figure B.7 shows the relationship between total mortality and unemployment during the period. For the cohorts under study, there is no significant correlation between the two series.

penicillin was widely used after then.

Following Bhalotra and Venkataramani (2015), I also control for diseases not treatable by sulfa drugs, like tuberculosis and cancer, to control for other health improvements that may affect $Post_t$. I also include the under-2 diarrhea mortality base to control for better water quality and sewage treatment, which may affect mortality. Maternal mortality is also included as a control because sulfa drugs also considerably reduced maternal mortality, so our coefficients may be capturing these effects of the reduction in these diseases.

The regressions also include some health variables like the number of hospitals, the number of doctors, and province per capita health spending, this will help control as a proxy of access to sulfa drugs, given that we should expect that access to the drug will be easier in areas with more hospitals and health personnel.

I run the regression separately for men and women, this is important, because in the literature of the long-term impact of health shocks in infancy is essential to distinguish by gender. This is because it has been found that the effects for males are always larger than for females. This is because males are more vulnerable to side effects of maternal stress in utero. For example, Almond and Mazumder (2011) uses Ramadan as a natural experiment to see the impact of fasting on fetal health, and find that exposure to fasting in utero (especially during the first month of pregnancy) reduces the number of male births. Almond et al. (2007) find that fallout from the Chernobyl disaster had significant negative impacts on the percentage of live male births for cohorts that were in their second trimester during the disaster. Nilsson (2008) finds that lower alcohol prices, and the associated increase in consumption decreased the percentage of male births among cohorts conceived before the price decrease.

Summary statistics for the outcomes variables are presented in table C.2.

Table 6, panel A, reports the result for years of schooling for men born between 1933 and 1943, using data from the 1970, 1982, and 1992 censuses. My favorite specification is column 3, that shows that exposure to sulfa-drugs, and thereby less exposure to pneumonia in the birth year, led to a statistically significant improvement in years of education

for men. Column 3 suggests that a decrease of one standard deviation[15] in pneumonia exposure (mortality) is estimated to have resulted in an increase of 0.5 years of schooling for men.

Table 7, panels A and B, examine the effects on educational attainment, particularly the likelihood of completing primary and secondary school. Column 3 suggests that a one standard deviation decrease in pneumonia exposure (mortality) is estimated to have resulted in an increase of 7 percentage points in the probability of completing primary and a three percentage points in completing secondary school.

In table 6, panel B, I show the results for employment. The employment variable is an indicator that equals one if the person is employed. The coefficient, in this case, is significant and positive after all the controls cariables are included. Results show that being born in an environment with a lower incidence of pneumonia is associated with a 2.8% increase in the likelihood of being employed. Table 8 exhibit the results for physical and mental disability, however, these results are not significant.

In order of magnitude, for the US, Bhalotra and Venkataramani (2015) find a smaller effect on education and employment. They find that a one standard deviation decrease in pneumonia mortality was associated with 0.1 more years of schooling, a 1.5 percentage point increase in the probability of completing high school, and a 0.43 percentage point increase in the probability of being employed. The difference in results is consistent with higher mortality levels in Chile and with lower baseline levels on education and employment (for example, while the mean year of schooling in Chile was 6.6 years, in the US it was over 12).

The results for women are not significant, and in most cases, they show a negative and insignificant effect for all the variables studied (Results are in tables C 3-8). This outcome may be because men's pneumonia incidence in childhood (and, more specifically, under one year old) was much higher for men than for women; therefore, they benefited the most from sulfa drug availability. Figure B.7 shows the incidence of pneumonia by

[15]The mean of *Basepneumonia* mean was 160 deaths per 100,000 population, while the standard deviation is 78.5.

gender and mortality for male children under 1 year old was larger than for females. This finding is consistent with Bhalotra and Venkataramani (2015), who also find significant results only for men.

Additionally, to examine whether the start of the convergence of provinces in the outcomes coincide with the introduction of sulfa drugs, and to see if exposure to pneumonia at other ages matters, I estimate:

$$Y_{ibtc} = \alpha + \beta_t basepneumonia_b + \theta_b + \eta_t + \delta_c + e_{btc} \tag{8}$$

This also includes all the previous controls.

This equation provides birth-cohort-specific coefficient estimates of the introduction of sulfa drugs. I present these β_t in figures 7, 8, 9, and 10, only for men.

Figure 7 plots the cohort-specific coefficient estimates with years of education as a dependent variable. This chart shows that after 1938, the year sulfa drugs were introduced, there is a trend break; the coefficient gets larger, providing visual evidence of the positive long-run impact of sulfa drugs. The same effect is observed for the other variables[16].

Robustness

A potential threat to the identification strategy is that *BasePneumonia* correlates with other variables that affect long-term outcomes. In table 9, I show some of the correlation between *BasePneumonia* and other variables. Each cell is a different cross-section regression. For example, % working in agriculture represent the percentage of the population working in agriculture in each province in 1930. It can be seen that working in agriculture, percentage of the population illiterate, percentage rural, and percentage foreign are correlated with *BasePneumonia*. To correct for these factors, I add the significant variables to the previous regression. In particular, I include linear trends interacting with these variables. Table 10 exhibits the results. When I include these

[16] A small amount of pre-trend can be observed for the variable employment. This shift may be due to the fact that better health during the first 5 years of life matters, not only in utero and during the first year of life, this variable may be capturing this effect.

variables, the results remain significant, although some of the coefficients get slightly smaller.

As a second robustness check, I do a placebo test, changing the *Post* year from 1938 to the surrounding years. If these effects are driven by the decline in mortality caused by sulfa drugs, the change of year should result in coefficients that are not significant. Table C.9 presents these results. Every cell is a different regression, and all the previous controls are included. The results for years other than 1938 are not significant, providing evidence that the results are due to less pneumonia exposure in infancy driven by the availability of sulfa drugs.

Third, I control for parents' education. The results may be due to the fact that the cohorts born after 1938 are different than the previous cohorts for reasons other than sulfa drugs. One possibility is that the parents of the cohorts born after the sulfa drugs were of better quality than parents of the earlier cohorts. This possibility is relevant because Dehejia and Lleras-Muney (2014) study how babies conceived in times of high unemployment are healthier than babies conceived during other times, and what drives these health improvements is selection (changes in the type of mothers who conceive during recessions). Also, Brown and Thomas (2018) find that the negative impacts of being exposed to the 1918 influenza pandemic in the US were also due to selection into parenthood. They find that the 1918 cohort was more likely to be born to families of lower socioeconomic status relative to those who were not exposed. For example, the fathers of the 1919 birth cohort were less likely to be literate, worked in lower-earning occupations, had lower socioeconomic status, were older, were less likely to be White, and had higher fertility. After controlling for parents' characteristics, there is little evidence that individuals born in 1919 have worse socioeconomic outcomes in adulthood relative to the surrounding cohorts[17].

To control for this, I add the parents' education (father and mother education, measured as years of schooling) to the regressions. I present the finding in table 11. The first column is the previously preferred specification, with all the previous controls except

[17]See Grossman (2006) for a detailed review of the importance of parental education for child health.

parents' education. The second column controls for parents' education, and the third column repeats the regression of column 1 only using those people for whom parental education information is available. Again, each cell is a separate regression. Including parents' education changes the results; they are no longer significant, and sometimes they change signs. However, this shift is the result of restricting the sample to people for whom parental education data are available. The results of columns 2 and 3 are relatively similar[18].

Fourth, I extend the period under study to include cohorts born between 1929 and 1948 (instead of 1933–1943). Table C.10 shows these results. The significance of the coefficient remains, but its magnitude is reduced. In every case, we still observe the positive impact of being born in a birth year that was exposed to less pneumonia.

Fifth, I use the tuberculosis pre-sulfa-drug average (*basetuberculosis*) as a placebo disease. The results in table 12 show that places with higher tuberculosis mortality during infancy are associated with lower educational attainment in adulthood. This result should be expected because the geographical distribution of tuberculosis was very different than the distribution of pneumonia (see figures B.9 and B.10).

I also repeat the results for men separately for each census. Tables C.11, C.12, C.13 and C.14 presents the results for men. The results are consistent with the previous analysis. There is a positive impact on education and employment of being less exposed to pneumonia in the birth year.

[18]Ipums only have data on parents' education when families share a household.

Conclusion

This paper contributes to studying the impacts of short- and long-term medical advances. In particular, I examine how sulfa drugs led to a decline in mortality using a difference-in-difference estimation. The results suggest that sulfa drugs resulted in a drop of 10–28% in maternal mortality. They also led to a 25–50% decline in pneumonia mortality and a 10–40% decline in meningitis mortality.

Additionally, in the long run, sulfa drugs also lead to an increase in education and employment. Using an "intensity of treatment" research design, I find that, for men, a decrease of one standard deviation in pneumonia exposure (mortality) is estimated to have resulted in an increase of 0.5 years of schooling, seven percentage points in the probability of completing primary school, and three percentage points in the probability of completing secondary school. The results are also significant and positive for employment, showing that being born in an environment with a lower incidence of pneumonia is associated with a 2.8 percent point increase in the likelihood of being employed. I do not find significant results for physical disability, mental disability, or for any outcomes for women.

Also, these results are relevant for policymakers now. Even today, only one-third of children who have pneumonia can access antibiotics (WHO, 2019), despite their low cost. Given that sulfa drugs are still widely used in the developing world, understanding not only the short-term impacts on reducing mortality but also the long-term impacts is essential. Focusing only on the effects of sulfa drug availability in mortality underestimates the real long-term benefits.

References

Almond, D. (2006). Is the 1918 Influenza Pandemic Over? Long-Term Effects of In Utero Influenza Exposure in the Post-1940 U.S. Population. Journal Of Political Economy, 114(4), 672-712. doi: 10.1086/507154

Almond, D., Edlund, L., & Palme, M. (2009). Chernobyl's Subclinical Legacy: Prenatal Exposure to Radioactive Fallout and School Outcomes in Sweden. Quarterly Journal Of Economics, 124(4), 1729-1772. doi: 10.1162/qjec.2009.124.4.1729

Almond, D., & Mazumder, B. (2011). Health Capital and the Prenatal Environment: The Effect of Ramadan Observance During Pregnancy. American Economic Journal: Applied Economics, 3(4), 56-85. doi: 10.1257/app.3.4.56

Almond, D., Currie, J., & Herrmann, M. (2012). From infant to mother: Early disease environment and future maternal health. Labour Economics, 19(4), 475-483. doi: 10.1016/j.labeco.2012.05.015

Barker, D. (1990). The fetal and infant origins of adult disease. BMJ, 301(6761), 1111-1111. doi: 10.1136/bmj.301.6761.1111

Barreca, A. (2010). The Long-Term Economic Impact of In Utero and Postnatal Exposure to Malaria. Journal Of Human Resources, 45(4), 865-892. doi: 10.1353/jhr.2010.0027

Bhalotra, S., & Venkataramani, A. (2011). The Captain of the Men of Death and His Shadow: Long-Run Impacts of Early Life Pneumonia Exposure. SSRN Electronic Journal. doi: 10.2139/ssrn.1940725

Bleakley, H. (2010). Malaria Eradication in the Americas: A Retrospective Analysis of Childhood Exposure. American Economic Journal: Applied Economics, 2(2), 1-45. doi: 10.1257/app.2.2.1

Bozzoli, C., Deaton, A., & Quintana-Domeque, C. (2009). Adult Height and Childhood Disease. Demography, 46(4), 647-669. doi: 10.1353/dem.0.0079

Brown, R, & Thomas, D. (2018). On the long term effects of the 1918 US influenza pandemic. Unpublished Manuscript.

Crimmins, E., & Finch, C. (2005). Infection, inflammation, height, and longevity. Proceedings Of The National Academy Of Sciences, 103(2), 498-503.
doi: 10.1073/pnas.0501470103

Cutler, D., Fung, W., Kremer, M., Singhal, M., & Vogl, T. (2010). Early-life Malaria Exposure and Adult Outcomes: Evidence from Malaria Eradication in India. American Economic Journal: Applied Economics, 2(2), 72-94. doi: 10.1257/app.2.2.72

Cutler, D., Miller, G., & Norton, D. (2007). Evidence on early-life income and late-life health from America's Dust Bowl era. Proceedings Of The National Academy Of Sciences, 104(33), 13244-13249. doi: 10.1073/pnas.0700035104

Dehejia, R., & Lleras-Muney, A. (2004). Booms, Busts, and Babies' Health. The Quarterly Journal Of Economics, 119(3), 1091-1130. doi: 10.1162/0033553041502216

Díaz, J., Lüders. R. & Wagner, G. (2016). Chile 1810 – 2010. La República en cifras. Historical statistics. Santiago: Ediciones Universidad Católica de Chile

Eppig, C., Fincher, C., & Thornhill, R. (2010). Parasite prevalence and the worldwide distribution of cognitive ability. Proceedings Of The Royal Society B: Biological Sciences, 277(1701), 3801-3808. doi: 10.1098/rspb.2010.0973

Glied, S., & Lleras-Muney, A. (2008). Technological Innovation and Inequality in Health. Demography, 45(3), 741-761. doi: 10.1353/dem.0.0017

Jayachandran, S., Lleras-Muney, A., & Smith, K. (2010). Modern Medicine and the Twentieth Century Decline in Mortality: Evidence on the Impact of Sulfa Drugs. American Economic Journal: Applied Economics, 2(2), 118-146. doi: 10.1257/app.2.2.118

Lazuka, V. (2018). Infant Health and Later-Life Labor Market Outcomes. Journal Of Human Resources, 55(2), 660-698. doi: 10.3368/jhr.55.3.0817-9016r

Lesch, J. E. (2007). The first miracle drugs: How the sulfa drugs transformed medicine. Oxford: Oxford University Press.

Lowenburg, H., & Lowenburg, H. (1939). Sulfapyridine in the Treatment of Pneumonia in Children. Diseases Of The Chest, 5(8), 8-12. https://doi.org/10.1378/chest.5.8.8

Minnesota Population Center. Integrated Public Use Microdata Series, International: Version 7.2 [dataset]. Minneapolis, MN: IPUMS, 2019. https://doi.org/10.18128/D020.V7.2

Myrskylä, M. (2010). The effects of shocks in early life mortality on later life expectancy and mortality compression: A cohort analysis. Demographic Research, 22, 289-320. doi: 10.4054/demres.2010.22.12

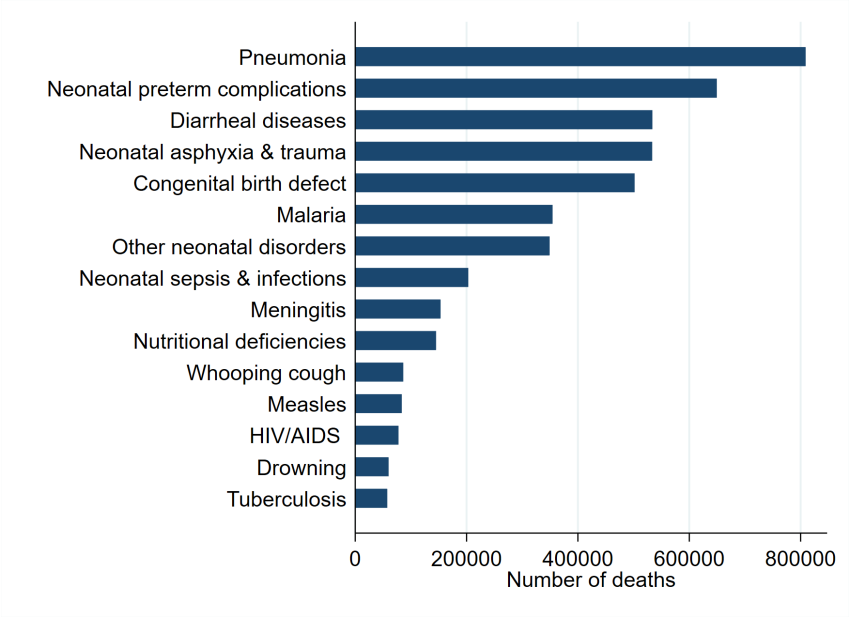
Nilsson, J. P. (2008). Does a Pint a Day Affect Your Child's Pay? Unintended and Permanent Consequences of a Temporary Alcohol Policy Experiment." CEMMAP Working Paper.

World Health Organization, Pneumonia Fact sheet. (2019). Retrieved 28 August 2020, from https://www.who.int/news-room/fact-sheets/detail/pneumonia

Venkataramani, A. (2012). Early life exposure to malaria and cognition in adulthood: Evidence from Mexico. Journal Of Health Economics, 31(5), 767-780.
doi: 10.1016/j.jhealeco.2012.06.003

Figures

Figure 1: Cause of death in children under 5, 2017



Source: IHME, Global Burden of Disease (GBD).

Figure 2

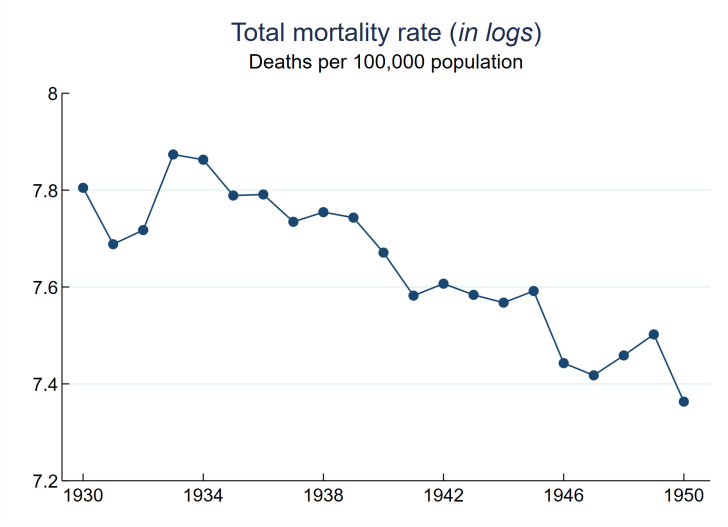

Total mortality rate (*in logs*)
Deaths per 100,000 population
8
7.8
7.6
7.4
7.2
1930
1934
1938
1942
1946
1950

Figure 3

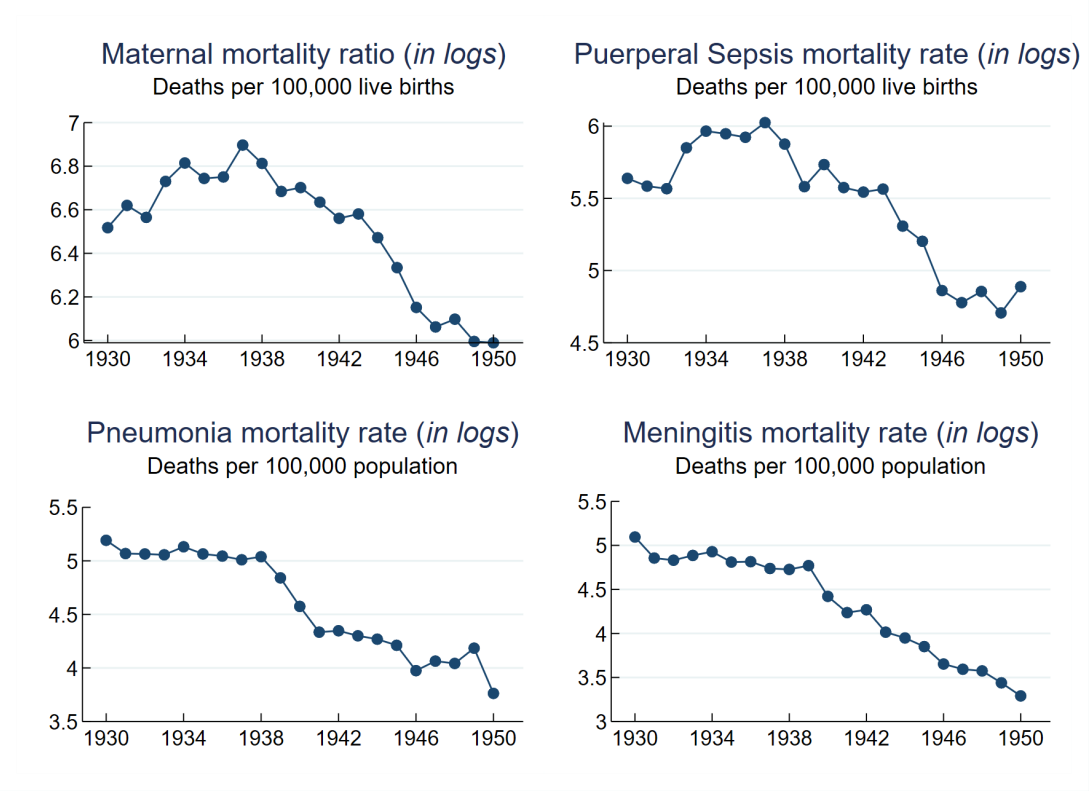

Maternal mortality ratio (*in logs*)
Deaths per 100,000 live births
7
6.8
6.6
6.4
6.2
6
1930
1934
1938
1942
1946
1950
Puerperal Sepsis mortality rate (*in logs*)
Deaths per 100,000 live births
6
5.5
5
4.5
1930
1934
1938
1942
1946
1950
Pneumonia mortality rate (*in logs*)
Deaths per 100,000 population
5.5
5
4.5
4
3.5
1930
1934
1938
1942
1946
1950
Meningitis mortality rate (*in logs*)
Deaths per 100,000 population
5.5
5
4.5
4
3.5
3
1930
1934
1938
1942
1946
1950

Figure 4

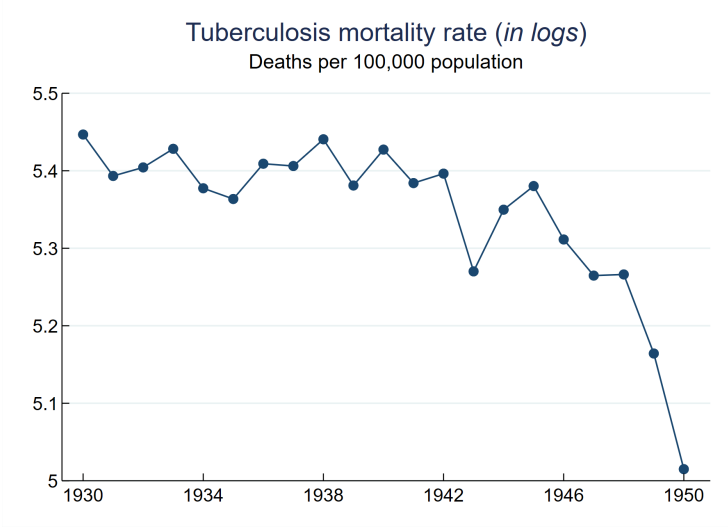
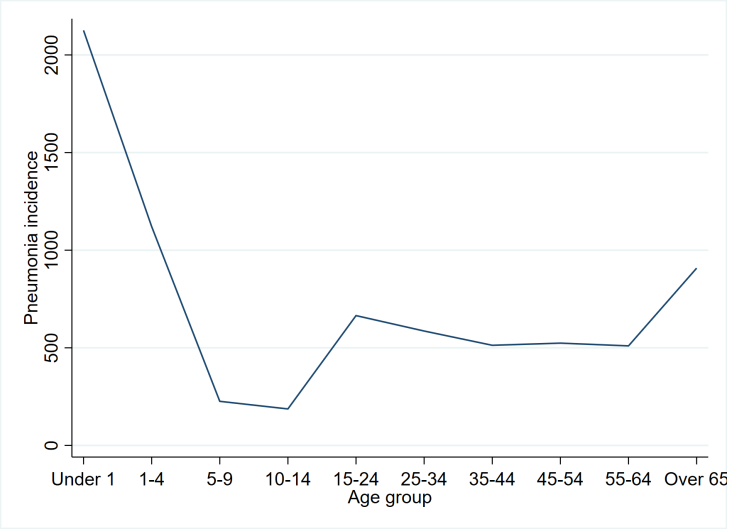
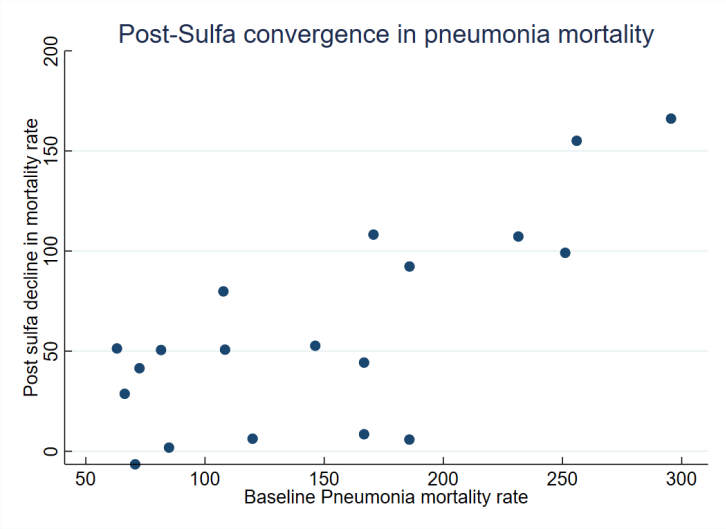

Tuberculosis mortality rate (*in logs*)
Deaths per 100,000 population
5.5
5.4
5.3
5.2
5.1
5
1930
1934
1938
1942
1946
1950

Figure 5: Age distribution of pneumonia incidence, 1935



Note: Pneumonia incidence is the number of deaths due to pneumonia for each age group.
Source: National Institute of Statistics (Chile).

Figure 6: Pneumonia convergence



Note: Base Pneumonia is the average of pneumonia mortality during 1933-1937. Pneumonia decline is the change in mortality between 1943 and base pneumonia.

Figure 7: Cohort-specific coefficient, primary schooling

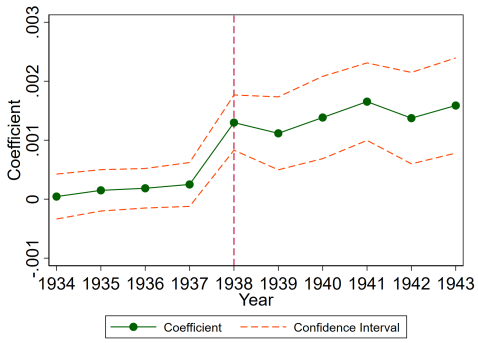


Figure 8: Cohort-specific coefficient, secondary schooling

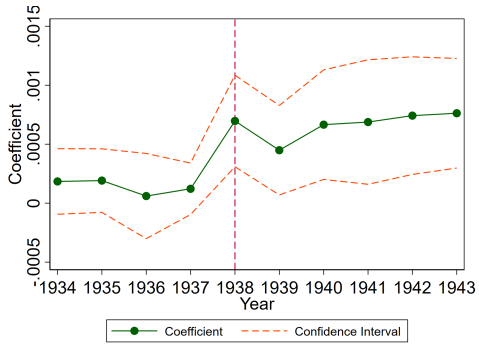


Figure 9: Cohort-specific coefficient, years of schooling

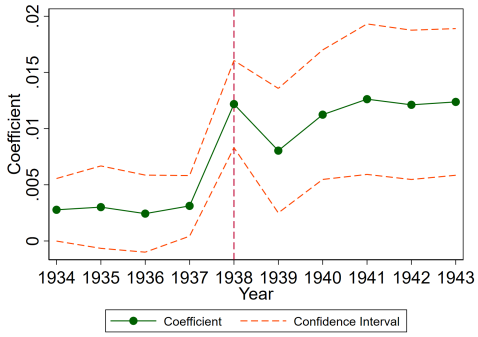
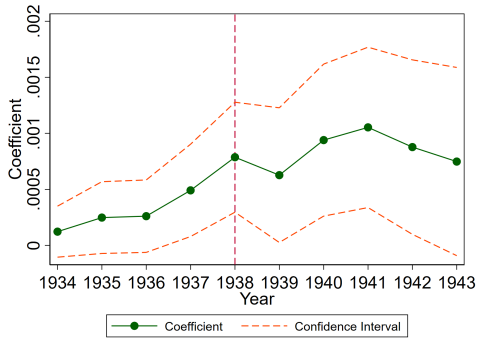


Figure 10: Cohort-specific coefficient, employment



Notes: Each point reflects the coefficient estimated on an interaction term between the birth year and the pre-intervention (base) level of the pneumonia mortality rate in the birth-state. All models includes the full set of previous control variables, and standard errours are cluesterd by province of birth.

Tables

Table 1: Summary Statistics, mortality rates

	Pre-sulfa	Post-sulfa	All period
	1930-38	1939-50	1930-50
All Causes Mortality	2395	1900	2113
Diseases treated with sulfa drugs			
MMR	831.9	596.5	697.4
Puerperal sepsis	341.3	196.5	258.6
Pneumonia	165.9	76.7	114.9
Scarlet Fever	2.13	0.65	1.3
Meningitis	129	55.4	86.9
Control disease			
Tuberculosis	223.2	201.7	210.9
Chronic diseases			
Diabetes	4.4	4.8	4.6
Circulatory system	184.7	194.2	190.1
Cancer	68.5	78.6	74.3
Infectious diseases not treated with sulfa			
Diarrhea (under 2 years old)	184.7	144.7	162.7
Accidents	100	87.6	93

Note: Mortality rates are calculated as number of deaths per 100,000 population for all the variables except maternal mortality and puerperal sepsis, for those variables rates are calculated as deaths per 100,000 live births.

Table 2

Total mortality and Control disease			
	Total mortality	Tuberculosis	Cancer
Post-1939	-0.142***	-0.0043	-0.00406
	(0.04)	(0.0348)	(0.0321)
Treated diseases			
	MMR	Pneumonia	Meningitis
Post-1939	-0.256**	-0.480***	-0.378***
	(0.116)	(0.0821)	(0.0545)

Notes: Estimates based on 1930-1943 national-level mortaity rates. To account for serial correlation I compute Newey-West standard errors. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Table 3

Total mortality and Control disease				
	Total mortality		Tuberculosis	
	(1)	(2)	(1)	(2)
Post-1939	-0.142***	-0.0968**	-0.0043	0.0880**
	(0.04)	(0.0393)	(0.0348)	(0.028)
Year x post-1939		-0.0217**		-0.0440***
		(0.00912)		(0.0105)
Treated diseases				
	MMR		Pneumonia	
	(1)	(2)	(1)	(2)
Post-1939	-0.256**	-0.107	-0.480***	-0.358***
	(0.116)	(0.0773)	(0.0821)	(0.0864)
Year x post-1939		-0.0710***		-0.0583**
		(0.0123)		(0.0214)

Notes: Estimates based on 1930-1943 national-level mortaity rates. To account for serial correlation I compute Newey-West standard errors. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Table 4: Difference-in-difference: National level series

	MMR		Pneumonia		Meningits	
	(1)	(2)	(1)	(2)	(1)	(2)
Treated x post-1939	-0.279***	-0.204***	-0.372**	-0.255**	-0.142	-0.0078
	(0.0899)	(0.0639)	(0.157)	(0.0923)	(0.167)	(0.0965)
Treated x year x post-1939		-0.0747***		-0.117***		-0.134***
		(0.0113)		(0.0308)		(0.0286)
Obs	28	28	28	28	28	28
R^2	0.998	0.999	0.999	0.999	0.998	0.999

Notes: Robust standard errors in parenthesis. clustered by disease-year, are shown in parentheses.
$^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table 5: Difference-in-difference: Province level series

	MMR		Pneumonia		Meningits	
	(1)	(2)	(1)	(2)	(1)	(2)
Treated x post-1939	-0.198***	-0.200***	-0.293*	-0.308***	-0.099	-0.118
	(0.0503)	(0.0575)	(0.169)	(0.0878)	(0.199)	(0.0925)
Treated x year x post-1939		-0.00901		-0.0982***		-0.130***
		(0.0163)		(0.0321)		(0.0297)
Obs	492	492	492	492	490	490
R^2	0.876	0.888	0.553	0.577	0.746	0.765

Notes: Robust standard errors, clustered by disease-year, are shown in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table 6

Panel A

Dependent variable: Years of schooling			
	(1)	(2)	(3)
Base Pneumonia	0.0022***	0.0020**	0.0066***
	(0.00057)	(0.00090)	(0.0016)
	[126027]	[126027]	[126027]
Mean of dep var	6.65	6.65	6.65
Standard deviation of dep var	4.22	4.22	4.22

Panel B

Dependent variable: Employment			
Base Pneumonia	0.000075	0.00028**	0.00036***
	(0.00013)	(0.00013)	(0.00012)
	[125313]	[125313]	[125313]
Mean of dep var	0.82	0.82	0.82
Standard deviation of dep var	0.39	0.39	0.39
Fixed Effects	Yes	Yes	Yes
Control diseases	Yes	Yes	Yes
Maternal mortality	Yes	Yes	Yes
Health variable	No	Yes	Yes
Trend by province	No	No	Yes

Notes: Robust standard errors, clustered by province of birth, are shown in parentheses, number of observations in square brackets. $*p < 0.1$, $**p < 0.05$, $***p < 0.01$. Regressions only for men born between 1933 and 1943. Fixed effects include province of birth, year and census fixed effects.

Table 7

Panel A

Dependent variable: Completed primary			
	(1)	(2)	(3)
Base Pneumonia	0.00024***	0.00019**	0.00091***
	(0.000055)	(0.000084)	(0.00012)
	[126314]	[126314]	[126314]
Mean of dep var	0.60	0.60	0.60
Standard deviation of dep var	0.49	0.49	0.49

Panel B

Dependent variable: Completed secondary			
Base Pneumonia	0.00017**	0.00021***	0.00041**
	(0.000061)	(0.000060)	(0.00017)
	[126314]	[126314]	[126314]
Mean of dep var	0.16	0.16	0.16
Standard deviation of dep var	0.37	0.37	0.37
Fixed Effects	Yes	Yes	Yes
Control diseases	Yes	Yes	Yes
Maternal mortality	Yes	Yes	Yes
Health variable	No	Yes	Yes
Trend by province	No	No	Yes

Notes: Robust standard errors, clustered by province of birth, are shown in parentheses, number of observations in square brackets. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$. Regressions only for men born between 1933 and 1943. Fixed effects include province of birth, year and census fixed effects.

Table 8

Panel A

Dependent variable: Physical disability			
	(1)	(2)	(3)
Base Pneumonia	0.000038	-0.000014	0.00016
	(0.000090)	(0.000094)	(0.00012)
	[39728]	[39728]	[39728]
Mean of dep var	0.04	0.04	0.04
Standard deviation of dep var	0.19	0.19	0.19

Panel B

Dependent variable: Mental disability			
Base Pneumonia	0.0000086	-0.000016	-0.000066
	(0.000028)	(0.000040)	(0.000052)
	[39728]	[39728]	[39728]
Mean of dep var	0.01	0.01	0.01
Standard deviation of dep var	0.07	0.07	0.07
Fixed Effects	Yes	Yes	Yes
Control diseases	Yes	Yes	Yes
Maternal mortality	Yes	Yes	Yes
Health variable	No	Yes	Yes
Trend by province	No	No	Yes

Notes: Robust standard errors, clustered by province of birth, are shown in parentheses, number of observations in square brackets. $*p < 0.1$, $**p < 0.05$, $***p < 0.01$. Regressions only for men born between 1933 and 1943. Fixed effects include province of birth, year and census fixed effects.

Table 9: Correlates with BasePneumonia

	BasePneumonia
% working in agriculture	1.9470**
	(0.7139)
% working in industry	-2.0583
	(2.4334)
% illiterate	4.8330***
	(1.4137)
Number of births	0.0006
	(0.0020)
% rural	1.6090**
	(0.6134)
Number of doctors	-0.0979
	(0.0621)
Number of hospitals	1.6091
	(2.5161)
Population density	1.0493
	(1.3767)
% foreigner	-10.4921***
	(2.8233)
Illegitimate births	-1.4961
	(2.4114)
N	18

Notes: Each cell reports coefficients for a cross section regression with *BasePneumonia* as the dependent variable. Percentage working in agriculture, % working in industry, % illiterate, percentage rural, %foreigner come from the 1930 census. For the number of births, doctors, hospital, population density and illegitimate births I use the 1930 Demographic yearbook. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table 10: Results for men

Years of schooling	0.0066***	0.0062**
	(0.0016)	(0.0027)
	[126027]	[126027]
Primary	0.00091***	0.00071**
	(0.00012)	(0.00024)
	[126314]	[126314]
Secundary	0.00041**	0.00049**
	(0.00017)	(0.00021)
	[126314]	[126314]
Employment	0.00036***	0.00030*
	(0.00012)	(0.00016)
	[125313]	[125313]
Previous control	Yes	Yes
% Rural	No	Yes
% working in agriculture	No	Yes
% illiterate	No	Yes
% foreigner	No	Yes

Robust standard errors, clustered by province of birth, are shown in parentheses, number of observations in square brackets. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$. Regressions only for men born between 1933 and 1943. Fixed effects include province of birth, year and census fixed effects.

Table 11

	Men		
	(1)	(2)	(3)
Years of schooling	0.0066***	-0.0023	-0.012
	(0.0016)	(0.0062)	(0.0081)
	[126027]	[5382]	[5382]
Primary	0.00091***	0.00074	-0.00015
	(0.00012)	(0.00058)	(0.00072)
	[126314]	[5392]	[5392]
Secondary	0.00041**	0.0011	0.00045
	(0.00017)	(0.00074)	(0.00086)
	[126314]	[5392]	[5392]
Employment	0.00036***	0.0026***	0.0026***
	(0.00012)	(0.00064)	(0.00064)
	[125313]	[5334]	[5334]
Previous control	Yes	Yes	Yes
Parents education	No	Yes	No

Notes: Each cell reports coefficient on $post * basepneumonia$ from a separate regression. Robust standard errors, clustered by province of birth, are shown in parentheses. The number of observations is shown in square parentheses. Regressions use IPUMS sampling weights.

$^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table 12: Results for men

	Pneumonia	Tuberculosis
Years of schooling	0.0066***	-0.012***
	(0.0016)	(0.0028)
Primary	0.00091***	-0.0018***
	(0.00012)	(0.00034)
Secondary	0.00041**	-0.00040
	(0.00017)	(0.00032)
Employment	0.00036***	-0.0013***
	(0.00012)	(0.00026)
Disability	0.00016	-0.00038
	(0.00012)	-0.0003

Notes: Each cell reports coefficient on $post * basetuberculosis$ from a separate regression. Robust standard errors, clustered by province of birth, are shown in parentheses. The number of observations is shown in square parentheses. Regressions use IPUMS sampling weights.
$^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

A Appendix: Data Sources and Variables description

The outcomes variables were taken from IPUMS international, (https://international.ipums.org/international/).

For most of my regressions I pooled data from the 1970, 1982 and 1992 censuses. The exception is the variable of disability and mental disability that is only available since 1992.

Years of schooling (YRSCHOOL): accounts for the number of years of study.

Completed Primary (EDATTAIN) records the person's educational attainment in terms of the level of schooling completed. In this case, is a dummy equals 1 if the person complete primary schooling.

Completed Secondary (EDATTAIN): is a dummy equals 1 if the person complete secondary schooling.

Employed (EMPSTAT): dummy equals 1 if the individual reports current employment and 0 otherwise.

Disability (DISABLED): indicates whether the person reported a disability of any kind. It is a dummy equal 1 if the person is disable.

Mental disability (DISMNTL): indicates whether the person suffered a mental disability in the form of diminished capacity. It is a dummy equal 1 if the person is disable.

B Appendix: Figures

Figure B.1: First Sulfonamide drug, Prontosil

Figure B.2: Data

Defunciones generales por provincias, indicando el % de certificados por médico	Total general	Total por sexo		Tarapacá		Antofagasta		Atacama		Coquimbo		Aconcagua		Santiago	
		H	M	H	M	H	M	H	M	H	M	H	M	H	M
Grupo I.—Enfermedades infecciosas y parasitarias	**26 313**	**13046**	**13267**	**329**	**301**	**325**	**240**	**185**	**160**	**716**	**739**	**1 182**	**1 213**	**3 306**	**3 230**
1 Fiebre tifoidea (tifus abdominal)	681	387	294	9	2	4	6	5	8	22	30	39	19	59	38
2 Fiebres paratifoideas (paratifus)	23	10	13	2	—	1	—	—	1	1	2	1	2	4	3
3 Tifus exantemático	790	493	297	—	—	—	—	1	—	18	9	19	10	219	135
7 Sarampión	514	265	249	—	—	2	—	1	—	1	5	56	56	151	142
8 Escarlatina	52	23	29	—	—	12	10	—	—	—	—	—	1	6	10
9 Coqueluche	2 110	914	1 196	14	13	17	23	10	12	65	59	78	96	293	394
10 Difteria	243	116	127	—	1	4	4	2	1	6	9	14	12	33	44
11a Grippe con complicaciones respiratorias mencionadas	1 835	919	916	32	34	23	15	26	17	106	104	77	84	73	76
11b Grippe sin complicaciones respiratorias mencionadas	5 805	2 871	2 934	35	33	15	16	23	17	162	204	85	64	116	111
13 Disenterías	202	108	94	6	10	3	4	3	2	5	7	6	3	39	22
15 Erisipela	83	43	40	2	2	3	2	1	—	1	2	4	5	21	17
16 Poliomielitis aguda y polioencefalitis aguda	4	1	3	—	1	—	—	—	—	—	—	—	—	1	—

Source: National Institute of Statistics (Chile).

Figure B.3

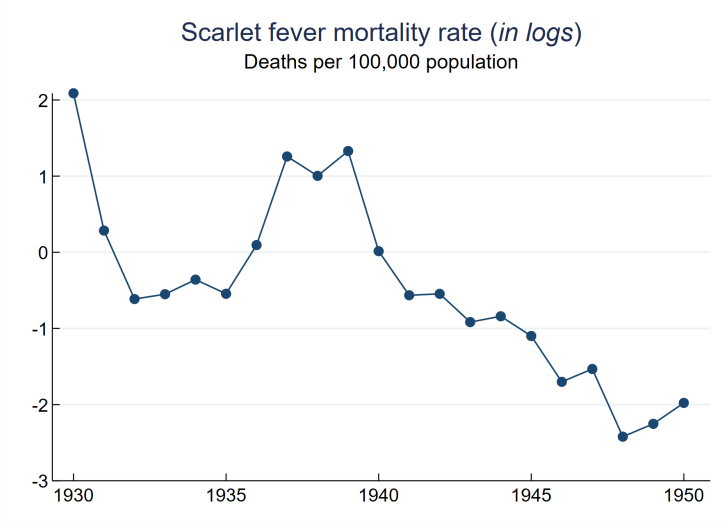
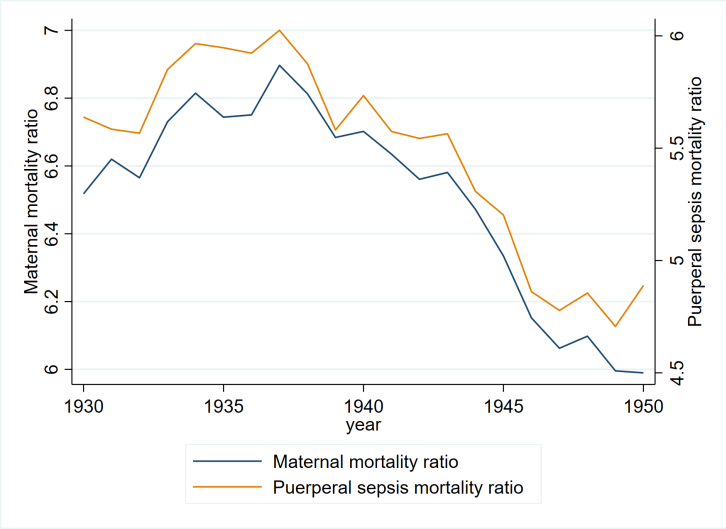

Scarlet fever mortality rate (*in logs*)
Deaths per 100,000 population
2
1
0
-1
-2
-3
1930
1935
1940
1945
1950

Figure B.4



Note: Mortality rates calculated as number of deaths per 100,000 live births. Series in natural logarithm. Maternal mortality on the left-axis, and puerperal sepsis mortality on the right axis.

Figure B.5

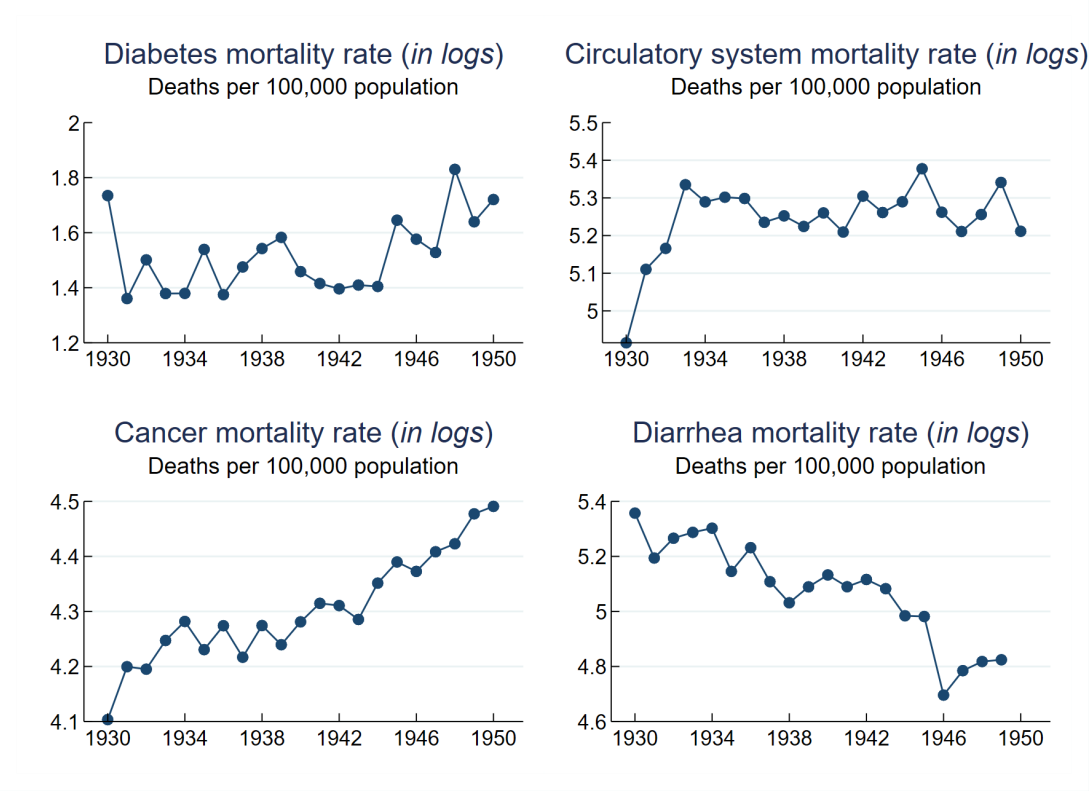
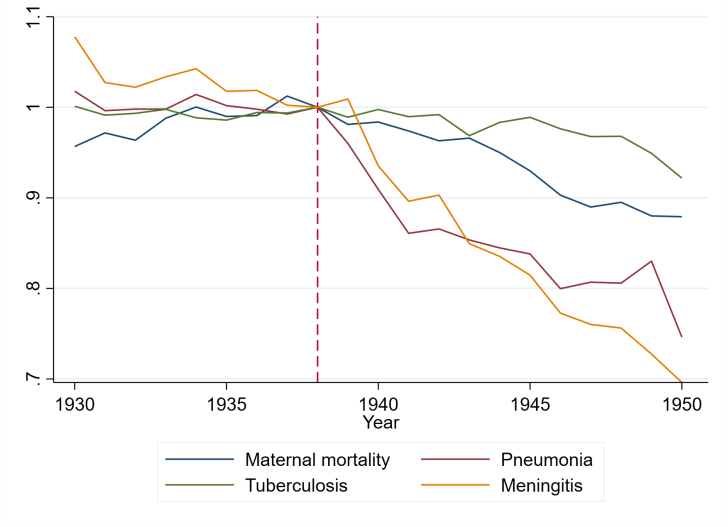
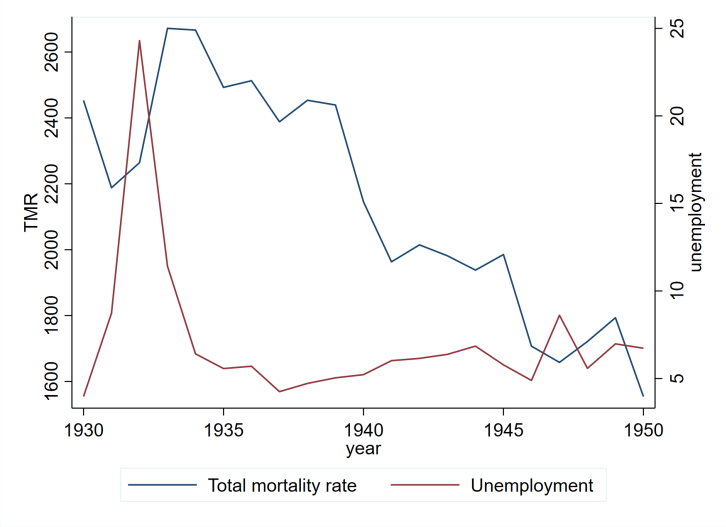

Diabetes mortality rate (*in logs*)
Deaths per 100,000 population
2
1.8
1.6
1.4
1.2
1930
1934
1938
1942
1946
1950
Circulatory system mortality rate (*in logs*)
Deaths per 100,000 population
5.5
5.4
5.3
5.2
5.1
5
1930
1934
1938
1942
1946
1950
Cancer mortality rate (*in logs*)
Deaths per 100,000 population
4.5
4.4
4.3
4.2
4.1
1930
1934
1938
1942
1946
1950
Diarrhea mortality rate (*in logs*)
Deaths per 100,000 population
5.4
5.2
5
4.8
4.6
1930
1934
1938
1942
1946
1950

Figure B.6



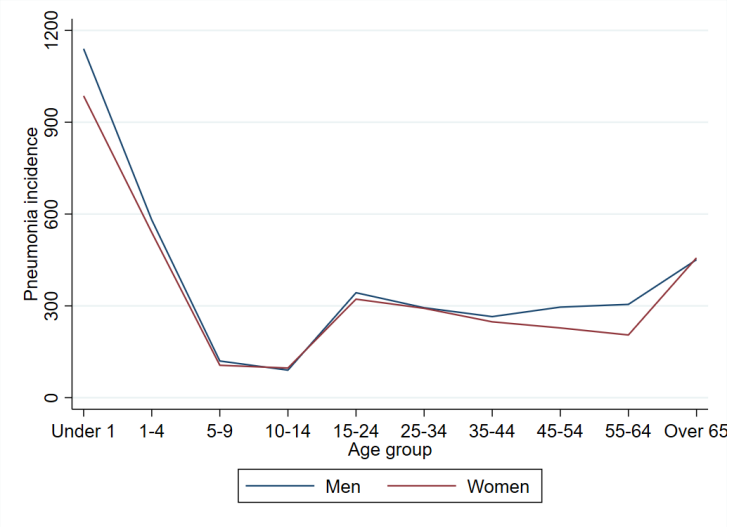
Note: Mortality rates per 100,000 population for all the variables except maternal mortality. For maternal mortality rate is calculated as deaths per 100,000 live briths. Mortality rates normalized to 1 in 1938.

Figure B.7



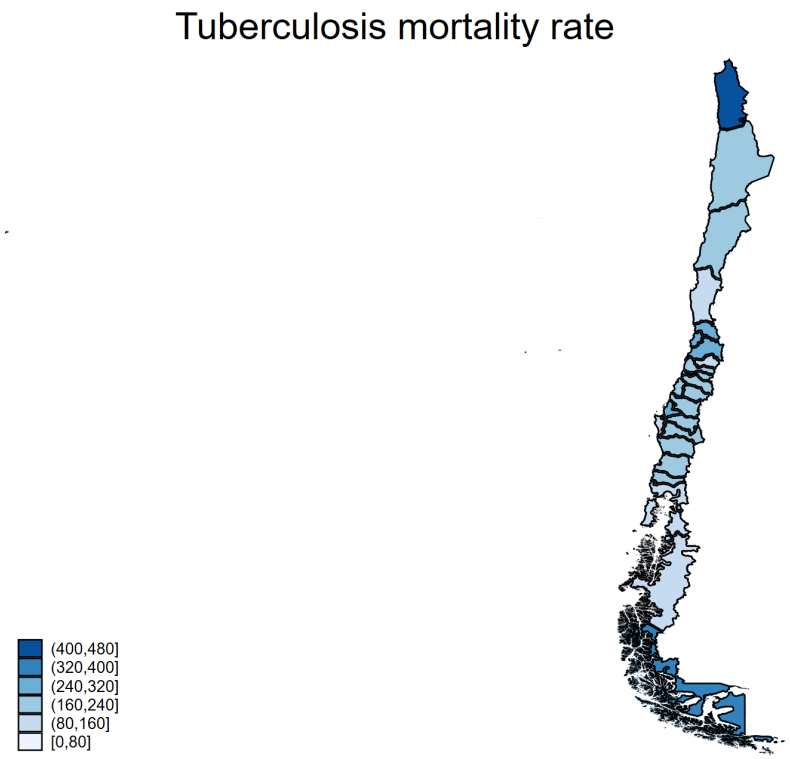
Note: Total mortality rates per 100,000 population in the left axis, unemployment rate in the right axis.
Source: Mortality : Demographic yearbook; unemployment: Díaz, J.; Lüders. R. y Wagner, G. (2016).

Figure B.8: Age distribution of pneumonia incidence by gender, 1935



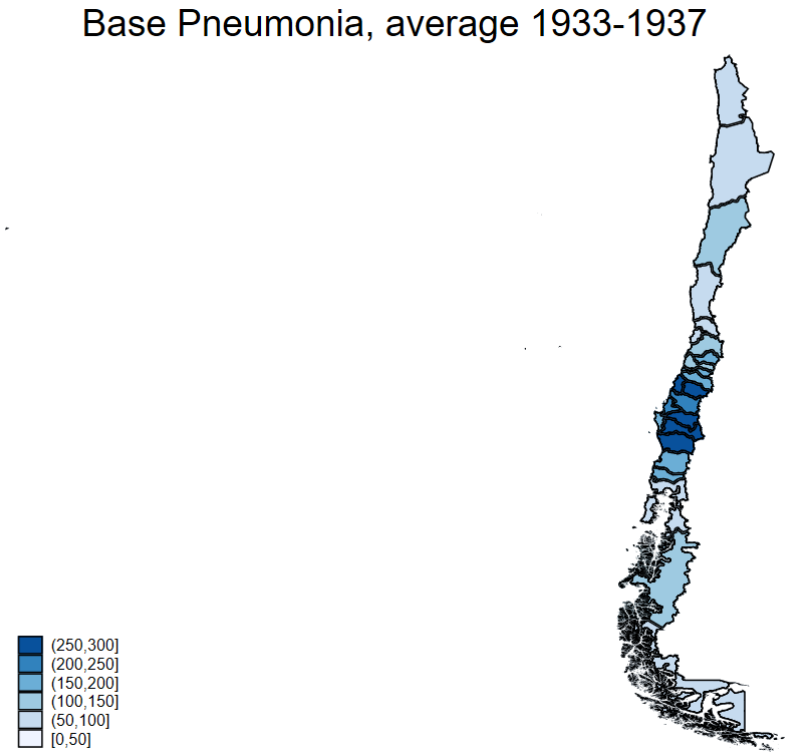
Notes: Pneumonia incidence is the number of deaths due to pneumonia for each age group.
Source: National Institute of Statistics (Chile).

Figure B.9



Notes: Base tuberculosis, average of tuberculosis mortality between 1933 to 1937.

Figure B.10



Notes: Base Pneumonia, average of pneumonia mortality between 1933 to 1937.

C Appendix: Tables

Table C.1: Summary statistics

	1933-1950		1933-1938		1939-1950	
	Mean	Std dev.	Mean	Std dev.	Mean	Std dev.
Hospitals	12.15	9.28	10.83	7.42	12.81	10.04
Doctors	94.43	182.95	67.52	120.05	107.89	206.35
Hospital beds	1436.84	2012.11	1164.91	1575.83	1572.81	2189.20
Relative values						
Hospitals	5.18	2.67	5.78	2.97	4.88	2.46
Doctors	24.36	20.72	22.31	20.16	25.39	20.98
Hospital beds	443.55	240.43	446.49	263.36	442.07	228.82

Notes: Relatives values per 100,000 population.

Table C.2

Men

Variable	Mean	Std. Dev.	Min	Max
Years of schooling	6.654	4.225	0	18
Primary	0.599	0.490	0	1
Secondary	0.164	0.370	0	1
Employment	0.817	0.387	0	1
Disability	0.037	0.188	0	1
Mental disability	0.005	0.073	0	1

Women

Variable	Mean	Std. Dev.	Min	Max
Years of schooling	6.281	3.974	0	18
Primary	0.573	0.495	0	1
Secondary	0.136	0.343	0	1
Employment	0.256	0.436	0	1
Disability	0.025	0.157	0	1
Mental disability	0.004	0.067	0	1

Notes: Summary statistics of pooled data from the 1972, 1982 and 1992 censuses. Only includes people born betwenn 1933 to 1943.

Source: IPUMS international.

Table C.3

Women					
Years of schooling					
	(1)	(2)	(3)	(4)	(5)
Base Pneumonia	-0.00034	-0.0014	-0.00064	-0.0018*	-0.0016
	(0.00092)	(0.0013)	(0.00076)	(0.00094)	(0.0024)
Number of observations	136601	136601	136601	136601	136601
Fixed Effects	Yes	Yes	Yes	Yes	Yes
Control diseases	No	Yes	Yes	Yes	Yes
Maternal mortality	No	No	Yes	Yes	Yes
Health variable	No	No	No	Yes	Yes
Trend by province	No	No	No	No	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table C.4

Women					
Completed primary					
	(1)	(2)	(3)	(4)	(5)
Base Pneumonia	-0.000012	-0.00027**	-0.00021**	-0.00034***	-0.00022
	(0.00010)	(0.000094)	(0.000093)	(0.000094)	(0.00023)
Number of observations	136903	136903	136903	136903	136903
Fixed Effects	Yes	Yes	Yes	Yes	Yes
Control diseases	No	Yes	Yes	Yes	Yes
Maternal mortality	No	No	Yes	Yes	Yes
Health variable	No	No	No	Yes	Yes
Trend by province	No	No	No	No	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table C.5

Women					
Completed secondary					
	(1)	(2)	(3)	(4)	(5)
Base Pneumonia	(1)	(2)	(3)	(4)	(5)
	-0.000069	0.000025	0.000066	0.000090	0.00025
Number of observations	(0.000075)	(0.000075)	(0.000061)	(0.000093)	(0.00021)
Fixed Effects	136903	136903	136903	136903	136903
Control diseases	No	Yes	Yes	Yes	Yes
Maternal mortality	No	No	Yes	Yes	Yes
Health variable	No	No	No	Yes	Yes
Trend by province	No	No	No	No	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table C.6

Women					
Employment					
	(1)	(2)	(3)	(4)	(5)
Base Pneumonia	0.000022	-0.000029	-0.000055	-0.00012	-0.00044***
	(0.000055)	(0.000061)	(0.000065)	(0.000089)	(0.00011)
Number of observations	136218	136218	136218	136218	136218
Fixed Effects	Yes	Yes	Yes	Yes	Yes
Control diseases	No	Yes	Yes	Yes	Yes
Maternal mortality	No	No	Yes	Yes	Yes
Health variable	No	No	No	Yes	Yes
Trend by province	No	No	No	No	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights.*$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Table C.7

Women					
Disability					
	(1)	(2)	(3)	(4)	(5)
Base Pneumonia	0.0000064	-0.000023	-0.000027	-0.000087	-0.00012
	(0.000042)	(0.000055)	(0.000065)	(0.000079)	(0.00014)
Number of observations	43610	43610	43610	43610	43610
Fixed Effects	Yes	Yes	Yes	Yes	Yes
Control diseases	No	Yes	Yes	Yes	Yes
Maternal mortality	No	No	Yes	Yes	Yes
Health variable	No	No	No	Yes	Yes
Trend by province	No	No	No	No	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights.*$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Table C.8

Women					
Mental disability					
	(1)	(2)	(3)	(4)	(5)
Base Pneumonia	-0.000024	-0.000023	-0.000031	-0.000047	-0.000022
	(0.000024)	(0.000026)	(0.000026)	(0.000036)	(0.000078)
Number of observations	43610	43610	43610	43610	43610
Fixed Effects	Yes	Yes	Yes	Yes	Yes
Control diseases	No	Yes	Yes	Yes	Yes
Maternal mortality	No	No	Yes	Yes	Yes
Health variable	No	No	No	Yes	Yes
Trend by province	No	No	No	No	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table C.9: Change of years

Panel A				
	Post=1935	Post=1936	Post=1937	Post=1938
Years of schooling	0.000098	-0.00044	0.00064	0.0066***
	(0.00078)	(0.0014)	(0.0012)	(0.0016)
Primary	-0.000023	-0.000013	0.000096	0.00091***
	(0.00010)	(0.0014)	(0.00012)	(0.00012)
Secondary	-0.000065	-0.00014	-0.000016	0.00041**
	(0.000081)	(0.00016)	(0.00013)	(0.00017)
Employment	0.00019	0.00018*	0.00029**	0.00036***
	(0.00015)	(0.000098)	(0.00011)	(0.00012)
Disability	0.0000053	-0.0000035	-0.0000068	0.00016
	(0.00016)	(0.000089)	(0.000056)	(0.00012)
	Post=1938	Post=1939	Post=1940	Post=1941
Years of schooling	0.0066***	-0.0023*	0.00081	0.00017
	(0.0016)	(0.0012)	(0.0012)	(0.0011)
Primary	0.00091***	-0.0000038	0.00013	0.000019
	(0.00012)	(0.00015)	(0.00011)	(0.00012)
Secondary	0.00041**	-0.00010	0.000094	0.000040
	(0.00017)	(0.00013)	(0.00013)	(0.000099)
Employment	0.00036***	-0.000055	0.000096	-0.00013
	(0.00012)	(0.000075)	(0.00013)	(0.000095)
Disability	0.00016	0.0001	-0.00017	-0.00009
	(0.00012)	-0.00013	(0.00015)	-0.000078

Notes: Each cell reports coefficient for $post * basepneumonia$ from a separate regression. Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights.*$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Table C.10

	Men	
	1933-1943	1928-1948
Years of schooling	0.0066***	0.0021***
	(0.0016)	(0.00061)
	[126027]	[255161]
Primary	0.00091***	0.00030***
	(0.00012)	(0.000098)
	[126314]	[255724]
Secondary	0.00041**	0.00020***
	(0.00017)	(0.000053)
	[126314]	[255724]
Employment	0.00036***	0.00034**
	(0.00012)	(0.00014)
	[125313]	[253659]
Previous control	Yes	Yes

Notes: Each cell reports coefficient on $post * basepneumonia$ from a separate regression. First column reports the results for people born between 1933 and 1943, while the second column show the results for people born between 1928-1948. Robust standard errors, clustered by province of birth, are shown in parentheses. The number of observations is shown in square parentheses. Regressions use IPUMS sampling weights.

$^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table C.11

Years of schooling			
	1970	1982	1992
Base Pneumonia	0.0015	0.0062***	0.012**
	(0.0023)	(0.0021)	(0.0044)
Number of observations	42344	43955	39728
Fixed Effects	Yes	Yes	Yes
Control diseases	Yes	Yes	Yes
Maternal mortality	Yes	Yes	Yes
Health variable	Yes	Yes	Yes
Trend by province	Yes	Yes	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table C.12

Completed primary			
	1970	1982	1992
Base Pneumonia	0.00095***	0.00079**	0.00099***
	(0.00015)	(0.00032)	(0.00030)
Number of observations	42631	43955	39728
Fixed Effects	Yes	Yes	Yes
Control diseases	Yes	Yes	Yes
Maternal mortality	Yes	Yes	Yes
Health variable	Yes	Yes	Yes
Trend by province	Yes	Yes	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table C.13

Completed secondary			
	1970	1982	1992
Base Pneumonia	0.000012	0.00049*	0.00078**
	(0.00018)	(0.00027)	(0.00032)
Number of observations	42631	43955	39728
Fixed Effects	Yes	Yes	Yes
Control diseases	Yes	Yes	Yes
Maternal mortality	Yes	Yes	Yes
Health variable	Yes	Yes	Yes
Trend by province	Yes	Yes	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. Regressions use IPUMS sampling weights. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table C.14

Employment			
	1970	1982	1992
Base Pneumonia	0.00058***	0.00012	0.00044
	(0.000098)	(0.00039)	(0.00028)
Number of observations	42467	43118	39728
Fixed Effects	Yes	Yes	Yes
Control diseases	Yes	Yes	Yes
Maternal mortality	Yes	Yes	Yes
Health variable	Yes	Yes	Yes
Trend by province	Yes	Yes	Yes

Notes: Robust standard errors, clustered by province of birth are shown in parentheses. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$