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Sonia Bhalotra
Martin Karlsson
Therese Nilsson

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Forschungsinstitut
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of Labor

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Sonia Bhalotra
University of Essex
and IZA

Martin Karlsson
University of Duisburg-Essen

Therese Nilsson
Lund University
and IFN

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IZA

P.O. Box 7240
53072 Bonn
Germany

Phone: +49-228-3894-0
Fax: +49-228-3894-180
E-mail: iza@iza.org

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ABSTRACT

Infant Health and Longevity: Evidence from a Historical Trial in Sweden*

This paper investigates the potential of an infant intervention to improve life expectancy, contributing to emerging interest in the early life origins of chronic disease. We analyse a pioneering program trialled in Sweden in the 1930s, which provided information, support and monitoring of infant care. Using birth certificate data from parish records matched to death registers, we estimate that the average duration of program exposure in infancy led to a 1.54% point decline in the risk of infant death (23% of baseline risk) and a 2.37% decline in the risk of dying by age 75 (6.5% of baseline risk).

NON-TECHNICAL SUMMARY

This paper investigates effectiveness of an infant intervention that, in addition to clinical services, provided universal access to "soft" inputs including information, monitoring and support with a large home-visiting component. The intervention was implemented as a trial in the early 1930s in Sweden, and was a precursor to legislation that rolled it out nationwide from 1937 as part of the expansion of the welfare state. Using birth certificate data from parish records matched to death registers, we find that the programme led to substantial improvements in infant survival and to improvements in longevity over and above this. This is consistent with the hypothesis that infant morbidities create structural changes that predict the onset or progression of chronic disease, but that are latent earlier in the life course. We estimate that an individual with the average duration of program exposure in infancy experienced a 23% reduction in infant mortality risk and a 6.5% reduction in the risk of dying by the age of 75 (an age by which almost 40% of their cohort had died). The results contribute new evidence on the potential benefits of postnatal care programs that are being introduced or modified in many contemporary settings. They are of particular relevance in today's poorer countries, which are carrying the double burden of infectious disease and chronic diseases.

JEL Classification: I15, I18, H41

Keywords: infant health, life expectancy, early life interventions, program evaluation

Corresponding author:

Sonia Bhalotra
Department of Economics
University of Essex
Wivenhoe Park
Colchster CO4 3SQ
United Kingdom
E-mail: srbhal@essex.ac.uk

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1 Introduction

Life expectancy in rich countries increased by thirty years in the course of the twentieth century, a historically unprecedented improvement that occurred largely on account of the control of infectious disease and infant mortality (Cutler et al., 2006). Murphy and Topel (2006) estimate that the cumulative gains in life expectancy after 1900 were worth over USD 1.2 million to the representative American in 2000. These estimates account for the direct contribution of infant survival gains, but not for the possibility that infant health improvements have spillover effects, increasing the chances of survival beyond infancy. Evidence of such spillovers would raise the benefits from infant health interventions for any given cost. While the evidence is scarce, there is a biological basis for these effects insofar as adverse conditions such as infection or poor nutrition early in life inhibit development of vital organs or modify endocrine processes with irreversible negative effects on health at later ages. Of particular interest is the hypothesis that infant morbidities result in the foetus being programmed with metabolic traits that predict the onset or progression of chronic disease but that are latent earlier in the lifecourse (Barker, 1990, 1994; Barker and Osmond, 1986). While there is growing interest in the question of whether childhood programs benefit adult health or longevity, the evidence base is small (Campbell et al., 2014).

The question is pertinent because the costs of a "war against infant mortality" are orders of magnitude smaller than the costs of a "war against chronic diseases". Chronic disease accounted for 68% (38 million) of all global deaths in 2012 and is on the rise. It exerts a major burden on individual wellbeing and work capacity, on state health expenditure, and on families' ability to invest in the younger generation.[1] Policies targeting chronic diseases tend to focus upon treatment or else on modifying adult behaviours to mitigate risks. But there is enormous unexplored potential to address the origins of chronic disease in infancy. This is especially relevant in poorer countries where child mortality rates, which symptomize the prevalence of infectious disease, continue to be unnecessarily high, with 1 in 10 children dying before their 5th birthday compared with 1 in 143 in richer countries. The toll in 2012 was estimated to be 6.6 million deaths, of which 74% occurred in infancy and 44% in the first month of life (Jones et al., 2003; Black et al., 2010).

This paper presents what would appear to be the first attempt to identify causal effects of a publicly-provided infant care program on longevity. We study a historical, pioneering trial that was implemented in the early 1930s in response to a cessation in the decline of infant mortality in Sweden, at a time when its incidence was similar to that in many of today's poor

[1] In the USA today, average healthcare costs for individuals with a chronic condition are five times greater than for those without, chronic diseases account for 75% of healthcare spending, and for 7 of 10 deaths; see `http://www.forahealthieramerica.com/ds/impact-of-chronic-disease.html`.

countries (Razavi, 2012). Trained health workers provided information, support and monitoring of newborn health through home visits alongside which there was an extension of services offered through local health clinics. The program was part of a broader Infant Welfare Movement of the time (Fildes et al., 2013), fueled by concerns about population decline, exacerbated by the First World War (Davis, 2011), and it constituted a significant step in the development of the modern welfare state in Sweden. Similar early childhood and home-visiting programs are are being introduced in developing countries but a recent survey notes that despite the surge in funding for these programs, there are few systematic evaluations (Engle et al., 2007). Thus, not only is there scarce evidence of their impacts on longevity, there is similarly scarce evidence of their more immediate impacts, i.e., of the extent to which information and monitoring influence infant health and survival.

Data pose a significant challenge to our task as we need to track a sufficiently large number of individuals over a lifetime. To address this challenge, we purposively digitized individual birth certificate data from historical parish records for a large and representative sample of roughly a quarter of a million individuals born during 1930-34 in 114 rural parishes and 4 cities.[2] We then matched births to the Swedish Death Index (Fischer et al., 2013) using name and date and place of birth. We validated our match of administrative birth and death records using burial (Swedish Genealogical Society, 2012) and tax records and, in addition, we used the 1970 census to cross-check names, especially for women who often changed surname upon marriage. We were able to track 96% of births for a period of 75 years. Among our sample cohorts, 36.5% did not survive to the age of 75, which makes it a good proxy for longevity. The availability and analysis of such data is fairly rare.

The other, more familiar challenge is to identify causal effects of the program. We are aided in this by three features of the program. First, it was time-delimited, starting 1 October 1931 and ending 30 June 1933, and it was announced as such, which will have limited selective migration and fertility. The narrow window of eligibility for the program also limits the possibility that program impacts are confounded by unobserved trends. Second, it was universal, in contrast to many contemporary programs in, for instance, the US, that target low-income women and children. Third, program documentation indicates that the National Board of Health selected the seven medical districts where the trial was implemented "randomly" to be representative of the country. We nevertheless use the 1930 census to create matched controls at the parish level, and we obtain estimates from comparison of exposed and unexposed siblings within mother. We also investigate endogeneity in the composition of births, and investigate the socioeconomic

[2]National vital statistics were available in Sweden before they were available in any other European country. All newborn children in Sweden automatically became members of the Church of Sweden. Parish records were linked to other administrative data, including records of in- and out-migration, births and baptisms, bans and marriages, and deaths and burials.

gradient in program uptake exploiting unusual access to utilization data.

Using exact birth date and birth parish to compute duration of exposure, we find large and significant impacts of eligibility for the infant-care program on the probability of the individual surviving to the ages of 1, 5, 40 and 75. We estimate that a child with the average duration of potential exposure to the program in infancy experienced a 1.54% point decline in the risk of infant death, which is 23% of baseline risk, and a 2.37% decline in the risk of dying by the age of 75, which is 6.5% of baseline risk. The estimates imply that the program accounted for between 20 and 50 per cent of the actual decline in infant mortality, and was associated with reductions in mortality which persist to this day. This is a fairly remarkable achievement given that a decade of stagnation in infant mortality preceded the intervention, and the intervention coincided with the Great Depression in Sweden.

There are no significant survival gains beyond infancy at age 5 or 40: the survival advantage of treatment over control groups at ages 5 and 40 is the same as at age 1. The finding that program impacts persist to the ages of 5 and 40 is meaningful given that a growing body of experimental evidence finds that program impacts often fade. For instance, the Perry and Abecederian preschool demonstrations, the Head Start program and Project STAR in the US show fade out of test score gains (see Almond and Currie (2011b) for a review). The importance of follow up is therefore increasingly recognized. In our context, it is conceivable that effects fade because of relapse of treated behaviours or because of endogenous behavioural responses that neutralize the effects of the intervention. However our results suggest that health gains in infancy dominate and persist. This is consistent with infancy being a critical stage of development and with dynamic complementarity across stages of development and, alternatively, with program-driven learning within mothers that persists over time.

Our finding that the gains in life expectancy exceed the gains in infant survival but that these additional gains emerge after the age of 40 is consistent with latent effects of program exposure on chronic disease, since the onset of chronic disease is typically after the age of 40. The biological mechanisms are not fully understood or documented, but it is argued that the early life environment may set the switch for genome expression later in life (Petronis, 2010). The effects we identify will tend to include a behavioural component over and above the biological mechanism referred to earlier to the extent that infant health improvements raise adult education and income (Almond and Currie, 2011a), and education and income are effective in averting and treating morbidities (Cutler and Lleras-Muney, 2010; Ettner, 1996). Moreover, the expectation of greater longevity may itself motivate increased investments in human capital by extending the horizon over which payoffs accrue (Ben-Porath, 1967; Soares, 2005).

Mother fixed effects estimates are not significantly different from estimates obtained on the same sample using pooled data. Using digitized records of program uptake and number of visits for a (representative) sub-sample of districts, we find little evidence of selection into the program. We find some evidence consistent with the hypothesis that parental investments favour treated children and thereby reinforce public investment, inasmuch as older siblings of treated children do worse than matched controls.

The program contributed to narrowing health inequality. For instance, children born to single mothers experienced a reduction in infant mortality of as much as 6.9 % points and a reduction in the chances of dying by age 75 of 11.44% points. They constituted 12% of all births at the time (Statistics Sweden, 1940). While the program was universally available in treated areas, program documents highlighted the importance of reaching this group (Steenhoff, 1931), so these results provide a crude consistency check on identification of program effects. Since the program coincided with the Depression, we used tax records to generate an indicator of the severity of the financial crisis at the parish level and to study heterogeneity in impact by this variable, but found no significant differences. We consistently control for coverage by a program providing antenatal care services in the treated districts and find no direct effects of this program. This result resonates with the conclusions from a recent survey of US programs which shows that postnatal care programs have a more consistent record of positive impacts than antenatal programs (Currie and Rossin-Slater, 2015).

This paper contributes to two streams of the literature. First, it contributes to a literature on the power of information (Dupas, 2011) and monitoring and feedback (Andrabi et al., 2013; Bandiera et al., 2009) to effect behavioural change. We provide some of the first evidence that an information and monitoring program for infant care can have long run effects. The second is a thriving literature evaluating the effects of early life health shocks (or interventions) on education and labour market outcomes (Almond, 2006; Bhalotra and Venkataramani, 2011; Bharadwaj et al., 2014), to which we contribute by estimating impacts on longevity.

Until recently, there was no analysis of the long run effects of the sort of infant-care and home-visiting program that we study, but emerging in parallel with our work are Hjort et al. (2014) and Bütikofer et al. (2014). These studies analyse the nationwide rollout of broadly similar programs in the late 1930s in Denmark and Norway respectively, which occurred alongside rollout of a Swedish program modelled upon the trial that we analyse. These studies model impacts on chronic disease incidence in a sample of survivors that are likely to be endogenously selected. In addition, because the survivors are relatively young at the time of measurement,[3] these studies

[3]In Hjort et al. (2014), chronic disease is measured among a sample of individuals who survived till 1980 i.e. about age 45 (and still resident in Denmark) and in Bütikofer et al. (2014) for a sample who survived till 1967

may be unable to capture the later onset of chronic disease or allow for program-driven increases in the probability of surviving chronic disease. In this paper we track individuals for an additional 20-30 years, focusing upon longevity, and we have the entire sample of births rather than births that survive to the age of observation.[4] The results of our study are clearly important for policy design in today's poorer countries, which are carrying the double burden of infectious disease and chronic diseases,[5] and they are also relevant to contemporary interventions in Europe and the United States (see section 6).

2 Infant Health and the Emergence of the Field Experiment

Infant mortality had started to decline in all Western countries by the turn of the 20th century (Loudon, 2000). Sweden experienced an impressive decline from above 10% in 1900 to 6.5% in 1917 (Corsini and Viazzo, 1997; Brändström, 1988). However, as is clear in Figure 1, from 1920 to 1930, there were no further substantive declines, and a similar stagnation was noted elsewhere, for instance, in Denmark and the USA (Moehling and Thomasson, 2014; Wüst, 2012). The deterioration of public health in the 1920s, coupled with falling birth rates (Wisselgren, 2005), gave rise to an intense public debate in Sweden on how to improve conditions for expectant mothers and newborns. The public concern in Sweden was part of an international phenomenon known as the Infant Welfare Movement (Fildes et al., 2013), fuelled by concerns about population decline, exacerbated by the First World War (Davis, 2011).

The rising concern about public health led, in 1929, to the Royal Commission being charged with the task of modernising maternal and neonatal care and it proposed the trial that we analyze. In December 1930, the trial was approved by the National Board of Health (*Medicinalstyrelsen*) and the final proposal was accepted in the Swedish Parliament on 29 May 1931. The trial started on 1 October 1931 and ended 30 June 1933, after a decision to extend it was taken in the summer of 1932. The state committed SEK 30,000 (USD 133,000 in current prices) to funding the trial. Seven health districts were selected to receive free and extended antenatal and infant care services for the delimited trial period.[6]

i.e. about age 35.

[4] Also, we analyse a short trial introduced all at once in seven medical districts while the Danish and Norwegian studies analyse rollout across the nation over 13 and 20 years respectively, which makes it harder in principle to isolate impacts of the infant-care program from other programs and events, and endogenous confounding behaviours like fertility and migration.

[5] Three-quarters of all chronic disease related deaths today occur in low and middle-income countries, where many individuals cannot afford the costs of treatment.

[6] The participating districts were Lidköping, Hälsingborg, Harad, Råneå, Jokkmokk, Pajala and Mörtfors.

At the time some services were already being provided in most Western countries, but these tended to be local or private initiatives in larger cities, often targeting certain groups. For instance, although the UK Maternal and Child Welfare Act in 1918 required local authorities to provide antenatal care clinics, there was considerable fragmentation in maternity services across the the UK until the start of World War II when it became routine practice to visit every newborn in the UK at home (Chamberlain, 2006; Loudon, 2000). Home visiting programmes were available in the USA in 1920-1929. In Sweden, before the trial was initiated in 1931, there were two types of institutions for infant and family care at scattered locations. One relied mainly on visits to physicians and the distribution of cow milk powder to poor mothers (*Mjölkdroppen*) and the other involved home visits by nurses to families across the socioeconomic status distribution but mostly in larger cities (*Barnavårdscentralsystemet*; cf. Wallgren, 1936; Stenhammar et al., 2001).

2.1 Organisation of the Activities

The trial intervention of 1931-1933 was monitored by the National Board of Health but implementation was decentralized to the district level, adapted to heterogeneous local conditions, and led by physicians. In order to ensure uniform standards, an educational event was organised in the capital Stockholm in July 1931, during which staff from all participating districts attended lectures and courses for five days, in which the objectives of the project were detailed and staff visited existing health clinics. Particular emphasis was placed on the importance of providing care on equal conditions to all mothers.

In each test district, a health centre for infant care with regular office hours was started,[7] and in all locations there were intense outreach activities to inform people about the available services. Historical documents indicate that there were recurrent announcements concerning the availability and opening hours of the health centres in local newspapers and in churches in the test districts (Lindsjö, 1934). Figure 2 provides an example from the local newspaper in the district of Hälsingborg.

The project focused on preventive care and involved three main types of activities, all provided free of charge, which were guidance services and examinations at the surgeries, home visits and information campaigns. Children were weighed and monitored at the clinics, and sickness was diagnosed early, with children being referred to doctors as needed. Mothers were given guidance and encouragement to breastfeed and provided with illustrated advice on when and how to give the child proper nutrition at various stages of development, and how to monitor

[7] In the more remote areas, advice was also given by telephone and urine samples sent by mail.

newborn health (see e.g. Swedish Red Cross, 1928). Nutritional advice for all children included recommendation of cod-liver oil and fruit juice rich in vitamin C, and a *blutsaft* rich in iron for premature children. Figure 3 shows an extract from a leaflet given to all enrollees. A focal point in the public debate was the situation of single mothers and children born out of wedlock, who had significantly worse health prospects than the rest of the population (Steenhoff, 1931).

Home visits were seen as an important part of the project (Lindsjö, 1934). Visits were made by nurses and aimed at ensuring that families followed the recommendations on infant and child care given by physicians, but also at acquiring knowledge of the household's environment and circumstances. At the time many families lived in cramped conditions, sanitary standards were poor and pests, such as lice, mange and rats were rife; see (Nordström, 1938). Advice was given on hygiene, sanitation and cleanliness in the household. Intervention of this sort are likely to be more cost-effective than disease-specific interventions as, by educating and supporting mothers' preventive behaviours and investments, they tend to generate externalities.

2.2 Utilisation and Evaluation

Despite some initial scepticism,[8] auditing reports suggest that the intervention induced behavioural changes amongst participating families leading to improvements in diet and hygiene (Steenhoff, 1934). Participating physicians attributed substantial improvements in child and maternal health to the intervention, which motivated the roll-out of a similar scheme in all parts of the country from 1937 and the passage of a bill in the national parliament legislating free maternal and infant care with equal access for all. The field trial may hence be seen as a seed of an emerging welfare state. It seems likely that the trial also influenced the decision to rollout similar programs in Norway in 1936 and in Denmark in 1937 (Moehling and Thomasson, 2014; Wüst, 2012; Bütikofer et al., 2014).

Program eligibility was determined by birth date: all children less than one year of age were eligible at the beginning of the trial, and all children born during the trial became eligible (see Figure 4). Participating doctors and nurses were obliged to keep records of their activities using standard forms for each mother and child. We were able to acquire and digitize these forms for four of the seven participating districts (they appear to have been destroyed for the others). These data indicate that, on average, there were 2.8 visits to clinics and mothers received 3.9 home calls per enrolled child and that, in all, around 2,600 children were enrolled in the trial,

[8]There was scepticism at the national level- in 1914 two motions for better care of childbearing women and their newborns and for the establishment of maternity care facilities had been rejected in the National Parliament.

which represents a large proportion of eligible families in the chosen locations (see Table B.1).[9]

3 Data Sources and Matching Procedure

3.1 Matching Procedure

Administrative documents describing the introduction of the program underline that the intervention districts were chosen to reflect the diversity in local conditions with respect to *inter alia*, population density, demographic structure, and living standards (Propositioner, 1930). Importantly, infant mortality was given no consideration in selection of participating districts. We nevertheless generate matched controls at a decentralized level. The seven medical districts assigned to participate in the trial comprised two cities and 57 rural parishes. We matched these to two cities and 57 rural parishes which were not exposed to the intervention, and these control locations belonged to 38 different medical districts.

The matching was done using a range of observable parish characteristics drawn from the 1930 census. The best matches (denoted $\mathcal{J}_M(i)$) were identified using the Mahalanobis distance metric [10], defined as

$$\mathcal{J}_M(i) = \arg\min_j \sqrt{(X_i - X_j)' S^{-1} (X_i - X_j)} \tag{1}$$

where X_i is the vector of observable characteristics for a parish belonging to a test district, namely, average income, net wealth, employment shares in manufacturing and agriculture, population density, proportion of fertile married women, and a dummy variable for urban locations, and S denotes the covariance matrix of the vector of observable characteristics. The matching was done in random order and without replacement. Observations from the control group were weighted based on their population size relative to the population size of the treated locations they were matched to. Thus, each control observation matched to parish c is weighted according to $w_{\mathcal{J}_M(c)} = \sqrt{N_c / N_{\mathcal{J}_M(c)}}$ where N_c ($N_{\mathcal{J}_M(c)}$) denotes the 1930 population size in the test (control) district. This ensures covariance balance for the variables used in the matching procedure. The reduction in bias comes at the cost of reduced efficiency of estimates. However, the reduction

[9] See Figure 5 for a histogram of utilization.

[10] King et al. (2011) compare the effectiveness of alternative matching estimators for causal inference. They argue that the commonly used propensity score matching estimator often approximates random matching but that, contrary to conventional wisdom, random matching is not benign i.e. it can degrade inferences relative to not matching at all. They show that the Mahalanobis metric does not have this problem.

in efficiency is likely to be small since half of the observations (the treated group) have weights equal to one and 90 per cent of individuals have weights within the range $(0.46, 1.98)$. Robustness checks showed that the results are insensitive to the weighting scheme used. Below, we assess match quality by conducting tests of differences in means for a set of observable covariates, discussed below. Figure 6 shows a map of the treated and matched-controls districts.[11]

3.2 Data Sources

The data are purpose-built using myriad sources. We digitized individual level data from administrative records maintained by the historical parish for the 57 parishes and two cities in the seven trial districts and for the 59 areas that they were matched to for the years 1930-1934. The sample contains 24,710 deliveries resulting in 24,374 live births.[12] The parish records contain exact date of birth, whether stillbirth or live birth, child gender, mothers' age and marital status, parents' occupation (transformed into professional classes based on the HISCO classification (Leeuwen et al., 2002)), and subsequent mortality by cause for mothers and infants covering all deaths for the period up until 1946. This allows us to track mortality during the first 10-15 years of life for the sample individuals born during 1930-34. To identify mortality beyond this, and to validate information on mortality during childhood, we used the Swedish Death Index (Fischer et al., 2013) which includes the universe of all deaths occurring between 1901–2009.

A very detailed and strict reporting procedure regarding death causes was introduced in 1911, which improved reporting from rural areas. Local clergymen had to make monthly reports to Statistics Sweden on the likely cause of death of persons in cases where no doctor had been involved. These notes and reports were then reviewed and confirmed by a GP who reported the final cause of death to the bureau.[13] Individual records were matched based on date of birth, sex, forename, surname and birth parish. To validate the matching, we use a dataset containing burial records (Swedish Genealogical Society, 2012). As a second source of validation of adult mortality we used tax records from the 2002–2013 period. We also used the 1970 census to get information on names, which is pertinent for women as they traditionally changed their surnames upon marriage. Whenever we found conflicting information between data sources, for example,

[11] The administrative unit used for the map is the parish which is a lower level than the medical district. For example, in the Norrbotten region, there were four test districts containing seven parishes in total.

[12] In the 17th century, the Swedish clergy created an information system that included all individuals in their parishes older than 6 to 7 years. By the middle of the 18th century, this registration included the entire population. The information system was based on the annual catechetical examination of every household, where the clergy examined knowledge of the catechism as well as the reading ability of all household members. The Office of the Registrar General (Tabellverkskommissionen), founded in 1749, compiled national statistics from the ecclesiastical registry (Högberg, 2004). Membership of the Church of Sweden could actively be cancelled if an individual wanted to enter another denomination, but church records nevertheless covered all citizens.

[13] For details on the reporting of deaths, see the introductory chapter in *Dödsorsaker 1911* (Statistics Sweden, 1915), Hyrenius (1914) and Hultkvist (1940).

when we found individuals recorded as earning an income after being deceased, we checked the records manually. Thus, the mortality information in our dataset is very carefully assimilated and checked. We investigated migration and we find that our sample cohorts were very unlikely to migrate, for example, only a handful of individuals migrated during the first ten years of life (evident in the parish data), and these individual were dropped from the dataset. Table B.2 presents summary statistics, and Appendix A provides variable definitions.

Of all births in the sample cohorts, 1930-34, we identified a certain death date for 9,793, we noted that 16,217 were recorded as having an income in 2002 tax records, 7 individuals emigrated before 1950 according to the parish records and 1,002 individuals had no death date and no tax records but we assume that they are still alive (note that an individual can have a known death date and an income reported in 2002). The death register also contains deaths occurring outside Sweden- while the information is particularly reliable for the Nordic countries, there are several entries from Germany and North America and, in total, 2-3% of recorded deaths in the relevant cohorts occurred abroad. We checked that our results are robust to dropping emigrants from the analysis.

We created another dataset at the district level using annual reports of each of the 447 medical districts of Sweden (*provinsialläkardistrikt*). District physicians provided yearly summary statistics on deliveries, the number of midwives, child mortality, delivery complications and maternal mortality, amongst other variables, based on the local midwives' standardized diaries. We collect this information for the years 1930–1934; see Table B.3 for summary statistics.

We recovered utilization data from archived physician and nurse records from four districts and matched them to individual birth records. These cover about half of the eligible sample of women and children and, as is clear from Table B.4, summary statistics for this subsample are similar to those for the overall for all available relevant characteristics. We use these data below to investigate gradients in uptake by indicators of socio-economic position, and to gauge impacts of treatment on the treated.

4 Empirical Strategy

We identify program impacts by interacting birth-cohort based eligibility with being born in a treated parish or city, conditioning on cohort and parish fixed effects. All children aged 0-12 months during the trial period 1 October 1931 to 30 June 1933 in a treated area were eligible.

The baseline specification is

$$y_{icj} = \alpha + \beta T_c D_j + \gamma_j + \delta_c + \lambda X_i + \epsilon_{icj} \tag{2}$$

where y_{icj} is a survival outcome of child i born on date c in parish j, T_c is the duration in months of eligibility for a child born on day c, γ_j are parish fixed effects and δ_c are fixed effects for quarter of birth times year of birth. The parameter β estimates the intent-to-treat (ITT) effect, which is the effect, for each additional month of eligibility, of making the services available. This estimate is the relevant parameter in cost-benefit analysis when policy makers are unable or unwilling to make utilisation of services mandatory. Individual heterogeneity is absorbed by covariates X_i that include the sex of the child, an indicator for whether it is a singleton or multiple birth, the marital status of the mother at birth and dummies capturing old (> 32.3) and young (< 25.8) mothers, with mothers in the middle age category constituting the omitted case, and indicators for the occupational class of the father. We also include parish-specific linear trends.

To allow for time-invariant unobserved heterogeneity at the mother level which may determine selection into programme uptake or fertility, we re-estimate the model including mother fixed effects. This is a substantial advantage but it comes at the cost of using a smaller sample, which is that of women with at least two births in the sample period. As this is likely to be a selected sample, we report estimates on it with and without mother fixed effects so that we can isolate changes in coefficients created by controlling for mother-level unobserved heterogeneity from changes created by sampling this group of women.

At the same time as the infant intervention that we study, the Government trialled an intervention for pregnant women. Women who were at any stage of pregnancy between October 1931 and June 1933 were provided with improved antenatal care services. Therefore, some of the infants treated by the infant intervention were treated in the fetal period by the antenatal intervention and it is important that we take account of its effects. The raw correlation of duration of eligibility for the antenatal and the postnatal interventions is 0.32 and, conditional on participation in any one intervention, this falls to 0.13. Still, in every specification we consistently control for duration of eligibility of the mother of the index child for the antenatal intervention.

5 Results

We now present the survival equation estimates, followed by a discussion of potential threats to identification, and an investigation of heterogeneity in program impact. The main estimates are

intent-to-treat estimates using all births in the sample-areas. For the sub-sample for which we have data on uptake and utilisation, we estimate impacts of utilisation, instrumenting this with program exposure. Under the assumption that the duration of eligibility (which depends only on birth date) is orthogonal to unobservable differences in the health of children (conditional upon birth quarter and birth year), these 2SLS estimates identify the treatment effect on participating children (ATT). We also estimate program impacts on siblings to assess whether the reform may have induced parents to reinforce investments in index children at the expense of unexposed children.

5.1 Infant Survival

Table 1 presents impacts of duration of potential exposure to the intervention on survival to ages 1, 5, 40 and 75. The odd-numbered columns present estimates from a parsimonious specification in which the controls are the main effects (an indicator for treated region and dummies for quarter×year of birth) and duration of exposure to the antenatal care program. In the even-numbered columns we introduce parish fixed effects, parish-specific linear trends and the covariates listed in the preceding section.

Here we discuss estimates from the richer specifications. The intervention is estimated to have reduced infant mortality by 1.54% points for individuals with the average duration of exposure conditional upon non-zero exposure, which is 23% of the mean of the pre-intervention infant mortality rate. The addition of controls enlarges the coefficient but the change is not significant, which raises our confidence that the treatment indicator is picking up exogenous variation in program eligibility rather than unobserved heterogeneity in treated parishes or differential pre-trends between treated and control parishes. The coefficient on the infant program exposure variables is similarly invariant to dropping the control for the antenatal care program. The antenatal care intervention has no direct impact on infant survival: the coefficient is small and imprecisely determined. These findings are consistent with the evidence surveyed in Currie and Rossin-Slater (2015) who show that infant care programs including home visiting programs in the US have clearer long run benefits than prenatal programs.[14]

[14] A possible explanation in our context is that much of the information provided to expectant mothers was already available prior to the 1930s. Sweden was a forerunner in obstetric care, and had professionalized birth assistance in the early 18th century, achieving a maternal mortality rate in 1900 that was half that in England and Wales and less than half that in the United States (Högberg, 2004). Also, we investigated our utilization data for both programs and found that, while utilization of the infant program was high and occurred across the SES distribution, uptake of the antenatal program was lower, more varied across regions and on average skewed in favour of married women and women of higher socioeconomic status, possibly because of the social stigma associated with illegitimate pregnancies, and the greater weight of this among low-SES women. This will have further limited any effects of the antenatal care intervention.

A study of underprivileged mothers in the USA shows that they systematically underestimate the extent to which child development responds to maternal behaviours (Cunha et al., 2013), which suggests the scope for programs such as ours that combine information and salience to improve child outcomes. In general information trials show mixed results, see Dupas (2011), but a recent study which randomized information on nutrition and diet to mothers in Malawi shows significant improvements in child nutrition (Fitzsimons et al., 2014). This is relevant given that an important component of the Swedish intervention we analyse was provision of information on nutrition and breastfeeding to mothers.

5.2 Longevity

We modelled longer range survival rates of the sample cohorts to assess whether individuals who survived infancy lived longer as a result of program exposure. We chose age 5 because the hazard of mortality tends to be highest at birth when individuals have limited immunity and to then decline exponentially to age 5, after which it flattens noticeably. We chose age 40 as an age by which maternal mortality risk has been realized, and we chose age 75 as this was a "ripe old age" to achieve for the 1930s cohorts in our sample, 36.5% of whom were dead by this age. We model survival to ages 5, 40 and 75 conditional upon birth rather than conditional upon survival to age 1 because survival to age 1 is endogenous.

We find significant effects of program exposure duration on survival to each of the chosen age thresholds (see Table 1). The coefficients on survival to ages 5 and 40 are very similar to the coefficients for survival to age 1 which suggests persistence of the survival advantage accruing to cohorts exposed to the program in infancy, but no additional survival advantage to those who survived infancy, up until age 40.[15] There is however a substantial rise in the coefficient associated with survival to age 75 (from -0.20 to -0.31), an increase of 55%, which suggests that individuals who survive infancy carry an advantage from exposure to the infant-intervention that makes them hardier late in life. For eligible children with the mean duration of exposure, the program is associated with a 2.37% point reduction in the probability of dying by the age of 75, which relative to the mean of 36.5% is a 6.5% reduction. While each of these regression coefficients is statistically significant, the difference between the coefficients is not. Nevertheless the increase in the coefficient is substantial and probably meaningful, and the pattern of results suggests that post-infant survival gains from the intervention are concentrated in late adulthood (after age 40), consistent with the onset of chronic disease being later in life. As for infant survival, so for

[15]The persistence of effects to age 40 allays the potential concern that infant survival improved because of intensive monitoring, feedback and home-visiting, but that when this ceased upon the child reaching the age of twelve months, the mother reverted to "old habits" pertaining to nutrition and health so that, by the age of five, the exposed birth cohorts showed smaller gains in survival rates than the control-group.

survival into adulthood, we find no discernible impacts of the antenatal care intervention.

A handful of existing studies estimate impacts of the infant environment on chronic disease in adulthood using historical data on (selected) survivors. The two studies of infant health interventions were discussed in the Introduction. In addition are studies of early life exposure to recession (Yeung et al., 2014; Van den Berg and Modin, 2013), to a pre-school intervention (Campbell et al., 2014), and to the introduction of antibiotics (Bhalotra et al., 2015). The only other study that attempts to model changes in longevity (rather than in chronic disease among survivors) flowing from a change in early life conditions is Aizer et al. (2014), who study a cash transfer program directed at single mothers in early 20th century America, but matching births to death records was much harder in their data.

5.3 Within Mother Estimates

We introduce mother fixed effects to control for any selection on time-invariant mother-level traits, such as for instance time preference, altruism towards children or ability. This restricts the sample to mothers with at least two births which is about half of the full sample, so we also present pooled estimates on this subsample, see panel B Table 1. The coefficients estimated on the pooled sample are much larger than in panel A, and the only difference is that they are on the subsample. This is consistent with mothers who have closely spaced births (and hence two within the period 1930-34) being negatively selected (see Table B.5), it being well established that births preceded by a short interval suffer higher infant mortality risks (Bhalotra and Soest, 2008). Thus larger program impacts amongst these women probably arise from their children being more "treatable". Importantly, the fixed effects estimates in panel C, identified from variation in program exposure among siblings, are not significantly larger than the pooled estimates. This suggests that there is no significant selection.

5.4 Threats to Identification

In this section we discuss tests of matching, common pre-program trends, and behavioural responses to the program that may be correlated with the survival outcomes.

5.4.1 Match Quality

For each of the parishes in the trial, the nearest neighbour in terms of observable characteristics was identified. Table 2 presents summary statistics for observable characteristics from the 1930 census on which the matching was done. We show the standardised difference ('Std. Dif.') (Imbens and Wooldridge, 2009) between the treatment group and the matched controls (labelled "matched"). The lower panel of the table compares treated and matched-control areas on characteristics that were not used to create the matches, data for which were assimilated by us from annual medical district reports. In particular these characteristics include the primary outcome, infant mortality. Tests for balance indicate that the treated and matched-controls are balanced on characteristics, which validates the matching procedure. We also show the standardised difference between the treatment group and the rest of Sweden (labelled "control"), which suggests that the treated regions were, as the implementers of the trial intended, representative of Sweden, and this corroborates the external validity of our results.

5.4.2 Common Time Trends

Trends in infant mortality prior to the intervention for an extended time period stretching back to 1910 are shown in Figure 7 for treated and control regions, and there is no evidence of differential pre-trends.

5.4.3 Anticipation, Selection

In this section we investigate behavioural responses to program announcement, investigate whether the composition of post-program cohorts was influenced by persistence or spillover of the program, and whether there is any evidence of confounders i.e. omitted events that coincided with our treatment. We also investigate whether the antenatal program (that we consistently control for exposure to) impacted the birth conditions of children exposed to the infant care program. Results are presented in Table 3.

The Parliamentary decision to implement the trial was announced in May 1931 and the program was effective October 1931. In order to address the possibility of anticipation effects that may, for instance, have led families to change the timing of fertility, we define a new treatment variable, capturing the time period during which information on the intervention was in the public domain, which is 1 for 1 April 1932 to 15 November 1933, and 0 otherwise. The idea

is that children who were born on the first of April 1932 were the first that could have been conceived after the May 1931 announcement if we allow a month to conceive. Likewise, there was no incentive to time a birth to occur after 15 November 1933, since these women and births would be covered during less than half of the pregnancy, and ineligible for the infant care services. So as to test whether children born after the intervention ended are a valid control group, we define a second new variable which identifies these cohorts. We interact both variables with an indicator for treated parish (or city). So as to investigate whether anything else was going on that coincided with the infant program and changed the composition of births, we estimate regressions including the "usual" treatment variable, measuring exposure to the infant program.

We find no evidence of endogenous heterogeneity in fertility responses to the program by age of mother, her wedlock status or the occupational status of the father (coefficient on *treated parish*cohorts exposed to the announcement*, Panel A). Since we use women with at least two births in the sample in the mother fixed effects models, we investigated endogenous selection of these women by testing whether the probability of having a further birth after the index birth was a function of program variables, and we find not. We used district medical records to analyse the (quarterly) birth rate in the local area and the (annual) percentage of births attended by a midwife (Pettersson-Lidbom, 2014). We find no evidence of changes in fertility levels or of access to midwives (Panel B).[16] All coefficients on the indicator for post-intervention cohorts (*treated parish*cohorts born after the end of the program*) are statistically not significantly different from zero, which confirms that there were no post-intervention changes in the level or composition of births.

We also find insignificant coefficients on the baseline measure of exposure to the infant program with one exception. Panel A shows that the infant program coincided with a decrease in the proportion of in-wedlock births of 2.2 percentage points (2.5 per cent), relative to a mean of 89%. This is not a pre-program difference between the treatment and control group as our tests of the matching procedure confirm balance on this characteristic. It suggests that the composition of births shifted towards higher-risk births during the program, a selection effect that will bias our estimates of causal effects of the program on survival, but downward. In the next section, we estimate impacts separately for in-wedlock and out-of-wedlock births, and other sub-groups.

In Panel C we test whether the antenatal care program which some mothers of the infants in our sample were exposed to modified their birth outcomes, in particular, we examine maternal mortality and stillbirth at the individual level and complications during delivery at the district

[16]The discrepancies in "baseline" between Table 3 and Table 2 are mainly due to seasonal fluctuations in the variables: in Table 3 we use the exact daily (quarterly) definition of the baseline period, whereas Table 2 uses the baseline year 1930.

level. The negative signs on the coefficients are possibly indicative of some improvement but they are not statistically distinguishable from zero.

5.5 Treatment Effect Heterogeneity

In this section we explore the impact of the infant care intervention in different population subgroups, identified as those among whom we may expect to find elevated risks of infant mortality (see Table 4). As we estimate intent to treat effects, any difference in impact will reflect both "treatability" and differences in take-up. Below we show that take up does not, in general, vary significantly across the groups we consider.

We first examined whether the program had different impacts for children born in and out of wedlock because children born out of wedlock were a particular concern among policymakers involved in instituting the trial and they continue to be a concern today, for instance, they have worse indicators at birth than other children (Currie and Rossin-Slater, 2015). We find significantly larger program impacts for out of wedlock births. A child with the average duration of exposure is estimated to have experienced a reduction in infant mortality risk of 6.91 percentage points, which is close to 70% of the pre-intervention infant mortality rate of 9.9 percent in this group. By the age of 75, the cumulative effect is as large as 11.44 percentage points, which is a reduction of almost 30% of the baseline risk. This suggests substantial post-infant survival gains although, as for the population average, the difference in the point estimates is not statistically significant.

We defined "young mothers" as mothers who, at the time of birth, were in the bottom third of the age distribution, below the age of 25.8 years at the index birth. There is some evidence that they benefited more than older mothers from the intervention, although in this case, there is no persistence in the advantage accruing to exposed births beyond the age of five.[17] We then analysed impacts for women who were recorded in the data as having experienced a child death before the program was introduced, because previous research has shown that children who have an older sibling who has died are at much greater risk of dying young (see Arulampalam and Bhalotra, 2006; Bhalotra and Soest, 2008). We find no significant difference in child survival for

[17]This difference in our findings for women who give birth young vs women who give birth unmarried may suggest that births out of wedlock suffer lower investments over the lifecycle while births to younger women have different survival prospects for primarily biological reasons- which are evident early in life but then fade. Without further investigation, however, it is hard to be certain. The pre-intervention infant survival rates for younger women were on average better, so a likely explanation of the larger benefit to children of younger women is that they are much more likely to have been first births, and first born children are known to face higher mortality risks. We could in principle investigate this by studying heterogeneity by age of mother conditional on birth order (parity) and by birth order conditional on age, but we do not have information on birth order in our data.

this group but, at the same time, identify significantly larger chances of surviving to age 75.

There is no statistically significant difference in program impact by child sex, neither for survival to age five when we might expect larger impacts for boys as they have higher baseline risks (Waldron, 1983), nor for survival to age 40 when we may have expected larger gains for girls if treated girls were less likely to suffer death during childbirth. We examined heterogeneity in impact by the infant mortality rate in the mothers birth year and parish, on the premise that this proxies disease conditions in her birth year (Bozzoli et al., 2007) and so reflects the stock of her health which is relevant to the survival of her births (Bhalotra and Rawlings, 2011). We find no evidence that this matters for program impacts or even for baseline survival chances. Finally, as the trial years coincided with the Great Depression, we created an indicator for parishes disproportionately affected by the Great Depression, defined as the drop in local taxable earnings being larger than median. We find no difference in impact in these as opposed to less affected parishes.

In conclusion, the evidence indicates that the intervention was successful in reducing health inequalities insofar as relatively vulnerable individuals experienced disproportionately large survival gains. Given evidence that early life health improvements translate into higher levels of education and income in adulthood (Almond, 2006; Bleakley, 2007; Cutler et al., 2010; Lucas, 2010; Bhalotra and Venkataramani, 2011) it seems likely that the programme also led to a narrowing of economic inequalities.

5.6 Programme Utilisation

The estimates discussed thus far are of intent-to-treat effects, which is what most studies in this domain deliver and which is what is relevant to policy formulation. It is nevertheless of interest to study variation in utilisation among the enrolled and, in particular, how this was graded by socioeconomic status as this may contribute to determining where in the distribution program gains lie. Rather unusually, we have access to detailed information on service utilization, measured at the child level as number of visits received at home, plus number of visits to a clinic to see a nurse or doctor. We regress this upon eligibility in a "first stage" regression and then estimate impacts of the program on children who took it up, weighted implicitly by the intensity of their utilization of the program in a series of "second stage" regressions of survival outcomes on utilization, with utilization instrumented by eligibility duration.

The results are in Table 5. We report results for a linear model (column 1) and also for a linear

probability model in which the dependent variable is a binary indicator of enrolment rather than the continuous indicator utilization (column 2). We model utilization conditional on enrolment (i.e. removing cases of zero visits for eligible children) to focus upon intensive margin responses in column 3. In column 4 we present a first stage regression of number of visits (including zero visits) on duration of treatment eligibility. Column 4 includes both eligible and control cohorts and parishes but, in columns 1-3, the sample is restricted to eligible individuals so that we can assess the socioeconomic gradient in take up amongst those who were eligible. As we may expect, the duration of treatment eligibility is a powerful determinant of treatment intensity (utilisation). However, there is no systematic evidence of a socio-economic status gradient in utilization.

5.7 2SLS Estimates

We estimated the impact of actual utilisation on survival rates using two stage least squares and we show estimates using first enrollment (0/1) and then utilization (number of visits including the zeroes), instrumented with eligibility (0/1) and duration of eligibility. See Table 6, where the F tests in the lower panel indicate that our instruments are sufficiently strong.[18] Enrolment in the infant care intervention is associated with a reduction in infant mortality of 6.6 percentage points, and a marginal change in utilisation (an additional visit) is associated with a reduction in infant mortality of 1.1 percentage points. The pattern of results for survival beyond infancy is similar to the pattern of IIT effects, the coefficient on enrolment dipping slightly but insignificantly for survival to ages 5 and 40 but rising to 9.1% percentage points for survival to age 75. The coefficients on number of visits are sizeable and follow the same pattern of rising for the age 75 threshold, but they are less well-determined for post-infant survival.

5.8 Parental Investments

There is considerable interest in the extent to which parental behaviour reinforces or compensates acts of nature or the state (Almond and Mazumder, 2013). If parental investments made during or after the program reinforced the improvement in infant health created by the intervention, then they will have contributed to the divergence of the trajectories of treated and untreated individuals. We investigate this using sibling data and using matching to control for fertility or birth-spacing effects.

[18] We report the statistic for a standard F test and also the variable-specific statistic as suggested by Angrist and Pischke (2008). The negative coefficients on eligibility in the first stage are intercept effects, the coefficients on duration indicate increments in utilization with duration and these are positive.

We select older siblings of index children, focusing on children born before 1 October 1930 who were ineligible because they had crossed infancy when the program was introduced on 1 October 1931. Amongst this group, we compare those with and without *younger* siblings born in the eligibility period. Our sample contains 846 children who were ineligible but had an eligible younger sibling. We match each of these children to three other individuals born in the pre-intervention period: one born in a treatment district but with no eligible younger sibling, a second born in a control district with an eligible younger sibling and a third born in a control district without an eligible younger sibling. The matching procedure was executed with replacement and based on a propensity score. The propensity score was estimated using the birth date, the mother's age and marital status, socioeconomic group of the household head and the maternal disease environment. Table B.6 presents averages of these covariates for the four groups.

By comparing pre-intervention children in the treated and control areas, we estimate the effect of having a younger sibling who was eligible for the infant-care programme. By comparing with control areas, we get rid of 'normal' differences between children with and without younger siblings, including a tendency for children with a sibling in our sample to be closely spaced. The double difference then captures the effect of the younger sibling's eligibility for services. See Figure 8. The DID estimate indicates a survival disadvantage for children with an eligible younger sibling of at least two percentage points, which manifests itself during the first years of life and persists for at least two decades. The effect kicks in around the time when the younger sibling becomes eligibile for the services (typically by being born, or on the 1 October 1931 if born before that date). This suggests that parents may have reinforced the improved health of the index child at the cost of resources expended on the older child. This is consistent with theory (Heckman, 2007; Cunha and Heckman, 2007; Becker and Tomes, 1976) and empirical evidence (Bhalotra and Venkataramani, 2013; Chay et al., 2009). It may also explain why the coefficients estimated with mother fixed effects are larger (though not significantly larger) than coefficients estimated without mother fixed effects. We conducted a similar analysis of impacts on younger siblings of eligible children and found no significant difference, which we interpret as consistent with learning effects from an intervention that had a large information component.

6 Conclusion

The poor state of reproductive and early childhood health in developing countries is regarded as one of the greatest failures of development. In recognition of this, at the 2010 United Nations Summit reviewing progress towards the Millennium Development Goals (MDGs) for 2015, a sum

of over 40 billion USD over five years was pledged to support a Global Strategy for Women's and Children's Health, a concerted worldwide effort initiated by United Nations Secretary-General Ban Ki-moon who said: *We know what works to save women's and children's lives, and we know that women and children are critical to all of the MDGs* (see United Nations, 2010).[19] In fact knowledge of causal effects at a population level is limited. We evaluate a rather rare intervention, designed as a trial and implemented over a short horizon in the 1930s in Sweden, which emphasised "soft" inputs including information, monitoring and support with a large home-visiting component.

We find large effects on infant and longer term survival and the results are especially compelling as infant mortality rates had been more or less constant for about a decade before the trial started, and the trial coincided with the Great Depression. The trial was instrumental in the decision of the Swedish government to rollout a similar program nationwide in 1937, and it seems plausible that it influenced a similar rollout in the other Scandinavian countries at the same time. Our findings have the potential to influence current global health priorities by highlighting that large gains in infant health and, at no further cost, in chronic disease reduction, may be achieved by a relatively low-cost and scale-able intervention. The findings have wider contemporary relevance as a number of recent programs in, for instance, the USA and the UK, involve targeting high-risk mothers with similar programs, for instance, in 2010 the US Congress established the Maternal, Infant, and Early Childhood Home Visiting Program and in 2007 the UK government instituted the Family Nurse Partnership (inspired by the Nurse Family Partnership in the USA).

[19] Child mortality is widely used as an indicator of child health, being much easier to measure and track than a host of morbidities.

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Tables

Table 1. Infant and Future Survival Chances

	d_{0-1}		d_{0-5}		d_{0-40}		d_{0-75}	
A. All Mothers	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treated parish*duration of eligibility	-0.1414**	-0.1997***	-0.0872	-0.1574**	-0.1295*	-0.1973**	-0.2972**	-0.3070**
	(0.061)	(0.064)	(0.057)	(0.066)	(0.076)	(0.094)	(0.143)	(0.152)
AITTI	-1.0890	-1.5387	-0.6717	-1.2128	-0.9980	-1.5199	-2.2894	-2.3651
Pre-Mean	6.617	6.617	8.257	8.257	11.221	11.221	36.535	36.535
r2	0.004	0.031	0.003	0.031	0.003	0.032	0.004	0.042
N	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374
B. Repeat Mothers Pooled								
Treated parish*duration of eligibility	-0.2776***	-0.3516**	-0.1684*	-0.2556	-0.2059*	-0.3204**	-0.3450*	-0.4645**
	(0.101)	(0.140)	(0.095)	(0.155)	(0.120)	(0.130)	(0.203)	(0.225)
AITTI	-2.1388	-2.7086	-1.2971	-1.9692	-1.5862	-2.4685	-2.6578	-3.5788
Pre-Mean	6.612	6.612	8.251	8.251	11.223	11.223	36.491	36.491
r2	0.007	0.049	0.006	0.047	0.006	0.047	0.005	0.061
N	11,406	11,406	11,406	11,406	11,406	11,406	11,406	11,406
C. Mother Fixed Effects								
Treated parish*duration of eligibility	-0.3759***	-0.4948***	-0.2756**	-0.4528***	-0.3052**	-0.4660***	-0.4382**	-0.6691***
	(0.113)	(0.151)	(0.113)	(0.151)	(0.147)	(0.161)	(0.196)	(0.243)
AITTI	-2.8959	-3.8120	-2.1232	-3.4882	-2.3511	-3.5901	-3.3761	-5.1543
Pre-Mean	6.612	6.612	8.251	8.251	11.223	11.223	36.491	36.491
r2	0.012	0.052	0.010	0.048	0.010	0.051	0.005	0.067
N	11,406	11,406	11,406	11,406	11,406	11,406	11,406	11,406

Standard errors clustered at the parish level in parenthesis. Treated parish*duration of eligibility denotes the DID term for exposure to the infant program. d_{0-x} denotes mortality before age x. Each specification includes the number of months of eligibility for each of the interventions and quarter-of-birth dummies for each of the 20 quarters (*QOB Effects*). The odd-numbered columns present the basic DiD specification in which the controls are a treatment-district dummy, quarter*year of birth dummies, as well as an indicator for exposure to the antenatal care intervention (which is an interaction of treated district with duration of eligibility for the antenatal program). The even-numbered columns include a richer set of controls including covariates, parish fixed effects and parish specific trends. $AITTI$ is the intent-to-treat effect for the average eligible individual (i.e. the product of the DID point estimates and the average eligibility period conditional on enrollment). $Pre-Mean$ represents the mortality rate for children born before the start of the eligibility period (starting 2 October 1930). Variable definitions and sources are provided in Appendix A.

Table 2. Characteristics of Matched and Control Districts

	All (1)	Treated (2)	Other (3)	Std. Dif. (2) vs. (3)	Matched (5)	Std. Dif. (2) vs. (5)
Panel A: Matching Characteristics from the 1930 Census.						
Agriculture	0.340	0.324	0.340	-0.040	0.302	0.054
Manufacturing	0.318	0.340	0.318	0.096	0.345	-0.018
Fertile Women	0.121	0.101	0.121	-0.135	0.100	0.060
Income	811	839	810	0.042	847	-0.013
Wealth	2,525	2,703	2,521	0.080	2,655	0.022
Urban	0.334	0.439	0.331	0.158	0.437	0.003
Population	6,271,266	258,418	6,004,052		160,987	
Panel B: Other Pre-Intervention Characteristics.						
Live Birth	0.973	0.974			0.979	-0.024
Wedlock	0.836	0.888			0.884	0.008
Infant Mortality	0.055	0.063			0.064	-0.002
Perinatal Mortality	0.030*	0.017			0.021	-0.017
Infectious Disease	0.005*	0.005			0.006	-0.004
Other Causes	0.020*	0.041			0.038	0.011
Maternal Mortality	348.1	417.275			381.785	0.004
Mother's Age	29.45	29.455			29.610	-0.017
Professional, technical		0.049			0.038	0.037
Administrative, managerial		0.025			0.016	0.046
Clerical		0.016			0.025	-0.045
Sales worker		0.029			0.023	0.031
Service worker		0.022			0.010	0.071
Agricultural		0.297			0.307	-0.015
Production worker		0.426			0.460	-0.048

Panel A contains local characteristics from the 1930 census, which were used to match treated parishes to control parishes. *Panel B* contains other local characteristics in the year 1930 which were not available in the 1930 census. Whenever possible, these characteristics are compared with the national averages; however * signifies that national and local statistics not directly comparable. '*Std Dif.*' presents the standardised difference (cf. Imbens and Wooldridge, 2009); a standardised difference of less than 0.25 is generally viewed as acceptable.

Table 3. Selection and Confounders

Panel A	Mother's Age		Wedlock		SES Agriculture		SES Manufacturing	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treated Parish*Cohort exposed to announcement	-0.0408		-0.0081		0.0041		0.0285	
	(0.213)		(0.012)		(0.030)		(0.021)	
Treated Parish*Cohorts born after the end of program	-0.0339		0.0016		0.0299		0.0162	
	(0.230)		(0.008)		(0.035)		(0.025)	
Treated Parish*duration of eligibility for infant program		0.0093		-0.0022**		-0.0009		0.0008
		(0.018)		(0.001)		(0.002)		(0.002)
Pre-Mean	29.576	29.576	0.881	0.881	0.280	0.280	0.452	0.452
r2	0.002	0.002	0.002	0.002	0.003	0.003	0.002	0.002
N	25,029	25,029	25,029	25,029	25,029	25,029	25,029	25,029

Panel B	Has Younger Sibling		Birth Rate		Midwife Share	
	(1)	(2)	(3)	(4)	(5)	(6)
Treated Parish*Cohort Exposed to announcement	-0.0113		-0.1042		1.9853	
	(0.024)		(0.165)		(3.064)	
Treated Parish*Cohorts born after the end of program	-0.0223		-0.0619		2.9423	
	(0.049)		(0.187)		(3.320)	
Treated Parish*duration of eligibility for infant program		0.0011		0.0162		0.2061
		(0.002)		(0.016)		(0.258)
Pre-Mean	0.432	0.432	5.084	5.084	67.836	67.836
r2	0.202	0.202	0.628	0.628	0.906	0.906
N	25,039	25,039	2,127	2,127	235	235

Panel C	Maternal Mortality	Stillbirth	Complication
	(1)	(2)	(3)
Treated Parish*Eligibility for maternal intervention	-7.2133	-0.0243	-0.0454
	(21.385)	(0.074)	(0.045)
Pre-Mean	400.266	1.990	2.967
r2	0.010	0.015	0.674
N	24,710	25,029	235

See Notes to Table 1. Each specification also includes all the terms used to build the interaction terms of interest.

Table 4. Effect Heterogeneity

	Single Mother				**Young Mother**				**Child Female**			
	d_{0-1}	d_{0-5}	d_{0-40}	d_{0-75}	d_{0-1}	d_{0-5}	d_{0-40}	d_{0-75}	d_{0-1}	d_{0-5}	d_{0-40}	d_{0-75}
Treated parish*duration of eligibility×_$Variable$	-0.8079***	-0.8806***	-0.8843***	-1.3574*	-0.1874*	-0.2509**	-0.0990	-0.1537	0.2070	0.1834	0.1385	-0.0488
	(0.246)	(0.288)	(0.266)	(0.720)	(0.113)	(0.106)	(0.152)	(0.273)	(0.138)	(0.175)	(0.163)	(0.251)
Treated parish*duration of eligibility	-0.0894	-0.0392	-0.0649	-0.1280	-0.0735	0.0031	-0.1010	-0.1866	-0.2709**	-0.2172*	-0.2223	-0.2433
	(0.063)	(0.066)	(0.098)	(0.167)	(0.081)	(0.077)	(0.133)	(0.206)	(0.113)	(0.128)	(0.141)	(0.219)
AITTIVariable	-6.913	-7.086	-7.313	-11.443	-2.010	-1.909	-1.540	-2.622	-0.492	-0.260	-0.645	-2.251
AITTI	-0.689	-0.302	-0.500	-0.986	-0.566	0.024	-0.778	-1.438	-2.087	-1.673	-1.712	-1.874
Pre-MeanInter	9.891	10.948	12.878	39.054	6.312	7.554	10.420	36.564	5.559	7.170	9.474	29.043
Pre-MeanAll	6.617	8.257	11.221	36.535	6.617	8.257	11.221	36.535	6.617	8.257	11.221	36.535
r2	0.029	0.029	0.030	0.040	0.028	0.029	0.030	0.040	0.028	0.029	0.030	0.040
N	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374
	Previous Mortality				**Maternal IMR**				**Crisis**			
	d_{0-1}	d_{0-5}	d_{0-40}	d_{0-75}	d_{0-1}	d_{0-5}	d_{0-40}	d_{0-75}	d_{0-1}	d_{0-5}	d_{0-40}	d_{0-75}
Treated parish*duration of eligibility×_$Variable$	-0.9111	-1.4540*	-1.3856	-2.0156**	0.2485	0.1441	0.2049	-0.2365	-0.0084	-0.0593	0.0347	-0.4158
	(0.890)	(0.811)	(0.914)	(0.798)	(0.212)	(0.200)	(0.253)	(0.268)	(0.125)	(0.130)	(0.185)	(0.286)
Treated parish*duration of eligibility	-0.0872	-0.0369	-0.0674	-0.1948	-0.2908**	-0.1958	-0.2539	-0.1484	-0.1702*	-0.1031	-0.1708	-0.0663
	(0.070)	(0.072)	(0.099)	(0.147)	(0.142)	(0.124)	(0.181)	(0.198)	(0.088)	(0.086)	(0.141)	(0.210)
AITTIVariable	-7.691	-11.486	-11.193	-17.029	-0.326	-0.398	-0.377	-2.965	-1.375	-1.250	-1.048	-3.714
AITTI	-0.672	-0.284	-0.519	-1.501	-2.240	-1.508	-1.956	-1.143	-1.311	-0.794	-1.316	-0.511
Pre-MeanInter	96.688	97.024	97.024	97.976	6.471	7.937	10.763	37.300	7.737	10.006	13.533	39.013
Pre-MeanAll	6.617	8.257	11.221	36.535	6.617	8.257	11.221	36.535	6.617	8.257	11.221	36.535
r2	0.152	0.125	0.095	0.054	0.029	0.029	0.030	0.040	0.028	0.028	0.030	0.040
N	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374	24,374

Standard errors clustered at the parish level in parentheses. Treated parish*duration of eligibility is the indicator to exposure to the infant intervention. $Treatedparish * durationofeligibility \times \_Variable$ denotes the interaction with the variable in the column heading. $AITTIVariable$ represents the intent-to-treat effect for the average eligible individual with the characteristics given in the column head. $Pre-MeanInter$ represents the pre-intervention mortality rate for the corresponding group. *Previous Mortality* is a dummy variable which takes on the value one if the mother had a child in the pre-intervention period which died before their first birthday. *Maternal IMR* is a dummy variable which takes on the value one if the infant mortality rate in the mother's birth parish was above the median for her birth year. Each specification includes the richer set of controls including covariates, parish fixed effects and parish specific trends. Variable definitions and sources are provided in Appendix A. Also see notes to Table 1.

Table 5. Utilisation & First Stage

	(1)	(2)	(3)	(4)
	OLS	LPM	Conditional on Enrollment	1st
Duration of eligibility(months)	0.2884***	0.0457***	0.0271	0.2502***
	(0.097)	(0.003)	(0.090)	(0.072)
Female child	0.2427	0.0012	-0.1743	0.0590
	(0.197)	(0.018)	(0.173)	(0.053)
Twin birth	0.1135	0.0827	-1.4014*	0.0185
	(0.407)	(0.073)	(0.804)	(0.104)
In-wedlock birth	0.4566	-0.0181	1.4442*	0.1442
	(0.479)	(0.031)	(0.758)	(0.157)
Birth to young mother	0.0728	-0.0118	-0.4452	0.0167
	(0.115)	(0.022)	(0.433)	(0.031)
Birth to old mother	-0.0301	-0.0346	-0.3329	-0.0156
	(0.137)	(0.022)	(0.327)	(0.037)
High Socio-economic status	0.1561	0.0047	1.2994	0.0340
	(0.182)	(0.030)	(0.984)	(0.054)
Low Socio-economic status	0.0140	-0.0793	0.1001	0.0056
	(0.407)	(0.068)	(0.407)	(0.094)
Infant mortality in Mother's birth year	1.3543	-0.2178	7.4428	0.4538
	(2.407)	(0.266)	(4.703)	(0.810)
Crisis area	-4.0298	-0.2395	-4.8022*	-0.9633
	(4.213)	(0.163)	(2.806)	(0.933)
r2	0.065	0.206	0.035	0.167
N	2,574	2,574	624	9,792

Standard errors clustered at the parish level in parenthesis. The first three columns include individuals who were born in the treated districts for which we have utilisation data in a period that implies eligibility for the programme. The fourth column is a first-stage estimate, for which births from other periods and from the matched locations are also included. Variable definitions and sources are provided in Appendix A.

Table 6. Two Stage Least Squares Estimates

Second Stage	d_{0-1}		d_{0-5}		d_{0-40}		d_{0-75}	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Enrolment Infant	-6.6905***		-3.6463		-5.6649**		-9.0833*	
	(2.225)		(2.378)		(2.332)		(5.074)	
Utilisation Infant		-1.1409***		-0.6445		-1.0494*		-1.7833
		(0.420)		(0.455)		(0.620)		(1.147)
Pre-Mean	8.476	8.476	10.083	10.083	12.988	12.988	37.798	37.798
r2	0.067	0.057	0.079	0.076	0.107	0.103	0.337	0.327
N	9,792	9,792	9,792	9,792	9,792	9,792	9,792	9,792
First Stage	Infant							
	Enrollment	Utilization						
Treated parish*duration of eligibility	0.0449***	0.2518***						
	(0.003)	(0.090)						
Eligible infant	-0.0174	-0.3594**						
	(0.053)	(0.142)						
Ftest	136.0	21.4						
pValueF	0.000	0.000						
APFtest	126.1	6.9						
pValueAP	0.000	0.001						
r2	0.51	0.17						
N	9,792	9,792						

Standard errors clustered at the parish level in parenthesis. *Enrolment Infant* is a dummy and *Utilisation Infant* is a count variable capturing the number of visits during infancy including zero visits. Enrollment and utilization are instrumented with program eligibility. Treated parish*duration of eligibility represents the length of the eligibility period in months. $Ftest$ ($pValueF$) present the test statistic (p value) for a first-stage F test of excluded instruments; $APFTest$ ($pValueAP$) represent the corresponding test statistic (p value) for Angrist and Pischke's test for weak identification (cf. Angrist and Pischke, 2008).

Figures

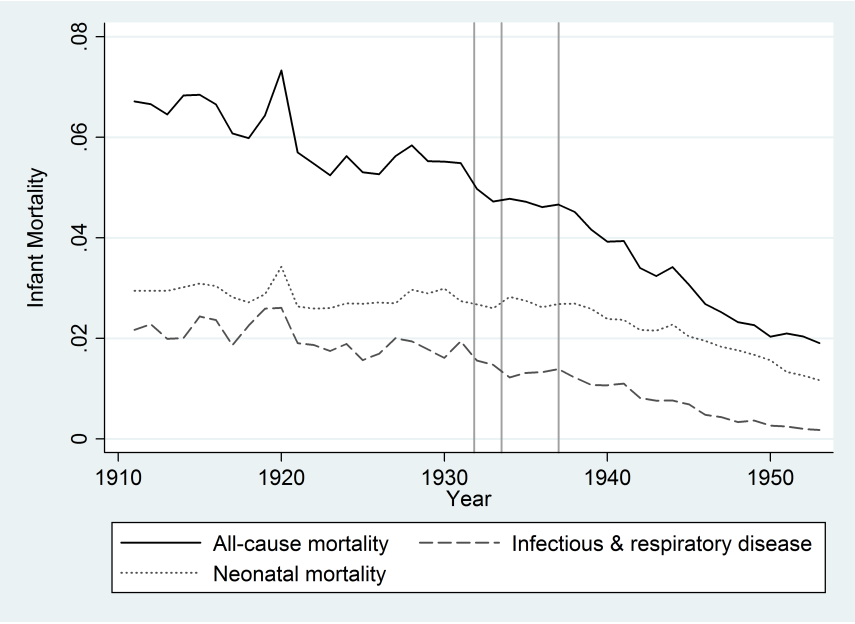

Figure 1. Infant (All-Cause and Cause Specific) Mortality Rates in Sweden 1910–40.


Brunns Råsvaruaffär.
Södra Storg. 43. (Betaniakapellets hus). Tel. 2713. Helsingborg.

MEDDELANDE

angående

moderskaps- och barnavård.

Med anledning av Riksdagens beslut kommer hädanefter — tills vidare intill 1 juli 1932 — s. k **för- och eftervård vid barnsbörd** att anordnas för blivande mödrar och spädbarn från Helsingborg med omnejd. — Vården är **kostnadsfri.**

Mottagningar:

För blivande mödrar å Helsingborgs stads barnbördshus (D:r E. Jerlov): Måndagar kl. 6—7 e. m., onsdagar kl. 12—1 e. m.

För spädbarn å Mjölkdroppens barnavårdscentral, Karl Krookegatan 60 (D:r D. Lindsjö): onsdagar, torsdagar kl. 4—5 e. m.

Annonsera i Helsingborgs Dagblad.

Figure 2. Advertisement from Daily *Helsingborgs Dagblad* (1931) announcing the services and opening hours of the centres for infant care services.

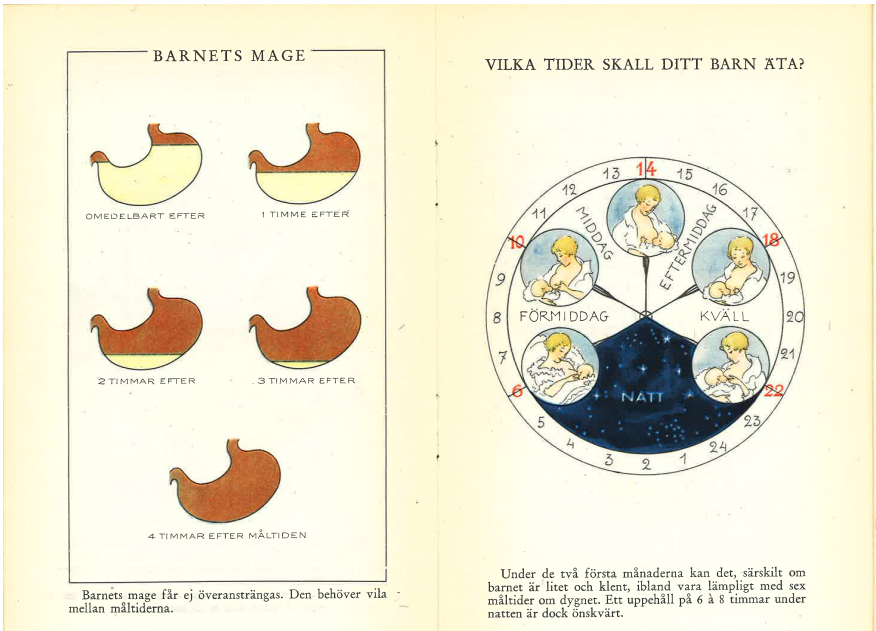

Figure 3. Advice on appropriate feeding of infants from leaflet provided within the Infant Care Intervention. The left panel shows how the stomach of the infant empties in the 4 hours following a feed and the right panel suggests intervals for breastfeeding.

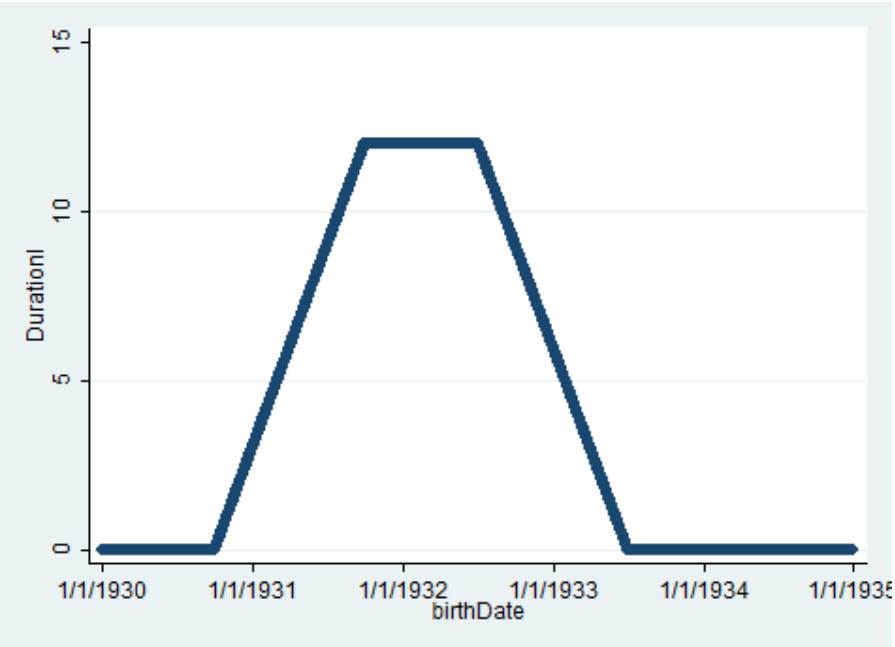

Figure 4. Eligibility duration by birth date of child.

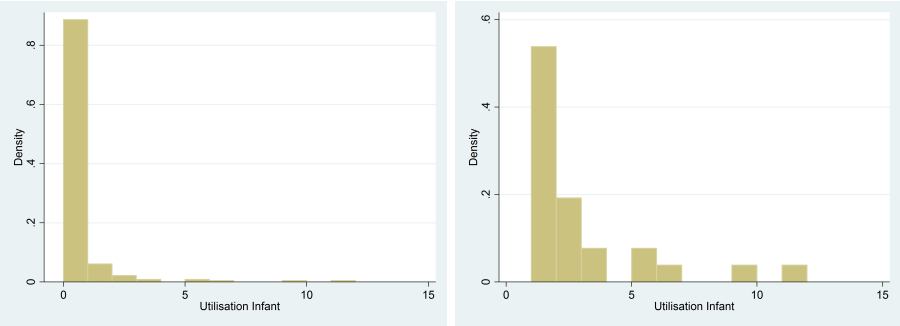

Figure 5. Histograms of utilisation (Number of Visits). Left panel includes zero visits and right panel excludes them.

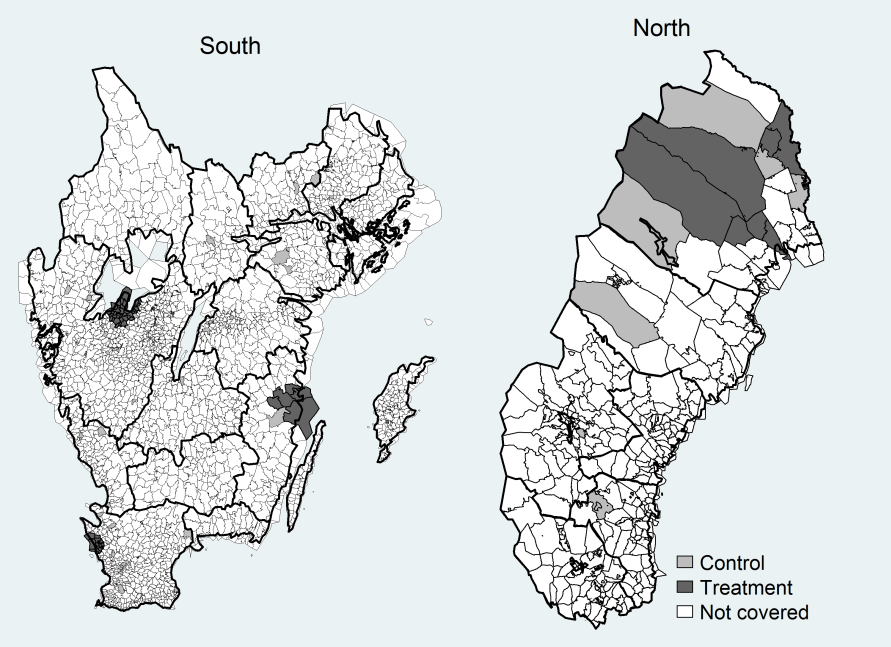

Figure 6. Municipalities containing treated and control districts.

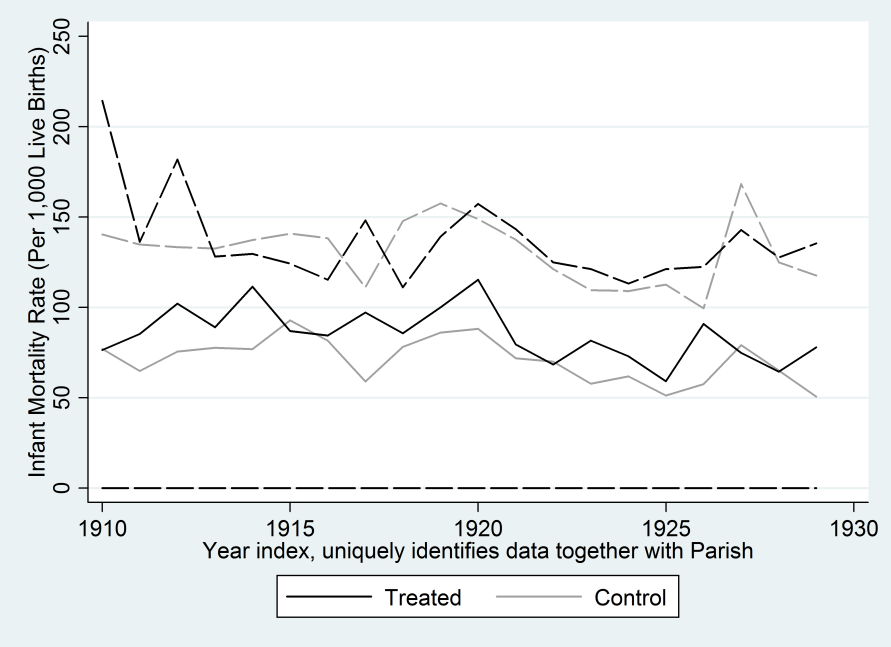

Figure 7. Pre-intervention trends in infant mortality rates in treated and control locations – Median, 10^{th} and 90^{th} Percentiles. Black lines (solid or dashed) correspond to treated areas, while grey lines (solid or dashed) correspond to control areas. For the 10th percentile the lines for treated and control areas coincide.

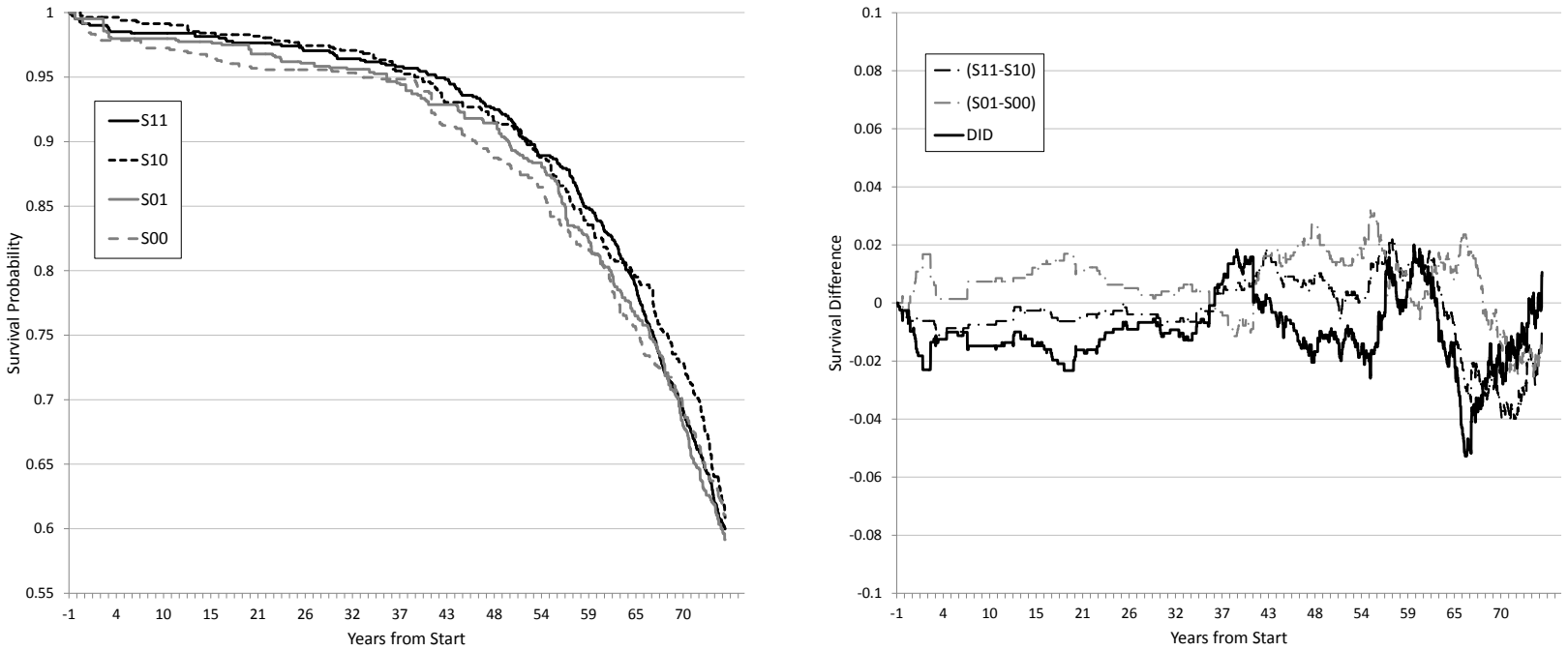

Figure 8. Survival Curves for Children Born Before the Intervention. (Also see Table B.6). The left figure plots survival curves for sub-groups of children as follows. Black curves refer to children born in treated areas and grey curves to children born in control areas. Solid curves refer to children with younger siblings born in the eligible period and dashed curves refer to children without younger siblings born in the eligible period. The x axis measures time in years with reference to the start of sibling eligibility: if the sibling's conception was before 1 October 1931, the start is on that day, and otherwise at the time of the sibling's conception. The right figure shows single and double differences: when subtracting the curve $S10$ (treated area, has no eligible sibling) from the curve $S11$ (treated area, has eligible sibling) we get the difference in survival prospects between the two groups, and by calculating the double difference $S11 - S10 - (S01 - S00)$, we capture the effect of sibling eligibility.

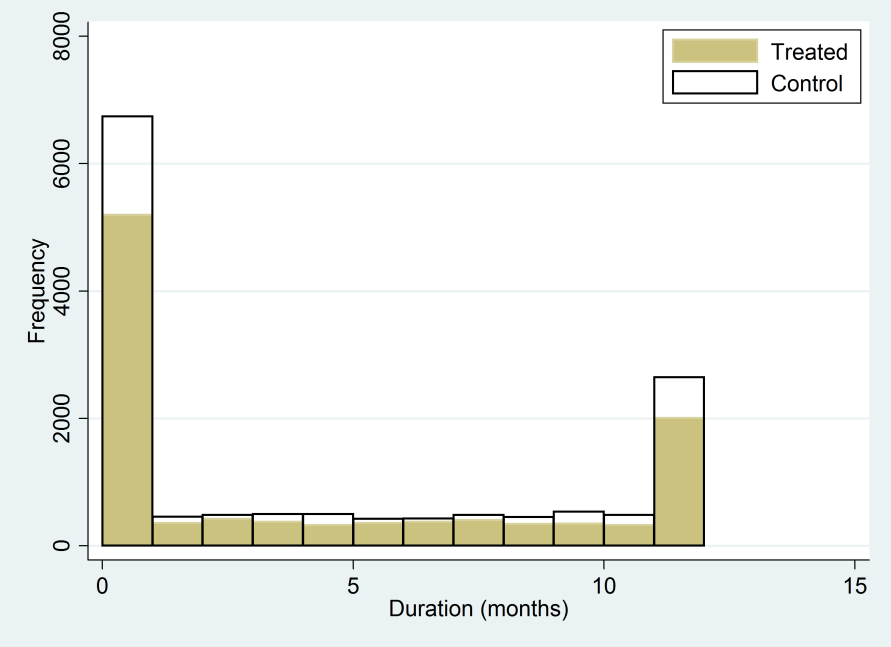

Figure 9. Histogram for eligibility in months, and the corresponding theoretical eligibility for the control group.

Appendix

A Variable Definitions

A.1 Information from Parish Records

Infant Mortality (d_{0-1}): dummy variable taking on value one for live births who die within a year of birth.

Child Mortality (d_{0-5}): dummy variable taking on value one for live births who die within five years of birth.

0-40 Mortality (d_{0-40}): dummy variable taking on value one for live births who die within 40 years of birth.

0-75 Mortality (d_{0-75}): dummy variable taking on value one for live births who die within 75 years of birth.

Female Dummy variable taking on the value one for female births.

Twin Dummy variable taking on value one for (mono- and dizygotic) twins.

Wedlock Dummy variable taking on value one for children born to married mothers.

Mother's Age The mother's age at the time of birth.

MYoung Dummy variable taking on value one if the mother belongs to the youngest third of mothers (age < 25.8) at the time of the birth.

MOld Dummy variable taking on value one if the mother belongs to the oldest third of mothers (age > 32.3) at the time of the birth.

Maternal IMR The infant mortality rate in the parish and year of birth of the mother (defined as deviation from local average and overall time trend).

Treated Dummy variable taking on value one for children born in treated areas.

DurationI Duration in months of potential eligibility for infant care intervention.

SES Classification of head of household profession according to HISO 9-point scale (Leeuwen et al., 2002).

Birthrate Births in a quarter per fertile married woman in the parish.

A.2 Information from Other Sources

Crisis Parish-level shortfall in taxable incomes per capita compared to 1930 level (%), measured in the birth year. Source: official tax records.

Midwife Share Proportion (in per cent) of births in health district attended by a midwife. Source: district physicians.

Complications Proportion (in per cent) of midwife-attended births in health district with complications reported. Source: district physicians.

Enrolment Infant Equals one if the child had at least one visit/home call during infancy. Source: official records from the intervention.

Utilisation Infant Number of visits during infancy. Source: official records from the intervention.

B Appendix Tables

Table B.1. Mothers and Children Enrolled in the Test Programme.

	Infant Care		
District	Enrolled	Eligible	Ratio
Harads	225	269	.84
Hälsingborg	646	2025	.32
Jokkmokk	227	298	.76
Lidköping	550	1060	.52
Mörtfors	363	445	.82
Pajala	344	552	.62
Råneå	230	369	.62

Table B.2. Descriptive Statistics, Individual Level.

Variable Name	Mean	Std. Dev.	Min	Max	*N*
Infant Mortality	5.821	23.41	0.00	100.00	24,374
Child Mortality	7.343	26.08	0.00	100.00	24,374
Died40	10.536	30.70	0.00	100.00	24,374
Died75	34.661	47.59	0.00	100.00	24,374
Maternal Mortality	318.752	5,636.93	0.00	100,000.00	24,710
Stillbirth	2.509	15.64	0.00	100.00	25,029
Female	0.487	0.50	0.00	1.00	25,029
Twin	0.031	0.17	0.00	1.00	25,029
Wedlock	0.893	0.31	0.00	1.00	25,029
Maternal Age	29.495	6.57	13.97	51.36	25,029
Maternal IMR	-0.001	0.02	-0.16	0.33	25,029
Treated	0.510	0.50	0.00	1.00	25,029
DurationI	4.274	4.82	0.00	12.00	25,029
SES Professional/Technical	0.046	0.21	0.00	1.00	25,029
SES Administrative/Managerial	0.022	0.15	0.00	1.00	25,029
SES Clerical	0.019	0.14	0.00	1.00	25,029
SES Sales	0.030	0.17	0.00	1.00	25,029
SES Service	0.017	0.13	0.00	1.00	25,029
SES Agricultural	0.319	0.47	0.00	1.00	25,029
SES Production	0.432	0.50	0.00	1.00	25,029
SES Unknown	0.115	0.32	0.00	1.00	25,029
Crisis	0.045	0.19	-0.59	0.61	25,029
YOB 1930	0.210	0.41	0.00	1.00	25,029
YOB 1931	0.206	0.40	0.00	1.00	25,029
YOB 1932	0.204	0.40	0.00	1.00	25,029
YOB 1933	0.189	0.39	0.00	1.00	25,029
YOB 1934	0.191	0.39	0.00	1.00	25,029

Variable definitions and sources are provided in Appendix A.

Table B.3. Descriptive Statistics, District Level.

Variable Name	Mean	Std. Dev.	Min	Max	*N*
Birthrate	5.34	2.82	0.65	23.83	2,127
Midwife Share	69.81	20.96	15.28	100.00	240
Complications	2.31	2.01	0.00	8.71	240

Variable definitions and sources are provided in Appendix A.

Table B.4. Descriptive Statistics, Utilisation Sample.

Variable Name	Mean	Std. Dev.	Min	Max	*N*
Enrolment Infant	0.122	0.33	0.00	1.00	9,793
Utilisation Infant	0.523	2.61	0.00	100.00	9,793
Infant Mortality	5.085	21.97	0.00	100.00	9,793
Child Mortality	6.562	24.76	0.00	100.00	9,793
Died40	9.674	29.56	0.00	100.00	9,793
Died75	32.939	47.00	0.00	100.00	9,793
Female	0.487	0.50	0.00	1.00	9,793
Twin	0.030	0.17	0.00	1.00	9,793
Wedlock	0.909	0.29	0.00	1.00	9,793
Maternal Age	29.896	6.65	14.31	51.36	9,793
Maternal IMR	-0.003	0.03	-0.16	0.33	9,793
Treated	0.472	0.50	0.00	1.00	9,793
DurationI	4.203	4.80	0.00	12.00	9,793
SES Professional/Technical	0.047	0.21	0.00	1.00	9,793
SES Administrative/Managerial	0.024	0.15	0.00	1.00	9,793
SES Clerical	0.013	0.11	0.00	1.00	9,793
SES Sales	0.022	0.15	0.00	1.00	9,793
SES Service	0.011	0.10	0.00	1.00	9,793
SES Agricultural	0.434	0.50	0.00	1.00	9,793
SES Production	0.347	0.48	0.00	1.00	9,793
SES Unknown	0.102	0.30	0.00	1.00	9,793
Crisis	-0.014	0.13	-0.59	0.45	9,793
YOB 1930	0.202	0.40	0.00	1.00	9,793
YOB 1931	0.204	0.40	0.00	1.00	9,793
YOB 1932	0.201	0.40	0.00	1.00	9,793
YOB 1933	0.196	0.40	0.00	1.00	9,793
YOB 1934	0.197	0.40	0.00	1.00	9,793

Variable definitions and sources are provided in Appendix A.

Table B.5. Descriptive Statistics, Mothers in the Fixed Effects Sample.

Variable Name	Mean	Std. Dev.	Min	Max	*N*
Infant Mortality	7.285	25.99	0.00	100.00	11,406
Child Mortality	9.182	28.88	0.00	100.00	11,406
Died40	12.707	33.31	0.00	100.00	11,406
Died75	36.744	48.21	0.00	100.00	11,406
Maternal Mortality	98.372	3,135.03	0.00	100,000.00	11,198
Stillbirth	0.829	9.07	0.00	100.00	11,494
Female	0.488	0.50	0.00	1.00	11,494
Twin	0.063	0.24	0.00	1.00	11,494
Wedlock	0.939	0.24	0.00	1.00	11,494
Maternal Age	29.700	6.14	15.76	50.95	11,494
Maternal IMR	-0.000	0.03	-0.13	0.33	11,494
Treated	0.522	0.50	0.00	1.00	11,494
DurationI	4.278	4.84	0.00	12.00	11,494
SES Professional/Technical	0.041	0.20	0.00	1.00	11,494
SES Administrative/Managerial	0.017	0.13	0.00	1.00	11,494
SES Clerical	0.011	0.11	0.00	1.00	11,494
SES Sales	0.023	0.15	0.00	1.00	11,494
SES Service	0.014	0.12	0.00	1.00	11,494
SES Agricultural	0.390	0.49	0.00	1.00	11,494
SES Production	0.410	0.49	0.00	1.00	11,494
SES Unknown	0.092	0.29	0.00	1.00	11,494
Crisis	0.071	0.22	-0.59	0.61	11,494
YOB 1930	0.209	0.41	0.00	1.00	11,494
YOB 1931	0.195	0.40	0.00	1.00	11,494
YOB 1932	0.211	0.41	0.00	1.00	11,494
YOB 1933	0.190	0.39	0.00	1.00	11,494
YOB 1934	0.195	0.40	0.00	1.00	11,494

The summary statistics presented are for the subsample of women who gave birth to at least one child during the study period. Variable definitions and sources are provided in Appendix A.

Table B.6. Ineligible Children Born Before the Intervention With and Without Younger Siblings.

	Treated		**Control**	
Variable	S11	S10	S01	S00
Birthdate	1930.377	1930.357	1930.386	1930.382
Maternal Age	28.866	29.005	28.924	28.270
Wedlock	0.936	0.929	0.928	0.916
Maternal IMR	0.001	0.001	-0.001	0.003
SES High	0.071	0.085	0.092	0.077
SES Low	0.159	0.136	0.158	0.155

The table show observable characteristics of children born before the intervention who were matched according to the protocol described on page 21. $S11$ denotes children who were born before the intervention in a treated region, who had an eligible younger sibling. $S10$ denotes children who were born before the intervention in a treated region who did *not* have an eligible younger sibling. $S01$ denotes a child born in a control region with a younger sibling born in the intervention period, and $S00$ denotes a child born in a control region without a younger sibling born during the intervention period.