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Essays on Economic History and Health

By

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DISSERTATION

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Abstract

My dissertation focuses on health outcomes for Chile during the 20th century.

My first chapter examines 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 year of birth. I focus on Chile during the first half of the twentieth century.

The chapter starts by studying the contribution of sulfa drugs to the decline of mortality in Chile. 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 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 chapter, 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, there are also long-term benefits associated with a healthier overall population because of this low endemicity.

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 increased 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.

For employment, men 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.

My second chapter focuses on the impacts on the decline in mortality on fertility, labor markets, and marriage outcomes, using as a natural experiment the introduction of sulfa drugs in Chile in 1938.

Literature studying the effects of mortality declines on fertility is mixed (Soares (2005) argues that mortality decline will generate fertility decline, however Galor (2011) show more inconclusive results).

In theory, declines in infant and child mortality can have an ambiguous effect on fertility. For example, reductions in infant mortality may have a negative impact on fertility if parents have a preference for the target number of live births. If fewer children die, the probability of survival increases, and less precautionary childbearing is needed to get to the target number of children. But at the same time, a decline in infant mortality can positively affect fertility

by reducing the price of a child's quality.

The effects of a decline in maternal mortality will also have ambiguous results in theory. For example, if maternal mortality declines, women's risk of dying will be lower, and this may increase fertility because the cost of having children is lower. This can also be interpreted as a decline in the price of child quantity. However, fertility can decline given that a lower risk of dying also increases expected life expectancy, so the benefits of getting educated increased. This is because the incentives of getting educated increase as the investment return also increases.

Using the 1960 and 1970 Censuses of Chile, I identify women of reproductive age during the period around the introduction of sulfa drugs and examine those cohorts of women when they have completed their fertility. I estimate models for the total number of children, distinguishing between the extensive and intensive margins of fertility. I also use a similar estimation strategy to analyze the impacts of mortality decline on labor and marriage market outcomes.

I show that child mortality decline, measured as pneumonia decline because of sulfa drugs availability, can decrease fertility by stimulating labor force participation. At the same time, a drop in maternal mortality also decreases fertility and increases the likelihood of remain childless. These results imply that the opportunity cost aspect because of longer life expectancy and higher returns of education and employment is more important than reducing the risk of dying during childbearing.

In particular, for intensive margin, an interquartile decline in pneumonia mortality (a movement from the 75th percentile to the 25th percentile), evaluated at the average number of reproductive years of exposure to sulfa drugs, led to 1.01 fewer total births for the average woman. The decline in maternal mortality led to 0.98 fewer births. While, for the extensive margin, a reduction in pneumonia mortality led to a 0.11 percentage point increase in the probability of being childless, while the decrease in maternal mortality a 0.26 percentage point increase.

Also, I show that declines in mortality from pneumonia increases labor force participation and employment status.

In terms of the marriage market, a decline in pneumonia and maternal mortality reduces the likelihood of a woman ever having married, consistent with the higher probability of being childless.

Finally, my last chapter studies the determinants of infant and maternal mortality for the period 1930 to 1960. Infant mortality rate (IMR), defined as infant deaths under one year old over 1,000 live births, is one of the most critical health outcomes. Moreover, it is not only important as a health outcome but as a social and economic development indicator.

Every country saw declines in infant mortality during the 20th century; however, the magnitude of the reductions was quite different. While there are still countries, like India, with infant mortality rates over 40 or countries in Africa over 50, there are some that have more successful histories. Chile is a clear example of this, the infant mortality rate in 1940 was one of the highest in the world, with over 200 babies dying before their first year of life. However, since then, IMR has declined significantly, Chile's IMR nowadays is similar to those of the developed countries (and lower than any other Latin American country).

Using panel data and an instrumental variable approach, with yearly hand-collected data from the Demographic Yearbooks of the National Institute of Statistics for infant mortality, maternal mortality, births, illegitimate births, number of hospitals, number of deliveries occurring in a hospital, number of deliveries with the assistance of a midwife/physician, for the period 1933-1960. I estimate a fixed-effect model of the impact of access to health care on infant mortality rate.

Results show that being born in a hospital reduces neonatal mortality. However, it doesn't have the same strong effects reducing total infant mortality, and it shows no effect on maternal mortality. The same holds for more presence of doctors or midwives. Also, results are stronger for urban than rural areas.

Contents

Chapter 1

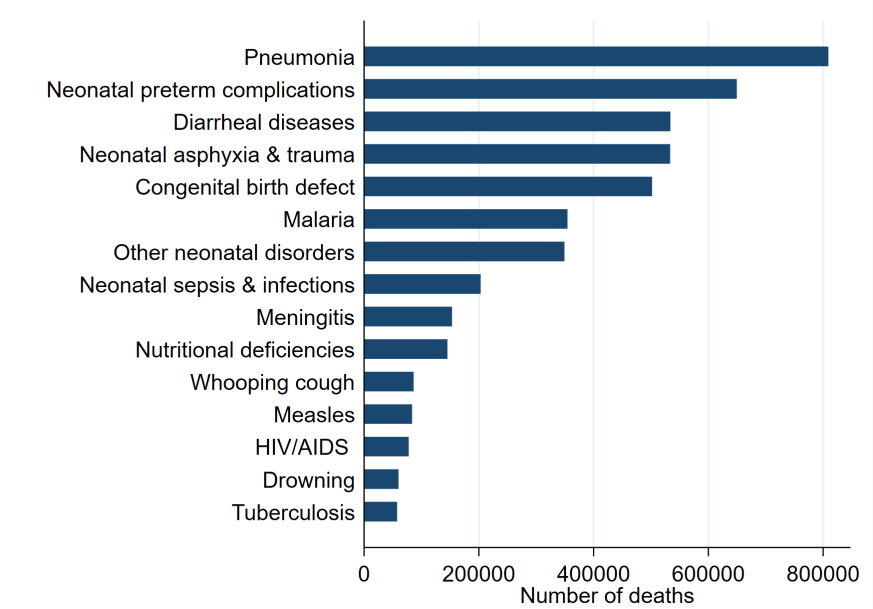
Long-term effects of early life exposure to pneumonia

1.1 Introduction

Pneumonia is the most significant cause of death for children under five globally (figure 1.1), 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].

[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.

Figure 1.1: Cause of death in children under 5, 2017



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

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 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. (2009), 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.

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

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.

1.2 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 A.1.1), 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 used after that.

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

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.

1.3 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 A.1.2). 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 1.1 reports summary statistics of national mortality rates for 1930 and 1950. It

[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.

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.

Table 1.1: 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

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1952 Chilean Census, and author's calculations.

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.

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

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

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.

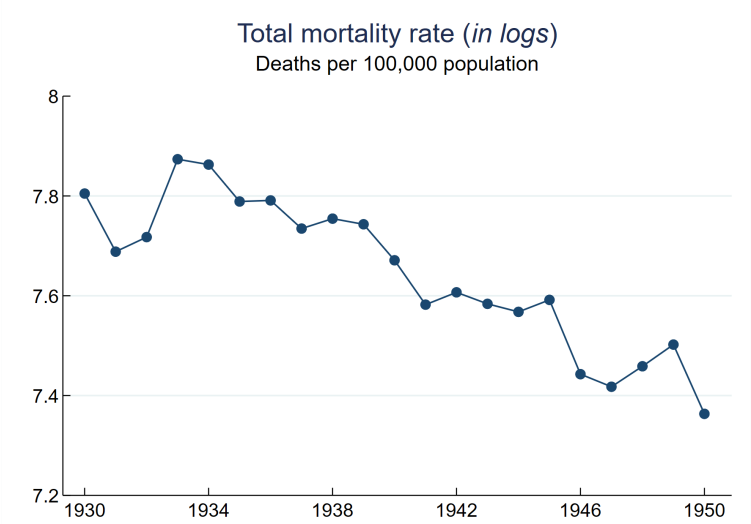
I also hand-digitized data on the number of hospitals, the number of doctors, and province-specific health expenditures from the Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile. Statistics from these variables are in the table A.1.1.

1.4 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 1.2 plots total mortality between 1930 and 1950 and figure 1.3 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].

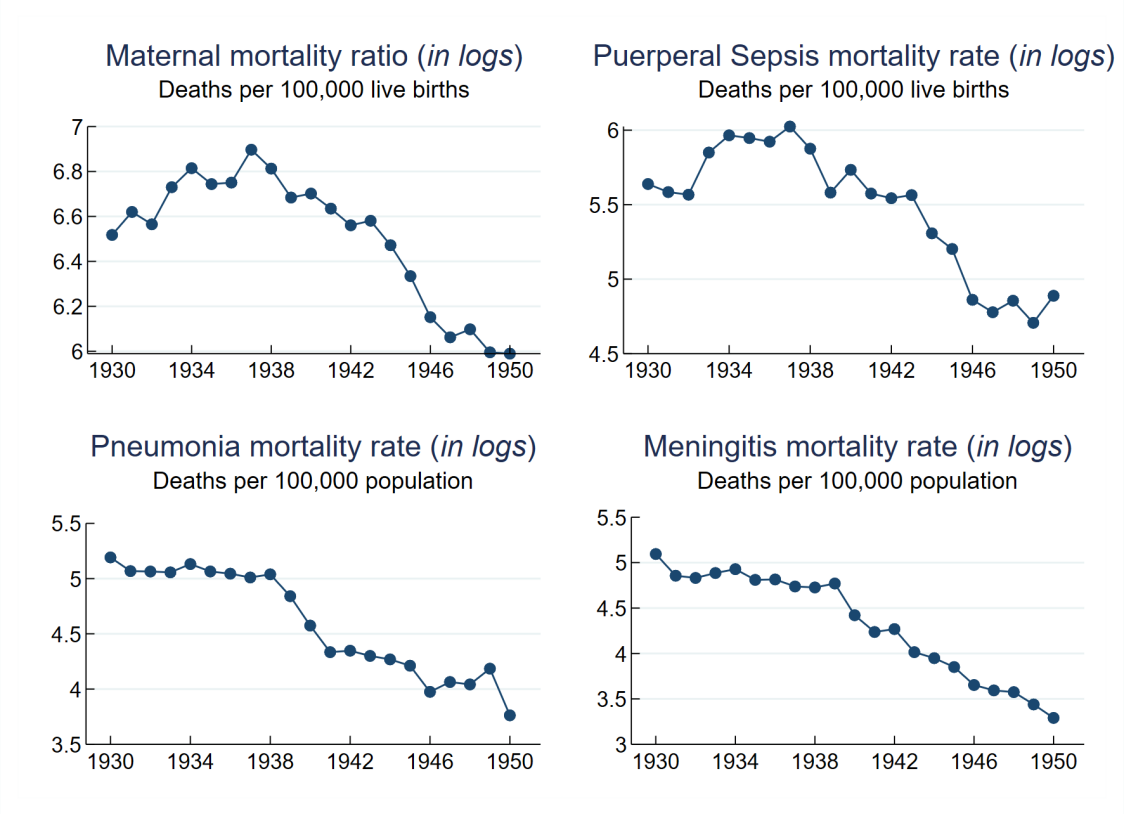
Figure 1.2: Total Mortality Rate



Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1950 Chilean Census, and author's calculations.
Note: Mortality rates calculated per 100,000 population.

[8]Scarlet fever was also treated by sulfa drugs, however, figure A.1.3 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 A.1.4 shows the trajectory of both series, as it can be seen, they move similarly, so for simplicity I will only focus on maternal mortality.

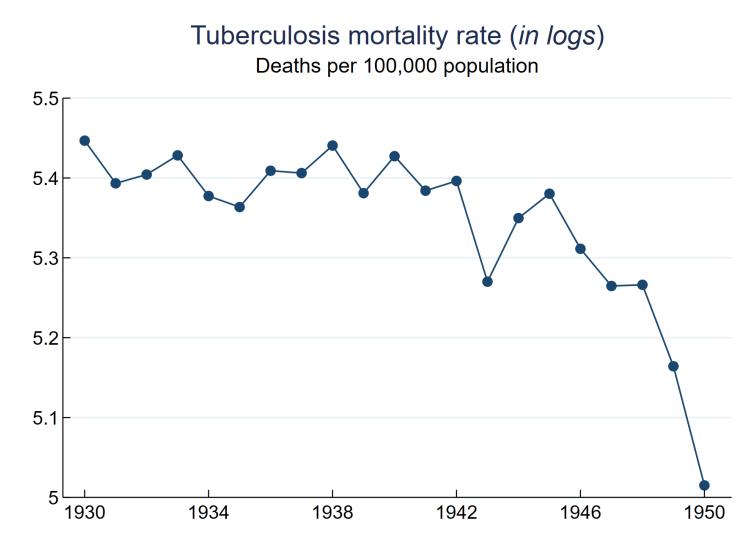
Figure 1.3: Mortality Rate: Treated diseases



Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1950 Chilean Census, and author's calculations.
Note: Pneumonia and meningitis mortality rates calculated per 100,000 population, Maternal and puerperal Mortality rate calculated per 100,000 live births.

Figure 1.4 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).

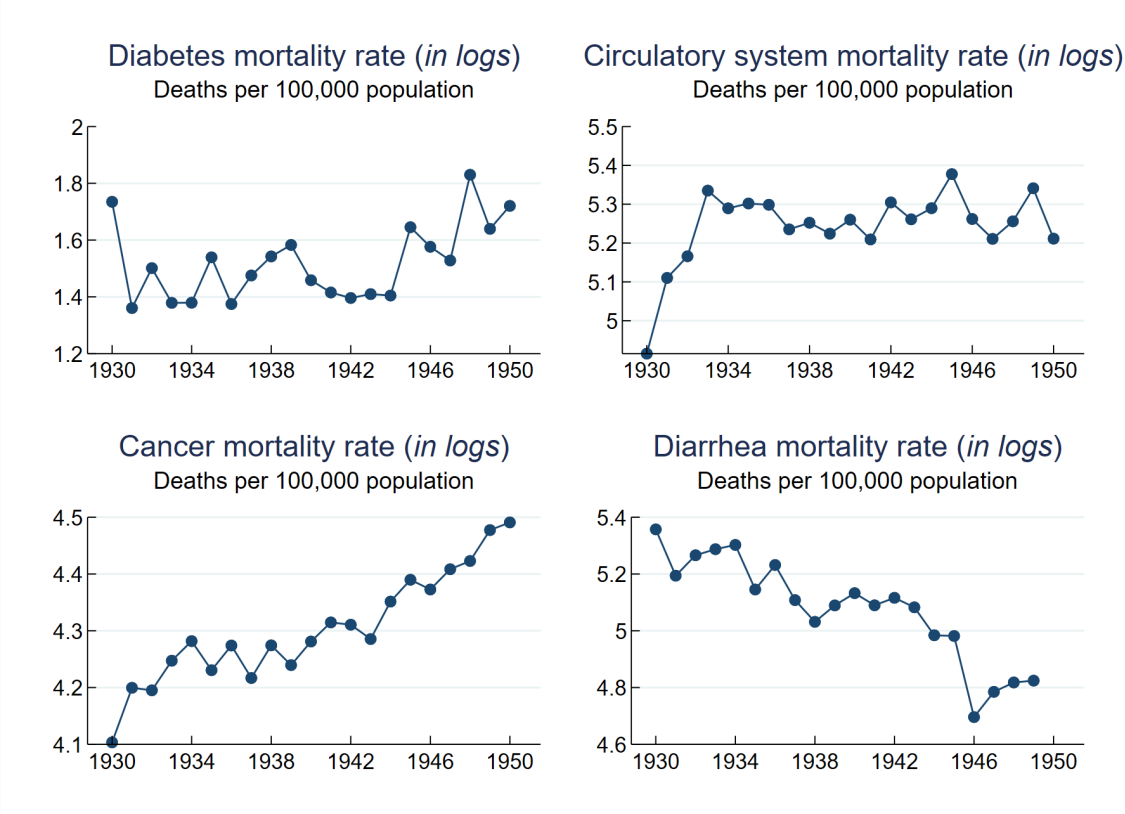
Figure 1.4: Mortality Rate: Tuberculosis



Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1950 Chilean Census, and author's calculations.
Note: Mortality rates calculated per 100,000 population, Maternal and puerperal Mortality rate calculated per 100,000 live births.

The same pattern can be seen from figure 1.5 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.

Figure 1.5



Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1950 Chilean Census, and author's calculations.
Note: Mortality rates calculated per 100,000 population, Maternal and puerperal Mortality rate calculated per 100,000 live births.

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

$$log(M)_t = \beta_0 + \beta_1 Year_t + \delta_0 Post\text{-}1938 + e_t \tag{1.1}$$

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:

$$log(M)_t = \beta_0 + \beta_1 Year_t + \delta_0 \, Post\text{-}1938 + \delta_1 \, Post\text{-}1938 \times Year_t + e_t \tag{1.2}$$

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

Table 1.2 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.

Table 1.2: Effect of sulfa drug on mortality

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

The second model results are in table 1.3. 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.

Table 1.3: Effect of sulfa drug on mortality

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. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$.

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:

$$\begin{aligned} log(M)_{dt} = \beta_0 + \beta_1 Treated_d \times Post1938_t \\ + \beta_2 Treated_d \times Year_t + \beta_3 Treated_d + \beta_4 Year_t + \beta_5 Post1938_t + \ e_{dt} \end{aligned} \tag{1.3}$$

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 A.1.5 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:

$$\begin{aligned} log(M)_{dt} = \beta_0 + \beta_1 Treated_d \times Year_t \times Post1938_t + \beta_2 Treated_d \times Post1938_t \\ + \beta_3 Treated_d \times Year_t + \beta_4 Treated_d + \beta_5 Year_t + \beta_6 Post1938_t + \ e_{dt} \end{aligned} \tag{1.4}$$

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 1.4. 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.

Table 1.4: 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. $^*p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Province level

I repeat the same exercise using province-level data, which allows us to control for

province-specific trends. I estimate the following:

$$\begin{aligned} log(M)_{idt} = \beta_0 + \beta_1 Treated_d \times Post1938_t \\ + \beta_2 Treated_d \times Year_t + \beta_3 Treated_d + \beta_4 Year_t + \gamma_{it} + \ e_{idt} \end{aligned} \tag{1.5}$$

and

$$\begin{aligned} log(M)_{idt} = \beta_0 + \beta_1 Treated_d \times Year_t \times Post1938_t \\ + \beta_2 Treated_d \times Post1938_t + \beta_3 Treated_d \times Year_t \\ + \beta_4 Treated_t + \beta_5 Year_t + \gamma_{it} + \mu_{it} \times Year_t + e_{idt} \end{aligned} \tag{1.6}$$

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 1.5. 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.

Table 1.5: 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 in parenthesis. clustered by disease-year, are shown in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

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 (table 1.5).

1.5 Long-term impacts of pneumonia exposure

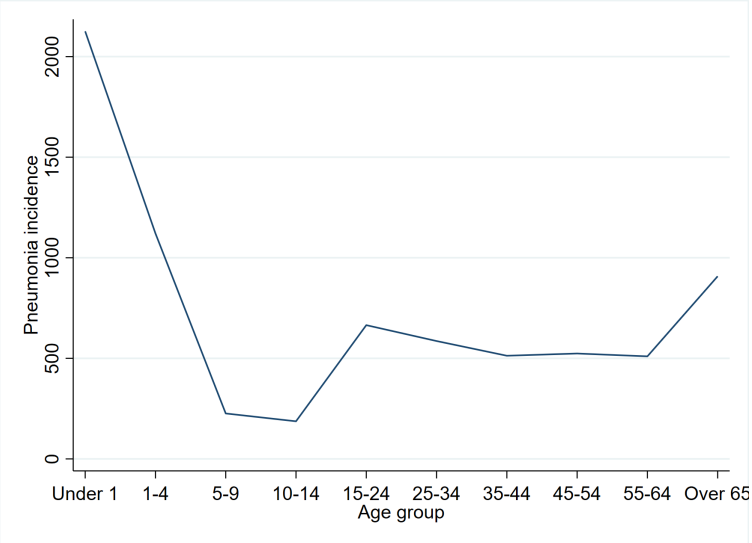
The second part of this paper, estimates the impacts of being born in an environment with less pneumonia, using individual-level outcome data from the census.

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 1.6 exhibits the incidence of pneumonia mortality by age group in 1935; the deaths are mostly concentrated in children under one-year old.

Figure 1.6: 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).

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]

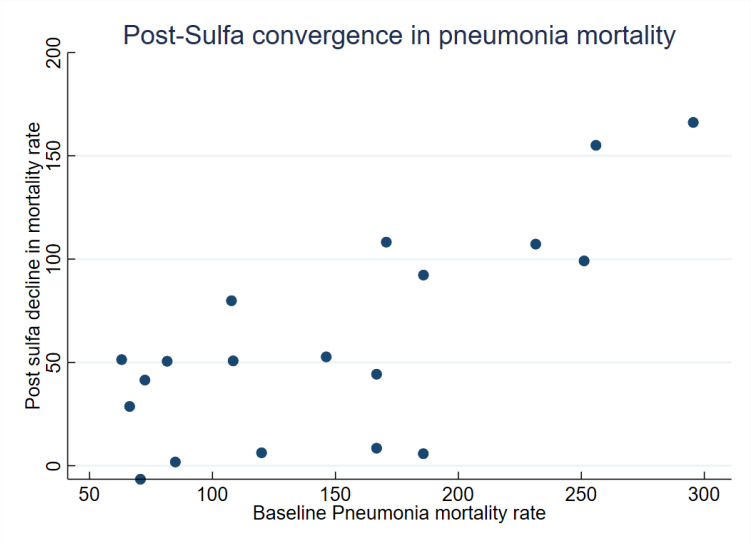
Because identification is challenging because of selectivity in infection, given that people who contracted pneumonia may be a selected sample of the population, my identification strategy uses the introduction of sulfa drugs in 1938, to determine the impact of sulfa drugs and pneumonia exposure on long-term 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

[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).

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 1.7, 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.

Figure 1.7: 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.
Source: National Institute of Statistics (Chile), and author calculations.

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

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

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:

$$Y_{ibtc} = \alpha + \beta Post_t \times basepneumonia_b + \theta_b + \eta_t + \delta_c + e_{btc} \tag{1.7}$$

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

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

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 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

[14]I don't have provincial-level data for economic activity or unemployment, but figure A.1.6 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.

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 A.1.2.

Table 1.6, 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.

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

Table 1.6

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 1.7, 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 seven percentage points in the probability of completing primary and a three percentage points in completing secondary school.

Table 1.7

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.

In table 1.6, 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 variables 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 1.8

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 1.8 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 A.1.3-A.1.8). 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 A.1.7 shows the incidence of pneumonia by 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.

Effects for cohort

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:

$$Y_{ibtc} = \alpha + \beta_t basepneumonia_b + \theta_b + \eta_t + \delta_c + e_{btc} \tag{1.8}$$

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 1.8, 1.9, 1.10, and 1.11, only for the sample of men.

Figure 1.8: Cohort-specific coefficient, primary schooling

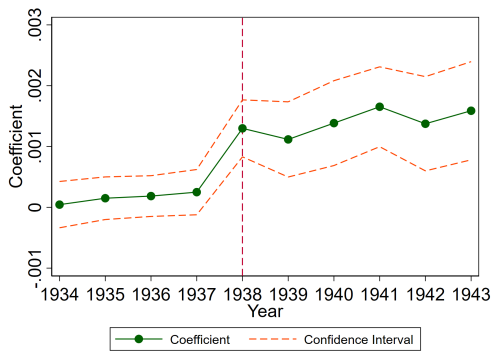


Figure 1.9: Cohort-specific coefficient, secondary schooling

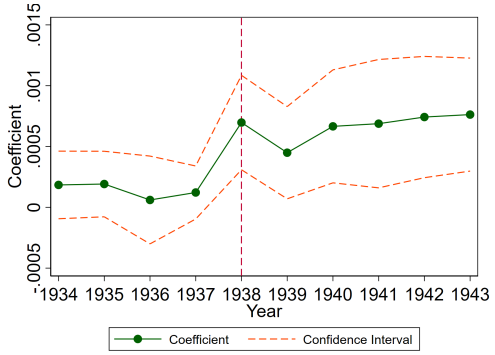


Figure 1.10: Cohort-specific coefficient, years of schooling

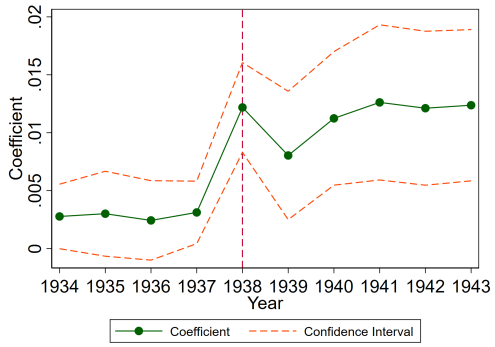


Figure 1.11: Cohort-specific coefficient, employment

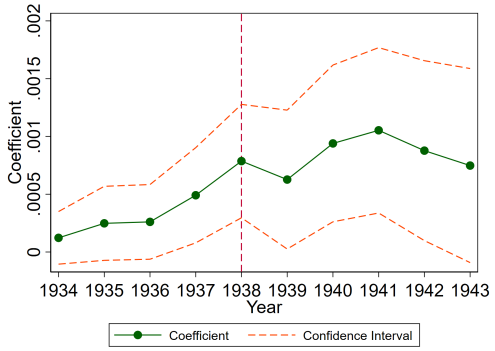


Figure 1.7 plots the cohort-specific coefficient estimates with years of education as a dependent variable. This chart shows that after 1938, the year sulfa drugs were introduced, there is a trend break; the coefficient gets larger, providing visual evidence of the positive long-run impact of sulfa drugs. The same effect is observed for the other variables[16].

[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.

1.6 Robustness

A potential threat to the identification strategy is that *BasePneumonia* correlates with other variables that affect long-term outcomes. In table 1.9, 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 1.10 exhibits the results. When I include these variables, the results remain significant, although some of the coefficients get slightly smaller.

Table 1.9: 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 1.10: 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.

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 A.1.9 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.

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 1.11. The first column is the previously preferred specification, with all the previous controls except 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[17].

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

Table 1.11

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

Fourth, I extend the period under study to include cohorts born between 1929 and 1948 (instead of 1933–1943). Table A.1.10 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 1.12 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 A.1.9 and A.1.10).

Table 1.12: 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 $post*basepneumonia$ 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.

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

I also repeat the results for men separately for each census. Tables A.1.11, A.1.12, A.1.13 and A.1,14, 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.

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.

1.7 Appendix

1.8 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.

1.9 Appendix: Figures

Figure A.1.1: First Sulfonamide drug, Prontosil

Figure A.1.2: 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 A.1.3

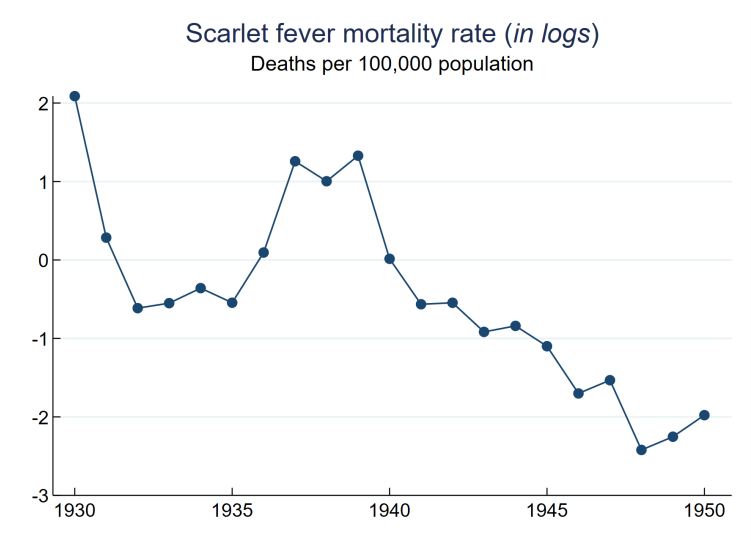
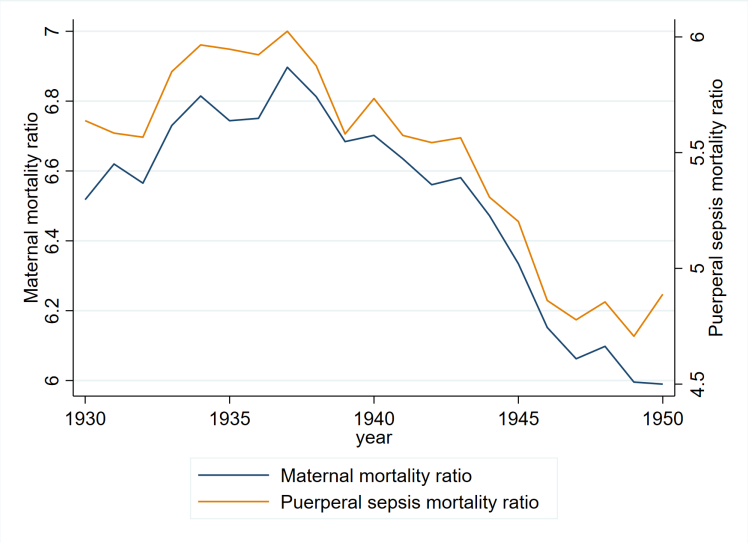
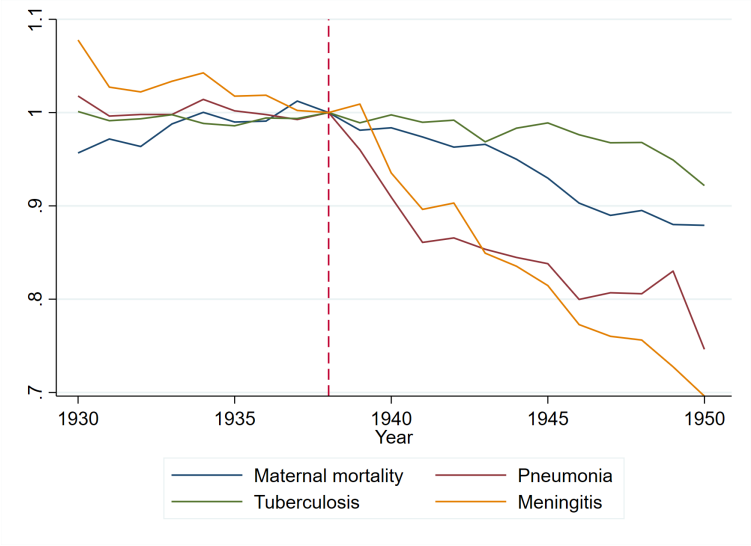

Scarlet fever mortality rate (*in logs*)
Deaths per 100,000 population
2
1
0
-1
-2
-3
1930
1935
1940
1945
1950

Figure A.1.4



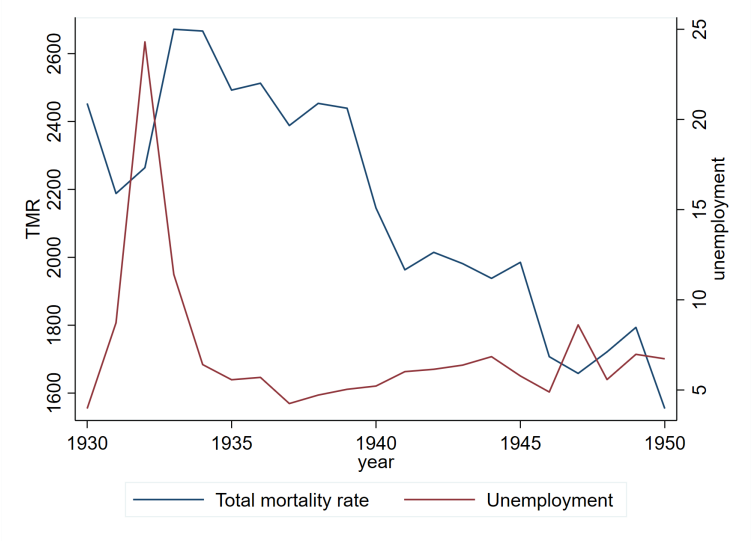
Note: Mortality rates calculated as number of deaths per 100,000 live births. Series in natural logarithm. Maternal mortality on the left-axis, and puerperal sepsis mortality on the right axis.

Figure A.1.5



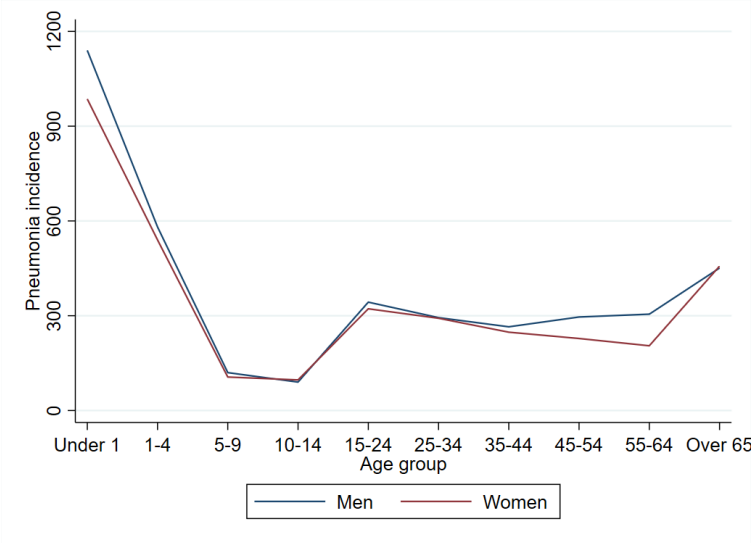
Note: Mortality rates per 100,000 population for all the variables except maternal mortality. For maternal mortality rate is calculated as deaths per 100,000 live briths. Mortality rates normalized to 1 in 1938.

Figure A.1.6



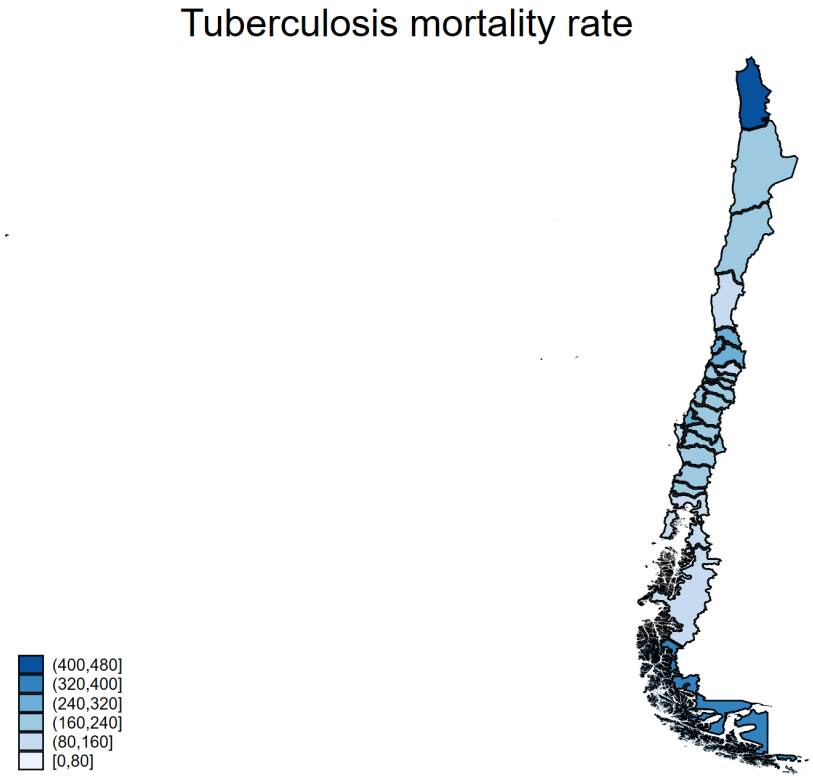
Note: Total mortality rates per 100,000 population in the left axis, unemployment rate in the right axis.
Source: Mortality : Demographic yearbook; unemployment: Díaz, J.; Lüders. R. y Wagner, G. (2016).

Figure A.1.7: Age distribution of pneumonia incidence by gender, 1935



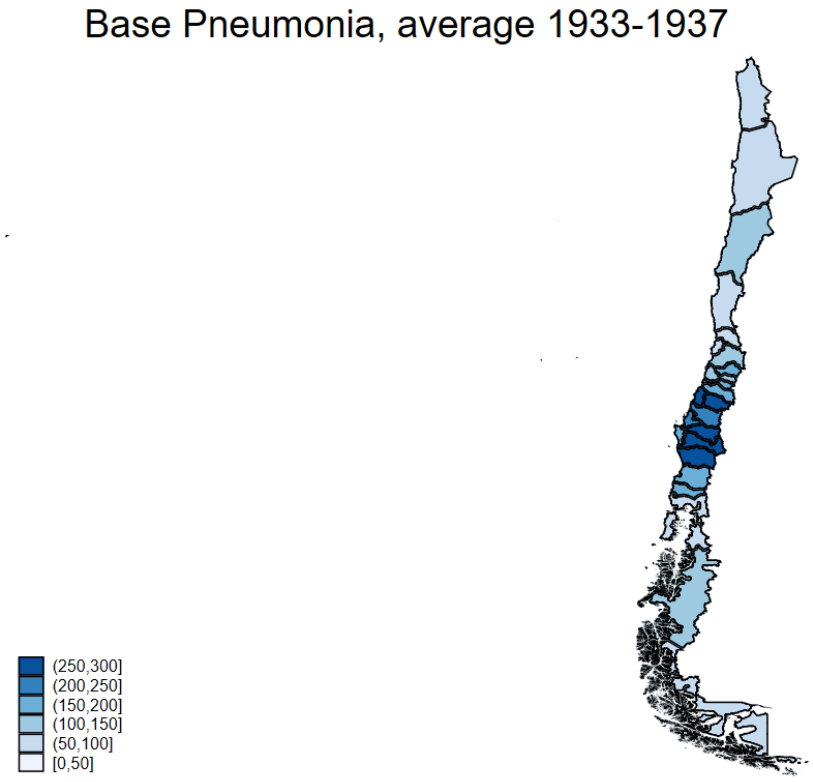
Notes: Pneumonia incidence is the number of deaths due to pneumonia for each age group.
Source: National Institute of Statistics (Chile).

Figure A.1.8



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

Figure A.1.9



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

1.10 Appendix: Tables

Table A.1.1: 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 A.1.2

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 A.1.3

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 A.1.4

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 A.1.5

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 A.1.6

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 A.1.7

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 A.1.8

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 A.1.9: 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 A.1.10

	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 A.1.11

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 A.1.12

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 A.1.13

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 A.1.14

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.

*$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Chapter 2

Fertility responses to decline in infant and maternal mortality: Evidence for Chile in the 20th Century

2.1 Introduction

The theory of demographic transition states that declines in mortality will cause reductions in fertility. And this decline in fertility has been attributed to the rise in education (Galor and Weil (2000, Galor (2005, 2011), Murtin (2013)). However, these theories omit the role played by increasing health standards.

This paper studies the impacts of the decline in mortality on fertility, labor markets, and marriage outcomes, using as a natural experiment the introduction of sulfa drugs in Chile in 1938.

Sulfa drugs were introduced in 1938, and after that, they were available nationwide. Because of their already available high supply and low cost, the diffusion was fast. The introduction of sulfa drugs causes a sharp decline in mortality levels in almost all the infectious diseases that were effectively treated by the drug, like puerperal sepsis, pneumonia,

and meningitis.

Literature studying the effects of mortality declines on fertility is mixed (Soares (2005) argues that mortality decline will generate fertility decline, however Galor (2011) show more inconclusive results).

In theory, declines in infant and child mortality can have an ambiguous effect on fertility. For example, reductions in infant mortality may have a negative impact on fertility (Kalemlu-Ozan, Ryder, and Weil (2000)) if parents have a preference for the target number of live births. If fewer children die, the probability of survival increases, and less precautionary childbearing is needed to get to the target number of children. But at the same time, a decline in infant mortality can positively affect fertility by reducing the price of a child's quality (for example, because the lower mortality also implies a better disease environment for the child).

Most papers have focused on the effect of mortality on total fertility. However, it is crucial to determine if there are distinct effects on the extensive (probability of having a child) and the intensive margin (number of children conditional on having at least one). This is because the quantity-quality model of Becker and Lewis (1973) does not give a different prediction for the fertility transition along the intensive and extensive margin.

The Backer and Lewis (1973) model focus exclusively on the intensive margin. Hence, the quantity and quality of children are substitutes; therefore, reductions in the price of a child's quality will reduce the desired number of children. However, if we focus on the extensive margin, a decrease in the cost of investing in children (for example, due to better health because of sulfa drugs or better access to education) predicts an increase in the probability that a woman will have at least one child. In this case, we see some complementarity between the quality and quantity of children.

Moreover, declines in infant mortality have also been associated with reductions in maternal mortality. The effects of a decline in maternal mortality will also have ambiguous results in theory. For example, if maternal mortality declines, women's risk of dying will

be lower, and this may increase fertility because the cost of having children is lower. This can also be interpreted as a decline in the price of child quantity. However, a lower risk of dying also increases expected life expectancy, so the benefits of getting educated increased. If a woman is expected to have a longer life expectancy, the incentives of getting educated increase as the investment return also increase (Jayachandran and Lleras Muney (2009)). Suppose, for example, that women became more educated and enter the labor market; in that case, the opportunity cost of having children will increase, then women should be seen their fertility reduced along both intensive and extensive margin.

In this paper, I use the introduction of sulfa drugs to test the effects of a decrease in child and maternal mortality over fertility, labor market, and marriage market decisions.

Sulfa drugs led to a sharp decline in mortality from pneumonia, which was the leading cause of child mortality; hence it was like a reduction in the price of investing in children, increasing child quality by improving child health.

However, this decline was not the same across the country. Areas with a high level of pre-sulfa mortality levels saw more significant decreases. Hence, besides the cohort exposure variation created by the introduction of sulfa drugs, 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 most significant mortality declines after introducing 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 (see Bleakley (2007), Lucas (2010), and Bhalotra and Venkataramani (2015)).

Using the 1960 and 1970 Censuses of Chile, I identify women of reproductive age during the period around the introduction of sulfa drugs, and examine those cohorts of women when they have completed their fertility. I estimate models for the total number of children, distinguishing between the extensive and intensive margins of fertility. I also use a similar estimation strategy to analyze the impacts of mortality decline on labor and marriage market outcomes.

I show that child mortality decline, measured as pneumonia decline because of sulfa drug availability, can decrease fertility by stimulating labor force participation. At the same time, a drop in maternal mortality also decreases fertility and increases the likelihood of remain childless. These results imply that the opportunity cost factor because of longer life expectancy and higher returns of education and employment are more significant than reducing the risk of dying during childbearing.

In particular, for intensive margin, an interquartile decline in pneumonia mortality (a movement from the 75th percentile to the 25th percentile), evaluated at the average number of reproductive years of exposure to sulfa drugs, led to 1.01 fewer total births for the average woman. The decline in maternal mortality led to 0.98 fewer births. While, for the extensive margin, a decline in pneumonia mortality led to a 0.11 percentage point increase in the probability of being childless, while the decrease in maternal mortality a 0.26 percentage point increase.

Also, I show that declines in mortality from pneumonia increase labor force participation and employment status.

In terms of the marriage market, a decline in pneumonia and maternal mortality reduces the likelihood of a woman ever having married, consistent with the higher probability of being childless.

My paper contributes to the literature on the effects of child and maternal mortality decline on fertility (Galor (2011), Albanesi and Olivetti (2014)) but also focus on the distinction between intensive and extensive margin.

Literature distinguishing between both effects is scarce. Aaronson and Mazumder (2014) examine fertility along the extensive and intensive margins in response to a large school-building program. They find that women facing better schooling opportunities for their children were more likely to have at least one child but chose to have smaller families overall.

Bahlotra et al. (2018) investigate women's fertility, labor, and marriage market responses to significant declines in child and maternal mortality. They find that in the US,

women delayed childbearing and had fewer children overall in response to the decrease in child mortality. They also find that reductions in child mortality increased women's labor force participation, improved their occupational status, and reduced their chances of ever having married. However, they find that maternal mortality decline had opposing effects on all of these outcomes.

It also contributes to the literature on the reasons behind childlessness (Baudin, de la Croix, and Gobbi (2015), Currie and Schwandt (2014)).

Finally, this paper is also related to literature arguing that better women's health increases women's labor force participation or education and, thereby lower fertility (Jayachandran and Lleras-Muney (2009), Bloom, Kuhn, and Prettner (2020)).

The rest of the paper is structured as follows. Section 2 provides a brief history of sulfa drugs and describes the mortality environment in Chile. Section 3 describes the data used. Section 4 explains the empirical strategy used. Section 5 shows the results, and finally, section 6 concludes.

2.2 Mortality rates and Sulfa drugs

Mortality rates in Chile during the first half of the twentieth century were high. Infant mortality rates were over 200 for every 1,000 live births, with pneumonia the leading cause of death for children under 1-year-old.

Maternal mortality was also high. The maternal mortality ratio was over 800 per 100,000 live births, with puerperal sepsis representing over 40% of all maternal deaths.

Sulfonamide drugs were the first drug that effectively treated a series of bacterial diseases. Before the arrival of sulfa drugs, 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, discovered that a dye, later called sulfas, was helpful in treated streptococci bacterial infections.

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, an ample 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 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.

The arrival of sulfa drugs, the first widely used antibiotic, drastically reduced mortality rates. Pneumonia mortality decline by over 50%, while maternal mortality around 30% (puerperal sepsis mortality even more). Table 2.1 reports summary statistics of national mortality rates for 1930 and 1950. 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%. It can be also seen that puerperal sepsis represents around 41% of the total maternal deaths during the period before sulfa drugs.

Table 2.1: 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

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1952 Chilean Census, and author's calculations.

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.

2.3 Data

Causes of deaths were obtained 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. The data include information on deaths, broken down by cause of death by province, year, and gender.

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

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.

To determine the impact on the decline in child and maternal mortality, I used individual-level data from the census of 1960 and 1970, available from the Integrated Public Use Microdata Series, International. I pooled these censuses to have a larger sample size and more precise estimates. In particular, I use the variable of "children ever born[1]" to create my fertility variables, "years of schooling" to control for education, also variables related to employment status and marriage market.

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.

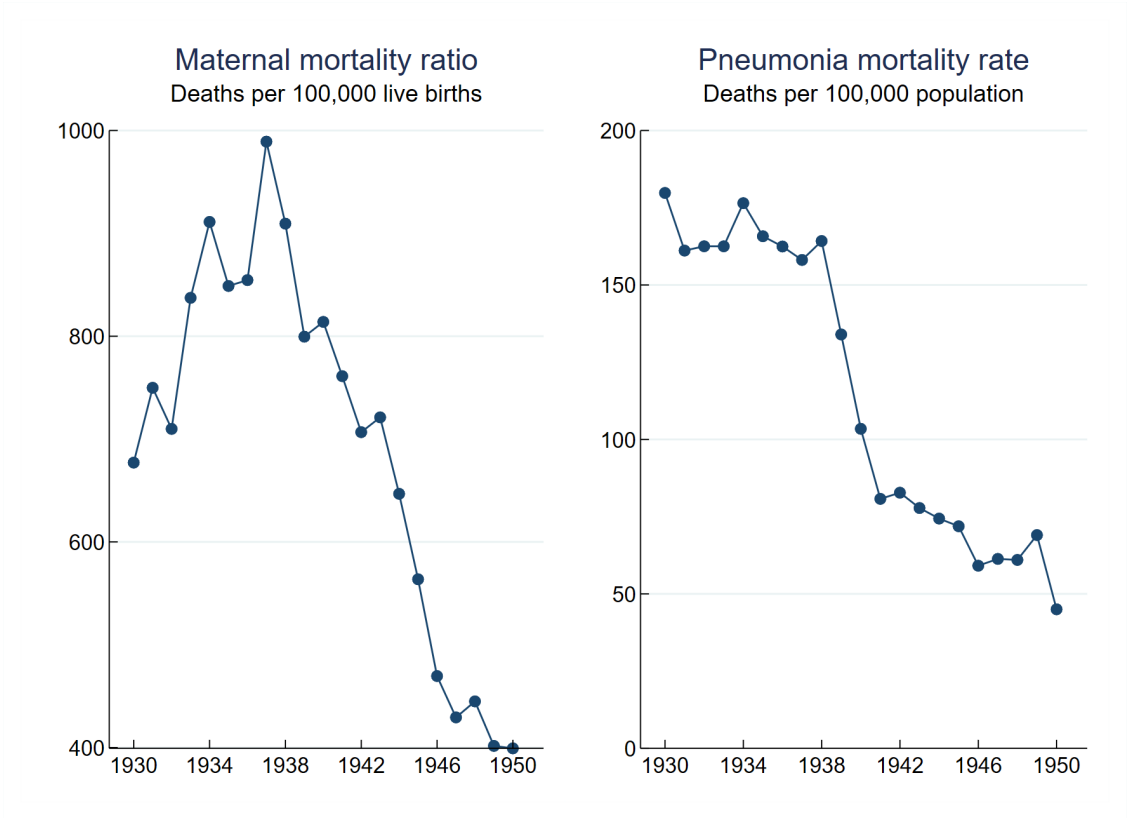
[1]This include the total number of live births; however, some of these children may have died during early life, so this variable may be over-estimating the effective level of fertility, and under-estimate childness.

2.4 Research Strategy

Sulfa drugs were introduced in 1938, and after that, they were available nationwide. Because of their already available high supply and low cost, the diffusion was fast. The introduction of sulfa drugs causes a sharp decline in mortality levels in almost all the infectious diseases that were effectively treated by the drug, like puerperal sepsis, pneumonia, and meningitis.

Figure 2.1 shows the trajectory of pneumonia and maternal mortality. It can be seen that post introduction of sulfa drugs, mortality decline significantly. For example, maternal mortality decreases by around 30% and pneumonia mortality more than 50% in the post-sulfa drugs period.

Figure 2.1: Maternal and Pneumonia mortality rates



Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1950 Chilean Census, and author's calculations.
Note: Pneumonia mortality rates calculated per 100,000 population, Maternal Mortality rate calculated per 100,000 live births.

However, this decline was not the same across the country. Areas with a high level of pre-sulfa mortality levels saw more significant decreases. Hence, besides the cohort exposure variation created by the introduction of sulfa drugs, 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 most significant mortality declines after introducing 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 (see Bleakley (2007), Lucas (2010), and Bhalotra and Venkataramani (2015)).

Figure 2.2 and 2.3 show the post-sulfa convergence in pneumonia and maternal mortality rates in the Chilean provinces. The x-axis has the value of *basemortality*, which is the pre-sulfa mortality rate in the birth province before sulfa drugs were introduced (period 1933-1937[2]). The y-axis shows the decline in mortality after sulfa drugs were introduced (hence is *basemortality* minus the value of mortality in 1943), while the x-axis has the average mortality level before the introduction of sulfa drugs. As the graph shows, there was a strong convergence in mortality levels for pneumonia and maternal mortality. Provinces with higher pneumonia and maternal mortality levels pre-sulfa drugs experience the largest declines.

[2]I started in 1930 to avoid potential confounding factors related to higher mortality levels because of the Great Depression, and stopped in 1943, because of the introduction of penicilin.

Figure 2.2: Convergence in Pneumonia mortality rate

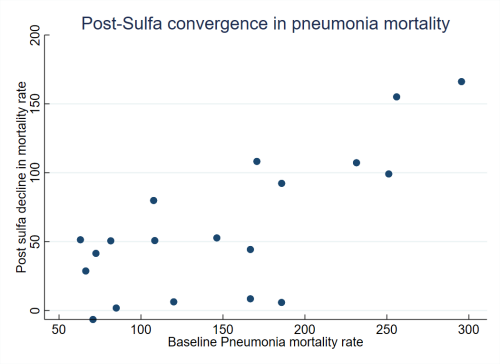
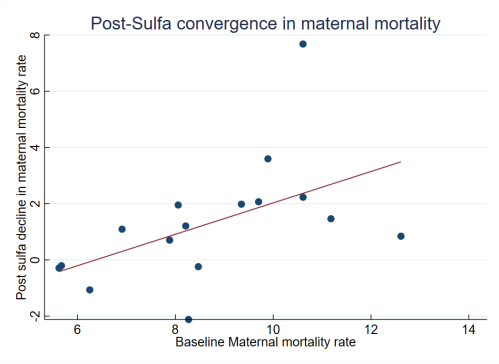
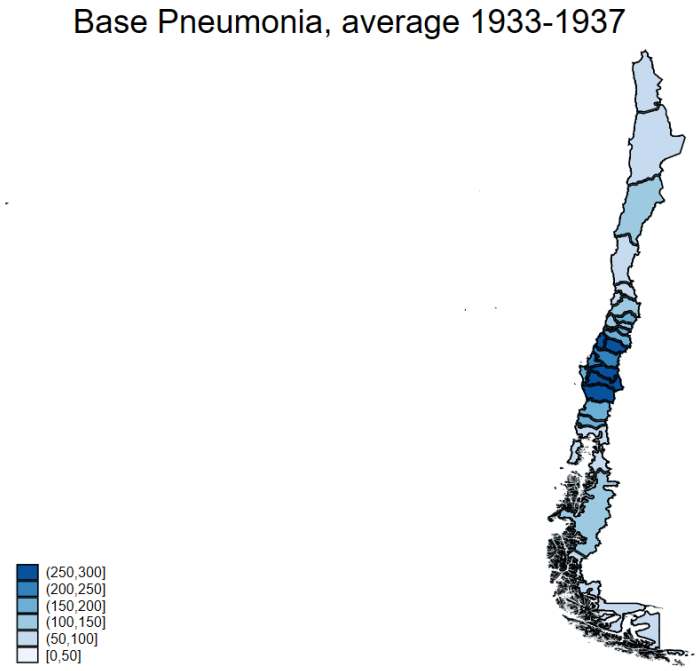


Figure 2.3: Convergence in Maternal mortality rate



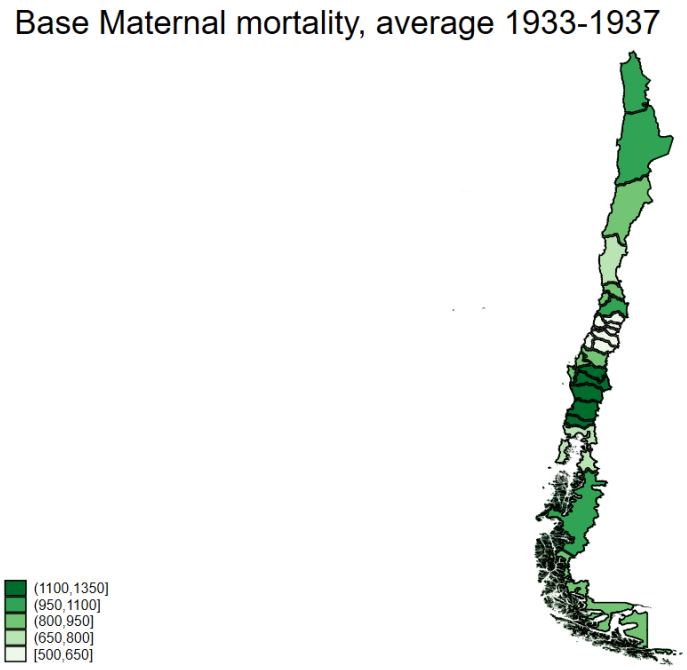
Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1952 Chilean Census, and author's calculations.
Note: Pneumonia mortality rates calculated per 100,000 population, Maternal Mortality rate calculated per 100,000 live births. Post-sulfa decline in mortality refers to the decline in mortality after sulfa drugs were introduced (hence is basemortality minus the value of mortality in 1943), while Baseline mortality is the average mortality level before the introduction of sulfa drugs (period 1933 to 1937), for both diseases.

Figure 2.4: Base Pneumonia Mortality Rate



Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1952 Chilean Census, and author's calculations.
Note: Mortality rates calculated per 100,000 population. Post-sulfa decline in mortality refers to the decline in mortality after sulfa drugs were introduced (hence is basemortality minus the value of mortality in 1943), while Baseline mortality is the average mortality level before the introduction of sulfa drugs (period 1933 to 1937), for both diseases.

Figure 2.5: Base Maternal Mortality rate



Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, 1930, 1940 and 1952 Chilean Census, and author's calculations.
Note: Mortality rates calculated per 100,000 population. Post-sulfa decline in mortality refers to the decline in mortality after sulfa drugs were introduced (hence is basemortality minus the value of mortality in 1943), while Baseline mortality is the average mortality level before the introduction of sulfa drugs (period 1933 to 1937), for both diseases.

The pre-sulfa levels of pneumonia and maternal mortality exhibited different levels across the various provinces (Figure 2.4 and 2.5).

Effects of sulfa drugs on fertility

In this part, I estimate the effect of being exposed to sulfa drugs on fertility in terms of the total number of children (children ever born), extensive margin (if a woman ever becomes a mother), and the intensive margin (impact on the number of children, conditional on having one).

To do this, I use a sample of only women of reproductive age from 1930 to 1943 (so women of reproductive age[3] around the time of the introduction of sulfa drugs) observed in

[3]We consider that women are fertile between the ages of 15 to 40.

the 1960 and 1970 population Census. Women need to be at least 40 years old at the time of census enumeration to be in the sample, to be sure that they have plausibly completed their fertility.

However, to see if some women have delay fertility (and not just stop having childrens), I also focus on an alternative sample that only includes women aged 18 to 40 at census enumeration (so, women still on childbearing age).

I measure exposure to sulfa drugs as the number of fertile years spent in the post-sulfa era, interacting this with the pre-sulfa mortality levels.

I estimate the following equation:

$$\begin{aligned} Y_{jsc} = \alpha + \alpha_1 \times prePneumonia_s \times sulfayears_j + \alpha_2 \times preMMR_s \times sulfayears_j \\ + \alpha_3 \times birthyear_c + \alpha_4 \times education_j + \alpha_5 \times birthprovince_s \\ + \alpha_6 \times sulfayear_j \times provincecontrols_s + e_{jsc} \end{aligned} \tag{2.1}$$

Where Y_{jsc} denotes the total number of births to woman j born in province s in cohort c as recorded at the time of the census and $sulfayears_j$ is the number of fertile years that women j was exposed to sulfa drugs.

To focus on the intensive margin, this equation is estimated restricting the sample to women with at least one birth.

I also include fixed effects for the woman's birth year and birth state, also education (measure as years of schooling), and some province-specific variables (number of doctors, number of hospitals, and health expenditure per capita) interacted with $sulfayears_j$.

I also control for other diseases like tuberculosis, diarrhea, meningitis to ensure that I am not capturing the effects of an overall better disease environment. I also include some non-contagious diseases like cancer and circulatory system disease to control factors such as better health care access and quality. For all of these variables, I use the pre-sulfa province-level mortality rate and interact this with the measure of exposure to sulfa drugs.

Finally, I cluster the standard errors at the province level.

To focus on the extensive margin instead of the intensive, Y_{jsc} becomes a dummy variable equals to one if the woman is childless and zero otherwise.

Effect on labor and marriage market outcomes

To study the effect of sulfa drugs on labor decisions and the marriage market, I estimate the following equation:

$$\begin{aligned} L_{jsc} = \beta + \beta_1 \times prePneumonia_s \times sulfayears_j + \beta_2 \times preMMR_s \times sulfayears_j \\ + \beta_3 \times birthyear_c + \beta_4 \times education_j + \beta_5 \times birthprovince_s \\ + \beta_6 \times sulfayear_j \times provincecontrols_s + e_{jsc} \end{aligned} \tag{2.2}$$

Where L_{jsc} measure labor market outcomes (if the woman is in the labor force, and whether she is working) and marriage market outcomes (whether currently married or ever married). The rest of the variables are the same as in the previous equation.

In this part, I focus on women aged 18-50 at the census, thus covering women of childbearing age and those who have completed fertility.

2.5 Results

Results: Effects of sulfa drugs exposure on fertility

Table 2.2 present the results for the effects of the decline in child and maternal mortality on fertility outcomes. Columns 1-3 show the results for women during childbearing age, and columns 4-6 for women with completed fertility at the moment of enumeration.

For women still at childbearing age, the results show that the reduction in pneumonia mortality had a negative effect on fertility for the intensive and extensive margin, although the results are not significant (Table 2.2, columns 1-3). The intensive margin shows that a decline in pneumonia mortality is associated with a smaller number of children.

In terms of maternal mortality, reductions in maternal mortality led to women having more children; however, it also increases the probability of being childless (Table 2.2, columns 1-3). In this case, my results are significant both at the intensive and extensive margin level.

This can be explained in terms of intensive margin because declines in maternal mortality reduce the risk of a woman dying during childbirth, hence increasing fertility. However, it also will increase the probability of childlessness because women now can expect to have, on average, a longer life expectancy; hence the incentives to get educated and join the labor force will be more significant. Therefore they will delay or avoid having children.

Table 2.2: Effects on Fertility

	Childbearing age women			Completed fertility		
	Number of children	Intensive Margin	Extensive Margin	Number of children	Intensive Margin	Extensive Margin
$PrePneumonia \times Sulfayears$	-0.089	-0.033	0.017	-0.067***	-0.045***	0.0050**
	(0.13)	(0.076)	(0.011)	(0.015)	(0.0058)	(0.0019)
$PreMaternalmortality \times Sulfayears$	0.13	0.34***	0.041**	-0.064***	-0.030***	0.0081***
	(0.18)	(0.11)	(0.015)	(0.021)	(0.0083)	(0.0027)
Observations	798023	651371	798023	1528071	1322660	1528071

Note: Number of children corresponds to the total of children ever born. Intensive margin is the number of children, conditional on having at least 1, and extensive margin correspond to a dummy variable equal to 1 if the woman is childless (have zero births) and zero otherwise.
Regressions only include women of reproductive age from 1930 to 1943 observed in the 1960 and 1970 population Census of Chile.
For childbearing age woman, I consider woman age 18 to 40 years old at the moment of the census enumeration. For completed fertility, I focus on women that are at least 40 years old at the time of enumeration.
All regressions include year of birth fixed effect, census fixed effect, province of birth fixed effect, years of education, number of hospitals in each province, number of doctors, health expenditure per capita, other diseases mortality rates (tuberculosis, uncer 2-diarrhea, meningities, cancer and circulatory system disease).
Standard errors in parenthesis, are clustered by the province of birth.
Regressions use IPUMS sampling weights.
*$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Table 2.2, columns 4-6 exhibit the results for women with completed fertility. A decline in pneumonia and maternal mortality is associated with a significant decline in fertility in both extensive and intensive margins.

For intensive margin, an interquartile decline in pneumonia mortality (a movement from the 75th percentile to the 25th percentile), evaluated at the average number of reproductive years of exposure to sulfa drugs, led to 1.01 fewer total births for the average woman. The decline in maternal mortality led to 0.98 fewer births.

For the extensive margin, a decline in pneumonia mortality led to a 0.11 percentage point increase in the probability of being childless, while the decrease in maternal mortality a 0.26 percentage point increase.

This is consistent with Aaronson et al. (2014) and Bhalotra et al. (2018), who found that fertility transition was characterized in the US by a reduction in fertility on both the intensive and extensive margin.

Comparing the results for women with completed fertility and women of childbearing age, the coefficients for women with completed fertility are always larger than those for women of childbearing age (and always significant). This implies that the estimated impacts of pneumonia and maternal mortality on childlessness are not coming from delayed childbearing but an actual increase in childlessness.

In the childbearing age, I find a significant impact on maternal mortality decline caused an increase in the intensive margin (women having more children, conditional of having at least one), and an increase in the likelihood of being childless. However, for the sample of women with completed fertility, a decline in maternal mortality is associated with a lower number of children and a higher likelihood of childlessness.

A problem with my results can be survivorship bias. I only observe mothers who survive; if a mother died due to childbirth is not observed in the census. It makes sense to think that women who die during childbirth have higher fertility and probably live in a province with higher levels of maternal mortality. Hence, my results will overestimate the increase in

fertility because of the decline in maternal mortality.

The same concern will be about infant mortality. When infant mortality is high, the variable of children ever born will overestimate surviving births. This is important in Chile, where even in 1940, over 200 babies die for every 1,000 live births. This can be eventually compensated using the variable of surviving children that is only available in the 1970 census.

Results: Effects of sulfa drugs exposure on labor market

This section estimates the impact of reducing pneumonia and maternal mortality due to sulfa drug introduction on women's labor market choice.

For this, I use a sample of all women aged 18-50 at the time of the census and 6-44 in 1937 (pooling both women of childbearing age and completed fertility).

Results in table 2.3 show that pneumonia mortality decline led to improvement in both the probability of being employed and the probability of participation in the labor force.

A fall in maternal mortality had opposing effects; it caused a decline in labor force participation and in the probability of being employed. These results are consistent with what Bhalotra et al. (2018)) finds for the US, but contrary to what Albanesi and Olivetti (2014) find (they find that a decline in maternal mortality increase fertility and female labor force participation).

Table 2.3: Effect on Labor Market

	Employed	Labor force participation
$PrePneumonia \times Sulfayears$	0.0065***	0.0067**
	(0.00017)	(0.00019)
$PreMaternalmortality \times Sulfayears$	-0.00063**	-0.00020
	(0.00028)	(0.00029)
Observations	1522479	1523166

Note: Employed is a dummy variable equal to one if the woman reports to be employed at the time of the census and zero otherwise.

Labor force participation is a dummy variable equals to one if the woman is in the labor force and zero otherwise.

Sample includes women age 6-44 in 1937, and 18-50 at the time of the census. All regressions include year of birth fixed effect, census fixed effect, province of birth fixed effect, years of education, number of hospitals in each province, number of doctors, health expenditure per capita, other diseases mortality levels (tuberculosis, under 2 diarrhea, meningities, cancer and circulatory system disease).

Standard errors in parenthesis, are clustered by the province of birth.

Regressions use IPUMS sampling weights.
$^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Results: Effects of sulfa drugs exposure on marriage market

Table 2.4 presents the results for marriage outcomes. In this section, I also use a sample of women age 18 to 50 years old at the moment of the census enumeration.

A reduction in pneumonia mortality reduces the probability of a woman ever having married. Maternal mortality decline has the same effects, a decrease in maternal mortality reduces the likelihood of being married.

Table 2.4: Effect on Marriage Market

	Currently Married	At least once married
$PrePneumonia \times Sulfayears$	0.0010	-0.0027***
	(0.0037)	(0.00053)
$PreMaternalmortality \times Sulfayears$	-0.00041	-0.0040***
	(0.0055)	(0.00078)
Observations	808813	1522751

Note: Currently married is a dummy variable equal to one f the woman is married at the time of the census, and zero otherwise

At least once married is a dummy variable equals to one if the woman has ever been married in her lifetime and zero otherwise. Sample includes women age 6-44 in 1937, and 18-50 at the time of the census. All regressions include year of birth fixed effect, census fixed effect, province of birth fixed effect, years of education, number of hospitals in each province, number of doctors, health expenditure per capita, other diseases mortality levels (tuberculosis, under 2 diarrhea, meningities, cancer and circulatory system disease).

Standard errors in parenthesis, are clustered by the province of birth.

Regressions use IPUMS sampling weights.

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

2.6 Conclusion

This paper examines the impacts on the decline in mortality on fertility, labor markets, and marriage outcomes, using as a natural experiment the introduction of sulfa drugs in Chile in 1938.

Sulfa drugs were introduced in 1938, and after that, they were available nationwide. Because of their already available high supply and low cost, the diffusion was fast. The introduction of sulfa drugs causes a sharp decline in mortality levels in almost all the infectious diseases that were effectively treated by the drug, like puerperal sepsis, pneumonia, and meningitis.

Theoretically, declines in infant and maternal mortality can have ambiguous effects on fertility. According to the Backer and Lewis (1973) model, the quantity and quality of children are substitutes; therefore, reductions in the price of a child's quality will reduce the desired number of children. However, if we focus on the extensive margin, a decrease in the cost of investing in children (for example, due to better health because of sulfa drugs or better access to education) predicts an increase in the probability that a woman will have at least one child. In this case, we see some complementarity between the quality and quantity of children.

The effects of a decline in maternal mortality will also have ambiguous results in theory. For example, if maternal mortality declines, women's risk of dying will be lower, and this may increase fertility because the cost of having children is lower. This can also be interpreted as a decline in the price of child quantity. However, a lower risk of dying also increases expected life expectancy, so the benefits of getting educated increased. This is because the incentives of getting educated increase as the investment return also increase (Jayachandran and Lleras Muney (2009)). Suppose women became more educated and enter the labor market. In that case, the opportunity cost of women will increase, then women should be seen their fertility be reduced along both intensive and extensive margin.

I show that child mortality decline, measured as pneumonia decline because of sulfa

drugs availability, can decrease fertility by stimulating women's labor force participation. At the same time, a reduction in maternal mortality also reduces fertility and increases the likelihood of remain childless. These results imply that the opportunity cost factor because of longer life expectancy and higher returns of education and employment are more significant than reducing the risk of dying during childbearing.

These results are relevant in the developing world today, where child and maternal mortality are still high. Also, in these regions, fertility remains high, and therefore human capital investment, especially in the case of women, is low. My results show that declining mortality reduces fertility across both margins, so understanding the factors for this requires more future research.

Chapter 3

Infant mortality and health care access: Evidence from Chile 1933-60

3.1 Introduction

Infant mortality rate (IMR), defined as infant deaths under one year old over 1,000 live births, is one of the most critical health outcomes. Maternal mortality is also relevant as a determinant of female health. Moreover, these variables are not only crucial as health outcomes but as social and economic development indicators.

Currently, infant mortality in the world is still on average above 30 infants deaths over 1,000 live births, and maternal mortality is still 211 per 100,000 live births. This number, even decreasing in most of the world, are still high in the developing world. One of the main reasons that developed and developing countries attribute to the decline in infant and maternal mortality is known as childbirth professionalization, i.e., the use of highly skilled medical personnel to attend deliveries.

Immediate professional care during labor and delivery can make the difference between life and death for both women and their newborns. Both maternal and neonatal mortality are lower in countries where mothers give birth with the help of skilled professionals (WHO,

2005). It has been argued that most northern European countries halved their maternal mortality in the early 20th century by providing professional midwifery care at childbirth.

During the nineteenth century, most European countries adopted a strategy for promoting skilled attendance at delivery, believed to be a result of an extensive collaboration between physicians and highly competent, locally available midwives. The nonseptic maternal mortality was reduced from 414 per 100,000 live births to 122 when the proportion of deliveries assisted by midwives in the rural areas increased from 30% to 70%. The risk of nonseptic maternal death was reduced fivefold (Hogberg (2004)). For Sweden and Norway, it has also been tested that increasing the number of trained midwives decrease maternal mortality but has no significant effect on infant mortality (Pettersson-Lidbom (2015) and Kotsadam et al. (2017)).

In more developing countries, it has also been attributed to childbirth professionalization the decline of maternal mortality. Malaysia and Sri Lanka are examples of successful stories of reducing maternal mortality. These countries halved their maternal mortality every 7-10 years since 1950. It has been argued that this decline began whit the popularization of skilled attendance at birth and with the improved access and development of health care facilities. More recently, Honduras brought maternal deaths down from 182 to 108 per 100,000 between 1990 and 1997 by opening and staffing seven referral hospitals and 226 rural health centers and by increasing the number of health personnel and skilled attendants. More recently, Egypt reduced its maternal mortality by more than 50% in eight years, from 174 in 1993 to 84 per 100 000 live births in 2000 by doubling the proportion of births attended by a doctor or nurse and improved access to emergency obstetric care (WHO, 2005).

Every country saw declines in infant and maternal mortality during the 20th century; however, the magnitude of the reductions was quite different. While there are still countries, like India, with infant mortality rates over 40 or countries in Africa over 50, some have more successful histories.

Chile is a clear example of this; the infant mortality rate in 1940 was one of the highest

in the world, with over 200 babies dying before their first year of life (Figure 3.1 and 3.2 [1].

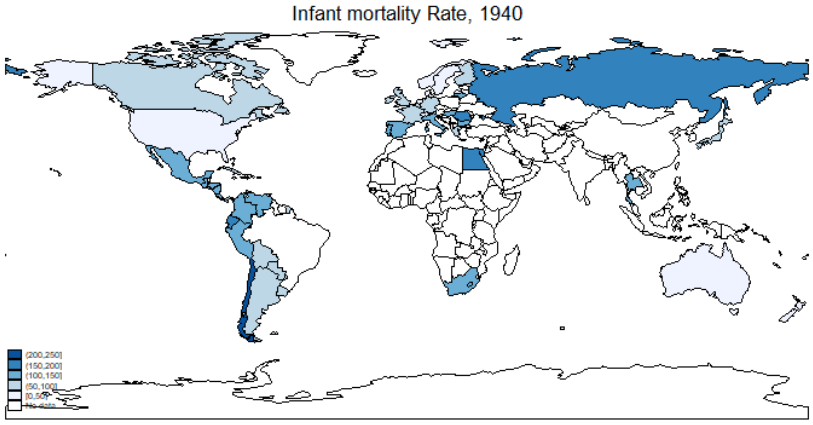


Figure 3.1: Infant mortality rate, 1940

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, Vital Statistics for United States, Abouharb, M.,& Kimball, A. (2007).

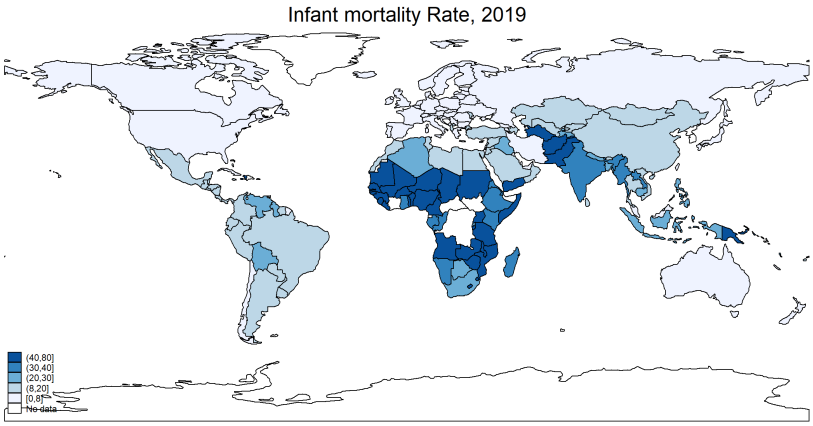


Figure 3.2: Infant mortality rate, 1940

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, Vital Statistics for United States, World Bank.

However, since then, IMR has declined significantly; Chile's IMR nowadays is similar to those of the developed countries (and lower than any other Latin American country). While in 1940, Chile has rates more than four times the infant mortality of the United States, now the difference is negligible. (Figure 3.1). At the same time, maternal mortality also declined

[1] As a reference, India Infant mortality rate in the 40s was around 160, and in other Latin American countries like Argentina was 90, Colombia 142, Mexico 126.

significantly, from more than 1,000 per 100,000 live birth in 1940 to 300 in 1960.

In this paper, I examine the role of health personnel on infant mortality in Chile for the period 1930-1966. In particular, I explore the effects of physicians' and midwives' assistance during birth and the impact of shifting of childbirth from home to hospitals on infant and maternal mortality ratio.

Literature studying infant mortality and demographic transition from a historical perspective is quite broad. Most of the research focuses on the nineteenth century for developed countries, and most of these studies use parish data. These studies explain IMR as a function of different variables, like fertility, mother's characteristics (mother's age and education), birth characteristics (birth order, birth interval, etc.), socioeconomic variables (income or unemployment), and illegitimacy status, between others.

Studies that focus on health care access are scarcer. In terms of health personnel (physicians or midwives) impact on health and mortality, historical research is limited and only based on high-income countries like Sweden, Norway, or the US. For more impoverished countries, there is only contemporary data. Most of these studies find evidence that being born with the assistance of a midwife/physician has a positive effect on maternal mortality, with more mixed conclusions for infant mortality. For Sweden, Pettersson-Lidbom (2015) investigates the impact on maternal and infant mortality and finds that increasing the number of trained midwives decreases maternal mortality. Lazuka (2018) analyzes the long-term effects of being born with the assistance of a midwife and finds that treatment by qualified midwives at birth reduced neonatal mortality. For Norway, Kotsadam et al. (2017), examine the relationship between health personnel and mortality for the period 1887-1921 and find that midwives reduced maternal mortality, but they find no effect for other types of health personnel or infant mortality. For the US, Thomasson and Treber (2007), study the impact on maternal mortality resulting from the shift of childbirth from home to hospitals during the first half of the twentieth century. Also, for US Anderson et al. (2016) uses the introduction of midwife licensing to identify the effect of professional birth help on maternal and infant

mortality and find significant decreases in maternal mortality but not in infant mortality.

The literature is a little scarcer for developing countries, and it focuses on the late 20th century. Also, survey data or Census are the primary sources of data to estimate IMR and maternal mortality. The main problem is the lack of yearly data in the case of the Census and surveys because it only shows you a moment and doesn't explain the trajectory of IMR.

Another problem with the literature of developing countries is that they usually focus on the country as a whole, not considering the regional differences in IMR. For Peru, Edmonston and Andes (1983) find that mortality variation exists: mortality is inversely related to community population size and is higher in the mountains than in the jungle or coast.

Similar to developed countries, most studies focus on birth characteristics [2], socioeconomic factors [3], urban-rural differences [4], clean water and sanitation [5], and health care [6].

Because of this, this paper fills the gap in the literature, using yearly data to study the impact of access to health care, measured as the number of birth deliveries in a hospital or under the assistance of a doctor or midwife in the context of a middle-income country like Chile. This has important policy implications for thinking in valuable ways that health interventions may reduce infant/maternal mortality.

Using panel data, with yearly hand-collected data from the Demographic and Social Assistance Yearbooks of the National Institute of Statistics, I estimate a fixed-effect model of the impact of access to health care on infant mortality rate. Panel data allows us to control for many confounding factors that may affect infant mortality, fixed effects capture geographical heterogeneity, and the year fixed effect captures aggregate shocks. To correct for potential endogeneity of the location of doctors/midwives, I also use an instrumental variable.

[2] Bangladesh, Koenig (1990); Malaysia, DaVanzo, and Habich (2016)
[3] For Philippines, Indonesia and Pakistan, Martin et al. (1983)
[4] For Brazil, Sastry (1984); for Guatemala, Haines et al. (1983); Costa Rica, Haines and Avery (1982)
[5] Malaysia, DaVanzo, and Habicht (2016); Estonia, Jaadla and Puur (2016), Brazil (Merrick (1985))
[6] Malaysia, Pais, and Lillard (1994), Costa Rica, Haines and Avery (1982).

My paper relates to the literature that studies the demographic and fertility transition. It also associated with the literature trying to explain the impact of medical care on children and women's health. I find that being born in a hospital impacts infant and neonatal mortality, especially for the period after 1940 and for urban areas where access to hospitals for childbirth is easier. The number of doctors and midwives in the province also significantly reduces infant mortality and neonatal mortality, but I find no effect on maternal mortality.

The rest of the paper is structured as follows. Section 2 provides a brief explanation of why Chile is an interesting country to test the effects of health personnel on IMR. Section 3 describes the data used. Section 4 gives a brief history of Health care in Chile during the period. Section 5 explains the empirical strategy used. Section 6 shows the results of the evidence between health care access and IMR, and finally, section 7 concludes.

3.2 Why Chile?

Chile has a history of data. Demographic data has been collected since the beginning of 19th century [7]. The vital statistics series has been collected by the Central Statistics Office (or its equivalent) since 1848. There has been variation in the consistency of these registers; before 1885, the Catholic Parishes reported births and marriage, but after this date, the Civil Register was created and took this responsibility.

Under-registration of births and deaths is a critical element to consider when studying infant mortality rates. In Chile, registration of deaths has been relatively complete since a death certificate has always been an indispensable preliminary to interment. However, births had been under-registered to some extent, especially before 1928. Also, it has been established that at certain times of the year, especially winter and in isolated parts of the country, registration is not carried out in the allocated time (60 days after birth). Nevertheless, most of the problem is related to a delay in the registration more than avoiding it.

Table 3.1 shows the completeness of the birth records for Chile for the period 1940 to 1955. For most of the period, under-registration has been close to 10%, to decrease to less than 5% after 1952.

Table 3.1: Births: Completeness of the record

1940	91.1
1941	90.4
1942	89.7
1943	88.7
1944	88.1
1945	88.2
1946	88.9
1947	86
1948	90.2
1949	91.6
1950	91.8
1951	91.2
1952	91.2
1953	95
1954	95
1955	95

Source: Chile, Demographic Yearbook, 1955.

[7] The first Census of Chile was taken in 1835, and have been continuing every ten years for almost all period since then.

Besides the relatively high-quality data available, the data is yearly, not a survey or estimated from census, like usually is available for other developing countries. This allows observing the determinants of the trajectory of infant mortality in detail. It also provides data at a provincial and circumscription level to see the cross-province difference and geographical variation in IMR.

Moreover, Chile has an exceptional performance in reducing infant mortality during the second half of the twentieth century, so it is an excellent example for other developing countries about policies that may contribute to declining infant mortality.

Figure 3.3 shows a scatterplot with the IMR for the years 1940 and 2000 for a selected group of Latin American countries and the US. As you can see, IMR in 1940 was the highest in Chile (compared to the other countries in the sample), but now it is one of the lowest in 2000. This implies that Chile decreases its IMR a lot more than any other of these countries.

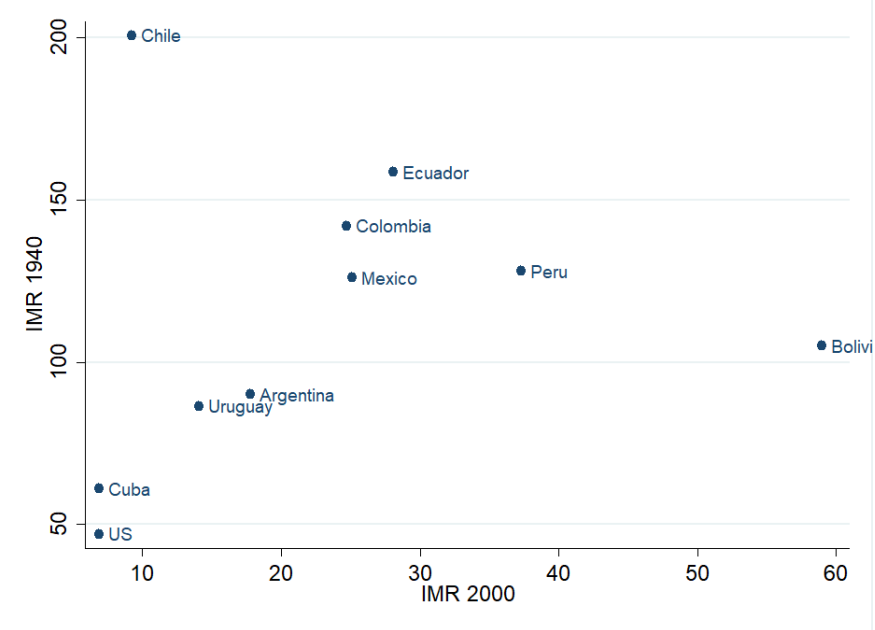


Figure 3.3: Infant mortality rate 1940 vs infant mortality rate 2000

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, Vital Statistics for United States, Abouharb, M.,& Kimball, A. (2007).

This decrease is not related to the increase in income during this period. If we plot

the percentage change in IMR during the same period, 1940 to 2000, versus the percentage change in Gross Domestic Product (Figure 3.4), we can see that the decline in IMR for Chile is more than what is expected given the increase in income.

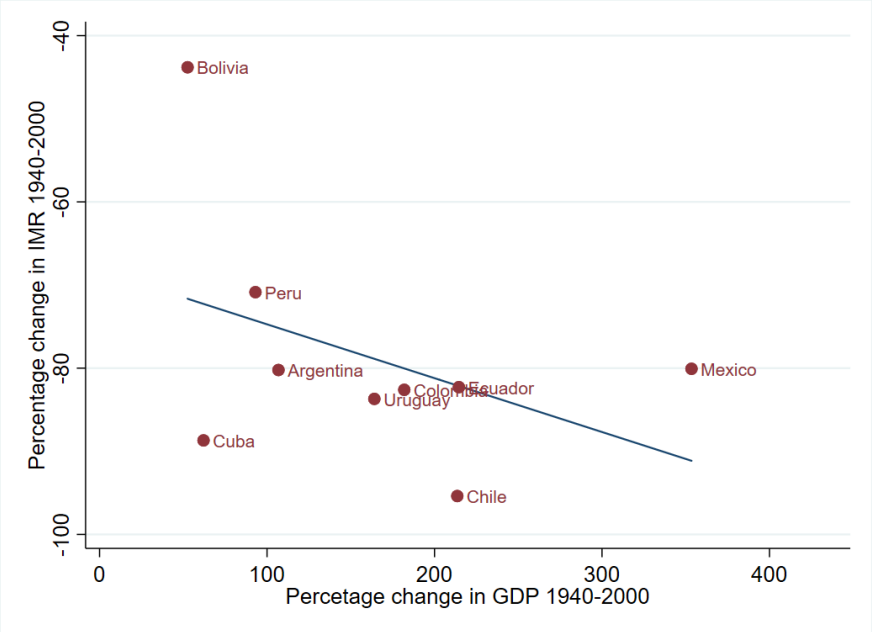


Figure 3.4: Percentage change in IMR vs. percentage change in GDP

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, Vital Statistics for United States, Abouharb, M.,& Kimball, A. (2007).

3.3 Data

Data comes from the Demographic and Social Assistance Yearbooks of the National Institute of Statistics. This information was produced by the National Health Service, including information on births, live births, death births, infant deaths (under one-year-old), total deaths, illegitimacy births, births under the assistance of midwife/physician, deliveries happening at a hospital, between others.

Information about births, deaths, infant deaths, illegitimacy, and deliveries in a hospital are available for all the period 1933-60. However, data on birth assistance is only available from 1952, and gender differences in mortality are only available until 1951.

Maternal mortality is available only at the provincial level, as well as other causes of death. Maternal mortality groups several causes of deaths related to childbearing, the most important being puerperal sepsis.

Data is available at the provincial and district ("circunscripcion") level depending on the variable since 195. For the years previous, only provincial data is available. I will do my analysis at the province and district levels, depending on data availability.

There are some changes in the number of provinces during the period studied. To avoid potential problems that may arise, I harmonized the districts, aggregating them when needed to form a unit that does not change over time.

I also use information from the 1930, 1940, 1952, and 1960 census. This provides population, the population living in urban and rural areas, illiteracy, among others.

3.4 Mortality and Health in Chile

At the beginning of the twentieth century, Chile's IMR was over 300. Figure 3.5 shows the time series of total infant mortality rate and by gender from 1933 to 1960. For most of the period, male infant mortality is higher than females, but both show almost the same trend. Infant mortality declines significantly during the period 1933-1953, to remain constant after.

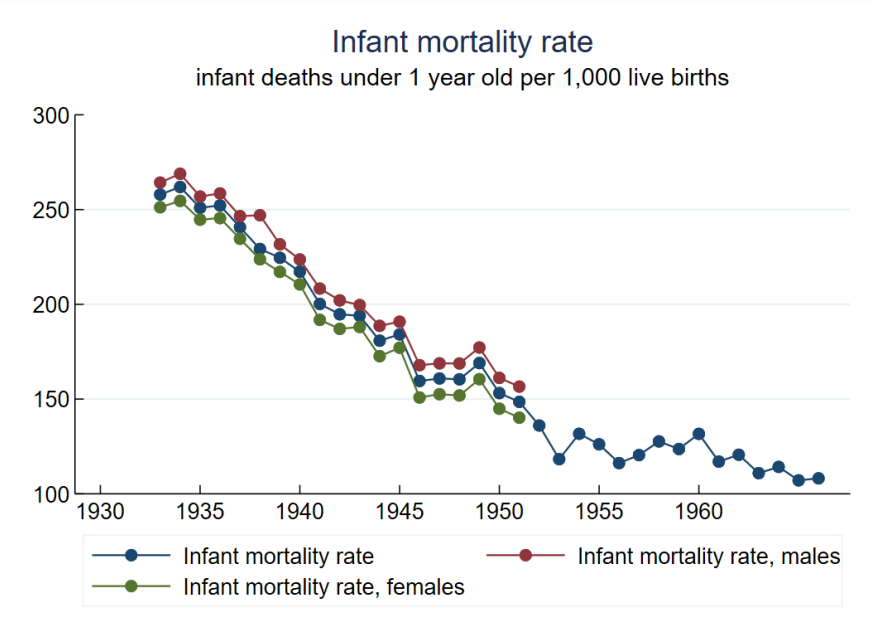


Figure 3.5

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, and Population Census.

Figure 3.6 show the trajectory of neonatal mortality rate (deaths of infants under 28 days per 1,000 live births) for the same period. Neonatal mortality falls more rapidly than total infant mortality, suggesting that access to medical care during childbirth may be a decisive factor to explain the fall in mortality.

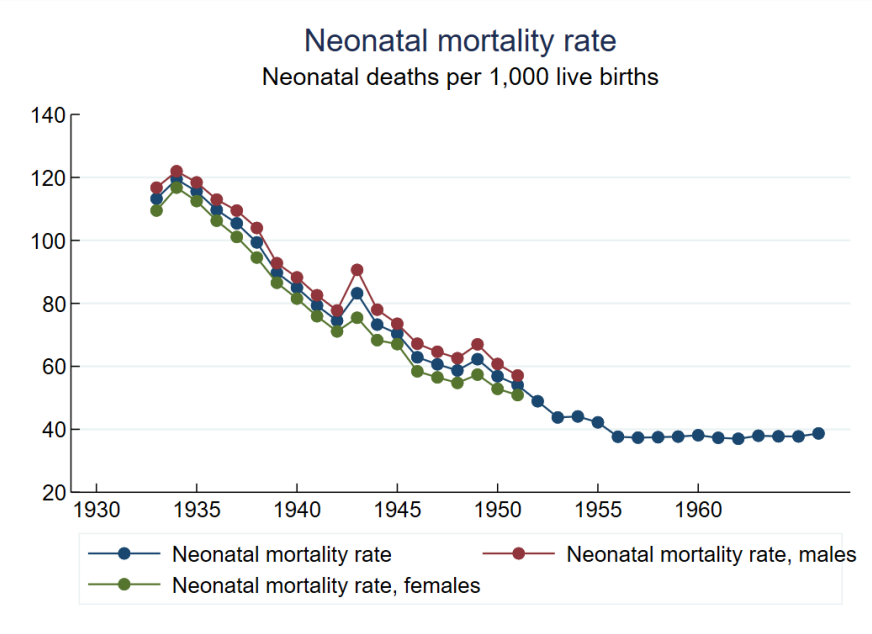


Figure 3.6

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, and Population Census.

The trajectory of maternal mortality ratio, maternal deaths over 1,000 live births, is shown in figure 3.7. As an infant and neonatal mortality, it experiences a significant decline during the period.

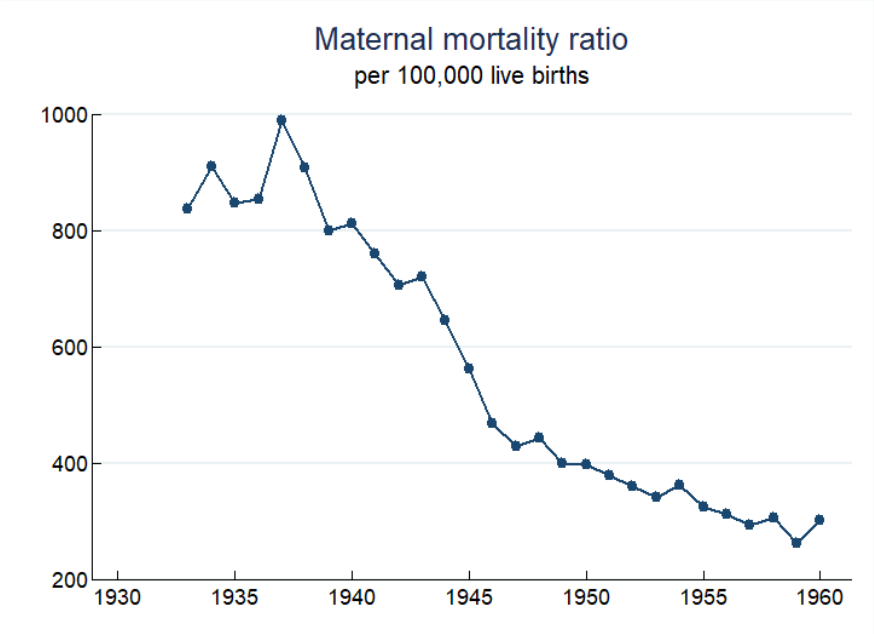


Figure 3.7

Source: Chile, Demographic and Social Assistance Yearbooks of the National Institute of Statistics, and Population Census.

In the early 20th century, most of the births were at home without assistance. Only poor women were having their babies in hospitals. It was not until 1940 that this start to changed, and hospitals become more prevalent. Figure 3.8 shows the percentage of births that occurred in a hospital. Before 1940, less than 20% of births were at hospitals; by 1960, this was over 60%. However, there was still significant geographical variation. Figure A.3.1 and A.3.2 shows this. In 1940, there were provinces with less than 10% of the births occurring in a hospital, while others like Santiago or northern provinces like Antofagasta and Atacama over 30%. By 1960, hospitals were a lot more frequent, but there was still a significant variation.

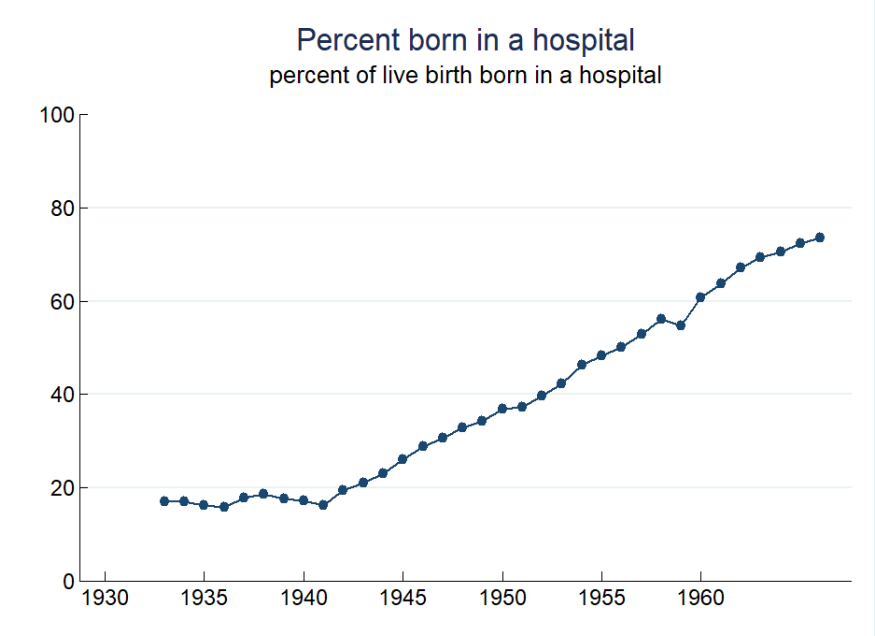


Figure 3.8

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, and Population Census.

Infant and maternal mortality were a critical issue for the Chilean authorities during the period, so they saw increasing the number of babies being delivered with medical assistance as a priority to fight the high levels at the moment. This includes not only more births happening in hospitals but also more deliveries with the assistance of physicians and midwives.

The number of hospitals and the number of maternity wards were inadequate. In 1917, for example, in Concepcion, one of the biggest cities with a population of 60,000 people, there was only one maternity ward, with only 29 beds. Smaller towns didn't have anything. Even the capital of Santiago only had three maternity wards (and two were in the same hospital). This change after the 1930s, the number, and size of hospitals (measure as total beds in hospitals) increase. Table 3.2 exhibits the number of hospitals, maternity beds, total beds in the hospital per 100,000 population (absolute numbers in table A.3.1). Chile's number of hospitals and the number of beds increased from 1933 to 1960; however, in terms of population, the number of hospitals and beds decreased during the 1950s. In 1935, the

number of hospitals per 100,000 habitants was 4.015, and in 1960 it was 3.128, a significant decrease [8].

Table 3.2

Year	Hospitals	Maternity beds	Total beds	Percent of births in a hospital
1933	3.249	39.864	390.430	17.01
1935	4.015	42.260	400.302	16.18
1940	3.941	48.770	443.592	17.13
1945	3.727	48.639	483.831	26.10
1950	3.818	51.601	480.755	36.94
1955	3.224	48.601	433.576	48.21
1960	3.128	47.259	393.287	60.82

Source: Chile, Demographic and Social Assistance Yearbooks of the National Institute of Statistics, and Population Census.

Health care in Chile during this period was free or available at a super low cost. However, until the creation in 1952 of the National Health Service (NHS), most hospitals were managed by philanthropic organizations. Hospitals were operated throughout the country by the local welfare boards or "juntas de beneficiencia." Initially supported by private contributions and the Church, these facilities gradually became semi-governmental institutions receiving public support and subject to limited governmental coordination and regulation. Collectively they accounted for almost all of Chile's hospital beds but provided little ambulatory care.

The NHS was born out of a fusion of the welfare hospital system, the network of ambulatory clinics for workers, the two-state agencies mentioned earlier, and other lesser institutions. Beneficiaries included workers formerly covered by the Workers' Obligatory Fund, dependents, employees of the NHS, and low-income families (medical indigents) not otherwise covered by social security. Besides economic benefits for insured workers, comprehensive curative and preventive health services were provided at low or no cost. Public health activities concerned with the environment and infectious disease control were carried out for the entire population, insured or not.

Another relevant aspect during this period, is total births. Births increased during

[8] As a reference, the number of hospitals per 100,000 population was 4.9 in 1935 and 4.7 in 1940 in the United States

the period, but the trend increase after 1955 (Figure A.3.3). It is essential to notice that illegitimate births were quite prevalent; figure 3.9 shows the illegitimacy rate, calculated as a percentage of total births. Even with Chile being a mostly catholic country, this number was close to a 30% in 1935, but it declined substantially after that[9].

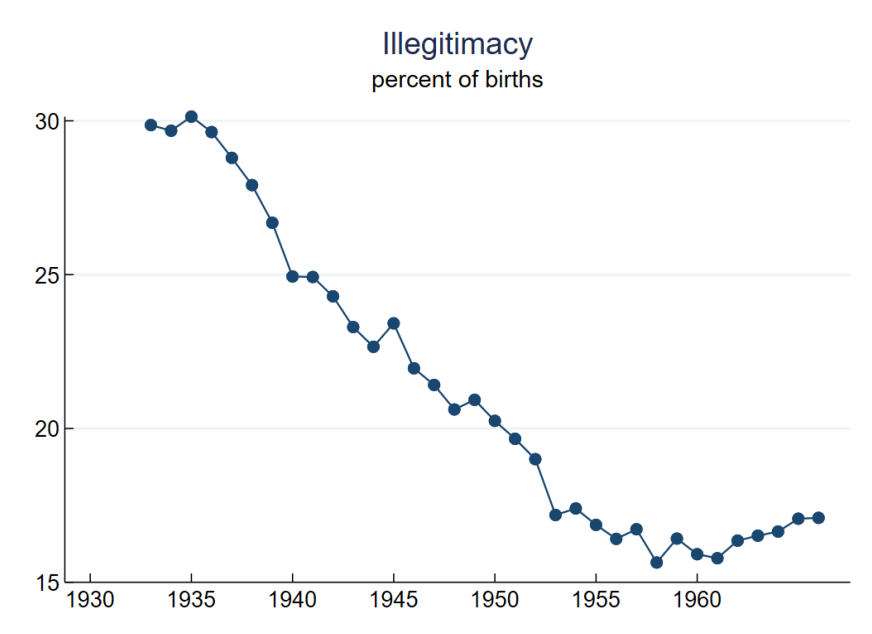


Figure 3.9

Source: Chile, Demographic and Social Assistance Yearbooks of the National Institute of Statistics, and Population Census.

Abortion was also quite spread; estimations are between a 16-23% of the women commit an abortion. Evidence shows that marital status was not related to the probability of inducing abortion, neither with religiosity or educational achievement, but mostly with the number of previous children. Women used abortion almost as a contraceptive.

Infant mortality was high for all the early 20th century, on average 20% of infants die before their first year. Table 3.3 shows the average infant, neo, post neo, and total mortality. While neonatal mortality was closed to half total infant deaths by 1935, it was less than a third by 1960. Regional and time variation was quite significant. Table 3.3 show the median, maximum, and minimum value for the whole period.

[9]It is interesting to notice, that a high number of these people having children out of wedlock eventually legitimize their children. For example, from the marriages happening in 1937, 25.8% legitimize their children.

Table 3.3

	Infant mortality rate	Neonatal mortality rate	Post-neonatal mortality rate	Total mortality rate	Maternal mortality
Median	166.2	58.1	102.6	1701.1	526.40
Max	333.96	200.8	179.83	7720.89	1670
Min	69.3	22.86	32.14	693.04	55.84

Source: Chile, Demographic and Social Assistance Yearbooks of the National Institute of Statistics, and Population Census.

3.5 Empirical Strategy

I want to test if the impact of health personnel on infant and maternal mortality. There are potential factors that may be linked with both infant/maternal mortality and the amount of health personeel in a district. For example, richer areas, may have lower IMR and higher quantity of personnel. To control for these potential factor, we estimate a province-year panel with the following specification:

$$y_{dt} = \alpha + \beta Percentborninhospital_{dt} + \gamma_t + \theta_d + \epsilon_{dt} \tag{3.1}$$

and,

$$y_{dt} = \alpha + \beta healthpersonnel_{dt} + \gamma_t + \theta_d + \epsilon_{dt} \tag{3.2}$$

Where y, the dependent variable can be total infant mortality, neonatal mortality, or maternal mortality, d is the province, and t is the year. Health personnel is the number of doctors (per 100,000 population) or midwives (per 1,000live births). The time fixed effect (γ) captures aggregate shocks that affect all districts, while the province fixed effect (θ) captures geographical heterogeneity (for example, a region may be more educated than the others).

Following the standard in the literature, the dependant and independent variables are logged to make the estimates less sensitive to extreme observations.

A concern for my specification is endogeneity. What if physicians or midwives may be more likely to locate in areas with greater numbers of hospitals, then this may have increased the probability that a pregnant woman sought a physician's assistance during delivery and thus increased her chance of delivering in a hospital. Also, physicians and midwives may be more likely to be placed in regions with high infant/maternal mortality, biasing the results.

To control for these factors, I can use total beds and maternity beds as an instrument for doctors and midwives. It should be expected that more doctors to be associated with more beds in the hospital, a larger hospital with more beds should also have more medical personnel. However, the number of beds should not directly affect infant or maternal

mortality [10].

For this I will estimate:

$$y_{dt} = \alpha + \beta \widehat{healthpersonnel}_{dt} + \gamma_t + \theta_d + \epsilon_{dt} \tag{3.3}$$

Where $\widehat{healthpersonnel}$ will be instrumentalized with total beds and maternity beds, and the rest variables are the same described before.

[10]The exception may be in a super crowded hospital, where lack of beds prevents people from getting access to medical care

3.6 Results

Fixed effects

Table 4 shows the results for estimating the fixed effects model of equation (1). for the period 1933-1960. This will allow us to observe if the shift of childbirth from houses to hospitals positively or negatively impacts females and infant's healths. Being born in a hospital is related to lower neonatal infant mortality (infant deaths in the first 28 days of life). However, it increases the likelihood of infant, post-neonatal (infant deaths between 28 days and one year), and maternal mortality.

In the case of post-neonatal mortality, this is expected. Being born in a hospital with the assistance of medical personnel will have an effect on reducing neonatal mortality, especially in the first days of life. Being put in an incubator or receiving specialized medical urgent care at birth may increase the chances of a baby surviving.

In the case of maternal mortality, the results are a little unexpected. However, one reason that may explain this is that at the beginning of our period, during the 1930s, only women with a high risk of dying during childbirth attend hospitals. If we do the same regression but only include the period from 1940, the results change (table A.2). For this time period, being born in a hospital is associated with reducing infant, neonatal, and maternal mortality.

Table 3.4

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Post-neonatal mortality	(6) Post-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Percent born in a hospital	-0.103***	0.00999	-0.133***	-0.0493*	-0.0545**	0.0756***	-0.142***	0.166***
	(0.0200)	(0.0194)	(0.0216)	(0.0257)	(0.0255)	(0.0254)	(0.0444)	(0.0476)
Constant	8.170***	7.081***	4.703***	3.009***	9.887***	9.116***	16.93***	10.88***
	(0.641)	(0.599)	(0.692)	(0.793)	(0.819)	(0.782)	(1.425)	(1.466)
Observations	420	420	420	420	420	420	420	420
R-squared	0.770	0.876	0.859	0.885	0.508	0.721	0.672	0.785
Number of prov	15	15	15	15	15	15	15	15
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Fixed effect regression, significance $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

In the following table are the results from regressing equation (2) (table 5) , regress the logged mortality rate on doctors per 100,000 population as independent variable. More doctors in a province reduce neonatal mortality. Still, effects are mixed for maternal and infant mortality [11].

For midwives, an increase in the number of midwives, also decreases neonatal mortality (table 6). However, they also don't have an important effect over the other mortalities.

[11] Regression since 1940 are in table A.3, in this case, effects on doctors don't change

Table 3.5

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Pos-neonatal mortality	(6) Pos-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Doctors	-0.0722**	0.0140	-0.168***	-0.108***	0.00158	0.0863**	-0.0163	0.141**
	(0.0323)	(0.0270)	(0.0345)	(0.0355)	(0.0404)	(0.0354)	(0.0709)	(0.0668)
Constant	7.454***	7.172***	4.037***	2.731***	9.370***	9.891***	15.65***	12.80***
	(0.639)	(0.533)	(0.683)	(0.701)	(0.800)	(0.700)	(1.402)	(1.320)
Observations	420	420	420	420	420	420	420	420
R-squared	0.758	0.876	0.854	0.886	0.502	0.719	0.664	0.780
Number of prov	15	15	15	15	15	15	15	15
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Fixed effect regression, significance *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Table 3.6

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Pos-neonatal mortality	(6) Pos-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Midwives	0.00104	0.0165	-0.0671**	-0.0226	0.0582*	0.0500	0.135**	0.213***
	(0.0279)	(0.0235)	(0.0303)	(0.0312)	(0.0346)	(0.0309)	(0.0605)	(0.0574)
Constant	7.197***	7.066***	3.887***	2.488***	8.998***	9.761***	14.72***	11.26***
	(0.658)	(0.572)	(0.715)	(0.761)	(0.816)	(0.754)	(1.427)	(1.399)
Observations	420	420	420	420	420	420	420	420
R-squared	0.755	0.876	0.847	0.884	0.506	0.717	0.668	0.786
Number of prov	15	15	15	15	15	15	15	15
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Fixed effect regression, significance *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Another consideration is that the effect may differ for urban and rural areas. Access to hospitals and medical care will be easier in more urban areas than in rural areas. To see this, I declared a province as urban if more than 50% of the population lives in urban areas. The following table 7 shows the results for only urban provinces. In this case, being born in a hospital is associated with a lower infant, neonatal, post-neonatal, and maternal mortality.

More doctors (table 8) and more midwives (table 9), reduce infant and neonatal mortality, but show no significant impact on maternal mortality.

Table 3.7

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Pos-neonatal mortality	(6) Pos-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Percent born in a hospital	-0.336***	-0.190***	-0.124**	-0.0794	-0.440***	-0.249***	-0.630***	-0.171
	(0.0475)	(0.0529)	(0.0500)	(0.0715)	(0.0599)	(0.0677)	(0.102)	(0.129)
Constant	7.477***	8.384***	6.786***	7.937***	6.826***	7.699***	16.59***	12.02***
	(0.995)	(1.053)	(1.046)	(1.425)	(1.254)	(1.348)	(2.134)	(2.580)
Observations	180	180	180	180	180	180	180	180
R-squared	0.784	0.897	0.789	0.835	0.688	0.848	0.729	0.833
Number of prov	8	8	8	8	8	8	8	8
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Fixed effect regression, significance $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table 3.8

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Pos-neonatal mortality	(6) Pos-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Doctors	0.0148	-0.132***	-0.110*	-0.189***	0.0711	-0.108*	0.124	-0.143
	(0.0628)	(0.0485)	(0.0585)	(0.0627)	(0.0798)	(0.0631)	(0.131)	(0.117)
Constant	6.863***	6.823***	6.859***	7.553***	5.886***	5.531***	15.18***	10.66***
	(1.140)	(0.945)	(1.061)	(1.223)	(1.447)	(1.231)	(2.374)	(2.275)
Observations	180	180	180	180	180	180	180	180
R-squared	0.720	0.894	0.786	0.843	0.590	0.837	0.669	0.832
Number of prov	8	8	8	8	8	8	8	8
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Fixed effect regression, significance *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$

Table 3.9

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Pos-neonatal mortality	(6) Pos-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Midwives	-0.0670	-0.150***	-0.119**	-0.257***	-0.0396	-0.0959	0.148	-0.0146
	(0.0584)	(0.0474)	(0.0544)	(0.0600)	(0.0745)	(0.0624)	(0.122)	(0.116)
Constant	7.169***	7.828***	7.048***	9.353***	6.229***	6.127***	14.91***	10.50***
	(1.148)	(1.015)	(1.069)	(1.286)	(1.465)	(1.336)	(2.395)	(2.477)
Observations	180	180	180	180	180	180	180	180
R-squared	0.722	0.895	0.787	0.852	0.589	0.836	0.670	0.831
Number of prov	8	8	8	8	8	8	8	8
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Fixed effect regression, significance $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Using total beds in a hospital (per 100,000 population) to instrumentalize the number of doctors (per 100,000 population), Table 10 show the results for the second stage of the regression. We see that more doctors decrease infant and neonatal mortality. For maternal mortality, using year-fixed effects cause the sign of the coefficient to change; hence maternal mortality increases with the number of doctors. This may be because, at the beginning of our period, doctors only delivered the more complicated births in hospitals, where the mother's risk was higher.

Table 3.10

	Infant mortality	Infant mortality	Neonatal mortality	Neonatal mortality	Maternal mortality	Maternal mortality
Doctors	-1.412***	-0.209	-1.968***	-0.574*	-1.974***	1.866**
	(0.159)	(0.199)	(0.219)	(0.347)	(0.246)	(0.915)
Constant	9.397***	6.013***	10.09***	6.072***	12.22***	1.730
	(0.483)	(0.524)	(0.666)	(0.913)	(0.747)	(2.409)
Observations	446	446	446	446	446	446
Number of prov	16	16	16	16	16	16
Country FE	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Instrumental variable regression, significance $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table 11 shows the results for midwives per 1,000 live births (instrumentalized by the number of maternity beds per 100,000 population). We can see midwives increase mortality. Again, this can be because, at the earlier time of our period, births with the assistance of a doctor or a midwife were scarce. Hence, people only use these services when there were complications in the pregnancy or during childbirth. If we restrict our sample to after 194, were births were more likely to be professionally assisted independently of the risk, we see that infant and neonatal mortality decrease; however, maternal mortality is still positive.

Table 3.11

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Maternal mortality	(6) Maternal mortality
Midwives	-0.829***	0.0431	-1.143***	0.0826	-1.333***	0.255*
	(0.0891)	(0.0599)	(0.115)	(0.0917)	(0.168)	(0.147)
Constant	5.474***	5.460***	4.622***	4.558***	6.810***	6.615***
	(0.0413)	(0.0266)	(0.0532)	(0.0407)	(0.0781)	(0.0654)
Observations	444	444	444	444	444	444
Number of prov	16	16	16	16	16	16
Country FE	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Instrumental variable regression, significance $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

3.7 Conclusion

There is a broad consensus that access to health care is essential and that decreasing infant and maternal mortality should be a priority across countries. In fact, the United Nations, Millenium Development Goals always emphasize a reduction in these indicators.

This paper shows the evolution of infant and maternal mortality in Chile for the mid-20th century, a period of high infant mortality, the highest in the world, and high population growth. It explores the shift from house deliveries to hospital deliveries. I found that being born in hospitals reduces the likelihood of dying during the first 28 days of life for babies. However, it doesn't have the same strong effects reducing total infant mortality, and it shows no effect on maternal mortality. The same holds for more presence of doctors or midwives.

This is a little different from the results found in articles observing this in the developed world. For Sweden, Pettersson-Lidbom (2015) investigates the impact on maternal and infant mortality and finds that increasing the number of trained midwives decreases maternal mortality. Lazuka (2018) analyzes the long-term effects of being born with the assistance of a midwife and finds that treatment by qualified midwives at birth reduced neonatal mortality. For Norway, Kotsadam et al. (2017), examine the relationship between health personnel and mortality for the period 1887-1921 and find that midwives reduced maternal mortality, but they find no effect for other types of health personnel or infant mortality. This is contrary to my results, where I found an impact on infant mortality but not maternal mortality. More research is needed in this area in more developing, high mortality rates countries to achieve a more consistent conclusion.

3.8 Appendix

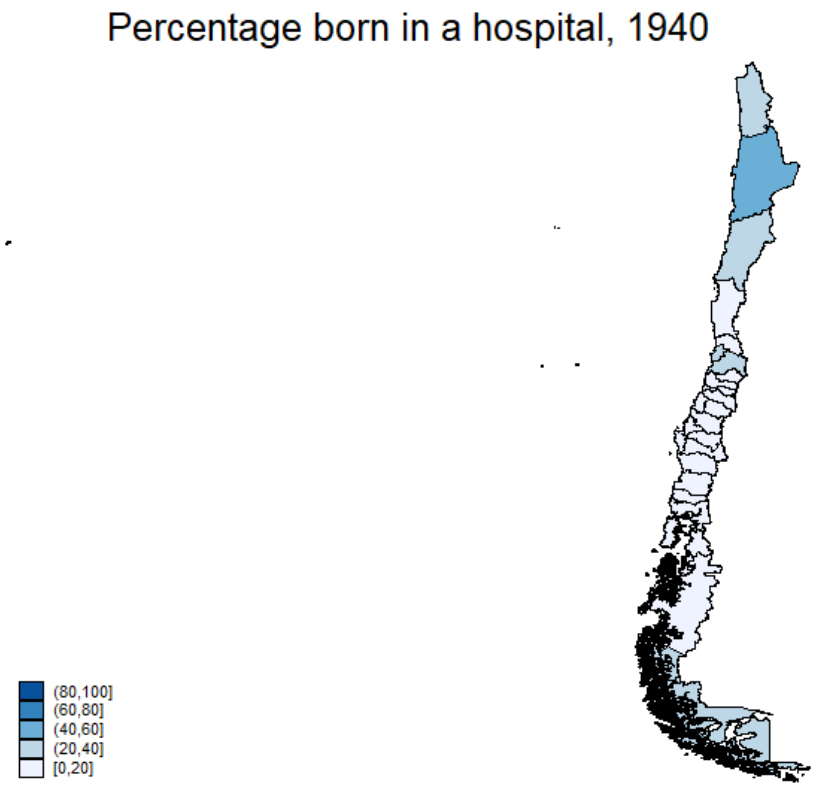


Figure A.3.1

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, and Population Census.

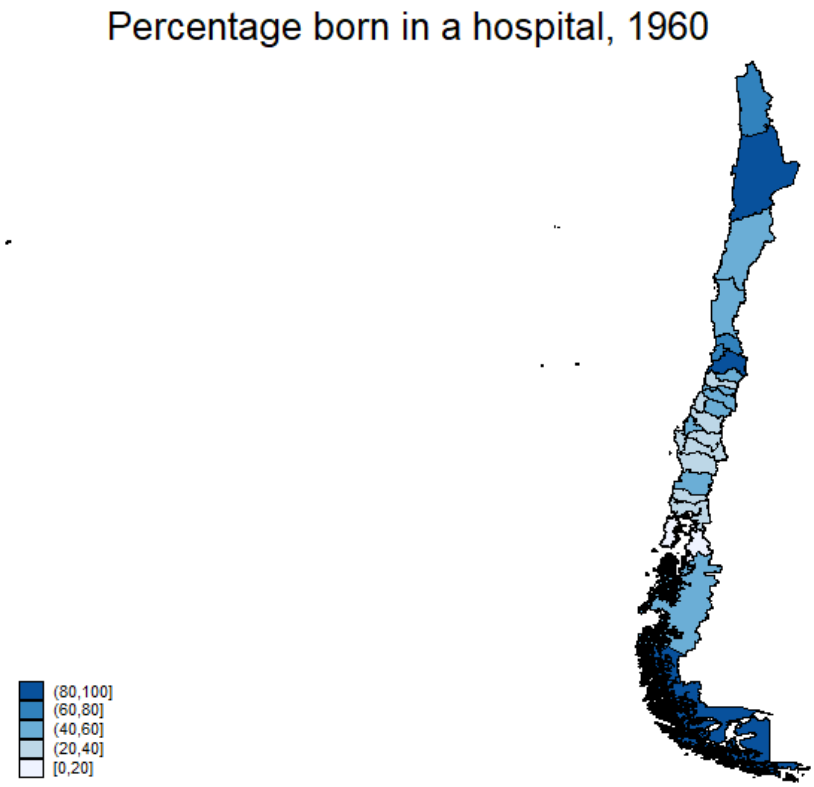


Figure A.3.2

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, and Population Census.

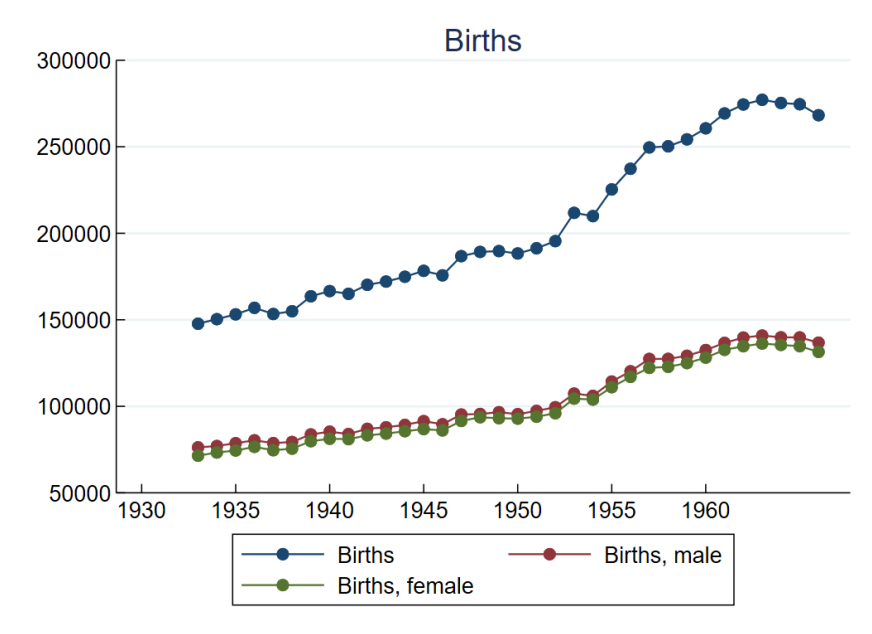


Figure A.3.3

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, and Population Census.

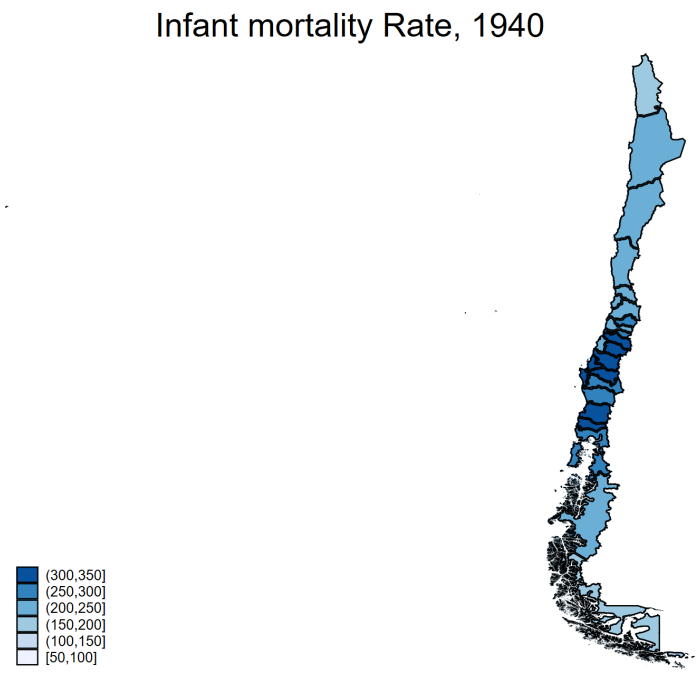


Figure A.3.4

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, and Population Census.

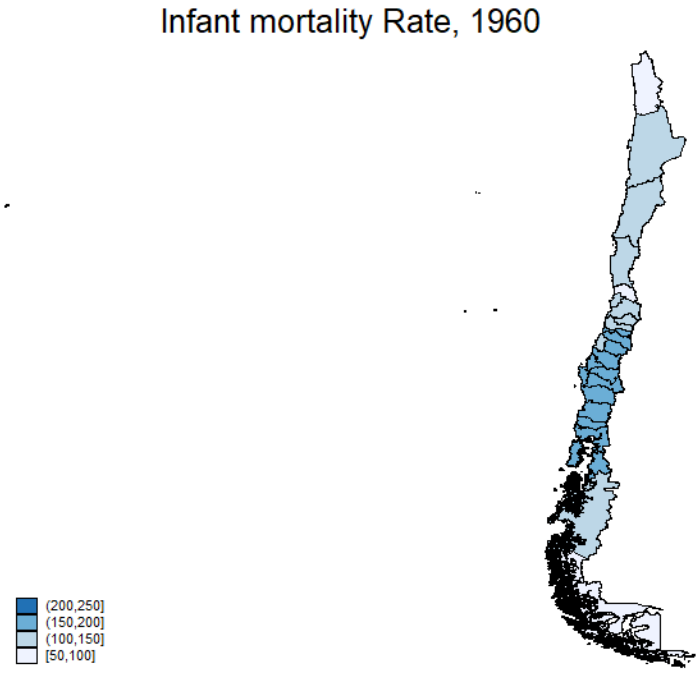


Figure A.3.5

Source: Demographic and Social Assistance Yearbooks of the National Institute of Statistics in Chile, and Population Census.

Table A.3.1

Year	Number of Hospitals	Number of doctors	Number of midwives	Number of maternity beds	Number of total beds	Number of births occurred in a hospital
1933	144	943	175	1,767	17,306	25,132
1935	181	1006	215	1,905	18,045	24,777
1940	198	1418	277	2,450	22,284	28,536
1945	201	1611	272	2,623	26,092	46,531
1950	224	2205	312	3,027	28,202	69,566
1955	218	3413	465	3,286	29,315	108,640
1960	242	3724	632	3,656	30,425	158,531

Source: Chile, Demographic and Social Assistance Yearbooks of the National Institute of Statistics, and Population Census.

Table A.3.2: Reg:1940-1960: Percent born in a hospitals

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Pos-neonatal mortality	(6) Pos-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Percent born in a hospital	-0.118***	-0.0474*	-0.152***	-0.0938***	-0.0842**	-0.00604	-0.239***	0.111
	(0.0258)	(0.0269)	(0.0280)	(0.0348)	(0.0332)	(0.0342)	(0.0630)	(0.0718)
Constant	7.617***	8.102***	5.896***	4.871***	8.021***	9.388***	14.81***	9.823***
	(0.668)	(0.713)	(0.724)	(0.923)	(0.856)	(0.908)	(1.626)	(1.906)
Observations	315	315	315	315	315	315	315	315
R-squared	0.656	0.798	0.803	0.835	0.333	0.615	0.594	0.713
Number of prov	15	15	15	15	15	15	15	15
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Instrumental variable regression, significance $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$

Table A.3.3: Reg:1940-1960: Doctors

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Pos-neonatal mortality	(6) Pos-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Doctors	-0.0448	-0.0117	-0.0658	-0.0217	-0.0454	-0.0239	-0.158*	0.0807
	(0.0379)	(0.0312)	(0.0416)	(0.0407)	(0.0475)	(0.0395)	(0.0911)	(0.0831)
Constant	7.058***	7.551***	5.178***	3.779***	7.623***	9.333***	13.68***	11.08***
	(0.678)	(0.643)	(0.743)	(0.838)	(0.850)	(0.813)	(1.630)	(1.713)
Observations	315	315	315	315	315	315	315	315
R-squared	0.633	0.796	0.785	0.831	0.321	0.615	0.578	0.712
Number of prov	15	15	15	15	15	15	15	15
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Instrumental variable regression, significance $*p < 0.1$, $**p < 0.05$, $***p < 0.01$

Table A.3.4: Reg:1940-1960: Midwives

	(1) Infant mortality	(2) Infant mortality	(3) Neonatal mortality	(4) Neonatal mortality	(5) Pos-neonatal mortality	(6) Pos-neonatal mortality	(7) Maternal mortality	(8) Maternal mortality
Midwives	-0.0185	-0.0570*	-0.108***	-0.0833**	0.0372	-0.0400	0.117	0.290***
	(0.0324)	(0.0294)	(0.0351)	(0.0383)	(0.0406)	(0.0374)	(0.0779)	(0.0770)
Constant	7.128***	8.136***	5.565***	4.631***	7.498***	9.733***	13.29***	8.116***
	(0.688)	(0.708)	(0.745)	(0.921)	(0.862)	(0.900)	(1.654)	(1.855)
Observations	315	315	315	315	315	315	315	315
R-squared	0.632	0.798	0.790	0.834	0.321	0.616	0.577	0.725
Number of prov	15	15	15	15	15	15	15	15
Country FE	YES	YES	YES	YES	YES	YES	YES	YES
Year FE	NO	YES	NO	YES	NO	YES	NO	YES

Note: Infant mortality corresponds to infant deaths under 1 year per 1,000 live births, neonatal mortality corresponds to infant death under 28 days per 1,000 population, post neonatal mortality is infant death between 29 days and 1 year, per 1,000 population.

Instrumental variable regression, significance $*p < 0.1$, $**p < 0.05$, $***p < 0.01$

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