Infant Health Monitoring, Infant Mortality and Adult Outcomes: Evidence From a Universal Reform in Sweden

Job Market Paper - Under Revision

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Abstract

In this paper, I evaluate a universal preventive health program in Sweden. The program provided well visits to pregnant women and infants. I find that access to the program reduced infant mortality by 7%. These improvements did not translate to meaningful gains in adulthood. I find no robust evidence that adult income, education or mortality were affected. To understand how the program was effective in reducing infant mortality, I use detailed contemporary cause of death data. The program did not reduce mortality in infectious diseases but decreased deaths from pneumonia by 22% among infants. This suggest that the program mainly facilitated early detection and improved access to treatment of pneumonia. The findings of this paper shed light on the importance of access to infant health care and monitoring. However, some doubts emerge on the generalizability of the long-term health benefits described in the previous literature.

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1 Introduction

A small but growing literature has taken interest in historical evaluations of early life health promoting programs (Bütikofer et al., 2015; Hjort et al., 2017; Wüst, 2012; Bhalotra et al., 2015; Moehling and Thomasson, 2014). This paper is the first to investigate a nationwide well-visit program aimed at improving infants' health. Starting in 1938 local health centres, providing well visits to pregnant women and infants free of charge, where opened all over Sweden. I evaluate the effect of this universal health program on infant mortality, adult health and labour market success. Using newly digitized cause of death data, I also examine the underlying mechanism.

I find that the program decreased infant mortality by close to 7%. Using detailed cause of death data on infant mortality, I show that the program did not affect deaths from infectious diseases but reduced deaths from pneumonia by 22%. These results do not suggest that it was increased breastfeeding or improved infant health in general that reduced infant mortality. More likely, increased monitoring, detection and treatment of pneumonia were the most important contributions of the program[1]. In contrast to the existing literature, I do not find any robust evidence supporting long-term gains on labour market outcomes or health.

Other Nordic countries introduced similar programs as in Sweden at around the same time (Bütikofer et al., 2015; Wüst, 2012). However, the Swedish setting has some clear advantages. Sweden was not militarily engaged in the second world war while both Denmark and Norway were occupied by Nazi Germany. The Swedish program was rapidly introduced over eight years which reduce the likelihood that other shocks bias the estimates. This rapid expansion also allow me to discard the years after the war from the analysis. A revolutionary time period in medicine as modern antibiotics were widely introduced[2].

[1]The introduction of Sulfapyridine in Sweden as of 1939 could further have facilitated this effect. This drug revolutionized pneumonia treatment at the time. It was more efficient that already existing treatments, could be prescribed by any physician and administered at home. It was also between $30-60$ times cheaper than other treatments (Thunberg, 1940).

[2]Also treatment for TBC, nutritionally adequate infant formulas and other advances where introduced after WWII (Wennerberg, 2012). If diffusion of these technologies where correlated with the Danish or Norwegian program roll outs, their results could be biased. If the programs

In a similar setting, Wüst (2012) show that infant mortality decreased when introducing regular nurse home visits during the first year in life in Danish towns and cities between $1937 - 1949$. Low power and imprecise data do not allow the author to be very explicit about the mechanism. Bhalotra et al. (2015) evaluate a well-visit (and home visit) trial in Sweden that later motivated the present program. They find that infant mortality decreased for those exposed to treatment. The authors also found that mortality risk was even more reduced at older ages pointing to further benefit in adulthood. The trial was though small and short lived[3].

By using detailed cause of death data, I add to these papers by establishing a credible mechanism for the program effects. I extend the external validity of these papers by using a nationwide introduction. Information on exact age at death allow me to construct mortality outcomes for any age, further strengthening the internal validity of the paper, as placebo outcomes can be constructed.

Two studies investigate historical well-visit programs and evaluate long-term outcomes (Hjort et al., 2017; Bütikofer et al., 2015). They both find that well-visit programs for infants improved adult health. The authors attribute these positive health effects mainly to improved disease environment and better nutrition. The two studies find different results on labour market outcomes, both positive (Bütikofer et al., 2015) and mixed (Hjort et al., 2017) effects. In these papers, only a subset of municipalities where ever treated, mainly in urban areas. Using the full population in Sweden, I come closer to the population effect of the intervention. Potentially explaining the differences in results.

The rest of the paper is structured as follows. Section 2 details the reform under study and the Swedish historical setting. In part 3, I describe the data sources used, variables and empirical framework. Following this, section 4 include the results and section 5 concludes the paper.

interacted with these technologies, that could explain differences in results.

[3]The Swedish trial included seven medical districts and cities that where treated for around two years. Around 4% of the population had access to the treatment.

2 Institutional Setting and the Health Centre Program

In the following section, I describe the universal preventive health program under study. Some additional information is also provided on the existing health environment, health institutions and contemporary medical technologies.

Prior to the program some infant health surveillance was already practised. Mainly through philanthropic organizations called *The milk drop*[4]. These operated in the major cities and was financed through donations and in some cases support from municipalities[5]. Just before the program started in 1938, there where atleast 48 cities, municipalities or medical districts that had some form of organised infant health monitoring in place. Still, Sweden lagged behind many other countries in Europe in terms of infant health monitoring (Medicinalstyrelsen, 1935).

To assess the interest, costs and effectiveness of universal free access to infant health centres, a trial was set up in seven medical districts (of around 450 in Sweden during the time) and cities starting in 1931 (Medicinalstyrelsen, 1935). The trial ended in 1933 and experiences from that trial guided the further planning of a nationwide program[6].

In 1937, it was decided that health clinics where to be subsidized using state grants with the ambition of nationwide coverage. Counties and cities outside counties could apply to enter the program by submitting a plan for complete coverage (SFS 745) (Medicinalstyrelsen, 1940). If the plan was approved, only centres and stations specified therein could be eligible for grants. While in the program, and complying with the standards specified there, state grants would cover around half of expenses related to the health centres. The rest would be covered by counties and municipalities[7].

[4]Mjölkdroppen in Swedish.

[5]The first of these centres started in Stockholm in 1901 and began as a milk distribution organisation for poor mothers whom could not breastfeed. As time went by, the operations shifted towards parental support, information on child care, breastfeeding encouragement and infant health monitoring. During the 1920s there where more than 20 cities and towns in Sweden serviced by these infant health centres.

[6]See Bhalotra et al. (2015) for more information on the trial and an evaluation.

[7]Since much of the actual work would be preformed by physicians and nurses already employed

To be eligible for the grant, certain requirements had to be fulfilled. These where related to staff quality as well as the facilities where the receptions where to take place. There also had to be antenatal health screening and the services had to be free of charge (Stenhammar, 2001).

The program implementation was staggered as counties entered the program in different years[8]. Also within counties the expansion was staggered due to limiting constraints in terms of interest, nurses and facilities[9].

The main components of the program included physician visits and home visits by nurses. Also nurses receptions and phone consultations where prevalent in some districts. Information about the new centres and reception hours where advertised at delivery clinics as well as in local newspapers[10].

Home visits where an important part of health centre nurses daily work. Relative utilization and supply of physician visits and home visits by nurses differed a lot between districts and could be due to differences in availability of nurses or geographical circumstances. At receptions (or home visits) the child was examined, parents could ask questions and where informed of proper child care practices accepted at the time. A written home visit instruction to nurses from Stockholm in the 1940s provide some information on what was included.

Nurses where to inspect the child and examine it. Inspect the home and infant sleeping environments. If the family was poor, nurses should inform them about available social services and report parents to the health board if the home environment was unsuitable for the infant. They where to provide advise and encourage breastfeeding also in written form. Encourage studies in child care and child psychology. (Authors translation from Stenhammar, 2001, pp.108)

by counties, the actual cost at the local level would have been much lower (Justus, 1941).

[8]In 1940, five counties where not in the program and the last one entered with an approved plan in 1942. Still, in 1940 one county with an approved plan from 1938 had not started implementing the program.

[9]Local physicians mention in their annual reports that lack of nurses both hinder further expansion in some areas and in one case even led to the dismantle of an existing health centre. The fraction of the population with access to monitoring in 1940, for counties already in the program, varied from $22 - 100\%$. At this time four counties in the program covered less than half of their populations (Justus, 1941).

[10]Receptions where open but a few physicians scheduled mothers and infants to specific dates to avoid overcrowding. Overcrowded receptions where initially common in many places as demand for physician visits, both at the intensive and extensive margins, where difficult to anticipate.

At physician visits, infant where weighted and measured besides being thoroughly examined. Breastfeeding was encouraged and the consensus recommendation at the time was very rigid feeding hours (normally five times per day). Despite this major promotion campaign of breastfeeding, increased maternal employment and better industrial infant formulas was part of an observed decrease in breastfeeding in Sweden at least since 1944 (Zetterström, 2005). Being supervised normally included $2-7$ physician visits as well as $3-12$ home visits during the first year in life (Medicinalstyrelsen, 1947). More contacts could be warranted if the infant seemed unhealthy.

The first year of the program (in 1938) only 13% of all children where supervised (see table 1). Despite the fact that many already existing philanthropic health centres changed management and entered the program. In 1945, 83% of the children born where supervised. Starting in 1942 grants was also provided for surveillance of older children (first $1-2$ years and later $3-7$ years). In 1945 around $40,000$ two to seven year old children where supervised while $111,000$ children below age one were supervised. Treatment intensity was also much lower for older children.

At this time, counties were responsible for hospital care while the central government organised primary care[11]. A local physician (or nurse) would often be the first health care contact if ill and could refer to hospital care if necessary. Patients normally paid a hospital fee of around 25% of total costs (Medicinalstyrelsen, 1947)[12]. Health insurance was not universal at this time. In 1948 around 45% of the population above 15 year of age had health insurance through their employer. For those insured, reimbursement for health care was around 57% of the cost (Socialstyrelsen, 1948). The number of hospital beds, deliveries at clinics and the number of physicians increased during the 1930s and 40s (see table 2).

During the 1930s, few medical treatments existed or where discovered. Only after the second world war would modern antibiotics be available in Sweden and

[11]At the local level, each county was divided into several medical districts and cities where normally a single physician was in charge, aided by nurses and midwives. Health care staff where employed by the state and operated with a pre specified maximum fee. These districts would in most cases include one or more municipalities with a population of around $10,000$ inhabitants. There where also many privately practising physicians available for those with more resources.

[12]In 1937, the average patient paid 45SEK for a 21 days visit (around 150USD in 2017).

later still, treatments for tuberculosis, nutritionally adequate infant formulas and safe routine Caesarean sections (Wennerberg, 2012). One existing class of treatments for infectious diseases were Serum treatments[13]. These treatments existed for pneumonia, diphtheria, scarlet fever, epidemic meningitis, plague and a few others infectious diseases.

One major contemporary innovation was the discovery and later introduction of sulphonamides in 1936[14]. These drugs revolutionized treatments for gonorrhoea, pneumonia and a few other bacterial diseases prior to the discovery of modern antibiotics. Sulphonamide drugs inhibit bacterial growth and thereby allow the immune system to more effectively fight the infection. These drugs where only effective against cocci type bacteria which made them useless in treating tuberculosis, the common influenza and many other viral and bacterial infections (Wennerberg, 2012). Perhaps the most famous sulphonamide drug, highly effective against bacterial pneumonia, was called M&B693 (Sulfapyridine) by the company that produced the drug, May & Baker. It was described in The Lancet in May 1938 and was rapidly embraced by physicians[15]. The clinical effect of Sulfapyridine was similar to or better than the already existing Serum treatments against pneumonia but the cost was $60-30$ times lower (Thunberg, 1940)[16]. Serum treatments required type testing and individual dosage delivered during lengthy hospital stays (Bullowa, 1929), while Sulfapyridine could be prescribed by any physician, where supplied at local pharmacies and could be administered at home (Thunberg, 1940).

[13]By extracting antibodies from an animal or human host, previously exposed to the infection, the disease could in many cases be treated (Bergman, 1944).

[14]The patent for the antibacterial molecules in sulphonamide drugs had expired at this time, making the drug open to exploration and production world wide. As of 1945 there where more than 5000 types of sulphonamides on the market.

[15]In Sweden, Sulfapyridine was produced and distributed since 1939 (Lazuka, 2017). Reports from physicians all over the country in 1939 confirm the availability and effectiveness of Sulfapyridine against pneumonia (Medicinalstyrelsen, 1942). A sharp introduction and wide spread availability is also confirmed in reports from the Swedish armed forces at the time (Kungliga arméförvaltningens sjukvårdsstyrelse, 1942). Mortality from pneumonia (in relation to incidence) in the army, dropped discontinuously with almost 11 percentage points from 1938 to 1939. Before 1939 mortality had been between $6-12\%$, while after 1938 it was only between $0.6-1.3\%$.

[16]Serum treatments would cost around 300SEK per treatment while Sulfapyridine cost only $5-10$SEK.

3 Data Sources, Variables and Design

This paper is built on several data sources. In this section, I detail the data used in the analysis. I further describe the design and make explicit the main assumptions that allow for a causal interpretation of the results. I use mainly on five different data sources. Mortality data from both summary reports sent to Statistics Sweden by parish officials and data from the Swedish Genealogical Society. Also summary population data from Statistics Sweden as well as recently digitized health center data for all stations within the program has been utilized. High quality Swedish register data was used to construct long-term outcomes. In this data, unique personal numbers allow the full population to be linked over several registers.

Around 3500 yearly reports from the local health stations where digitized (the original materials can be found at the Swedish National Archives)[17]. In each report, the uptake area of the health centre is described. Inhabitants in the municipalities specified therein where eligible to attend the station and use its services[18]. The unit of observation used in this paper is the smallest consistent collection of municipalities over time[19].

The main source of mortality data comes from the Swedish Genealogical Society and has been published in The Swedish Death Index (Sveriges släktforskarförbund, 2014). There, members and volunteers have gathered information about each death in Sweden between 1901 and 2013 using official death records complimented by manually collecting information about all other deaths among residents in Sweden (through the project "Namn åt de döda"). The data include all observable deaths in Sweden and the raw number of deaths correspond well to official statements (see

[17] These reports originate from a specific centre or station that was funded by the program. Each report contain information on the operations with respect to infants supervised, pregnant women supervised, vaccinations, blood tests, breastfeeding and other information. The report sheets changed over time such that much of the information therein is not available for the whole time period.

[18] Although compliance was not always perfect, see e.g. (Justus, 1941).

[19] As these uptake areas changed over time as the program expanded and new stations where established, each district (the unit of analysis) can contain more than one centre. If a specific municipality belong to two different health centres in different years, these are merged to one district. In 1945 there where 736 reports but in the end these have been merged to around 400 districts. The drawback being that treatment status for a district does not necessarily mean that the entire district is treated but only part of it.

table 3). It contain information on the exact date of birth, date of death, parish of birth, parish of death (parish of residence) and marital status. I use this data from $1930-1945$ and construct district level mortality rates. Diseased infants are placed in their parish of death and not parish of birth[20].

The second source of mortality data comes from parish death reports to Statistics Sweden. These reports are reprints of church death records and contain individual level death information on date of birth, year of death and cause of death[21]. It contains few missing observations. The coding allow for more than 100 different death causes. To keep cell sizes reasonably large, the data has been divided into four main categories. These mutually exclusive and exhaustive categories are diseases related to infancy and birth, infectious diseases, respiratory diseases and other. Except for the "other" category, these groups correspond to those described in the Statistics Sweden contemporary nomenclature (Statistiska Centralbyrån, 1934)[22]

I use population summaries at the parish level from Statistics Sweden with information on the number of births and population[23]. These data are only available until 1940 so I can not use the data for the whole time period. Most information used is from 1936 which I use to create constant scales for the outcomes. This will introduce some degree of measurement error in the outcome variables that are scaled. But at the same time it is motivated as scaling year to year could introduce bias as the denominator is potentially an outcome.

As long-term outcomes I have constructed three main measures from Swedish register data. The first is educational attainment measured in years. Long-term income is measured as the inflation adjusted average net income between ages $40-45$ (in SEK 1970). These ages are used such that individuals are not too old when

[20]At this time, parish of birth was generally registered as where the birth actually took place. Although officials where to double register births also in their parish of residence, there is an increasing scope for measurement error as institutional births increased during the 1930s and 40s (see table 2).

[21]For a full specification of causes of death see Statistics Sweden annual reports "Dödsorsaker" between the years $1931-1945$. The cause of death coding was changed in 1931 to accommodate a more precise coding. I do not use data from 1930 here as the comparability of the different coding regimes is not perfect. The data span until 1943 which is the last year I can use. Source pictures can be found at the Swedish National Archives

[22]The most common specific causes of death as well as the fraction of each cause within each group is listed in tables 4-7.

[23]See "Summariska folkmängdredogörelser" available at the Swedish National Archives

measured but recognize that the oldest cohorts are 40 years old when first observed in the income data (those born in 1930).

As a measure of long-term health, I use mortality between ages $45-57$. I choose this narrow age group to be able to replicate earlier work (see Hjort et al. (2017)). The measure describe the fraction of all individuals that died within the age span out of valid observations conditional on survival until age 45[24]

Few sample selections are made in this analysis. The main mortality data set, from the Swedish Genealogical Society, has extremely few missing values on crucial variables. This data is, conditional on its original quality, as good as complete[25].

A small sample is lost in the cause of death data. First of all, importing the crude data from imputed excel sheets, some of these could not be matched to a parish (around 2.5% of observations). This is mainly a consequence of human error in the imputation of identification codes for each sheet.

Around 4% of the existing municipalities could not be matched to a district. Many of these are treated at some point in time but not specified in the yearly reports[26]. I omit municipalities with unclear treatment status from the main analysis.

In the register data I have $1,476,937$ unique observations with valid parish of birth born between $1930-1945$. These are individuals born in Sweden that where alive in 1960. Around $13,000$ individuals observed in 1960, i.e. within the register data, can not be matched to income data in 1970. These individuals have most likely died between $1960-1970$ or emigrated out of Sweden. Furthermore, in the education data around $80,000$ individuals can not be matched to a parish of birth[27].

Empirical Specification

In this part of the paper, I detail my estimation strategy. I show the regression equations that I use in the empirical section and describe under which assump-

[24]A valid observation further require here that a parish of birth exist in the data and that the individual was alive in 1960. All data are aggregated to the district level as described above.

[25]For a comparison to official mortality statements, see table 3

[26]It can be noted in some reports that the municipality defined as the uptake area, often a small city, has a much larger population size than the city alone. Some of these municipalities could also have been never treated during the time period.

[27]Education is measured in 1990, the earliest year when I have access to this educational attainment measure. The attrition is mainly due to mortality prior to this year.

tions they produce causal estimates. I further motivate the choices I make when estimating the models in terms of both identification and inference.

The experimental benchmark implies randomization to either access to the program, or no access. In that case, a simple OLS regression would consistently estimate the causal effect of the program. Without actual randomization, stronger assumptions have to be invoked.

As the treatment vary at level higher than the individual, aggregated data at the district level is used. Districts that get access early might be different to begin with than those that enter the program later. More educated mothers can be better informed about the importance of regular well visits. Mothers of unhealthy infants can on the other hand demand more well-visits. Including district fixed effects will by construction align the pre-treatment levels of an outcome between early and late policy adopters. In this specification the districts are hence allowed to differ on characteristics as long as they are not time varying. The fixed effects regression model can be described using dummy variables as following.

$$\text{Outcome}_{dt} = \beta \cdot \text{Health Centre}_{dt} + \sum_{d=1}^{D} \pi_d + \sum_{t=1}^{T} \rho_t + v_{dt} \tag{1}$$

Where the outcome is observed at the district (d) and year (t) level. By also including year fixed effects, common shocks in time can be accounted for. Estimating β can be interpreted as the intent-to-treat effect of having access to (but not necessarily attending) Health Centres. Although interest primarily lies in evaluating if Health Centre attendance affected health, the intent-to-treat parameter is in some sense more policy relevant as forcing compliance is often neither feasible nor desirable.

The model is now a difference-in-differences in specification but non standard in that the treatment is staggered. As in any panel data setting, the pre-treatment trends are a potential source of bias. Identification relies on parallel trends prior to treatment. If districts that introduced the program early on have different growth in the outcome already before treatment, β will be biased.

To assess the parallel trends assumption when treatment is staggered I use an

event study approach. This method is in essence similar to a fully flexible difference-in-difference approach when treatment is fixed in time (Mora and Reggio, 2012). But here I restrict the fully flexible form k time periods around each groups treatment initiation. For each group, dummy variables are defined for each year k time periods before, up to k time periods after treatment started (treatment start at $k = 0$).

$$\text{Outcome}_{dt} = \sum_{i=-k}^{k} \gamma^i \lambda_{di} + \sum_{d=1}^{D} \pi_d + \sum_{t=1}^{T} \rho_t + v_{dt} \tag{2}$$

Equation 2 is similar to lagging and leading treatment k periods (years below and above k and $-k$ are collected in $k, -k$). Investigating the pattern of the estimated γ^i parameters, I assess if trends seem to be parallel prior to program initiation and if there is a trend shift corresponding in time with treatment.

A common way to redefine the parallel trends assumption is to introduce unit specific time trends. Using linear time trends, the identifying assumption of parallel trends can now be defined as parallel growth (or parallel trends in first differences) (Mora and Reggio, 2012). Including linear trend variables in equation 1 with time fixed effects, the model can be specified as:

$$\text{Outcome}_{dt} = \beta \cdot \text{Health Centre}_{dt} + \sum_{d=1}^{D} \pi_d + \sum_{t=1}^{T} \rho_t + \sum_{d=1}^{D} \text{time} \cdot \pi_d + v_{dt} \tag{3}$$

If the pre program outcome trends are parallel in growth (e.g. linear), the program effect can now be estimated consistently. Also higher order trend polynomials can be tested to assess the credibility of the parallel growth identifying assumption (requiring different assumptions of parallel trends). Recent critique of the use of time trend variables has emerged, atleast in a setting with treatment effects that are growing with time (Meer and West, 2015), or when the outcome is behaving in non-standard ways in response to treatment (Wolfers, 2006).

While it is not likely that the outcomes would behave very odd in response to treatment, clearly it is more likely that the health centre operations could be

more effective over time. Linear time trends could then lead to an attenuation bias according to Meer and West (2015).

Some districts include, as previously mentioned, several health centres with different program start years. The district is still assumed treated when the first centre enter, leading to a possible gradual effect. Gaining experiences from this new way of screening infants as well as increased take up over time could also strengthen these dynamics. In many cases, districts started operations late in the year (after summer) making large health gains less likely to be achieved during the year of initiation. To accommodate this empirically, I let the treatment start one year after the first contact with the program.

$$\text{Outcome}_{dt} = \beta \cdot \text{Health Centre}_{d(t-1)} + \sum_{d=1}^{D} \pi_d + \sum_{t=1}^{T} \rho_t + v_{dt} \tag{4}$$

As this paper address multiple outcomes, I use the event study approach for my main outcomes and otherwise include linear time trends in each specification. When pre-treatment trends does not seem to be stable, specifications with district specific linear time trends are likely more reliable and will be given higher weight.

An other way to parametrise a pattern of treatment effect growth is to include a trending treatment measure (Wooldridge, 2010). A linearly growing treatment effect can then be described in the model in the following way.

$$\delta \cdot \text{Health Centre}_{dt} + \gamma \cdot \text{Health Centre}_{dt} \cdot \text{time} \tag{5}$$

Here (γ) captures the (linear) change in effect over time conditional on the average reduced form effect over time (δ). These treatment variables will be used in the main specification to check if the treatment effect can be well approximated as linearly increasing over time.

Standard errors will normally be clustered at the district level to take into account the serial correlation of residuals over time often present in panel data settings (Cameron and Miller, 2015)[28]

[28] As counties entering the program was a pre-condition for a district to enter, the county level is also a candidate level of clustering. As there are only 25 counties in this data and as clustering on county does not have a very large impact on standard errors (up to 20% increase and even

Channels

There are several ways that the program can affect infant health. Below, I explicitly discuss a few ways related to the infant health part of the program. For infants, the program consisted of physician visits and home visits by nurses.

Access to health care was improved. Both as the program was free of charge and as receptions where decentralised at so many locations. If an infant was ill and a reception available, the cost of attending was very low. Although sick-visits were not the intention of the program, it was not uncommon.

Regular monitoring can enable physicians or nurses to early detect and treat infants that where sick. Observing infants regularly, and also in the home environment, means that health care staff more often can detect more common illnesses early on and provide treatment and advise. This was particularly important among parents who where less likely to seek formal care.

General prevention through vaccinations, information and breastfeeding support can improve the infants nutritional and hygiene status. It can also enable parents to make better decisions regarding child care and health care contacts. Vaccinations where though not supplied by all centres and where also available through other channels.

It is not obvious that monitoring, general prevention or access to health care must be very important in improving infant health. Detection of disease is only important if a treatment is available. At this time, not all diseases could effectively be treated with drugs. Information is productive if it is new, health promoting and complied with. Access to health care (when ill) could even have deteriorated as local physician resources where redirected from health care to preventive care. It is thus an empirical question if and how the program improved infant health.

decreases for some outcomes), I will use the district level and show how clustering at the county level affects precision for the main outcome.

4 Program Effects on Direct and Long Run Outcomes

Below I present the descriptive, graphical and regression results. I start by describing the main results using infant mortality as the outcome. Following this I use cause of death data to investigate the mechanism and discuss the findings. After that, I present results for the long-term outcomes. I then challenge the robustness of these findings in several ways and relate the effect sizes to other literature.

In table 1, the program expansion is described at the national level. Coverage increased from 26% to 97% between $1938-1945$. Most of the increase occurred until 1943 when already 94% had access and the last county had entered the program. Although access was high, take up was lower and increased more gradual from 13% in 1938 to 83% in 1945. The antenatal monitoring program had much lower take up. In 1945 only 58% of becoming mothers attended the services at least once[29]. In 1945 infants in the program had on average 8 contacts with the program (see table 8), four home visits by nurses, and three physician reception visits (as well as one nurse reception visit) during their first year of life. They where serviced by 1233 stations and centres all around Sweden.

In figure 1, I show event study results using the full sample for the base line specification (see equation 1). Both for the infant mortality rate and for the log infant mortality rate (using mortality between ages $8-365$ days). The timing of the program is confirmed and the pre program trends are reassuringly stable and close to zero in both specifications. While most of the post-treatment estimates are negative and significant, none of the parameter estimates pre-treatment are close. It is also clear that the first year of program initiation was not very productive further motivating the use of the reduced form lag as the treatment indicator.

Using regression models, I continue investigating how the program affected infant mortality. Table 10 show results from several different specifications using the Infant mortality rate as outcome. This measure is defined as the number of infants

[29]Some physicians note in the yearly reports that many mothers only attended the antenatal care program to receive documentation of their pregnancy. This was required to apply for a benefit program aimed at poor mothers giving birth and required that a physician signed it.

below age one that died each year divided by the number of births in 1936. In the first column, the estimated program effect comes from a standard difference-in-differences model (base line). The estimated effect, -0.0035, can be translated to around 7% of the pre program infant mortality mean. In columns 2 to 5, district specific linear and quadratic time trends are introduced in the model. In these models, I further use weighted least squares, include a larger sample and change the level of clustering[30]. The estimated effects are very stable over all specifications and with similar precision.

To deepen our understanding on how the program affected infant health, cause specific mortality data has been used. I have created four categories which are described further above and in tables 4-7. These are "infectious diseases", "infancy/birth", "respiratory diseases" and finally "other" which include all other deaths not categorized in the other categories[31].

In table 11, I show regression output using cause of death mortality rates as outcomes. Reassuringly, using the full scope of this data I find similar results on infant mortality as with the other data source. Even though infant mortality here is defined between ages $0-2$ years. Looking at the specific causes, most of the program effect on mortality comes from the category "respiratory diseases". Infectious diseases enter with a positive point estimate, although indistinguishable from zero. Some of the total effect comes from the "other" category which mainly include deaths of unknown causes.

Finding a null effect on infectious diseases does not lend support for prenatal health improvements or that parents where taking better care of their children as the main mechanism. Breastfeeding support, child care advise, vaccinations and most other interventions that the program supplied would have affected mortality

[30]In column 3, I use weighted least squares with births in 1936 as weights and in column 4, I include parishes that could not be matched to a district (the parishes where aggregated by county to 16 districts). Too show the importance of clustering at different levels, in column 5, I use the county level for clustering standard errors.

[31]Infectious diseases include TBC, the flu, the plague and enteritis among others. Respiratory diseases include mainly pneumonia. Infancy diseases include preterm birth, general weakness, deformities and deaths occurring during or shortly after birth. Many of the others category are of unknown cause, but also accidents and violent deaths are included there. Except for the other category, these follow the categories set of by Statistics Sweden in their cause of death nomenclature (Statistiska Centralbyrån, 1934).

also from infectious diseases. Naturally, these mechanisms can operate through the program but not at a margin detectable while investigating infant mortality.

If general health among infants improved, and given that boys are less resilient than girls, we would expect to see larger effects on boys than girls. In table 12 I show that boys and girls where equally affect. Compared to pre program mortality mean, infant girls even responded more. This finding further strengthen the claim that general health improvements like nutrition, where not likely the main mechanism in this setting.

The effects found on long-term outcomes do not support large general health improvements due to the program. Still, access to medication against infectious diseases in childhood could have beneficial long-term effects on those exposed (Lazuka, 2017; Bhalotra and Venkataramani, 2011). It is thus possible that better access to treatments for pneumonia in the present setting could be beneficial also in adulthood.

There are at least three major explanations consistent with the data. Either the program supplied very effective information targeted at pneumonia prevention or treatment, the program reduced the cost of pneumonia health care or the program enabled early detection and diagnosis of pneumonia among infants through regular monitoring. The selectivity of the mechanism in terms of cause of death could be due to that there existed effective treatments for pneumonia in contrast to most other viral and bacterial infections.

A pure information effect does not seem likely as there appears to be no spill over effects to older children (see tables 14 and 16). If a crucial piece of information was supplied that dramatically could reduce pneumonia mortality among infants, this information should also have been used on older children such as siblings, neighbours or other relatives in the treated areas. Previous results suggest that there where no spill over effects to other age categories.

The cost of health care and monitoring decreased in many ways due to the program. Infants could be taken to see a physician free of charge in (almost) any condition. Early signs of serious diseases could then be detected. The distance to travel to see the physician decreased as the program was highly decentralised.

This could have been extremely important for infants as they are more sensitive to serious disease.

The newly discovered Sulfapyridine drug made pneumonia treatment simpler and dramatically decreased the cost of treatment. Since Sulfapyridine was available in all of Sweden, the program could have generated a link between pneumonia sick infants and the treatment, not available outside the program. Improving the matching between sick infants and treatment. Either by relaxing budget constraints, by regular monitoring or both.

I continue to investigate if the infant health program had any effect beyond the direct impact on infant mortality. Three outcomes are used that entail information about labour market success and health. These estimates can provide more information about the total value of the program[32].

Event study graphs (see figure 2) suggest that pre-treatment trends are parallel for educational attainment but not convincingly so for the other outcomes. In table 13, regression results are presented for each outcome over four specifications. A standard difference-indifferences model, a model including district specific linear time trends, a weighted model using cell size and a model including district specific quadratic time trends. The estimated program effects are positive and generally small. The program effect on educational attainment is between 0.09 to 0.023 and insignificant in all specifications[33]. Even scaled by uptake, this is a very small effect. For income and mid-age mortality the event study figures motivate inclusion of linear time trends. Log Income is marginally significant, even with linear time trends, with an estimate of around 1.3%. This estimate is further attenuated using weights and including higher order time trend polynomials. Mortality is always positive and insignificant. In some specifications, the estimated effect is sizeable compared to the mean (up to a 10% increase). These estimates are likely lower bounds of their true effects. The marginal survivors in treated districts most likely

[32]The outcomes are average net discounted income between ages $40-45$, educational attainment measured in seven levels and mid-age mortality measured between ages $45-57$. This mortality measure is scaled by the cell size meaning all individuals observed in the register data born in the time period conditional on survival until age 45 for comparability between cohorts. To be in the register data you are required to be alive in 1960.

[33]I also try using education in seven levels to retain as many observations as possible with very similar results.

have poorer health and weaker labour market prospects than the average infant.

Robustness

Below I address some of the most pressing issues relating to the internal validity of the results. I investigate if fertility was related to, or affected by, the program as well as if miss-measured parish of birth bias the long-term outcomes. Also if the effect was generated solely by the diffusion of Sulfapyridine, any other health shock or the rapid expansion of hospital deliveries.

The program effect could still be an artefact of some positive health shock that is sufficiently correlated with the gradual program implementation. For example the introduction of Sulfapyridine in 1939. To address this issue, I create mortality outcomes that are not affected by the program (see table 14). The first is mortality in the first week of life ($0-7$ days). This measure could naturally be affected by the antenatal care program so it can not be viewed as a strict placebo test. Still, in the first week of life, few children would be directly exposed to the program[34].

Consistent with the previous line of reasoning, there is no program effect on mortality for infants in their first week of life. Neither for children between age one and seven or for mortality above age seven (see columns $1, 3$ and 4 of table 14). However, the target group of the program, infants younger than one year but older than one week, shows a 10% decrease in mortality. These results suggest that the estimated effect on infant mortality is actually generated by the program. The introduction of some medical technology or environmental change (e.g. change in average income) would benefit also others than infants at this particular age. These regression results are also supported by corresponding event study graphs (see figure 3).

To parametrize a gradually increasing effect, as previously described, the model in column 6 of table 14 include the reduced form treatment indicator and a linear (in time) treatment variable (see equation 5). The model support the assumption

[34]In 1940 some 75% of women gave birth at a clinic and stayed there on average for more than ten days (Medicinalstyrelsen, 1943). Information provided by the yearly health centre reports shows that only around a third of infants where listed prior to one month of age. It is hence unlikely that this group would benefit very much directly from the infant well-visit program.

of a gradual increasing effect, although precision is lower in this specification. This result is coherent with the event study graph.

With this information in mind, the following tables (where applicable) the outcome will be infant mortality between $8 - 365$ days. Mortality in the first week of life only introduce noise in the estimates.

In table 16 these results are strengthened by models similar to triple differences. The outcomes are now mortality differences between $8 - 365$ days and the other mortality rates, within the same district and year (here also including mortality in the second year of life). The estimates are, as expected, extremely similar to the previous estimated effects.

As it is likely that general improvements in the disease environment is most beneficial for infants, using other age categories as placebos as above might be insufficient. I there for use mortality in respiratory diseases among the elderly (+65 years) as a placebo outcome, to see if I am only estimating the effect of Sulfapyridine diffusion. In this group, mortality from respiratory diseases account for 10% of all cases. In 15, I show that mortality from respiratory diseases among the very old was not correlated with the program, either in relative or absolute terms.

At the end of the second world war, starting already in 1942, fertility increased dramatically in Sweden[35]. These changes in fertility could create compositional changes in birth characteristics that potentially could be correlated with the program. As I scale outcomes by a constant level of births, this increase could generate spurious regression results. However, as the results in table 17 show, the estimated effect on infant mortality is not affected in any meaningful way by omitting the last few years of the sample time period. The striking homogeneity in the effect over time also suggest that the effect comes in fast and is very similar between different districts entering at different times.

Using the long-term outcomes and removing the last few years, the results are not as stable (see table 18). Education is very volatile while log income and mortality are somewhat more stable. The program effect on educational attainment

[35]From around 100′000 births in 1941, there where almost 140′000 births in 1945 (Statistiska Centralbyrån, 1945; Medicinalstyrelsen, 1947).

seem to be generated in the very last few years of the analysis. This is a time when most districts where already treated and I have very little variation in the treatment variable. Higher incomes and increased mid-age mortality could still be actual effects of the program. However, precision is generally too low to claim any long-term gain or loss with confidence.

There are at least two important ways that the program effect could be biased. First, it is likely that fertility responded to the program. If fertility increased, this could have affected infant mortality in different ways. Both as I scale outcomes by a constant and through compositional changes. Improved in-utero health due to the antenatal part of the program could also increase the number of children born alive. Secondly, there was a simultaneous program to increase hospital delivery care in Sweden. If this program correlated with the infant well-visit program or interacted with the program, the estimated effects could be biased.

To address these issues, I have collected county level data as many measures are not available at the district level. In table 19, I show estimates from regressions at both the district and county level. At the county level the variable describing treatment is defined as the fraction of districts in each county with access to a health centre. This measure most tightly mimic the reduced form at the district level as implementation within counties was also staggered.

In the first column, I show a model at the district level using data on births up until 1940. Although the treatment time period is short, the estimated effect is small, insignificant and negative. Suggesting that births did not increase as a result of the program. A similar regression at the county level, for the same time period, produces very similar results (see column 2). Also when I use the full time period at the county level, in column 3, I find a negative, insignificant point estimate. Together with the results on mortality in the first week of life (see table 14), there is no evidence of any fertility effects or compositional changes among mothers. Most likely the antenatal program was not very important in this setting.

The baseline county level program effect on infant mortality is shown in column 4 of table 19, where I replicate the district level estimates with great precision. The next column show "fraction of home births" as the outcome. At the county

level, this measure describe the fraction of births delivered by local midwives, the alternative to having a delivery at a clinic or hospital. I find no evidence that the program affected the number of infants delivered at hospitals or birth clinics as shown in column 5. Finally, I run the main specification with the potentially confounding variables as controls. They both affect the infant mortality rate as expected, but do not affect the point estimate of the program parameter in any meaningful way.

During this time period, registration of parish of birth was done both in the actual parish where the birth took place and also at the parish of residence. This allows for substantial measurement error in the outcome variables, introducing systematic bias if related to the program expansion. For example if the antenatal part of the program effectively induced becoming mothers to deliver at clinics, this error could be systematic. For infant mortality, I use parish of death which is not contaminated by this issue but for long-term outcomes it could be problematic.

I address this issue in two ways. First, I investigate if the number of observations in each long-term district-year cell is related to the treatment indicator (see table 20). If the cell size change much when entering the program, this could indicate that a systematic bias is present. It is clear that when a linear time trend is included, the program effect on cell size is small, negative and insignificant for both samples. As cell sizes are trending upwards already before treatment, due to increased cohort sizes, inclusion of linear time trends are warranted.

As a second approach, I replicate the results of table 13 at the county level. At this level, the measurement error is averaged out when collapsing the data. Counties where to a great deal responsible for health and delivery care and few deliveries would take place outside an individuals county of residence (see figure 4). These results are shown in table 21 where the district level results are confirmed. The suggested measurement error does not seem to be systematic but could still affect precision of the estimates.

Neither fertility nor hospital births seem to have affected, or been correlated with the rollout of the health centre program. Potential measurement error from miss-coded parish of birth does not seem to be systematic but could affect the precision

with which the parameters are estimated. These findings strengthen the claim that the program effects described above are in fact generated by infant well-visits (and home visits). Still, it is not clear how the program improved infant health. In the next section I discuss some potential mechanisms.

Comparison to the Existing Literature

The direct effect of the program on infant mortality found in this paper is very similar to that found in Wüst (2012). The intensity of these two programs where very similar although the Swedish program included also physician visits. The most similar program is described in Bhalotra et al. (2015), analysing the trial leading up to the Swedish national implementation. There, the reduction in infant mortality was around two times as great as found here, although at average exposure to treatment. The fairly similar estimates suggest that infant mortality can actually respond to preventive infant health interventions in a meaningful way.

Comparing the estimated long-term effect of an intensive preventive health program for infants to the literature there are some interesting differences. Bhalotra et al. (2015) find that mid-age mortality decreased. This could suggest a long-term health benefit of early life exposure. In this paper, I can not confirm that mid-age health improved measured as mortality between ages $45-57$[36].

Bütikofer et al. (2015) found that a similar Norwegian program increased educational attainment by 0.15 years and mid-age income by $1.6-2.3\%$ at the mean of income. The estimates for income are not very far from those found in this paper although I have less precision[37] although I find much smaller effects on educational attainment. I find effects of between $0.01-0.023$ years for the reduced form. Around one tenth the size of the effects found in Bütikofer et al. (2015) and far from significant. Further the authors find large health gains in adulthood. I can not replicate the health measures in that paper due to lack of data but do not find any effect on mid-age mortality.

[36] Other mortality measures have been tested such as mortality between ages $31-65$ and survival above age 65 but with very similar results.

[37] The stimates in this paper for income are between $1-1.3\%$ in the preferred specifications.

The mid-age mortality measure used in this paper is the main outcome in Hjort et al. (2017). They investigate long-term effects of the Danish home visiting program and find that mid-age mortality decreased by 0.3% points. This paper do not find any evidence supporting any negative change in mid-age mortality but if anything, I find positive estimated effects on mortality. Hjort et al. (2017) also find mixed effects of the program on labour market success which is not consistent with the findings of Bütikofer et al. (2015) and also in stark contrast to the results found here. The different results here suggest that we should be careful in drawing firm conclusions on the long run importance of early life preventive health interventions.

There are atleast three explanations to the different results found here and in the other literature. First of all, I use a setting with higher internal validity. The program was phased in fast in a less volatile context. Secondly, the Swedish program provides higher external validity. My estimates are based on almost the full population of Sweden and not only a sample of treated municipalities. Finally, the findings of this paper suggest that the well-visit program improved matching between sick infants and existing medical technologies. Later on, as new antibiotics where developed, health centers could have improved access to these medications. The longer time horizons of Bütikofer et al. (2015) and Hjort et al. (2017) could mean that new types of antibiotics introduced after the second world war is important for their results. These antibiotics where innovations even more important than Sulfa drugs.

5 Conclusions

In this paper I showed that the Swedish universal antenatal and infant well-visit program meaningfully reduced infant mortality. I further found no significant effects on outcomes measured in mid age.

Most of the reduction in infant mortality caused by the program came from deaths in respiratory diseases. There is no apparent evidence that the antenatal component of the program affected early infant health or fertility. Either by behavioural responses to the program of through selective survival if the program had

improved in-utero health.

I have argued that general infant health was not the main way with which the program improved health and that information alone is an unlikely candidate explanation for the program effect. Instead I suggest that the program increased early detection of pneumonia, a treatable disease, both by reducing parental cost of care and also by regular monitoring of infants. The newly discovered Sulfpyridine drug could have been important in enhancing the program effect as it allowed local physicians to treat pneumonia patients without referring them to hospital care. Early detection could then have been more important as the treatment was cheap, effective, available and could be administered at home.

There is plenty of support for a direct effect of infant well visits on infant mortality. The evidence supporting long-term gains is less convincing. This paper replicated earlier findings using a larger population and a more compelling setting. I found that the program affected infant health through a rather specific mechanism. At later points in time, when new medical technologies where available, the long run health effects could be very different. Potentially explaining the different findings in the literature.

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Appendix A: Tables

Table 1: Program Take up and Expansion over Time

Year	Coverage (%)	Mother supervised (%)	Infants supervised (%)	Infants Supervised | Treated (%)
1938	26	6	13	50
1939	55	20	30	55
1940	68	26	41	60
1941	78	37	54	69
1942	86	46	63	73
1943	94	50	75	80
1944	97	55	79	81
1945	97	58	83	86

Note: Source: Historisk statistisk för Sverige. Statistiska översiktstabeller. Stockholm 1960

Table 2: Health Care in Sweden during 30s and 40s

Year	1930	1935	1940	1945
Working Physicians	2239	2639	3024	4089
Working Nurses	2835	3870	5757	6751
Hospitals	92	92	96	101
Hospital Beds	15800	16740	19412	21343
Hospital Patients	223451	279874	318483	414103
Birth Clinics	11	11	10	8
Birth Clinics Beds	1065	1291	2517	3190
Birth Clinics Patients	29995	40644	68602	124415
Children's Hospitals	19	20	18	13
Children's Hospital Beds	2083	2097	1991	1649
Children's Hospital Patients	12315	12237	12785	14504

Note: Source: Statistisk årsbok för Sverige 1948. Stockholm 1948. Specific delivery clinics where few but numbers also include deliveries at Hospitals with delivery clinics. These where 85 in 1945.

Table 3: Consistency of Data: Swedish Genealogical Society (Swedish Death Index)

Source/Year	1930	1935	1940	1945
Official[1]	71790	72813	72748	71901
Data	72070	72916	72818	70879

Note: (1) Dödsorsaker i Sverige SCB 1930 – 1945. Total mortality counts by source.

Table 4: Mortality Counts 1931-1937, 0-2 years: Infancy Diseases

Cause	Name	Code	Deaths	Fraction of Total
Birth defects	*Vitia primae conformationis*	0001	1622	0.11
General weakness at birth	*Debilitas Congenita*	0100	8594	0.56
Preterm delivery	*Partus praematurus*	0200	1663	0.11
Other infancy causes	*Aliae causae mortis neonatorum*	0300	3486	0.23

Note: Source: Own calculations of data from official transcripts to Statistics Sweden. For interpretation see: Dödsorsaker 1931, SCB (1934)

Table 5: Mortality Counts 1931-1937, 0-2 years: Infectious Diseases

Cause	Name	Code	Deaths	Fraction of Total
Enteritis	*Gastro-enteritis acuta infectiosa*	1150	1395	0.29
Whooping cough	*Pertussis*	1100	768	0.16
Blood poisoning	*(Pyo)Septichaemia*	1220	510	0.11
Influenza	*Influeza sine aegrotatione pulmonis*	1121	449	0.09
TB - meningitis	*Tubercolosis meningum cerebri*	1410	378	0.08

Note: Source: Own calculations of data from official transcripts to Statistics Sweden. For interpretation see: Dödsorsaker 1931, SCB (1934)

Table 6: Mortality Counts 1931-1937, 0-2 years: Respiratory Diseases

Cause	Name	Code	Deaths	Fraction of Total
Bronchitis	*Bronchitis acuta*	3500	1018	0.16
Pneumonia with brochitis	*Bronchopneumonia acuta*	3520	3666	0.58
Pneumonia	*Pneumonia acuta lobaris*	3530	1423	0.23

Note: Source: Own calculations of data from official transcripts to Statistics Sweden. For interpretation see: Dödsorsaker 1931, SCB (1934)

Table 7: Mortality Counts 1931-1937, 0-2 years: "Other"

Cause	Name	Code	Deaths	Fraction of Total
Unspecified	*Alii casus*	9010	864	0.17
Unspecified nutrition related	*Alii morbi nutritionis*	2460	690	0.14
Convulsions (epilepsy)	*Eclampsia infantilis*	2640	323	0.07
Brain abscess or meningitis	*Abscessus cerebri Meningitis purulenta*	2610	319	0.06

Note: Source: Own calculations of data from official transcripts to Statistics Sweden. For interpretation see: Dödsorsaker 1931, SCB (1934)

Table 8: Program Utilization in 1945

		Average number of:			
Infants Born	Infants Supervised	Physician visits	Nurse visits	Home visits	Stations
133793	110802	3	0.9	4.2	1233

Note: Source: Allmän hälso och sjukvård 1945. Stockholm 1946

Table 9: Descriptive Statistics

	Mean	Sd	Min	Max	Observations
Panel A. Main Outcomes					
Infant Mortality Rate	0.047	0.030	0.000	0.667	6654
Infant Mortality Rate 8-365 days	0.027	0.022	0.000	0.333	6654
Educational Level	2.146	0.395	1.000	6.000	6481
Average Income ages 40-45	27246.632	3248.558	5638.707	56823.980	6479
Mortality rate 45-57 yrs	0.047	0.035	0.000	1.000	6480
Panel B: Cause of Death Outcomes					
Mortality Rate 0-2: All causes	0.051	0.030	0.004	0.667	5039
Mortality Rate 0-2: Infancy	0.027	0.021	0.000	0.667	5039
Mortality Rate 0-2: Infectious	0.007	0.011	0.000	0.333	5039
Mortality Rate 0-2: Respiratory	0.010	0.013	0.000	0.333	5039
Mortality Rate 0-2: Other	0.007	0.011	0.000	0.333	5039
Panel C: County Level Variables					
Fraction Home Deliveries	0.53	0.29	0.01	1.09	400
Births	4002.83	1961.20	850.00	14676.00	400
Panel D: Placebo Outcomes					
Infant Mortality Rate 1-7 days	0.020	0.019	0.000	0.667	6654
Mortality Rate 1-2 years	0.006	0.010	0.000	0.333	6654
Mortality Rate 1-7 years	0.015	0.015	0.000	0.333	6654
Mortality Rate 8-99 years	0.011	0.002	0.001	0.031	6654
Panel E: Cell sizes					
Cell Size Education	212	543	1	13314	6481
Cell Size Income	220	575	1	14400	6481

Note: Full sample described above.

Table 10: FE Reduced Form Effect on Infant Mortality Rate (0-365 days)

	Infant Mortality Rate					
	OLS	OLS	WLS	OLS	OLS	OLS
	(1)	(2)	(3)	(4)	(5)	(6)
Reduced Form Lag	-0.0035**	-0.0031**	-0.0025**	-0.0030*	-0.0031**	-0.0038**
	(0.0016)	(0.0015)	(0.0012)	(0.0016)	(0.0012)	(0.0017)
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Linear Time Trends	No	Yes	Yes	Yes	Yes	Yes
Quadratic Time Trends	No	No	No	No	No	Yes
Outcome mean	0.051	0.051	0.051	0.051	0.051	0.051
Clusters	400	400	400	416	25	400
R squared	0.043	0.168	0.253	0.162	0.168	0.233
Observations	5998	5998	5998	6238	5998	5998

Note: Each column describe a separate fixed effects regression. The first column is a standard difference-in-difference model (see equation 1). Column 2 also include district specific linear time trends. The third column (3) is WLS where the weights are births in 1936. Column 4 include districts with uncertain treatment status that are aggregated by county as never treated. In column 5, standard errors are clustered at the county level instead of the district level (all other models). In the last column (6) quadratic time trends are included.

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

Table 11: FE Reduced Form Effect on Infant Mortality (0-2 years): Cause of Death

Cause of Death	All	Infancy/Birth	Infectious	Respiratory	Other
	(1)	(2)	(3)	(4)	(5)
Reduced Form Lag	-0.0032*	-0.0002	0.0006	-0.0022**	-0.0014*
	(0.0017)	(0.0014)	(0.0010)	(0.0011)	(0.0007)
Year FE	Yes	Yes	Yes	Yes	Yes
Linear Time Trends	Yes	Yes	Yes	Yes	Yes
Outcome mean	0.051	0.027	0.007	0.010	0.007
Clusters	400	400	400	400	400
R squared	0.226	0.125	0.142	0.102	0.171
Observations	4998	4998	4998	4998	4998

Note: Each column describe a different fixed effects regression model. The cause of death data stretch only $1931 - 1943$. The first column shows the estimated effect using all data. Columns $2 - 5$ decompose the infant mortality response by cause of death. The categories are Infancy, Infectious, Respiratory and Other. Infancy causes of death include prematurity, general weakness at birth and birth deformities among other. Infectious diseases as cause of death is mainly enteritis, the flu and measles. Respiratory causes include pneumonia and bronchitis. Other causes are mainly unknown but also include some cases of accidents, violence, epilepsy and meningitis. Further information on the cause of death data can be found in the text, in tables 4-7 or at Statistiska Centralbyrån (1934). All models include district specific linear time trends as well as year and district fixed effects. Standard errors are clustered at the district level.

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

Table 12: FE Reduced Form Effect on Mortality: Gender Heterogeneity

	Boys			Girls		
Mortality at age:	$0 - 7$ days	$8 - 365$ days	$1 - 2$ years	$0 - 7$ days	$8 - 365$ days	$1 - 2$ years
	(1)	(2)	(3)	(4)	(5)	(6)
Reduced Form Lag	-0.0001	-0.0031**	-0.0009	0.0002	-0.0032*	0.0006
	(0.0017)	(0.0016)	(0.0007)	(0.0016)	(0.0016)	(0.0011)
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Linear Time Trends	Yes	Yes	Yes	Yes	Yes	Yes
Outcome mean	0.023	0.030	0.006	0.017	0.024	0.005
Clusters	400	400	400	400	400	400
R squared	0	0	0	0	0	0
Observations	5998	5998	5998	5998	5998	5998

Note: Each column describe a different fixed effects regression model. The outcomes are mortality rates between $0 - 7$ days, $8 - 365$ days and $1 - 2$ years separate for girls and boys. All models include district specific linear time trends as well as year and district fixed effects. Standard errors are clustered at the district level.

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

Table 13: Reduced Form Effect on Long-Term Outcomes

	OLS	OLS	WLS	OLS
Outcome:	(1)	(2)	(3)	(4)
Years of Education	0.0225	0.0123	0.0126	0.0094
	(0.0308)	(0.0331)	(0.0142)	(0.0345)
Log income 40-45 yrs	0.0235***	0.0130*	0.0008	0.0104
	(0.0081)	(0.0078)	(0.0032)	(0.0083)
Mortality 45-57 yrs	0.0016	0.0045	0.0001	0.0048
	(0.0028)	(0.0039)	(0.0010)	(0.0040)
Year FE	Yes	Yes	Yes	Yes
Linear Time Trends	No	Yes	Yes	Yes
Quadratic Time Trends	No	No	No	Yes

Note: Each cell describe a different fixed effects regression model. The first row use average educational attainment measured in years as outcome. The second row use log average net discounted income between ages $40-45$ as outcome. The last row use mortality between ages $45-57$ as outcomes. All measures are averaged using valid observations for each outcome conditional on being observed at time of measure. All regressions show standard errors clustered at the district level. The third column use weighted least squares where the weights are the average cell size in each district.

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

Table 14: FE Reduced Form Effect on Mortality: Different Ages

Mortality during	$0-7$ Days	$8-365$ Days	$1-7$ Years	$8-99$ Years	$8-365$ Days
	(1)	(2)	(3)	(4)	(5)
Reduced Form Lag	-0.0002	-0.0029***	0.0002	-0.0000	
	(0.0012)	(0.0011)	(0.0009)	(0.0001)	
Reduced Form					-0.0008
					(0.0011)
Years in Program					-0.0013*
					(0.0007)
Year FE	Yes	Yes	Yes	Yes	Yes
Linear Time Trends	Yes	Yes	Yes	Yes	Yes
Outcome mean	0.020	0.031	0.018	0.011	0.031
Clusters	400	400	400	400	400
R squared	0.100	0.186	0.126	0.273	0.199
Observations	5998	5998	5998	5998	6398

Note: Each column describe a different fixed effects regression model. The outcomes are mortality rates between ages $0-7$ days (first column), $8-365$ days (second column), $1-7$ years (third column) and $8-99$ years (fourth column). The fifth column also use $8-365$ days mortality rate but instead of the reduced form lag, it follows the parametrization in equation 5. All models include district specific linear time trends as well as year and district fixed effects. Standard errors are clustered at the district level.

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

Table 15: Sulfa Diffusion: +65 Mortality in Respiratory Diseases

Outcome	Relative Mortality		Mortality Rate	
	(1)	(2)	(3)	(4)
Reduced Form Lag	0.0028	0.0041	0.0000	0.0000
	(0.0048)	(0.0059)	(0.0000)	(0.0000)
Year FE	Yes	Yes	Yes	Yes
Linear Time Trends	No	Yes	No	Yes
Outcome mean	0.114	0.114	0.001	0.001
Clusters	402	402	400	400
R squared	0.052	0.161	0.070	0.164
Observations	5202	5202	5176	5176

Note: Each column describe a separate fixed effects regression. Two outcomes are used; the first two columns use relative mortality in respiratory diseases. There I scale the number of respiratory cases by all non-respiratory deaths for those above 65 years of age. In the last two columns I scale mortality in respiratory diseases by population in 1936.

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

Table 16: FE Reduced Form Effect on Mortality: Within District 1930 − 1945

	Infant Mortality 8 − 365 days vs:			
	0 − 7 Days	1 − 2 years	1 − 7 Years	8 − 99 Years
	(1)	(2)	(3)	(4)
Reduced Form Lag	-0.0027	-0.0029**	-0.0031**	-0.0029***
	(0.0016)	(0.0014)	(0.0015)	(0.0011)
Year FE	Yes	Yes	Yes	Yes
Linear Time Trends	Yes	Yes	Yes	Yes
Pre Mean 8-365 days Mort	0.031	0.031	0.031	0.031
Pre Mean Other Mort	0.020	0.007	0.018	0.011
Clusters	400	400	400	400
R squared	0.123	0.134	0.102	0.175
Observations	5998	5998	5998	5998

Note: Each column describe a different fixed effects regression model. The outcomes are similar to those in table 14 but here I subtract the "placebo" mortality rates from the mortality rate 8 − 365 days in each district/year cell. In this way I am using within district/year differences in mortality as outcomes. All models include district specific linear time trends as well as year and district fixed effects. Standard errors are clustered at the district level.

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

Table 17: Reduced Form Effect on Infant Mortality (8 − 365 days): Time Consistency

Years Included	All	< 1945	< 1944	< 1943	< 1942
	(1)	(2)	(3)	(4)	(5)
Reduced Form Lag	-0.0034***	-0.0032***	-0.0033***	-0.0032***	-0.0025*
	(0.0011)	(0.0011)	(0.0011)	(0.0012)	(0.0013)
Year FE	Yes	Yes	Yes	Yes	Yes
Outcome mean	0.031	0.031	0.031	0.031	0.031
Clusters	400	400	400	400	400
R squared	0.083	0.081	0.083	0.082	0.076
Observations	5998	5598	5198	4798	4398

Note: Each column describe a different fixed effects regression model. The outcome is the mortality rate between 8 − 365 days. From left to right in the table, I sequentially and cumulatively remove one year at the time. In column 5 data is only used up until 1941. All models include district specific linear time trends as well as year and district fixed effects. Standard errors are clustered at the district level.

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

Table 18: Reduced Form Effect on Long-Term Outcomes: Time Consistency

Years Included	All	< 1945	< 1944	< 1943	< 1942
	(1)	(2)	(3)	(4)	(5)
Years of Education	0.0123	0.0049	-0.0020	-0.0145	0.0008
	(0.0331)	(0.0354)	(0.0338)	(0.0339)	(0.0398)
Log income 40-45 yrs	0.0130*	0.0073	0.0102	0.0123	0.0010
	(0.0078)	(0.0082)	(0.0076)	(0.0081)	(0.0087)
Mortality 45-57 yrs	0.0045	0.0011	0.0024	0.0040	0.0051
	(0.0039)	(0.0028)	(0.0030)	(0.0033)	(0.0036)

Note: Each column describe a different fixed effects regression model. The outcomes are educational attainment in years, log income between ages $40-45$ and mortality between ages $45-57$. From left to right in the table, I sequentially and cumulatively remove one year at the time. In column 5 data is only used up until 1941. All models include district specific linear time trends as well as year and district fixed effects. Standard errors are clustered at the district level.

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

Table 19: FE County and District Level Regressions

	Only < 1941		Full Sample $1930-1945$			
Outcome	Log Births	Log Births	Log Births	Infant Mort Rate	Fraction Home Births	Infant Mort Rate
Level of Analysis	(District)	(County→)				
	(1)	(2)	(3)	(4)	(5)	(6)
Reduced Form Lag	-0.0108					
	(0.0157)					
Fraction Treated Districts Lag		-0.0044	-0.0096	-0.0038***	-0.0083	-0.0035**
		(0.0317)	(0.0166)	(0.0012)	(0.0283)	(0.0013)
Fraction Home Births						0.0177***
						(0.0038)
Log Births						0.0170**
						(0.0081)
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
County Time Trends	No	Yes	Yes	Yes	Yes	Yes
District Time Trends	Yes	No	No	No	No	No
Pre Outcome mean	210.268	3594.202	3594.202	0.050	0.740	0.050
Clusters	402	25	25	25	25	25
R squared	0.347	0.990	0.992	0.860	0.964	0.871
Observations	4002	275	399	399	399	399

Note: Each column describe a separate regression. Table show both district regressions (column 1) and county regressions (columns $2-6$). The first two columns use data only up until 1940. Columns $3-6$ use data from the full time period but only at the county level. The outcome in columns 4 and 6 is scaled by births in each year. All models include year and county (district) fixed effects as well as county (district) specific linear time trends. "Fraction Treated districts lag" is a treatment variable that average the number of treated district by each county and year. Fraction home births are those assisted by a district midwife divided by total born each year. These include home births and births taking place at the residence of the midwife.

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

Table 20: Treatment Correlation With Measurement Error

Log cell size for sample	District Level				County Level	
	Education		Income		Education	Income
	(1)	(2)	(3)	(4)	(5)	(6)
Reduced Form Lag	-0.0281	-0.0149	-0.0300	-0.0167		
	(0.0257)	(0.0267)	(0.0255)	(0.0266)		
Fraction Treated Districts Lag					0.0364	0.0381
					(0.0404)	(0.0412)
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
County Linear Time Trends	No	No	No	No	Yes	Yes
District Linear Time Trends	Yes	Yes	Yes	Yes	No	No
District Quadratic Time Trends	No	Yes	No	Yes	No	No
Outcome mean	214.085	214.085	221.504	221.504	3510.291	3426.138
Clusters	400	400	400	400	25	25
R squared	0.742	0.810	0.742	0.811	0.974	0.971
Observations	5985	5985	5985	5985	399	399

Note: Each column describe a separate regression using difference-in-differences models and also models augmented with linear time trends. The first four columns use the long-term cell size as outcomes in logs, both for education and for income (measured between ages $40-45$). These are the number of valid observations used, by district and year. With systematic attrition, through mortality or miss measured parish of birth, the treatment indicator should be able to predict cell size. In the last two columns the outcomes are the same but at the county level.

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

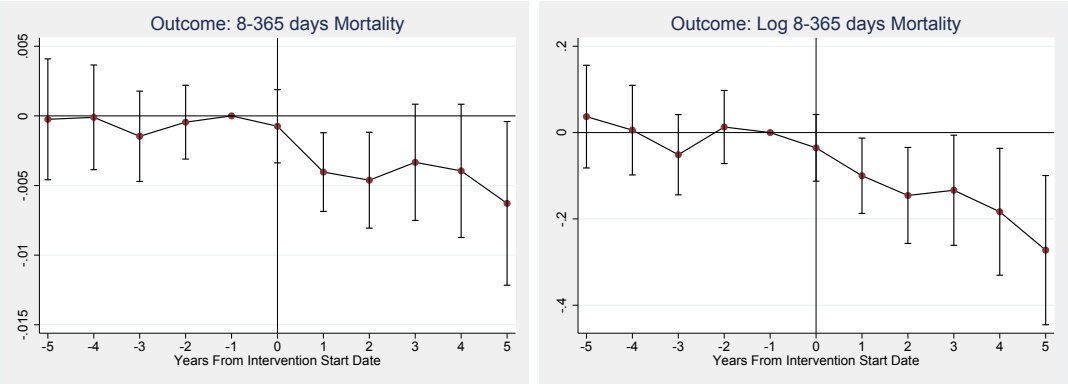
Table 21: Reduced Form Effect on Long-Term Outcomes: County Level

	Educational Attainment		Log Income $40-45$		Mortality $45-57$ Yrs	
	(1)	(2)	(3)	(4)	(5)	(6)
Fraction Treated Districts Lag	0.0377	0.0360	0.0096	0.0069	-0.0008	0.0004
	(0.0558)	(0.0272)	(0.0113)	(0.0071)	(0.0010)	(0.0012)
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Linear Time Trends	No	Yes	No	Yes	No	Yes
Outcome mean	1.861	1.861	28327.714	28327.714	0.047	0.047
Clusters	25	25	25	25	25	25
R squared	0.968	0.986	0.954	0.975	0.735	0.759
Observations	399	399	399	399	399	399

Note: Each column describe a different fixed effects regression model. The first two columns use average educational attainment measured years as outcomes. Columns $3-4$ use average net discounted income between ages $40-45$ as outcomes. The last two columns use mortality between ages $45-57$ as outcomes. All measures are averaged using valid observations for each outcome conditional on being observed at time of measure. All regressions use standard errors clustered at the district level.

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

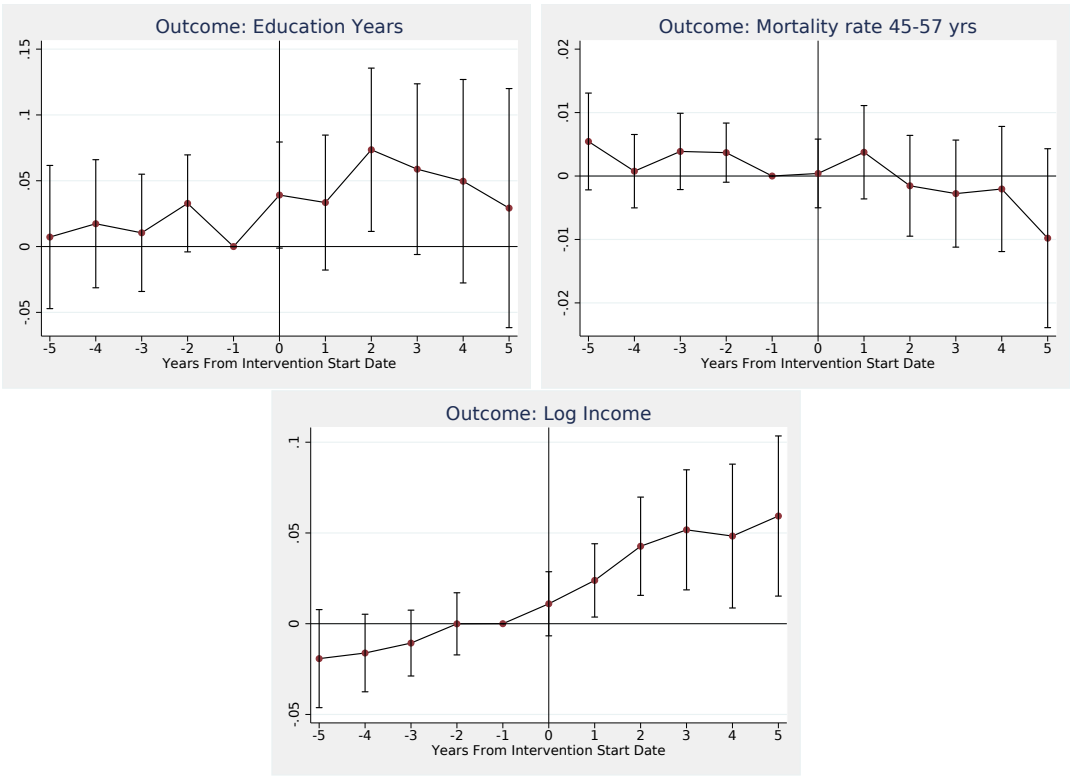
Figure 1: Event Study: DiD Specification Main Outcome



Note: The outcomes are infant mortality between $8 - 365$ days in life scaled by number of births in 1936 and the same in logs. Graphs show dummy variable estimates and 95% confidence intervals from regressions with year and district fixed effects. Displayed parameter estimates describe the kth year before and after treatment (year 0). The year -1 is the omitted category. Minus five and five includes all years before and after. All data included in regression. Standard errors are clustered at the district level.

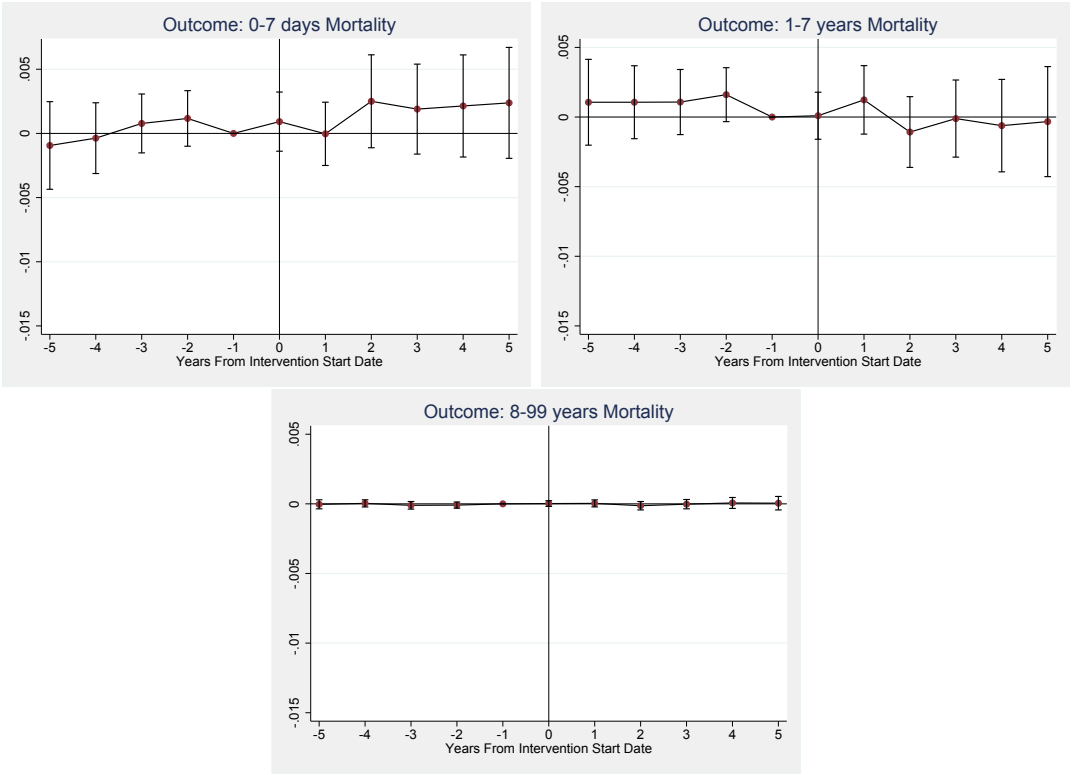
Appendix B: Figures

Figure 2: Event Study: DiD Specification



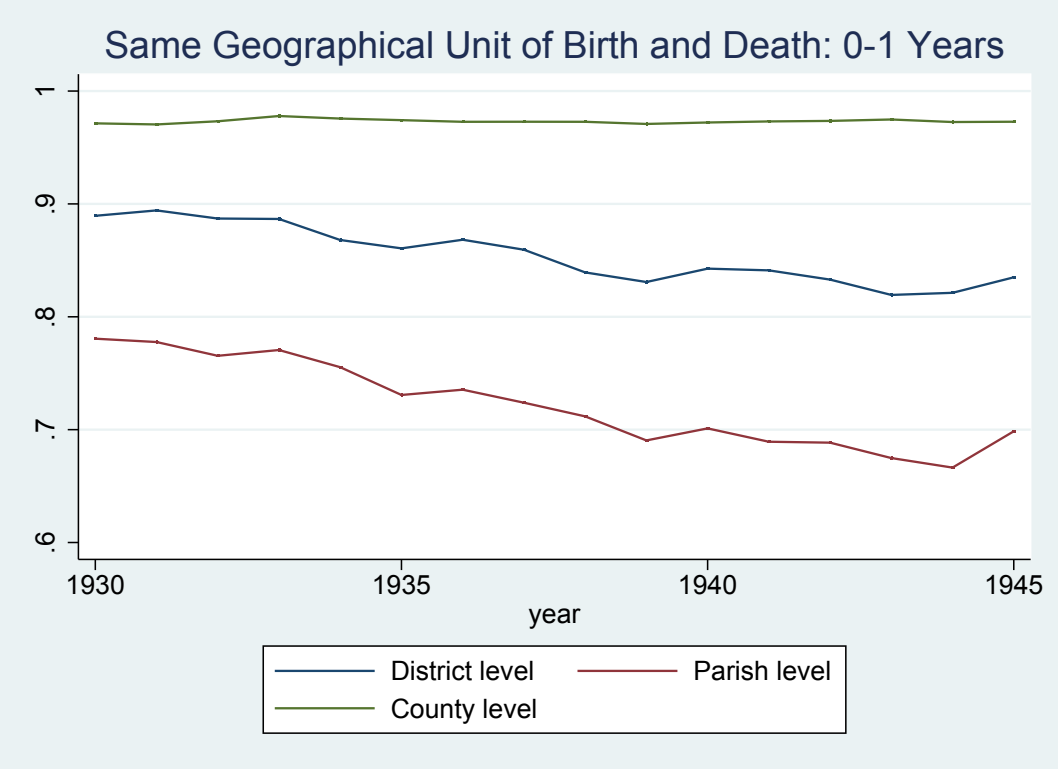
Note: The first outcome (top left) is educational attainment in 1970 (in 7 levels) aggregated to the district level. The second (top right) outcome is average inflation adjusted net income between ages $40-45$ aggregated to the district level. The last outcome (bottom) is mortality between ages $45-57$ scaled by the number of survivors at age 45 and aggregated to the district level. Figures shows dummy variable estimates and 95% confidence intervals from a regression with year and district fixed effects. Minus five and five includes all years before and after. All data included in regression. Standard errors are clustered at the district level.

Figure 3: Event Study: DiD Specification Placebo Outcomes



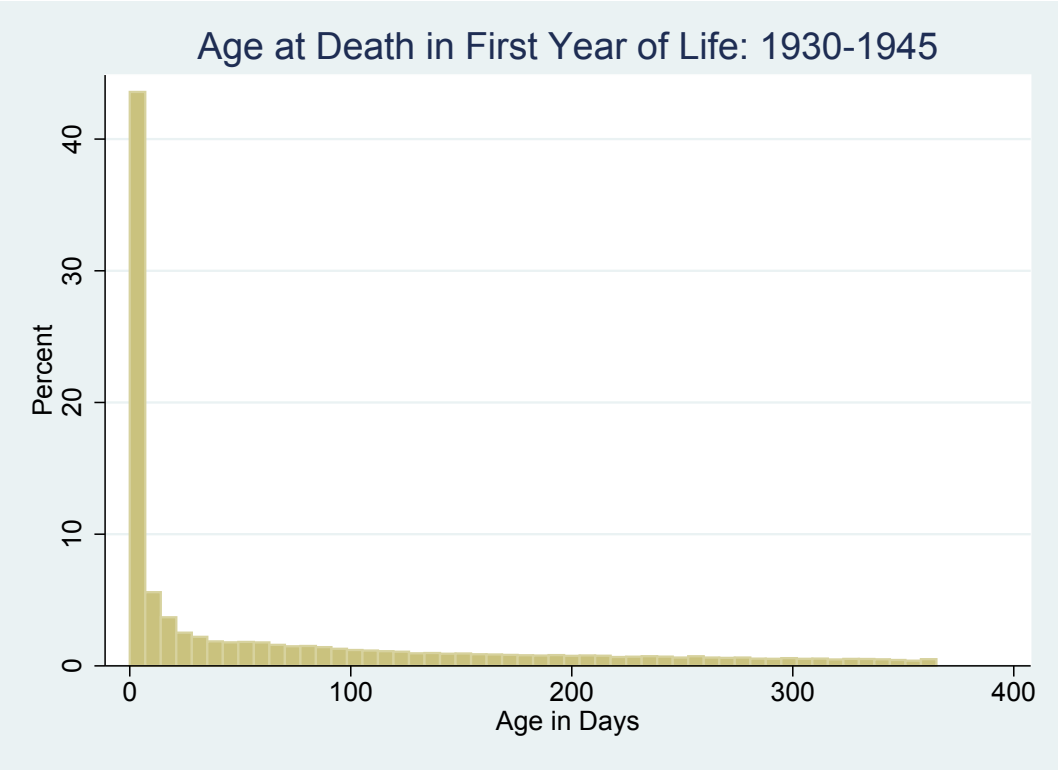
Note: The outcomes are mortality rate $0-7$ days, $1-7$ years and $8-99$ years. All scaled by either births in 1936 or population in 1936 (adult mortality). Graphs show dummy variable estimates and 95% confidence intervals from regressions with year and district fixed effects. Displayed parameter estimates describe the kth year before and after treatment (year 0). The year -1 is the omitted category. Minus five and five includes all years before and after. All data included in regression. Standard errors are clustered at the district level.

Figure 4: Infant Mortality fraction if same birth and death parish



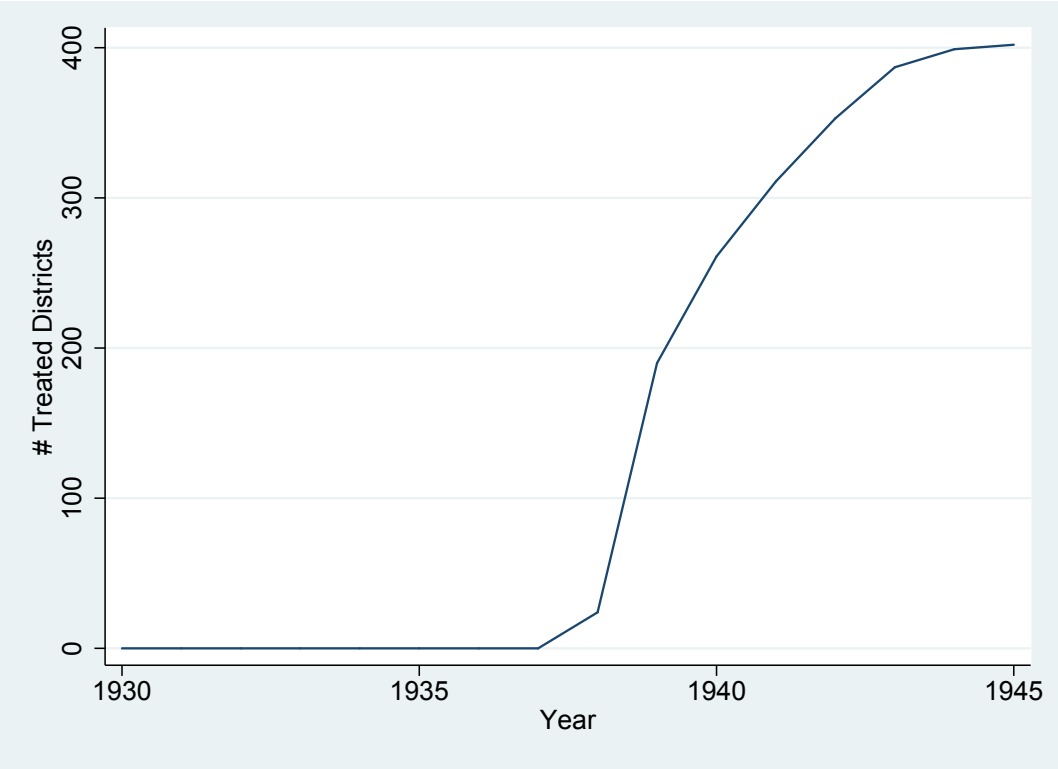
Note: The graph describe the change over time in the fraction of children that died in their first year of life that where born and died in the same geographical unit. At this time it was common that births where registered at the parish where the birth took place. District and county level based on parish of death which was based on parish of residence and not geographical parish of actual birth.

Figure 5: Infant Mortality Pattern During Intervention Time Period



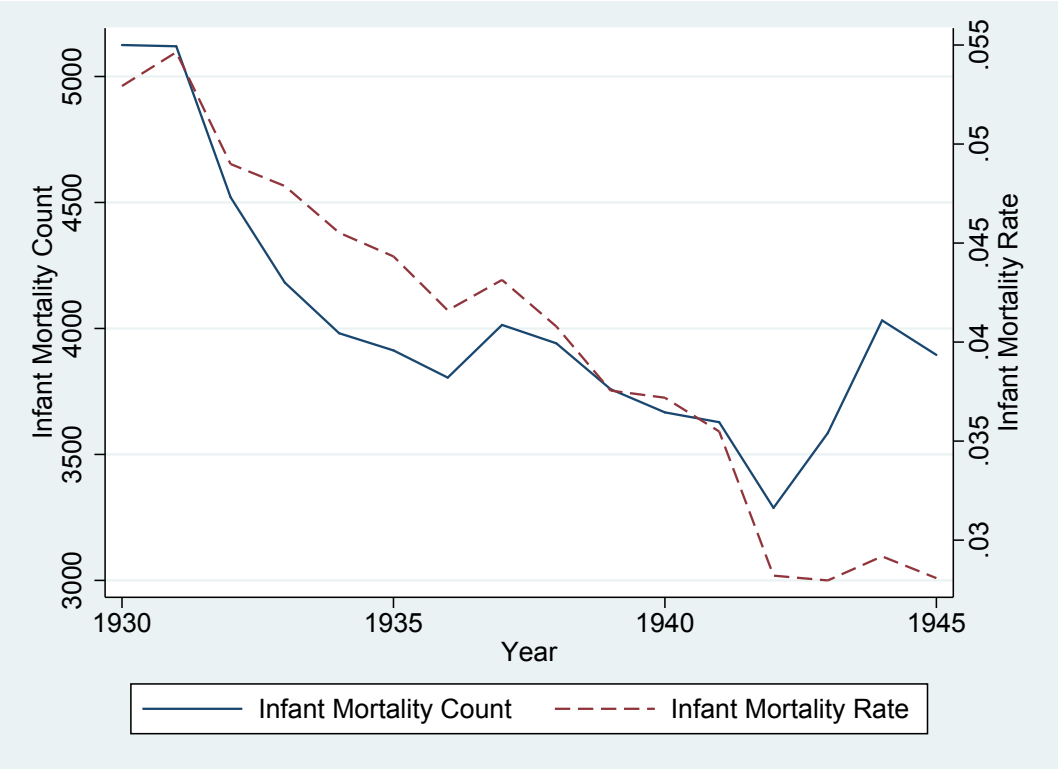
Note: Graph shows histogram described in percentages. Each bar represent one week (7 days).

Figure 6: Cumulative Number of Treated Districts



Note: Number of treated districts. Cumulatively by year.

Figure 7: Infant Mortality and Births



Note: Solid line is the number of infant deaths while the dashed line is the infant mortality rate. The discrepancy is caused by more children being born from 1942 while relatively more children survived their first year.

Table 22: Program Effect on Infant Mortality ($0 - 365$ days): IV

	Reduced Form	First Stage	IV
	(1)	(2)	(3)
Reduced Form Lag	-0.0035**	0.5080***	
	(0.0016)	(0.0256)	
Children Supervised			-0.0070**
			(0.0032)

Note: Each column describe a separate regression based on equation 1. The first column is the same as in column 1 of table 10. The second column use uptake defined as the number of children listed in the program at each district and year, divided by total births in 1936, as the outcome. The third column show the instrumental variables (IV) estimate. All models include year and district fixed effects. Standard errors are clustered at the district level.
* p < 0.1, ** p < 0.05, *** p < 0.01.

6 Appendix C: Supplementary Materials

Since take up among infants (and mothers) is available in the data, I can estimate the instrumental variables (IV) effect of being treated. This local average treatment effect (LATE) describe the average treatment effect of compliers (Angrist and Pischke, 2008). Since almost all of Sweden were eventually treated, the LATE will be close to the ATE (weighted up by time in program). The assumptions underlying this parameter is though more demanding. The exclusion restriction might not hold. The program included antenatal care, although I find that this part of the program was unproductive, primary health care was reorganised and there might have been interactions of treatment within the family. Still, I will show this parameter estimate, along with the first stage of infant uptake. This was the main component of the program with higher uptake and treatment intensity. The IV effect can provide a crude estimate of the treatment effect on the treated.

Using the base line specification, in table 22, I present the instrumental variables estimates. Here I find that the scaled treatment effect (treatment effect on the treated) is around -0.007, suggesting a 13% reduction in infant mortality from the pre program mean. The first stage, describing take up at the district level is strong, and shows that take up was around 50%. The seemingly low take up is likely due to the district aggregation and also from using the lag of the reduced form.