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Abstract

This paper investigates the potential of maternal and infant health programs to improve the life expectancy of women and children. We study a program trialed in 1931-1933 in seven Swedish medical districts before it was rolled out at the national level in 1937, digitizing and assembling individual data from parish records and birth and death registers and aggregate data from annual reports of medical districts. We estimate short run program effects on (first generation) maternal and infant mortality. In addition, we track individuals exposed to the program together with unexposed individuals from neighboring birth-cohorts so to establish whether they survive to age 40, an age by which maternal mortality of second-generation offspring is determined, and age 75, an age by which 35% of the sample cohorts had died. We find substantial and statistically significant impacts of exposure to the infant program on infant survival and on the probability of surviving to ages 5, 40 and 75, and these impacts are not differentiated by gender. The estimates suggest that the impact on life expectancy is largely driven by infant survival. The program narrowed health inequalities. In particular its impact was systematically larger among children of younger mothers and women who gave birth out of wedlock, who also exhibit higher baseline rates of infant mortality. There is no evidence of endogenous fertility responses or of selection into the program on a range of relevant observables. The evidence is consistent with parents reinforcing treatment by the public health intervention. We are unable to identify any impacts of program components delivered to mothers before birth on either maternal or infant mortality.

JEL Classification: I15, I18, H41.

Keywords: Maternal care, Infant care, Early life interventions, Barker Hypothesis, Program evaluation, Sweden.

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1 Introduction

Life expectancy in rich countries increased by 30 years in the course of the twentieth century, a historically unprecedented improvement that occurred largely on account of the control of infectious disease. As infectious disease tends to be most prevalent amongst infants and, conditional upon incidence, to be more likely to result in mortality for infants, twentieth century life expectancy gains mirrored improvements in infant and child survival \cite{Cutler2006}. We present what is possibly the first micro-data analysis that attempts to identify causal effects of publicly-provided maternal and infant care on infant survival and life expectancy using data on exposure to a trial initiated in a seven medical districts in Sweden in 1931–1933, after which the intervention was rolled out nationwide in 1937.

The current gap in life expectancy between rich and poor countries is, as it happens, about thirty years: a century’s worth. This cross-region difference reflects the failure of today’s poor countries to control infectious disease despite having the know-how that was critical to progress in the early twentieth century. In 2005, more than 10 million children under the age of five died in a year and more than half a million women died at birth or from pregnancy-related causes. In both cases, most of these deaths were preventable. The Partnership for Maternal Newborn and Child Health was set up by the WHO in 2005, in unison with UNICEF and UNFPA \cite{WHO}. In 2010, the United Nations Secretary-General launched the Global Strategy for Women’s and Children’s Health with the support of The Partnership, galvanizing further public and government support for interventions directed at maternal and newborn health (also see \cite{Engle2007, Bergsjo1997}).

Despite the enormity of the problem and the resources now available to potentially address it, the evidence base available to guide policy choice is small. A scatter of randomized control trials in developing countries establish large impacts of prenatal and newborn programmes on neonatal survival \cite{Bang1999} and recent studies set in richer countries exploit the discontinuity in access to post-partum neonatal care created by the low-birth weight threshold that defines access \cite{Freedman2009, Almond2011, Bharadwaj2011}. However we are unaware of any long term follow up that investigate impacts of early life exposure on life expectancy or other later life outcomes. Similarly not many studies have analysed long term effects of public health programmes: \cite{Hjort2014} is a notable exception that analyses the Danish Home Visiting Program.
Using individual birth and death records for the 57 parishes and two cities in the seven treated medical districts and corresponding data from two matched control cities and 57 matched control parishes for the years 1930-1934, we digitized and assimilated data on infant and maternal deaths along with relevant characteristics of the mother and child for more than 25,000 births of 15,000 mothers from church books. We matched the historical micro data to administrative data on mortality (death registers) by combining manual matching from the church books for deaths occurring during the first 10-15 years of life, and matching based on an algorithm (using place of birth, date of birth, and forenames) for all deaths occurring before 2010; thereby tracing individuals in our sample to the present date, roughly 80 years since the initiation of the reform. For some of the test districts, covering roughly half of the eligible population, we also obtained service utilization by date and component of the intervention. We also compiled data from annual medical district reports with municipality-level information on availability of midwives, complications at birth and child and maternal mortality for treated and control areas.

The trial included components delivered to mothers before giving birth and components delivered to infants. We define months of exposure to the programme using programme eligibility rules for pregnant women and newborn children, exploiting availability on the date of conception and birth. We then estimate the impacts of exposure duration (which is purely a function of semester and age for woman and child respectively) in treated vs control districts. To control for selection into fertility as well as selection into uptake driven by time-invariant maternal characteristics (preferences), we estimate models with mother fixed effects. Since only a minority of the women in the sample had more than one birth (in the period 1930–34), we also present estimates without mother fixed effects.

We find large and significant impacts of infant eligibility in treated areas on infant survival and the probability of the individual surviving to the ages of 5, 40 and 75, with the last proxying life expectancy. The activities funded by the Swedish Government in the 1930’s 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. The results suggest that programme-determined gains in the chances of surviving to age 75 are largely driven by programme-led improvements in the chances of surviving infancy. Relatively vulnerable groups with higher pre-intervention rates of infant (and maternal) mortality exhibit larger benefits from infant exposure; these include births out of wedlock and births to relatively young women. Our estimates suggest that children born to single mothers experienced a reduction in infant mortality by as much as 7
percentage points, and that vulnerable groups thus were responsible for a substantial share of the overall gain of roughly one percentage point. In this way, the programme caused a narrowing of inequalities in health. 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.

There is no evidence that fertility responded to the programme, either on average or by mother’s age, marital status at birth or occupation of household head, possibly because of the short duration of the programme. However, it was known from the outset that the programme would be discontinued, so short-term responses could in principle have been expected. Examining sibling data we find evidence that parental investments favour treated children, reinforcing public investments. This result is consistent with theory (Heckman, 2007; Cunha and Heckman, 2007; Becker and Tomes, 1976) and with the available empirical evidence (Almond and Mazumder, 2013). We are unable to identify any impacts on maternal mortality of either infant eligibility or of the mother’s eligibility for prenatal services.

2 Infant and Maternal 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) and Sweden was no exception, but an early starter. Infant mortality was above 10 per cent in 1900 and, by 1917, it had dropped to 6.5 per cent (Corsini and Viazzo, 1997; Brändström, 1988). However, from 1920 to 1930, there were no further substantive declines and a similar stagnation was noted in Denmark and the US (Moehling and Thomasson, 2014; Wiist, 2012). In particular, deaths occurring during the first few days of life, often because of delivery complications or imperfect expansion of the lungs, were increasing even in absolute terms. The number of stillbirths was constant, suggesting that these trends were not on account of selection in to live birth.

Maternal mortality was also rising in Sweden at this time, from 0.25 per cent in 1920 to 0.32 per cent in 1928 (Steenhoff, 1931). This too reflected an international pattern; Although maternal mortality declined substantially in the late nineteenth century, it did not decrease in any Western country between 1910 and 1935, and most countries had an upward trend (Loudon, 2000).
Figure 1 shows trends in infant mortality and maternal mortality in Sweden between 1910–1955. The vertical lines represent the duration of the trial (and the subsequent roll-out of a nationwide antenatal- and neonatal care scheme from 1937 onwards). The state of public health, indicated by infant and maternal mortality, in contemporary developing countries is broadly similar to that in Sweden in the early twentieth century (Razavi, 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, which led to the implementation of the trial that we analyze. This public attention in Sweden was part of an international phenomenon known as the infant welfare movement (Fildes et al., 2013) – a movement fuelled by concerns about population decline, exacerbated by the First World War (Davis, 2011).

A major policy focus in the 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). About 15 per cent of all children in Sweden were born out of wedlock, a share that was more or less constant over the 1920’s and 1930’s (Statistics Sweden, 1940). Another focal point related to the fact that less than 5 per cent of all pregnant women had been to the doctor before giving birth.

3 The Field Experiment

In 1929, a Royal Commission was charged with the task of modernising maternal and neonatal care and it proposed the trial we analyse which, in December 1930 was approved by the National Board of Health (Medicinalstyrelsen). The final proposal, which involved funding of SEK 30,000 (USD 133,000 in current prices), was accepted in the Swedish Parliament on 29 May 1931. Seven

1 A wide range of factors have been suggested in explaining the rising trend in maternal mortality appearing in Sweden and other countries suffering a similar experience, e.g. increasing primiparity, a decline in the health of mothers following from malnutrition and poor housing, increase in the virulence of the streptococcus, and lack of improvement in standard of obstetric care (Loudon, 1992). Another potential cause to the pattern is rising abortion rates. As noted by Högberg and Joelsson (1985) death rates due to abortion increased in Sweden, as elsewhere, through the 1920’s.

2 It was however not uncommon to give birth with the help of a midwife. A national system of midwives was introduced in Sweden in 1908. However the transition to institutionalized childbirth did not take off till the late 1930’s, after the maternal intervention that we analyse. In 1920, 12 per cent of all children were delivered in a confinement facility, in 1935 38 percent, and in 1940 65 percent.
health districts were selected to receive free ante- and neonatal care for a delimited period. The trial started on 1 October 1931 and ended June 1933, after an extension decision in the summer of 1932.

The intervention 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. The sample of seven districts was selected to reflect diversity in local conditions with respect to *inter alia*, population density, demographic structure, and living standards. Importantly, the outcome variables we analyse (infant, child and maternal mortality) were given no consideration in the selection of participating districts.

### 3.1 Organisation of the Activities

The participating districts were Lidköping, Hälsingborg, Harad, Råneå, Jokkmokk, Pajala and Mörfors. 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. Particular emphasis was put on the importance of providing care on equal conditions to all mothers. Participating staff were informed about the objectives of the project and the legislation applying to maternal care, and they visited existing care institutions.

In each test district, a health centre for ante- and neonatal care with regular office hours was started[^3] 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 district of Hälsingborg.

[^3]: In the more remote areas, advice was also given by telephone and urine samples sent by mail.

[^4]: For some districts these forms were digitised and used in the analysis of utilisation below.
Guidance services and examinations at the surgeries

Home visits

Information campaigns

The maternal care services included anamnensis, and antenatal examinations tested temperature, pulse, haemoglobin and urine samples. Pregnant women were given prescriptions, including a plan for prophylactic examinations and careful dietary recommendations (iron, vitamins, salt). The antenatal care also included recommendations for outdoor exercise, personal hygiene and sex, and patients received printed forms provided by the National Board of Health on maternal and infant care.

Visit to the neonatal care centres involved weighting of the child and checks on infant care. Focusing on preventive care, sick children were referred to doctors. 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 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 blutsaft rich in iron for premature children. Figure 3 shows an extract from a leaflet given to all enrollees.

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. Advice was given on hygiene, sanitation and cleanliness in the household.

Program eligibility for infants was determined by their birth date: all children less than one year of age were eligible at the beginning of the trial, and all children born during the duration of the trial became eligible. Expectant mothers were eligible throughout the intervention, irrespective of the stage of their pregnancy. Figure 4 plots eligibility by birth date and age. On average, the participating districts report having had roughly two visits per enrolled mother at the centres, and about 0.9 received a home call. For the infant care intervention utilisation was typically more intense with 2.8 visits and 3.9 home calls per enrolled child.

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].
3.2 Utilisation and Evaluation

Despite some initial scepticism, the project was well received in the test districts. In total, around 2,000 mothers and 2,600 children were enrolled in the trial, which represents a large proportion of eligible families. Two things evident from the descriptive statistics in Table 1 are that utilisation was higher for infant care than for maternal care in all locations, and that the two predominantly urban districts (Lidköping and Hälsingborg) had a lower utilisation rate than the rural districts. However, inside the urban districts – Hälsingborg and Lidköping – families from the cities exhibited higher utilisation than those from the surrounding countryside.

The project was well received among participating staff and physicians of the time attributed substantial improvements in child and maternal health to the intervention. Auditing reports suggest that the intervention induced behavioural changes amongst participating families leading to improvements in diet and hygiene (Steenhoff, 1934). Indeed the trial was so positively evaluated that it led to roll-out of a similar scheme in all parts of the country from 1937. This was reflected in a bill passed in national parliament legislating free maternal and infant care with equal access.

The field trial may hence be seen as a seed in an emerging Swedish welfare state. Before the experiment, there were two types of institutions for infant and family care at some locations in the country, one relying mainly on visits to physicians and the distribution of cow milk mixture to poor mothers (Mjölkdroppen) and the other relying more intensively on home calls by nurses (Barnavårdscentralsystemet; cf. Wallgren, 1936; Stenhammar et al., 2001). However, both institutions were based on local initiatives did not cover the entire population.

With its maternal and infant care initiative, Sweden was a forerunner on the international stage. At the time most Western countries provided some kind of services, but these were often local or private initiatives in larger cities, often targeting certain groups. For instance, antenatal

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6 There was scepticism at the national level too: already in 1914 two motions for better care of childbearing women and their newborns and for the establishment of maternity care facilities were rejected in the National Parliament.

7 Prior to 1937 Mjölkdroppen and Barnavårdscentralsystemet were only present in larger cities. The former primarily focused on families with low socio-economic status, while the latter institution did not have any restrictions with respect to eligibility.
care in the United States was dictated by local circumstances and 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 UK although it was routine practice to visit every newborn in the UK at home by the start of World War II. Home visiting programmes were available in the US in 1920-1929 and in Denmark a universal intervention similar to that in Sweden started in 1937.

4 Data and Empirical Strategy

4.1 Data Sources and Matching Procedure

We use individual level data from the test districts for the years 1930-1934. Using parish records we collected information on all births including stillbirths, children’s sex, mothers’ age and marital status, and subsequent mortality by death cause for both mothers and infants.

The dataset was purpose-built for the project, and the preparation of the dataset entailed several distinct steps. In a first step, we identified a suitable control group for the intervention areas, based on observable parish characteristics in the 1930 census. The best matches in terms of observable characteristics (denoted $J_M(i)$) were identified using the Mahalanobis distance metric, defined as

$$J_M(i) = \arg \min_j \sqrt{(X_i - X_j)' S^{-1} (X_i - X_j)}$$

(1)

where $X_i$ is a vector of observable characteristics for a parish belonging to a test district, in our case, 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. Match quality is assessed by conducting tests of differences in means for a set of observable covariates (see Table 16). It should be noted that the matching algorithm did not consider the outcome variables used in our analysis – so that these can also be used to cross-validate the performance of the algorithm. Figure 5 shows

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8 See Moehling and Thomasson (2014) and Wüst (2012) for evaluations of home visiting programs on infant mortality.

9 In total, the seven medical districts included in the trial comprised two cities and 57 rural parishes. These were matched to two cities and 57 rural parishes which had not been exposed to the intervention, and these control locations belonged to 38 different medical districts.
a map over the treated and control districts, and districts not covered in the analysis.\footnote{The administrative unit used for the maps are parishes, which are a lower level than the medical districts. For example, in the Norrbotten region, there were four test districts containing seven parishes in total.}

In order to get a control group which matches the treatment group on observable characteristics, we weighted observations from the control group based on their population size compared to the population size of the treated locations they had been matched to. Thus, each control observation matched to parish $c$ is weighted according to $w_{JM(c)} = \sqrt{N_c/N_{JM(c)}}$ where $N_c (N_{JM(c)})$ denotes the 1930 population size in the test (control) district. This weighting of observations assures covariance balance for the variables used in the matching procedure. The reduction in bias comes at the cost of reduced efficiency of estimates. However, this reduction in efficiency is likely to be very 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). Indeed, robustness checks showed that all results were insensitive to the weighting scheme used.

Once the parishes to be included – from treated as well as control areas – were identified, we digitised all births from these areas occurring during the five years 1930–34. There were a total of 25,029 individual children (fetuses) in the records, from 24,710 deliveries, resulting in 24,373 births. For each individual we recorded information on parish, date of birth, sex and stillbirth status, along a number of parent characteristics such as date of birth, marital status and profession.\footnote{The data used in our analysis comes from high-quality administrative church records covering individual-level information on a range of variables, such as births, deaths etc, collected by the parish. All newborn children in Sweden automatically became members of the Church of Sweden. Membership could actively be cancelled if an individual wanted to enter another denomination, but church records nevertheless covered all citizens. In the 1930’s, 97 per cent of the population were members of the Church of Sweden.}

The profession variable was later transformed into professional classes based on the HISCO classification (Leeuwen et al. 2002). The parish records also cover all deaths for the period up until 1946 and from this source we digitise information on mortality, along with death cause, during the first 10–15 years of life for our individuals.\footnote{Sweden has a long tradition of collecting and providing death statistics. A very detailed and strict reporting procedure regarding death causes was introduced in 1911, which improved the reporting also 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, for details see the introductory chapter in Dödsorsaker 1911 (Statistics Sweden 1915), [Hyrcius 1914] and [Hultqvist 1940].}

In a next step, the digitised parish records were matched with various other datasets. In order to record adolescent and adult mortality, and to validate information on mortality during childhood and adolescence, we used the Swedish Death Index (cf. Fischer et al. 2013). The
Swedish Death Index includes the universe of all deaths occurring between 1947–2009, and an overwhelming majority of the deaths occurring before 1947. Individual records were matched based on various combinations of date of birth, sex, forenames, surnames and birth parish. To validate mortality matches we use a dataset containing burial records (Swedish Genealogical Society [2012]). As a second source of validation we used tax records from the 2002–2013 period and the 1970 census. The 1970 census was used to get updated information on names; this issue is particularly pressing for females who traditionally change their surnames when getting married. Whenever there was conflicting information between data sources – for example, individuals recorded as earning an income after being deceased – the records were checked manually. Thus, the mortality information in the dataset is based on a careful comparison of several sources and a manual check of conflicting cases. The overall quality of the information should consequently be very high. It should also be noted that these cohorts were very unlikely to migrate. For example, only a handful individual emigrated during their first ten years of life, and these individual have been dropped from the dataset. Table 2 presents summary statistics of the main variables, while Appendix A provides information on variable definitions.

[Insert Table 2 about here]

In addition to the creation of the dataset covering individual-level information, we also drew data from annual reports of medical districts (provinsiallåkardistrikt). Every district physician in each of the 447 medical districts of Sweden 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; Table 3 provides some summary statistics.

[Insert Table 3 about here]

For about half of the eligible sample of women and children we also acquired the forms recording utilisation of services from archive records. The utilisation of the care service was recorded at the individual level by type of service (doctor/nurse) and date of visit. This information was digitised and matched to the individual births from the parish records. Table 4 provides summary statistics for this subsample. Clearly, the subsample for which utilisation data are available is very similar to the overall dataset.

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13 Archive records are unfortunately not complete with respect to individual utilisation cards, and the utilisation data corresponds to a nonrandom subset of locations.
The empirical analysis entails difference-in-differences to identify the intent-to-treat effect, and two-stage least squares (2SLS) to identify the treatment effect on the treated.

4.2 Intent-to-Treat Analysis

The baseline specification is designed to gauge the overall impact of the mother and baby interventions; it is followed by a more detailed analysis focusing on different subgroups.

Our estimating equation is

\[ y_{icj} = \alpha + \beta_M T_{Mj} + \beta_I T_{Ij} + \gamma D_c + \delta_M T_{Mj} D_c + \delta_I T_{Ij} D_c + f(j) + \epsilon_{icj} \]  

(2)

where \( y_{icj} \) is an outcome of child \( i \) born in district \( c \) on day \( j \), \( T_j \) is the duration (in months) of eligibility for a child born on day \( j \), \( D_c \) is a dummy variable for treated districts, and \( f(j) \) is a flexible time trend (quarter-of-birth times birth year dummies and sometimes also diverging time trends). Covariates that we condition upon include the sex of the child, a twin birth indicator, the marital status of the mother and dummies capturing old (> 32.3) and young (< 25.8) mothers.

Under the assumption that treated and control districts would have followed a common time trend in the absence of a treatment, the parameters \( \delta_M \) and \( \delta_I \) in equation (2) estimate 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.

To some extent the variables \( T_{Mj} \) and \( T_{Ij} \) are correlated: a subset of individuals were eligible for both interventions. The correlation coefficient in the entire dataset equals 0.32. However, this positive correlation is mainly driven by the many zeros. If we condition on eligibility for at least one of the interventions, the correlation coefficient between the two variables drops to 0.013. Table 6 shows the entire bivariate distribution of treatment eligibility. A majority of individuals have zero eligibility (upper left corner of the tables) and among those with positive eligibility, the two interventions are largely unrelated. Besides, by comparing with the (counterfactual) distribution of eligibility in the comparison regions (right part), we show that the treatment and control group exhibit common support at all levels of eligibility.

\[14\] Thus, in most cases \( T_j \) is collinear with the quarter dummies and suppressed from the regression tables.
4.3 Additional Specifications

To allow for selection into programme uptake, we re-estimate the model including mother fixed effects. This also allows for selection of mothers with different unobservable traits (potentially correlated with the outcome variables) into programme uptake and into fertility. We also conduct the analysis separately for subgroups of the population. First we isolate women who had the index birth out of wedlock as they were highlighted as a target group in public debates concerning the intervention. We also investigate heterogeneity by, inter alia, the age of the mother and the family’s socio-economic status.

As discussed above we acquired access to detailed utilisation data for close to half of the eligible population. Although the sub-sample for which we have these data are not necessarily representative of the entire sample, we use the utilisation data to estimate the treatment effect of actual utilisation by using program exposure as an instrument for utilisation. Under the assumption that the duration of eligibility (which depends only on birth date) is orthogonal to unobservable differences in the health of the children (conditional upon birth quarter and birth year), these 2SLS estimates identify the treatment effect (ATT) of utilisation.

5 Results

In this section we present estimates of the impact of the programme on various outcomes. We start with a descriptive analysis of utilisation, then present ITT estimates and finally we estimate the impact of actual utilisation. We then discuss and evaluate potential threats to our identification strategy.

5.1 Programme Utilisation

As discussed, the intent-to-treat estimates provide a policy relevant parameter. It is nevertheless relevant to study utilisation both to estimate impacts on families that were actually treated (viz our 2SLS estimates) and to study selection into uptake. We later control for selection into uptake using mother fixed effects, but information on who takes up the program is relevant for understanding program impacts on inequality.

In analysing program utilisation we bunch all types of service providers (nurse, doctor)
together but distinguish services provided to mothers from those provided to children. Figure 8 provides histograms of utilisation for infant and maternal care, respectively, for all individuals who were eligible for the services at some point. In both cases, the utilisation has considerable inflation of zeros and very long tails. Thus, a zero-inflated negbin model seems appropriate for the analysis. For comparison we also report results for a linear model.

[Insert Figure 8 about here]

In Table 5, presenting the regression results, the first four columns refer to the infant care intervention, and the last four columns to the maternal care intervention. The first column presents results from a zero-inflated negbin model, the second column presents LPM estimates (enrolment defined as a binary variable), the third column presents corresponding OLS results when using number of visits as the dependent variable, while the fourth column presents results from a first stage regression, where duration of treatment eligibility is used as an instrument for utilisation. The fourth specification also includes time periods and individuals who are not eligible for treatment. We return to this specification in the 2SLS analysis below.

[Insert Figure 5 about here]

As we may expect, the duration of treatment eligibility is a powerful determinant of utilisation for both the maternal and infant interventions. For pregnant women, each additional month of eligibility increase utilisation by around 0.12 visits. This effect is significant, and the negbin estimates (which also generate a significant marginal effect, not reported) suggest that it is on the extensive margin that the eligibility period matters, so we may interpret the 0.12 as a 12% point increase in participation for every month of exposure. Married women have, on average, 0.7 more visits than single women. Compared to the baseline of 1.01 visits per pregnancy, this is a large effect.

Utilisation of the infant care intervention exhibits some socioeconomic differences on the extensive margin: older mothers and those in higher socioeconomic groups were somewhat more likely than younger and working class women to take up the services. However, in most cases differences on the extensive margin are counteracted by movements on the intensive margin so that overall heterogeneity in uptake of the service is limited.
5.2 Intent-to-Treat Effects

5.2.1 Birth Outcomes

In a first set of specification, we present results for the effect of the maternal care intervention on some key birth outcomes. The first two columns of Table 7 present the effects on maternal mortality, measured as maternal deaths per 100,000 births. Maternal mortality is an extremely rare event – the baseline probability is 400 cases per 100,000 births. Thus, it is hardly surprising that the estimated effect comes out insignificant. Nevertheless, the sign is negative, suggesting that the intervention reduced the probability by around 50 deaths (12 per cent) for the average eligibility period. For the incidence of stillbirths, we also get an insignificant negative estimate. For the average eligible person, the reduction in stillbirth incidence equals 0.15 percentage points or around 7.5 per cent of the baseline risk. The third outcome presented in Table 7 is the incidence of complications among midwife-attended births. This variable is recorded at the health district level. Again there is evidence of an improvement with a reduction by some 10 per cent, but the effect is not statistically significant.

[Insert Table 7 about here]

5.2.2 Infant Mortality: Comparing Specifications

Table 8 presents programme impacts on infant mortality for a range of different specifications. We first investigate the total number of months of programme exposure of a child to both interventions. Then we isolate exposures (of the birth) to each of the maternal and child interventions (in the four rightmost columns). Controls are added cumulatively in sequence: the second controls for observable characteristics of the child; the third column adds health district fixed effects, and the final column adds linear group-specific time trends.

[Insert Table 8 about here]

The impact of eligibility aggregated over both arms of the intervention is imprecisely estimated but the coefficient is notably large, suggesting a 0.3–0.6 percentage point improvement in survival probabilities for individuals with the average months of exposure. The reason for this lack of precision becomes obvious once we allow independent coefficients on the components delivered before and after birth. The intervention targeted directly at children is estimated to have reduced infant mortality by as much as a statistically significant 1 percentage point on
average, but the corresponding coefficients on the duration of exposure to the maternal care intervention are smaller and insignificant. Following these results we model exposure to the two arms of the program separately and now investigate persistence of the impacts of the infant intervention.

5.2.3 Life Expectancy

Using administrative data on mortality (from parish records and death registers) to identify survival rates of the sample cohorts over time, we investigate whether the treated cohorts that we noted were less likely to die in infancy were also less likely to have died by ages 5, 40 and 75. We choose age 5 because mortality hazard tends to be highest at birth when individuals have limited immunity and to then decline exponentially to age 5, after which they flatten noticeably. We choose age 40 as an age by which maternal mortality risk has been realized, and we choose age 75 as this was a “ripe old age” to achieve for the 1930s cohorts in our sample, and hence a good indicator of life expectancy.

Estimates in Table 9 (top panel) suggest persistent effects: treated children born during the eligibility period in the early 1930s are more likely to have survived to each of the threshold ages – 5, 40 and 75. By the age of 75, the effect on survival has doubled since infancy. Consistent with our finding that the intervention for mothers had no discernible impact on infant mortality, we do not find any significant impact of the maternal care intervention on life expectancy.

5.2.4 Within Mother Estimates

Introducing mother fixed effects we control for potential selection into programme uptake and into fertility to the extent that this selection occurs on time-invariant mother-level traits, such as for instance time preference, altruism towards children or ability. Table 10 (upper panel) presents the results. These estimates, identified from variation in programme exposure among siblings, are substantially larger and consistently more significant. The coefficients obtained in specifications with covariates included indicate that for a child with the average duration of exposure to the infant care intervention, the probability of dying in infancy was 3 percentage points smaller, and the probabilities of dying by the ages of 5, 40 and 75 were of a similar magnitude. These point estimates suggest that the infant care intervention led to survival gains
beyond infancy and at least up to the age of 40. These are substantial effects and the infant intervention in the early 1930s contributed significantly to lowering morbidity and mortality amongst the elderly in present-day Sweden.

[Insert Table 10 about here]

For comparison, we also present estimates from pooled regressions on the subsample of children included in the fixed effects regressions. The larger programme impacts estimated using within-mother variation in exposure are consistent with selection bias contaminating estimates that derive from within and cross-mother variation in exposure if programme uptake and/or endogenous increases in fertility were larger amongst less “treatable” women, whom we may think of as women with better infant survival rates prior to the programme. As the available evidence on this is ambiguous, a potential explanation is that the larger coefficients indicate that the programme induced reinforcing investments in children. In particular, the evidence is consistent with parents investing more in children who were treated by the program than in their unexposed or less exposed siblings. We investigate this in the next section.

5.2.5 Parental Investments

In order to gain a deeper understanding of the results, we contrast different groups of ineligible children. This analysis is conducted in two steps. First, we focus on children born before 1 October 1930 who were ineligible because they had just crossed infancy when the program was introduced on 1 October 1931 and, amongst this group, we compare those with and without younger siblings born in the eligibility period. In order to make this comparison informative, we match each ineligible child with a younger eligible sibling – there were 846 such cases in total – to three other individuals born in the pre-intervention period: one individual born in a treatment district who did not have an eligible sibling; one individual born in a control district who did have a sibling born during the intervention period, and one individual born in a control district without a sibling born during the intervention period. 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 11 presents averages of these covariates for the four groups.

[Insert Table 11 about here]
By comparing pre-intervention children in the treated and control areas, we may estimate the effect of having a younger sibling who was eligible for the services. By comparing to control areas, we get rid of ‘normal’ differences between children with and without younger siblings. The double difference then captures the effect of the younger sibling’s eligibility for services. Figure 6 pictures the double difference where we plot survival curves against the left $y$ axis. Black curves refer to children born in treated areas and grey curves refer 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.

When subtracting the curve $S_{10}$ (treated area, no eligible sibling) from the curve $S_{11}$ (treated area, eligible sibling) we get the difference in survival prospects between the two groups, and by calculating the double difference $S_{11} - S_{10} - (S_{01} - S_{00})$, we capture the effect of sibling eligibility. The largely horizontal curves plotted against the right $y$ axis represent these differences. Children with siblings born in the intervention period seem to have a survival advantage in control areas (represented by the curve $S_{01} - S_{00}$): this advantage fluctuates around 1–2 percentage points from early childhood until thirty years after the ‘treatment’. In the treated areas, on the other hand, children with eligible siblings appear to have a survival disadvantage of similar or somewhat smaller magnitude. Thus, the net effect – our DID estimate – is a strong survival disadvantage of at least two percentage points, which manifests itself during the first years of life and then remains for at least two decades. The figure is thus indicative of reinforcing parental investments – since children with eligible siblings seem to fare worse than other children. This analysis thus supports the tentative conclusion from the comparison between pooled and fixed effects estimates in Section 5.2.4.

[Insert Figure 6 about here]

In the second step we turn to children born after the intervention, after 30 March 1933, and within this ineligible group we compare those with and without eligible older siblings. In our sample there are in total 873 children born in treated areas after the intervention, who have an

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\[15\] As we are now considering an indirect effect of the programme – i.e. the effect of having a treated sibling – we are not able to take variation in treatment intensity into account as it is unclear how one or more siblings’ eligibility maps into affectedness for the ineligible sibling.
older sibling who was eligible at some point in time. These are matched to their equivalents who do not have older eligible siblings, and to similar pairs in the control regions. In this case, the “treatment” is obviously induced from birth onwards, which somewhat simplifies the comparison. Table 12 presents the covariate match between the four groups.

Figure 7 shows the corresponding survival curves. Interestingly, we now fail to discern a clear effect of having an eligible sibling. In treated and control areas alike, those with older siblings born in the eligible period have worse survival prospects than the rest. This difference is almost the same in treated and control areas, and thus cancel out. This result suggests that there is no effect whatsoever of having an eligible sibling during the first twenty years of life. Beyond that age, it appears that there is an advantage of having an eligible sibling which amounts to some 3-4 percentage points.

In summary, the effect of having an eligible sibling appears to be quite different for children born before and after the intervention, respectively. For children born before the intervention, having a younger eligible sibling appears to be associated with a disadvantage. This disadvantage evolves quite rapidly after the younger sibling enters the stage and might thus be a sign of reinforcing parental investments. For children born after the intervention, on the other hand, there is no disadvantage of having an eligible sibling, and it might actually even entail an advantage at advanced ages. This evidence and the contrast between the two cases may in turn be seen as evidence of a learning effect which cancels out any reinforcing parental investment and leaves ineligible siblings born after the intervention better off than their counterparts born before the intervention.

5.2.6 Treatment Effect Heterogeneity

In this section we explore differences in the impact of the infant care intervention in different population subgroups. We here estimate intent to treat effects, so that any difference in impact may reflect not only “treatability” but also differences in take-up. We define six sub-populations of interest: single mothers; young mothers (below median age of 29.2 years at the index birth); female children; children born into families that had experienced infant mortality shortly before
the intervention; children born to mothers who had an adverse health environment as infants (captured by the Maternal IMR variable being above the median); and children born in areas disproportionately affected by the Great Depression (defined as the drop in local taxable earnings being larger than median). These are typically sub-populations with elevated risks of further infant mortality (see Arulampalam and Bhalotra, 2006; Bhalotra and Soest, 2008). Table 13 presents results for mortality over different time horizons.

Programme impact was larger among single mothers, each month of eligibility reducing infant mortality risk by almost a whole percentage point. For a child with the sample average duration of exposure this implies a reduction in infant mortality risk of 6.6 percentage points, close to the baseline mortality rate of 9.9 per cent in this group. By the age of 75, the cumulative effect is as large as 11.5 percentage points, a reduction of more than a third of the baseline risk. So although the average impact on life expectancy was only slightly larger than the average impact on infant survival for the vulnerable group of births to single mothers, there were substantial post-infant survival gains.

Young mothers benefited more than older mothers from the intervention, although in this case, the life expectancy gain appears to merely reflect child survival gain. The pre-intervention infant survival rates for younger women were on average better, so a likely explanation of their 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.

There is no statistically significant difference in program impact by child sex. For families with previous infant mortality experience, there is strong evidence that the impact is much larger than for the general population, consistent with their greater a priori risks, but this group is so small that it is difficult to get precise estimates. As regards maternal health environment during infancy and areas particularly affected by the crisis, we find no evidence of a heterogeneity in treatment effects.

16The difference viz single women 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 but without further investigation, identifying status as a combination of SES, age and marital status, it is hard to be certain.
In conclusion, it appears that the intervention was successful in reducing health inequality, since particularly vulnerable groups – single mothers and families with previous mortality experience – appear to have benefited disproportionately.

5.3 2SLS Estimates

In the next step, we estimate the impact of actual utilisation on various outcomes. In this part, we consider three different specifications. First, we examine the effect of enrolment in the two programmes, assuming that their effects are independent. Then we allow for a different impact when the two interventions are combined. Finally, we consider the marginal effect of each additional visit. Table 14 presents first-stage estimates for these three sets of specifications.

Clearly, program eligibility measured as months of potential exposure is a powerful instrument, affecting both enrolment and utilisation in the expected manner. The F tests suggest that our instruments have the necessary strength.17 Interestingly, we find that the programme takeup is systematically more selective for the maternal care intervention: takeup was particularly high for married women, young women and women of high socioeconomic status. For the infant care intervention, there are no observables which predict enrolment.

Allowing for three types of enrolment – maternal, infant and a combination of the two, reported under the heading ‘Exclusive’ – the pattern is even stronger. Individuals who only enrolled in the infant care intervention were more likely to be twins and less likely to be of high socioeconomic status: thus, they were negatively selected. Within the group that enrolled in both programmes, on the other hand, a high SES was strongly over-represented and mothers who had suffered from an adverse disease environment during infancy were underrepresented.

Table 15 presents the second-stage results. For each mortality horizon, we present one specification where the two interventions are assumed to be independent, one where we define three mutually exclusive treatments, and one where we estimate the marginal effect of utilisation. As far as infant mortality is concerned, we again identify a large and significant effect of the infant care intervention. Enrolment associates with a reduction in infant mortality by 6.6 percentage points. Interestingly, this effect appears to be entirely driven by the group that enrolled only in

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17 We report the statistic for a standard F test and also the variable-specific statistic as suggested by Angrist and Pischke (2008).
the infant programme, whereas the group which enrolled in both programmes did not experience any improvement in infant mortality. According to the estimates based on utilisation, each additional visit in the infant care programme was associated with a reduction in infant mortality of around one percentage point.

Also in the longer term, the effects of the infant care intervention persist. However, the 2SLS analysis also suggest a surprising positive mortality effect of the maternal care intervention in the very long run.

[Insert Table 15 about here]

5.4 Threats to Identification

We first evaluate our matching metric by comparing baseline observable characteristics between treated and control districts. Besides following standrad procedures comparing variables used for the matching, we also compare treated and control districts on variables collected after the matches were identified. Next, we assess the plausibility of the common time trends assumption. Third, we discuss possible confounding factors and assess whether they relate to our treatment variable in a way that could bias the results. We also estimate the effect of the intervention on outcomes which should not be affected by the program and implement placebo regressions.

5.4.1 Match Quality

For each of the parishes forming part of the experiment, the nearest neighbour in terms of observable characteristics was identified. Table 16 presents summary statistics for some observable characteristics, namely the sectoral shares of agriculture and manufacturing, the proportion of women of fertile age in the population, income, wealth and the degree of urbanisation. Columns 4 and 6 denoted ‘Std. Dif.’ present the standardised difference between the treatment group and the rest of Sweden, and the treatment group and the matched control group, respectively (cf. Imbens and Wooldridge 2009).

[Insert Table 16 about here]

The table shows that there are no significant differences between the treated and control parishes in a set of relevant covariates. We also note that the treated group is not very different on average from the rest of Sweden suggesting our findings have external validity. This is
especially relevant as the trial, which ceased in 1933, was followed by a nationwide roll out of a very similar mother-baby intervention programme in 1937.

5.4.2 Common Time Trend

To investigate the common trends assumption, we test for pre-trends, looking at infant mortality prior to the intervention, for an extended time period stretching back to 1910. We plot the time trends for the two groups of locations, showing the weighted median IMR, the 10th percentile and the 90th percentile. Clearly the common time trend appears to be satisfied.

[Insert Figure 9 about here]

5.4.3 Anticipation, Selection, Persistence.

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; the variable $T_{Sj}$

$$T_{Sj} = \begin{cases} 1 & \text{if } j \in [1 \text{ April 1932, 15 November 1933}] \\ 0 & \text{otherwise.} \end{cases}$$

(3)

The intuition is simply 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 one-month time to conceive. Likewise, there was little or no incentive to time a birth after 15 November 1933, since these women and births would be covered during less than half of the pregnancy and completely ineligible for infant care services. The interaction between $T_{Sj}$ and the treatment region indicator is denoted $DIDS$ in what follows. We also define a variable denoted $DIDP$, which is an interaction of being born after the intervention with the treatment region indicator. This variable tests whether children born after the intervention represent a valid control group.

Table 17 presents regression results for seven dependent variables: the mother’s age and marital status, two indicators of the father’s profession, a variable capturing whether the child has a younger sibling in the dataset (this variable would be indicative of composition effects), the local birthrate, and the proportion of births being attended by a midwife. The first rows
of the table reports the parameters of interest, and at the bottom of the two panels we provide baseline characteristics for comparison.\[18\]

The estimates confirm that the differences between treated and control groups are tiny. Moreover, there is no evidence that there are anticipation effects, persistent programme effects, or “undesired” treatment effects on variables that should not have been affected.

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).*

In fact knowledge of causal effects at a population level is limited. We supplement a small largely bio-medical literature that evaluates specific interventions using local randomized control trials by investigating population level effects of a packaged intervention affecting pregnant women and their children of the sort that is being delivered in many poor countries today. Our second contribution is to analyse long run effects on life expectancy.

While we are unable to identify any significant impact of services targeted at pregnant mothers, we find that postnatal services including nutritional guidance, home visits and diagnosis of disease targeted at infants were effective in reducing infant mortality and that these effects persist into adulthood, with exposed cohorts experiencing higher life expectancy. The results thus confirm [Heckman (2007)] on that early-life investments have high returns and highlight that it from a policy perspective is important to recall that the potential significance of interventions targeted to pregnant mothers and their children likely goes far beyond immediate effects. On the other hand, our results also deliver support for the observation of [Currie and Rossin-Slater](#).

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18 The discrepancies of baseline characteristics between Table 17 and Table 16 are mainly due to seasonal fluctuations in the variables.
that the evidence of beneficial early-life impacts of prenatal care is weak. Indeed, our findings show a striking absence of later-life benefits as well.

We also find evidence consistent with parents making reinforcing investments in treated children, which enhances the benefit-cost ratio for the intervention. For sub-groups such as out-of-wedlock births, the life expectancy gains reflect reductions in mortality risk beyond the infant period. Similar to contemporary discussions of health inequalities, a guiding policy concern in the debate preceding the Swedish intervention in the 1930’s was the situation of vulnerable groups, for instance, children born out of wedlock. The evidence suggests that the program narrowed health inequalities, acting as a pillar of the emerging welfare state.

A seeming puzzle is that the intervention targeting pregnant women was less effective than the intervention delivered to infants. The following are possible explanations. First, the maternal care intervention appears not to have reached the vulnerable groups that were the main targets of the interventions – married women and higher socioeconomic groups were strongly over-represented amongst the recipients of maternal care services, whereas there is no corresponding pattern in the takeup of infant care services. This difference may be associated with the social stigma associated with “illegitimate” pregnancies, and an implication is clearly that public interventions need to be designed with sensitivity for cultural norms in order to be effective. It may also be the case that the information provided to expectant mothers was already available and common knowledge among families prior to the 1930s. Another possibility is that the antenatal care intervention mainly involved information while the neonatal care intervention involved information but also monitoring and check-ups with respect to weight, vaccination and diagnosing of diseases.
References


Swedish Red Cross, 1928. Allt för barnet: Korta illustrerade råd och anvisningar i barnavård. Röda korset. URL http://books.google.se/books?id=rGCFmQEACAAJ


A Variable Definitions

A.1 Information from Parish Records

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

died5 Child Mortality \((d_{0-5})\): a dummy variable taking on the value one for live births who die within five years of birth.
died40 0-40 Mortality ($d_{0-40}$): a dummy variable taking on the value one for live births who die within 40 years of birth.

died75 0-75 Mortality ($d_{0-75}$): a dummy variable taking on the value one for live births who die within 75 years of birth.

Female A dummy variable taking on the value one for female births.

Twin A dummy variable taking on the value one for (mono- and dizygotic) twins.

Wedlock A dummy variable taking on the value one for children born to married mothers.

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

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

MOld A dummy variable taking on the 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 of birth and year of birth of the mother (defined as deviation from local average and overall time trend).

Treated A dummy variable taking on the value one for children born in treated areas.

Duration Duration in months of total theoretical eligibility for births in treated and control areas.

DurationM Duration in months of total theoretical eligibility of maternal care intervention for births in treated and control areas.

DurationI Duration in months of total theoretical eligibility of infant care intervention for births in treated and control areas.

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

BirthrateBirths 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 Maternal Equals one if the mother had at least one visit/home call during pregnancy. Source: official records from the intervention.

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

Utilisation Maternal Number of visits during pregnancy. Source: official records from the intervention.

Utilisation Infant Number of visits during infancy. Source: official records from the intervention.
Figure 1. Infant (All-Cause and Cause Specific) and Maternal Mortality Rates in Sweden 1910–40.
MEDDELANDE

angående
moderskaps- och barnavård.

Med anledning av Riksdagens beslut kommer hälsonefter — till vidare intill 1 juli 1932 — s. k. för- och eftervård vid barnabedr öfverblivande mödrar och spådbarn från Helsingborg med omnejd. — Vården är kostnadsfri.

MOTTAGNINGAR:

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

För spådbarn å Mjölkdroppens barnavårdscentral, Karl Krooksgatan 60 (Dir. D. Lindahl): onsdagar, torsdagar kl. 4—5 e. m.

Annonsera i Helsingborgs Dagblad.
Figure 3. Advice on Appropriate Feeding of Infants from Leaflet Provided within the Infant Care Intervention.

Figure 4. Eligibility at Different Ages of Children Born at Different Dates.
Figure 5. Municipalities Containing Treated and Control Districts.

Figure 6. Survival Curves for Children Born Before the Intervention. (Definitions on p. 16 and in Table 11).
Figure 7. Survival Curves for Children Born After the Intervention. (Definitions on p. 18 and in Table 12).

Figure 8. Histograms of Utilisation. Number of Visits.
Figure 9. Long-Term Trends in Infant Mortality Rates – Median, 10\textsuperscript{th} and 90\textsuperscript{th} Percentiles.
Table 1. Mothers and Children Enrolled in the Test Programme.

<table>
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<th>District</th>
<th>Infant Care</th>
<th>Maternal Care</th>
</tr>
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<td></td>
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<td>Eligible</td>
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<tr>
<td>Harads</td>
<td>225</td>
<td>269</td>
</tr>
<tr>
<td>Hälssingborg</td>
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<td>Jokkmokk</td>
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<td>298</td>
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<td>445</td>
</tr>
<tr>
<td>Pajala</td>
<td>344</td>
<td>552</td>
</tr>
<tr>
<td>Råneå</td>
<td>230</td>
<td>369</td>
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Table 2. Descriptive Statistics, Individual Level.

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<th>Variable Name</th>
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<th>Std. Dev.</th>
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<th>Max</th>
<th>N</th>
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<td>Child Mortality</td>
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<td>Died40</td>
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<td>30.70</td>
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<td>Died75</td>
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<td>100.00</td>
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<td>Maternal Mortality</td>
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<td>5,636.93</td>
<td>0.00</td>
<td>100,000.00</td>
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<tr>
<td>Stillbirth</td>
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<td>15.65</td>
<td>0.00</td>
<td>100.00</td>
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<td>Female</td>
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<td>0.00</td>
<td>1.00</td>
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<td>1.00</td>
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<tr>
<td>Wedlock</td>
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<tr>
<td>Maternal Age</td>
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</tr>
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<td>-0.59</td>
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Variable definitions and sources are provided in Appendix A.
Table 3. Descriptive Statistics, District Level.

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<th>Max</th>
<th>N</th>
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</table>

Variable definitions and sources are provided in Appendix A.

Table 4. Descriptive Statistics, Utilisation Sample.

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Variable definitions and sources are provided in Appendix A.
### Table 5. Estimation Results, Utilisation.

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<th>(OLS)</th>
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<th>(LPM)</th>
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<td>(0.003)</td>
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### Inflated

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<tr>
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| r2          | 0.064 | 0.033 | 0.100 | 0.206 | 0.065 | 0.167 |
| N           | 2.285 | 2.285 | 2.285 | 9.793 | 2.574 | 2.574 |

Standard errors clustered at the parish level in parenthesis. DurationTE represents the duration of treatment eligibility (in months) for the respective interventions. SES High is a dummy variable which takes on the value one if the household head belongs to higher professional classes (HISCO groups 1-5, which exclude farming, industrial production and those without a steady income), and SES Low is a dummy variable capturing household heads without a regular income or with missing information on occupation. The first three columns for each utilisation outcome cover 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 also births from other periods and from the matched locations have been included. The parameter estimates under the ‘Inflate’ heading are the results for the zero-inflated part of the Negbin model. Variable definitions and sources are provided in Appendix A.
Table 6. Frequency of Eligibility.

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<th>Control Districts</th>
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<td>9</td>
</tr>
<tr>
<td>(TI; months)</td>
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<td>700</td>
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<tr>
<td>(9, 12)</td>
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<td>0</td>
</tr>
<tr>
<td>12</td>
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<td>700</td>
</tr>
</tbody>
</table>

DurationM (DurationI) represents the number of months of eligibility for the maternal (infant) care programme an individual born on a certain day would have if born in a treated district. Each cell shows the (unweighted) frequency of individuals in the dataset within that range of (potential) eligibility for the two interventions.

Table 7. Estimation Results, Birth Outcomes.

<table>
<thead>
<tr>
<th>Maternal Mortality</th>
<th>Stillbirth</th>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
</tr>
<tr>
<td>DIDM</td>
<td>-8.4141</td>
<td>-7.2133</td>
</tr>
<tr>
<td>(21.383)</td>
<td>(21.385)</td>
<td>(0.070)</td>
</tr>
<tr>
<td>Female</td>
<td>96.2013</td>
<td>-0.5223**</td>
</tr>
<tr>
<td>Wedlock</td>
<td>-214.0328</td>
<td>-0.6012</td>
</tr>
<tr>
<td>(142.463)</td>
<td>(0.451)</td>
<td>(4.151)</td>
</tr>
<tr>
<td>Twin</td>
<td>-42.1631</td>
<td>3.3657**</td>
</tr>
<tr>
<td>(394.108)</td>
<td>(1.354)</td>
<td>(5.460)</td>
</tr>
<tr>
<td>MYoung</td>
<td>-80.5930</td>
<td>-0.9847**</td>
</tr>
<tr>
<td>MOld</td>
<td>697.8790**</td>
<td>1.5717***</td>
</tr>
<tr>
<td>QOB effects</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>SES effects</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>HealthDistrict FE</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>AITTM</td>
<td>-53.651</td>
<td>-45.995</td>
</tr>
<tr>
<td>Baseline</td>
<td>400.266</td>
<td>400.266</td>
</tr>
<tr>
<td>r2</td>
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<td>0.010</td>
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<tr>
<td>N</td>
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<td>24,710</td>
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</table>

Standard errors, clustered by health district, in parentheses. **DIDM** is the interaction between the eligibility period for maternal care services (in months) and the treatment location dummy *Treated*. Each individual-level specification also controls for the eligibility period and includes quarter-of-birth dummies for each of the 20 quarters (*QOB Effects*). *SES Effects* are fixed effects for the professional group of the household head, and *HealthDistrict FE* are fixed effects for the health district the individual lived in at the time of the birth. *AITTM* represents the intent-to-treat effect for the average eligible individual (i.e. they are the product of the DID point estimates and the average eligibility period for each of the interventions). *Baseline* represents the average of the outcome variable (per 100,000 for MMR, per cent for Stillbirth and Complication) in 1930. Variable definitions and sources are provided in Appendix A.
Table 8. Estimation Results, Infant Mortality.

<table>
<thead>
<tr>
<th></th>
<th>Total Eligibility</th>
<th>By Type of Service</th>
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<td>(2)</td>
</tr>
<tr>
<td>DID</td>
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<td>-0.0370</td>
</tr>
<tr>
<td></td>
<td>(0.050)</td>
<td>(0.049)</td>
</tr>
<tr>
<td>DIDM</td>
<td>0.1305</td>
<td>0.1020</td>
</tr>
<tr>
<td></td>
<td>(0.111)</td>
<td>(0.093)</td>
</tr>
<tr>
<td>DIDI</td>
<td>0.1020</td>
<td>0.0841</td>
</tr>
<tr>
<td></td>
<td>(0.061)</td>
<td>(0.065)</td>
</tr>
<tr>
<td>Treated</td>
<td>0.3422</td>
<td>-1.3837</td>
</tr>
<tr>
<td></td>
<td>(1.489)</td>
<td>(1.488)</td>
</tr>
<tr>
<td></td>
<td>(0.563)</td>
<td>(0.572)</td>
</tr>
<tr>
<td>Female</td>
<td>-1.9666***</td>
<td>-1.9678***</td>
</tr>
<tr>
<td></td>
<td>(0.330)</td>
<td>(0.326)</td>
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<tr>
<td>MYoung</td>
<td>0.4596</td>
<td>0.4914</td>
</tr>
<tr>
<td></td>
<td>(0.839)</td>
<td>(0.390)</td>
</tr>
<tr>
<td>MOld</td>
<td>1.3094***</td>
<td>1.2706***</td>
</tr>
<tr>
<td></td>
<td>(0.398)</td>
<td>(0.397)</td>
</tr>
<tr>
<td>Crisis</td>
<td>2.8563***</td>
<td>-1.2173</td>
</tr>
<tr>
<td></td>
<td>(0.938)</td>
<td>(1.015)</td>
</tr>
<tr>
<td>QOB effects</td>
<td>✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓</td>
<td></td>
</tr>
<tr>
<td>SES effects</td>
<td>✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓</td>
<td></td>
</tr>
<tr>
<td>HealthDistrict FE</td>
<td>✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓</td>
<td></td>
</tr>
<tr>
<td>Group-specific Trends</td>
<td>✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓</td>
<td></td>
</tr>
<tr>
<td>AITT</td>
<td>-0.3202</td>
<td>-0.3934</td>
</tr>
<tr>
<td>AITTM</td>
<td>-1.0884</td>
<td>-1.0062</td>
</tr>
<tr>
<td>r2</td>
<td>0.003</td>
<td>0.019</td>
</tr>
<tr>
<td>N</td>
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<td>24,373</td>
</tr>
</tbody>
</table>

Standard errors, clustered by health district, in parentheses. The left part of the table combines the two interventions into one measure, whereas the right part allows for a heterogeneous impact. DID is the interaction between the total eligibility period (in months) and the treatment location dummy Treated. DIDM and DIDI are the corresponding estimates for the maternal and infant care interventions. Each specification also controls for the eligibility period and includes quarter-of-birth dummies for each of the 20 quarters (QOB Effects). SES Effects are fixed effects for the professional group of the household head, and HealthDistrict FE are fixed effects for the health district the individual lived in at the time of the birth. Group-Specific Trends allows for divergent linear trends between treated and control districts. AITT, AITTM and AITTI represent the intent-to-treat effects for the average eligible individual (i.e. they are the product of the DID point estimates and the average eligibility period for each of the interventions). Baseline 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 9. Estimation Results, Different Time Horizons.

<table>
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<tr>
<th></th>
<th>$d_{0-1}$</th>
<th>$d_{0-5}$</th>
<th>$d_{0-40}$</th>
<th>$d_{0-75}$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DIDM</strong></td>
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<td>0.2057</td>
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<td>(0.111)</td>
<td>(0.103)</td>
<td>(0.132)</td>
<td>(0.179)</td>
</tr>
<tr>
<td><strong>DIDI</strong></td>
<td>-0.1413**</td>
<td>-0.0871</td>
<td>-0.1293*</td>
<td>-0.2964**</td>
</tr>
<tr>
<td></td>
<td>(0.061)</td>
<td>(0.057)</td>
<td>(0.076)</td>
<td>(0.143)</td>
</tr>
<tr>
<td><strong>Treated</strong></td>
<td>0.3173</td>
<td>0.7019</td>
<td>0.7169</td>
<td>-0.0791</td>
</tr>
<tr>
<td></td>
<td>(0.758)</td>
<td>(0.885)</td>
<td>(1.155)</td>
<td></td>
</tr>
<tr>
<td><strong>Twin</strong></td>
<td>9.1457***</td>
<td>9.4016***</td>
<td>9.1714***</td>
<td>5.7018*</td>
</tr>
<tr>
<td></td>
<td>(1.504)</td>
<td>(1.598)</td>
<td>(1.943)</td>
<td>(3.302)</td>
</tr>
<tr>
<td><strong>Wedlock</strong></td>
<td>-2.4410***</td>
<td>-2.1884***</td>
<td>-1.9571**</td>
<td>-3.3351***</td>
</tr>
<tr>
<td></td>
<td>(0.551)</td>
<td>(0.629)</td>
<td>(0.840)</td>
<td>(0.949)</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td>-1.9736***</td>
<td>-2.1862***</td>
<td>-3.7440***</td>
<td>-13.4925***</td>
</tr>
<tr>
<td></td>
<td>(0.331)</td>
<td>(0.328)</td>
<td>(0.343)</td>
<td>(0.669)</td>
</tr>
<tr>
<td><strong>MYoung</strong></td>
<td>0.4982</td>
<td>0.5375</td>
<td>1.1254**</td>
<td>1.8959*</td>
</tr>
<tr>
<td></td>
<td>(0.390)</td>
<td>(0.364)</td>
<td>(0.551)</td>
<td>(1.146)</td>
</tr>
<tr>
<td><strong>MOld</strong></td>
<td>1.2688***</td>
<td>1.6672***</td>
<td>2.2777***</td>
<td>2.5964***</td>
</tr>
<tr>
<td></td>
<td>(0.396)</td>
<td>(0.451)</td>
<td>(0.478)</td>
<td>(0.752)</td>
</tr>
<tr>
<td><strong>Maternal IMR</strong></td>
<td>-2.7394</td>
<td>-2.5900</td>
<td>-5.8325</td>
<td>8.1480</td>
</tr>
<tr>
<td></td>
<td>(0.519)</td>
<td>(7.588)</td>
<td>(9.906)</td>
<td>(16.695)</td>
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<td><strong>Crisis</strong></td>
<td>-1.3990</td>
<td>-1.6793</td>
<td>-0.6830</td>
<td>-2.5650</td>
</tr>
<tr>
<td></td>
<td>(1.065)</td>
<td>(1.018)</td>
<td>(1.738)</td>
<td>(1.888)</td>
</tr>
<tr>
<td><strong>QOB effects</strong></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td><strong>SES effects</strong></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td><strong>HealthDistrict FE</strong></td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td><strong>Group-specific Trends</strong></td>
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<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td><strong>AITTM</strong></td>
<td>0.8321</td>
<td>0.9178</td>
<td>1.3113</td>
<td>2.0394</td>
</tr>
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<td></td>
<td>(0.396)</td>
<td>(0.451)</td>
<td>(1.738)</td>
<td>(1.888)</td>
</tr>
<tr>
<td><strong>AITTI</strong></td>
<td>-1.0884</td>
<td>-1.4081</td>
<td>-1.2696</td>
<td>-2.2833</td>
</tr>
<tr>
<td></td>
<td>(0.617)</td>
<td>(0.827)</td>
<td>(1.122)</td>
<td>(1.888)</td>
</tr>
<tr>
<td><strong>Baseline</strong></td>
<td>6.617</td>
<td>6.617</td>
<td>12.211</td>
<td>36.535</td>
</tr>
<tr>
<td></td>
<td>(0.004)</td>
<td>(0.023)</td>
<td>(0.063)</td>
<td>(0.004)</td>
</tr>
<tr>
<td><strong>r2</strong></td>
<td>0.004</td>
<td>0.023</td>
<td>0.022</td>
<td>0.004</td>
</tr>
<tr>
<td></td>
<td>24.373</td>
<td>24.373</td>
<td>24.373</td>
<td>24.373</td>
</tr>
</tbody>
</table>

Standard errors, clustered by health district, in parentheses. **DIDM** denotes the DID estimate for maternal care, **DIDI** the corresponding DID estimate for infant care. $d_{0-x}$ denotes mortality before age x. Each specification also includes the number of months of eligibility for each of the interventions (**DIDM** and **DIDI** represent the interaction between this variable and **Treated**) and quarter-of-birth dummies for each of the 20 quarters (**QOB Effects**). **SES Effects** are fixed effects for the professional group of the household head, and **HealthDistrict FE** are fixed effects for the health district the individual lived in at the time of the birth. **Group-Specific Trends** allows for divergent linear trends between treated and control districts. **AITTM** and **AITTI** are the intent-to-treat effects for the average eligible individual (i.e. they are the product of the DID point estimates and the average eligibility period for each of the interventions). **Baseline** 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 10. Repeat Mothers, Different Time Horizons.

<table>
<thead>
<tr>
<th>Fixed Effects Regressions</th>
<th>$d_{0-1}$</th>
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<th>$d_{0-40}$</th>
<th>$d_{0-75}$</th>
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<tbody>
<tr>
<td>DIDM</td>
<td>0.2710</td>
<td>0.2817</td>
<td>0.2209</td>
<td>0.2827</td>
</tr>
<tr>
<td>(0.168)</td>
<td>(0.180)</td>
<td>(0.146)</td>
<td>(0.154)</td>
<td>(0.200)</td>
</tr>
<tr>
<td>DIDI</td>
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<td>-0.3486***</td>
<td>-0.2756**</td>
<td>-0.2448**</td>
</tr>
<tr>
<td>(0.113)</td>
<td>(0.117)</td>
<td>(0.113)</td>
<td>(0.115)</td>
<td>(0.147)</td>
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<tr>
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<td>18.0432***</td>
<td>17.8159***</td>
<td>11.0229**</td>
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<td>(2.664)</td>
<td>(2.835)</td>
<td>(4.869)</td>
<td></td>
</tr>
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<td>-2.1880**</td>
<td>-2.1629**</td>
<td>-2.6534***</td>
<td>-1.47785***</td>
</tr>
<tr>
<td>(2.664)</td>
<td>(2.941)</td>
<td>(2.936)</td>
<td>(3.168)</td>
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<td>1.2000</td>
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<td>1.0721</td>
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<td>(1.481)</td>
<td>(1.990)</td>
<td>(2.734)</td>
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<td>-3.2382</td>
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<td>(2.941)</td>
<td>(2.936)</td>
<td>(3.168)</td>
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</tr>
<tr>
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<td>(2.732)</td>
<td>(4.165)</td>
<td>(3.619)</td>
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<tr>
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<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
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<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>AITTM</td>
<td>1.7292</td>
<td>1.7959</td>
<td>1.4034</td>
<td>1.4585</td>
</tr>
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<td>-2.8959</td>
<td>-2.6855</td>
<td>-2.1232</td>
<td>-1.8861</td>
</tr>
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<td>Baseline</td>
<td>6.012</td>
<td>6.051</td>
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<td>8.525</td>
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<td>r2</td>
<td>0.012</td>
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</table>

Repeat Mothers Pooled

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<th>$d_{0-75}$</th>
</tr>
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<td>DIDM</td>
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</tr>
<tr>
<td>(0.172)</td>
<td>(0.172)</td>
<td>(0.160)</td>
<td>(0.172)</td>
<td>(0.209)</td>
</tr>
<tr>
<td>DIDI</td>
<td>-0.2776***</td>
<td>-0.3160***</td>
<td>-0.2614*</td>
<td>-0.2434*</td>
</tr>
<tr>
<td>(0.101)</td>
<td>(0.114)</td>
<td>(0.095)</td>
<td>(0.128)</td>
<td>(0.129)</td>
</tr>
<tr>
<td>Treated</td>
<td>0.7811</td>
<td>0.9078</td>
<td>1.2549</td>
<td>1.5139</td>
</tr>
<tr>
<td>(1.008)</td>
<td>(1.007)</td>
<td>(1.007)</td>
<td>(1.007)</td>
<td></td>
</tr>
<tr>
<td>Twin</td>
<td>7.6016***</td>
<td>7.9118***</td>
<td>7.5823***</td>
<td>3.8494</td>
</tr>
<tr>
<td>(1.364)</td>
<td>(1.734)</td>
<td>(2.096)</td>
<td>(3.871)</td>
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</tr>
<tr>
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<td>-2.8001</td>
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<td>(1.517)</td>
<td>(1.614)</td>
<td>(2.387)</td>
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</tr>
<tr>
<td>Female</td>
<td>-2.7650***</td>
<td>-3.0659***</td>
<td>-5.0216***</td>
<td>-15.4599***</td>
</tr>
<tr>
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<td>(0.503)</td>
<td>(0.499)</td>
<td>(0.954)</td>
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</tr>
<tr>
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<td>1.6430</td>
<td>2.3299*</td>
</tr>
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<td>(0.628)</td>
<td>(1.168)</td>
<td>(1.164)</td>
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<td>2.5510***</td>
<td>2.5838***</td>
<td>4.6859***</td>
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<td>(0.773)</td>
<td>(0.795)</td>
<td>(1.703)</td>
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</tr>
<tr>
<td>Maternal IMR</td>
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<td>-0.3194</td>
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<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>SES effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>HealthDistrict FE</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Group-specific Trends</td>
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<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
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<td>0.6221</td>
<td>0.7403</td>
<td>0.1895</td>
<td>0.7311</td>
</tr>
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<td>-2.1388</td>
<td>-2.4341</td>
<td>-1.2971</td>
<td>-1.8047</td>
</tr>
<tr>
<td>Baseline</td>
<td>6.612</td>
<td>6.612</td>
<td>8.251</td>
<td>8.251</td>
</tr>
<tr>
<td>r2</td>
<td>0.007</td>
<td>0.033</td>
<td>0.006</td>
<td>0.031</td>
</tr>
</tbody>
</table>

Standard errors, clustered by health district, in parentheses. DIDM denotes the DID estimate for maternal care, DIDI the corresponding DID estimate for infant care: $d_{0-1}$ denotes mortality before age x. Each specification also includes the number of months of eligibility for each of the interventions (DIDM and DIDI represent the interaction between this variable and Treated) and quarter-of-birth dummies for each of the 20 quarters (QOB Effects). SES Effects are fixed effects for the professional group of the household head, and HealthDistrict FE are fixed effects for the health district the individual lived in at the time of the birth. Group-Specific Trends allows for divergent linear trends between treated and control districts. AITTM and AITTI are the intent-to-treat effects for the average eligible individual (i.e. they are the product of the DID point estimates and the average eligibility period for each of the interventions). Baseline 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 B.
Table 11. Ineligible Children Born Before the Intervention With and Without Younger Siblings.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Treated</th>
<th></th>
<th>Control</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S11</td>
<td>S10</td>
<td>S01</td>
<td>S00</td>
</tr>
<tr>
<td>Birthdate</td>
<td>1930.377</td>
<td>1930.357</td>
<td>1930.386</td>
<td>1930.382</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>28.866</td>
<td>29.005</td>
<td>28.924</td>
<td>28.270</td>
</tr>
<tr>
<td>Wedlock</td>
<td>0.936</td>
<td>0.929</td>
<td>0.928</td>
<td>0.916</td>
</tr>
<tr>
<td>Maternal IMR</td>
<td>0.001</td>
<td>0.001</td>
<td>-0.001</td>
<td>0.003</td>
</tr>
<tr>
<td>SES High</td>
<td>0.071</td>
<td>0.085</td>
<td>0.092</td>
<td>0.077</td>
</tr>
<tr>
<td>SES Low</td>
<td>0.159</td>
<td>0.136</td>
<td>0.158</td>
<td>0.155</td>
</tr>
</tbody>
</table>

The table shows observable characteristics of children born before the intervention who were matched according to the protocol described on page 16. 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.

Table 12. Ineligible Children Born After the Intervention With and Without Older Siblings.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Treated</th>
<th></th>
<th>Control</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S11</td>
<td>S10</td>
<td>S01</td>
<td>S00</td>
</tr>
<tr>
<td>Birthdate</td>
<td>1934.621</td>
<td>1934.646</td>
<td>1934.636</td>
<td>1934.615</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>30.676</td>
<td>30.832</td>
<td>30.698</td>
<td>30.717</td>
</tr>
<tr>
<td>Wedlock</td>
<td>0.954</td>
<td>0.945</td>
<td>0.950</td>
<td>0.958</td>
</tr>
<tr>
<td>Maternal IMR</td>
<td>0.001</td>
<td>0.003</td>
<td>-0.000</td>
<td>-0.000</td>
</tr>
<tr>
<td>SES High</td>
<td>0.083</td>
<td>0.092</td>
<td>0.094</td>
<td>0.085</td>
</tr>
<tr>
<td>SES Low</td>
<td>0.050</td>
<td>0.028</td>
<td>0.050</td>
<td>0.047</td>
</tr>
</tbody>
</table>

The table shows observable characteristics of children born after the intervention who were matched according to the protocol described on page 18. S11 denotes children who were born after the intervention in a treated region, who had an eligible older sibling. S10 denotes children who were born after the intervention in a treated region who did not have an eligible older sibling. S01 denotes a child born in a control region with an older sibling born in the intervention period, and S00 denotes a child born in a control region without an older sibling born during the intervention period.
<table>
<thead>
<tr>
<th></th>
<th>Single Mother</th>
<th>Young Mother</th>
<th>Child Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$d_{0-1}$</td>
<td>$d_{0-5}$</td>
<td>$d_{40}$</td>
</tr>
<tr>
<td>DIDI × Variable</td>
<td>-0.7910**</td>
<td>-0.8584**</td>
<td>-0.8575**</td>
</tr>
<tr>
<td></td>
<td>(0.245)</td>
<td>(0.285)</td>
<td>(0.265)</td>
</tr>
<tr>
<td>DIDI</td>
<td>-0.0604</td>
<td>0.0002</td>
<td>-0.0450</td>
</tr>
<tr>
<td></td>
<td>(0.061)</td>
<td>(0.064)</td>
<td>(0.088)</td>
</tr>
<tr>
<td>Treated × Variable</td>
<td>0.5006</td>
<td>0.4213</td>
<td>0.6066</td>
</tr>
<tr>
<td></td>
<td>(1.822)</td>
<td>(1.987)</td>
<td>(2.390)</td>
</tr>
<tr>
<td>QOB effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>SES effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>HealthDistrict FE</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>AIITIVariable</td>
<td>-6.559</td>
<td>-6.611</td>
<td>-6.953</td>
</tr>
<tr>
<td>AIITI</td>
<td>-0.465</td>
<td>0.001</td>
<td>-0.347</td>
</tr>
<tr>
<td>r2</td>
<td>0.022</td>
<td>0.022</td>
<td>0.021</td>
</tr>
<tr>
<td>N</td>
<td>24,373</td>
<td>24,373</td>
<td>24,373</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Previous Mortality</th>
<th>Maternal IMR</th>
<th>Crisis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$d_{0-1}$</td>
<td>$d_{0-5}$</td>
<td>$d_{40}$</td>
</tr>
<tr>
<td>DIDI × Variable</td>
<td>-0.9292</td>
<td>-1.4550*</td>
<td>-1.3514</td>
</tr>
<tr>
<td></td>
<td>(0.877)</td>
<td>(0.797)</td>
<td>(0.891)</td>
</tr>
<tr>
<td>DIDI</td>
<td>-0.0721</td>
<td>-0.0117</td>
<td>-0.0606</td>
</tr>
<tr>
<td></td>
<td>(0.070)</td>
<td>(0.070)</td>
<td>(0.068)</td>
</tr>
<tr>
<td>Treated × Variable</td>
<td>-0.2220</td>
<td>0.2510</td>
<td>-0.2741</td>
</tr>
<tr>
<td></td>
<td>(4.178)</td>
<td>(4.344)</td>
<td>(4.488)</td>
</tr>
<tr>
<td>QOB effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>SES effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>HealthDistrict FE</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>AIITIVariable</td>
<td>-7.714</td>
<td>-11.290</td>
<td>-10.878</td>
</tr>
<tr>
<td>AIITI</td>
<td>-0.556</td>
<td>-0.090</td>
<td>-0.467</td>
</tr>
<tr>
<td>BaselineInter</td>
<td>96.688</td>
<td>97.024</td>
<td>97.024</td>
</tr>
<tr>
<td>BaselineAll</td>
<td>6.617</td>
<td>8.257</td>
<td>11.221</td>
</tr>
<tr>
<td>r2</td>
<td>0.146</td>
<td>0.118</td>
<td>0.080</td>
</tr>
<tr>
<td>N</td>
<td>24,373</td>
<td>24,373</td>
<td>24,373</td>
</tr>
</tbody>
</table>

Standard errors, clustered by health district, in parentheses. DIDI denotes the DID estimate for infant care. DIDI × Variable denotes the interaction with the variable in the column heading. AIITIVariable represents the intent-to-treat effect for the average eligible individual with the characteristics given in the column head. BaselineInter 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 IMR in the mother’s birth parish was above the median for her birth year. Each specification is fully interacted and also 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). SES Effects are fixed effects for the professional group of the household head, and HealthDistrict FE are fixed effects for the health district the individual lived in at the time of the birth. Variable definitions and sources are provided in Appendix [A].
<table>
<thead>
<tr>
<th></th>
<th>Overlapping</th>
<th>Enrolment</th>
<th>Utilisation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maternal</td>
<td>Infant</td>
<td>Maternal</td>
</tr>
<tr>
<td>DIDM</td>
<td>0.0303***</td>
<td>0.0294***</td>
<td>0.0225***</td>
</tr>
<tr>
<td></td>
<td>(0.002)</td>
<td>(0.005)</td>
<td>(0.002)</td>
</tr>
<tr>
<td>DIDI</td>
<td>0.0051***</td>
<td>0.0458***</td>
<td>-0.0046***</td>
</tr>
<tr>
<td></td>
<td>(0.002)</td>
<td>(0.004)</td>
<td>(0.001)</td>
</tr>
<tr>
<td>Eligible Maternal</td>
<td>0.0433**</td>
<td>-0.0577*</td>
<td>0.0309***</td>
</tr>
<tr>
<td></td>
<td>(0.018)</td>
<td>(0.031)</td>
<td>(0.011)</td>
</tr>
<tr>
<td>Eligible Infant</td>
<td>0.0102</td>
<td>0.0489</td>
<td>0.0259***</td>
</tr>
<tr>
<td></td>
<td>(0.012)</td>
<td>(0.084)</td>
<td>(0.008)</td>
</tr>
<tr>
<td>Eligible Both</td>
<td>-0.0253</td>
<td>-0.0472</td>
<td>0.1417</td>
</tr>
<tr>
<td></td>
<td>(0.022)</td>
<td>(0.044)</td>
<td>(0.075)</td>
</tr>
<tr>
<td>Treated</td>
<td>-0.0160***</td>
<td>-0.0293***</td>
<td>0.0020</td>
</tr>
<tr>
<td></td>
<td>(0.005)</td>
<td>(0.007)</td>
<td>(0.002)</td>
</tr>
<tr>
<td>Twin</td>
<td>0.0022</td>
<td>0.0253</td>
<td>0.0193</td>
</tr>
<tr>
<td></td>
<td>(0.019)</td>
<td>(0.026)</td>
<td>(0.013)</td>
</tr>
<tr>
<td>Wedlock</td>
<td>0.0230***</td>
<td>-0.0033</td>
<td>0.0222***</td>
</tr>
<tr>
<td></td>
<td>(0.007)</td>
<td>(0.007)</td>
<td>(0.007)</td>
</tr>
<tr>
<td>Female</td>
<td>0.0000</td>
<td>0.0002</td>
<td>0.0006</td>
</tr>
<tr>
<td></td>
<td>(0.004)</td>
<td>(0.004)</td>
<td>(0.003)</td>
</tr>
<tr>
<td>MYoung</td>
<td>0.0118**</td>
<td>-0.0023</td>
<td>0.0073**</td>
</tr>
<tr>
<td></td>
<td>(0.005)</td>
<td>(0.007)</td>
<td>(0.003)</td>
</tr>
<tr>
<td>MOld</td>
<td>-0.0014</td>
<td>-0.0089</td>
<td>0.0018</td>
</tr>
<tr>
<td></td>
<td>(0.006)</td>
<td>(0.006)</td>
<td>(0.004)</td>
</tr>
<tr>
<td>SES High</td>
<td>0.0149*</td>
<td>0.0008</td>
<td>-0.0014</td>
</tr>
<tr>
<td></td>
<td>(0.007)</td>
<td>(0.007)</td>
<td>(0.007)</td>
</tr>
<tr>
<td>SES Low</td>
<td>0.0048</td>
<td>-0.0188*</td>
<td>0.0107</td>
</tr>
<tr>
<td></td>
<td>(0.008)</td>
<td>(0.011)</td>
<td>(0.010)</td>
</tr>
<tr>
<td>Maternal IMR</td>
<td>-0.0353</td>
<td>-0.0617</td>
<td>0.0281</td>
</tr>
<tr>
<td></td>
<td>(0.056)</td>
<td>(0.075)</td>
<td>(0.053)</td>
</tr>
<tr>
<td>Crisis</td>
<td>-0.0541**</td>
<td>-0.0094</td>
<td>-0.0184</td>
</tr>
<tr>
<td></td>
<td>(0.026)</td>
<td>(0.055)</td>
<td>(0.021)</td>
</tr>
<tr>
<td>QOB effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Ftest</td>
<td>56.6</td>
<td>136.9</td>
<td>38.5</td>
</tr>
<tr>
<td>pValueF</td>
<td>0.000</td>
<td>0.000</td>
<td>0.000</td>
</tr>
<tr>
<td>APFtest</td>
<td>75.2</td>
<td>172.6</td>
<td>54.8</td>
</tr>
<tr>
<td>pValueAP</td>
<td>0.000</td>
<td>0.000</td>
<td>0.000</td>
</tr>
<tr>
<td>r²</td>
<td>0.26</td>
<td>0.51</td>
<td>0.15</td>
</tr>
<tr>
<td>N</td>
<td>9,792</td>
<td>9,792</td>
<td>9,792</td>
</tr>
</tbody>
</table>

Standard errors clustered at the parish level in parenthesis. DIDM and DIDI represent the length of the eligibility period in months whereas Eligible Maternal and Eligible Infant are dummy variables which take on the value one if the eligible period is greater than zero. In the column denoted ‘Exclusive’ we make eligibility indicators mutually exclusive and allow for a third category – Eligible Both – which takes on the value one if the child was eligible for both interventions. Each specification also includes quarter-of-birth dummies for each of the 20 birth quarters (QOB Effects). 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). Variable definitions and sources are provided in Appendix A.
<table>
<thead>
<tr>
<th>d0−1</th>
<th>d0−5</th>
<th>d0−40</th>
<th>d0−75</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.0163</td>
<td>-2.4393</td>
<td>1.3390</td>
<td>-7.1198</td>
</tr>
<tr>
<td>(4.659)</td>
<td>(7.436)</td>
<td>(5.340)</td>
<td>(8.994)</td>
</tr>
<tr>
<td>6.218***</td>
<td>-10.2796**</td>
<td>-3.7591</td>
<td>-9.4871**</td>
</tr>
<tr>
<td>(2.225)</td>
<td>(4.179)</td>
<td>(2.327)</td>
<td>(4.762)</td>
</tr>
<tr>
<td>5.1157</td>
<td>11.4076</td>
<td>12.2197</td>
<td>5.6382</td>
</tr>
<tr>
<td>(8.493)</td>
<td>(10.907)</td>
<td>(16.801)</td>
<td>(13.492)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>d0−1</th>
<th>d0−5</th>
<th>d0−40</th>
<th>d0−75</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.516**</td>
<td>1.9008**</td>
<td>1.5040**</td>
<td>1.3852</td>
</tr>
<tr>
<td>(0.767)</td>
<td>(1.344)</td>
<td>(0.921)</td>
<td>(1.278)</td>
</tr>
<tr>
<td>10.594***</td>
<td>21.1596***</td>
<td>12.0430*</td>
<td>-1.0771</td>
</tr>
<tr>
<td>(2.364)</td>
<td>(4.179)</td>
<td>(2.322)</td>
<td>(4.762)</td>
</tr>
<tr>
<td>-7.132***</td>
<td>-1.6593***</td>
<td>-2.0665**</td>
<td>-3.3640***</td>
</tr>
<tr>
<td>(0.446)</td>
<td>(0.465)</td>
<td>(0.809)</td>
<td>(0.841)</td>
</tr>
<tr>
<td>0.1881</td>
<td>0.1615</td>
<td>0.2642</td>
<td>0.1656</td>
</tr>
<tr>
<td>(0.450)</td>
<td>(0.544)</td>
<td>(0.542)</td>
<td>(0.809)</td>
</tr>
<tr>
<td>1.782***</td>
<td>1.8033***</td>
<td>2.2878***</td>
<td>2.7311***</td>
</tr>
<tr>
<td>(0.530)</td>
<td>(0.531)</td>
<td>(0.530)</td>
<td>(0.809)</td>
</tr>
<tr>
<td>-1.538**</td>
<td>-1.4555*</td>
<td>-0.9653</td>
<td>-0.6754</td>
</tr>
<tr>
<td>(0.710)</td>
<td>(0.756)</td>
<td>(0.720)</td>
<td>(0.768)</td>
</tr>
<tr>
<td>3.197***</td>
<td>3.2652***</td>
<td>3.1586***</td>
<td>2.6474*</td>
</tr>
<tr>
<td>(1.158)</td>
<td>(1.156)</td>
<td>(1.174)</td>
<td>(1.239)</td>
</tr>
<tr>
<td>-8.416*</td>
<td>-3.8938</td>
<td>-5.8211</td>
<td>-7.0903</td>
</tr>
<tr>
<td>(8.565)</td>
<td>(8.313)</td>
<td>(8.489)</td>
<td>(10.233)</td>
</tr>
<tr>
<td>0.2762**</td>
<td>1.0060**</td>
<td>0.7927</td>
<td>2.1409</td>
</tr>
<tr>
<td>(2.080)</td>
<td>(2.120)</td>
<td>(2.259)</td>
<td>(2.766)</td>
</tr>
<tr>
<td>✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓ ✓</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.617</td>
<td>6.617</td>
<td>6.617</td>
<td>6.617</td>
</tr>
<tr>
<td>8.257</td>
<td>8.257</td>
<td>8.257</td>
<td>8.257</td>
</tr>
<tr>
<td>11.221</td>
<td>11.221</td>
<td>11.221</td>
<td>11.221</td>
</tr>
</tbody>
</table>

Standard errors clustered at the parish level in parenthesis. Enrolment Maternal (Enrolment Infant) is the instrumented dummy variable capturing enrolment in the maternal (infant) care programme. Utilisation Maternal (Utilisation Infant) is a count variable capturing the number of visits during pregnancy (infancy). Consistent with the ‘exclusive’ part in Table 14 enrolment options in the second specification – Maternal, Infant, Both – are defined to be mutually exclusive. Each specification also includes quarter-of-birth dummies for each of the 20 birth quarters (QOB Effects). Variable definitions and sources are provided in Appendix A.
Table 16. Characteristics of Matched and Control Districts.

<table>
<thead>
<tr>
<th>Panel A: Matching Characteristics from the 1930 Census.</th>
<th>(1)</th>
<th>Treated</th>
<th>Control</th>
<th>Std. Dif. vs. (3)</th>
<th>Matched</th>
<th>Std. Dif. vs. (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agriculture</td>
<td>0.340</td>
<td>0.324</td>
<td>0.340</td>
<td>-0.040</td>
<td>0.302</td>
<td>0.054</td>
</tr>
<tr>
<td>Manufacturing</td>
<td>0.318</td>
<td>0.340</td>
<td>0.318</td>
<td>0.096</td>
<td>0.345</td>
<td>-0.018</td>
</tr>
<tr>
<td>Fertile Women</td>
<td>0.121</td>
<td>0.101</td>
<td>0.121</td>
<td>-0.135</td>
<td>0.100</td>
<td>0.060</td>
</tr>
<tr>
<td>Income</td>
<td>811</td>
<td>839</td>
<td>810</td>
<td>0.042</td>
<td>847</td>
<td>-0.013</td>
</tr>
<tr>
<td>Wealth</td>
<td>2,525</td>
<td>2,703</td>
<td>2,521</td>
<td>0.080</td>
<td>2,655</td>
<td>0.022</td>
</tr>
<tr>
<td>Urban</td>
<td>0.334</td>
<td>0.439</td>
<td>0.331</td>
<td>0.158</td>
<td>0.437</td>
<td>0.003</td>
</tr>
<tr>
<td>Population</td>
<td>6,271,266</td>
<td>258,418</td>
<td>6,004,052</td>
<td>160,987</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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 | 0.049 | 0.038 | 0.037 |
| Administrative, managerial | 0.025 | 0.025 | 0.016 | 0.046 |
| Clerical | 0.016 | 0.016 | 0.025 | -0.045 |
| Sales worker | 0.029 | 0.029 | 0.023 | 0.031 |
| Service worker | 0.022 | 0.022 | 0.010 | 0.071 |
| Agricultural | 0.297 | 0.297 | 0.307 | -0.015 |
| Production worker | 0.426 | 0.426 | 0.460 | -0.048 |

Panel A contains local characteristics according to 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 17. Estimation Results, Selection and Confounders.**

<table>
<thead>
<tr>
<th>PANEL A</th>
<th>Mother’s Age</th>
<th>Wedlock</th>
<th>SES Agriculture</th>
<th>SES Manufacturing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
</tr>
<tr>
<td>DIDS</td>
<td>-0.0408</td>
<td>-0.0081</td>
<td>0.0041</td>
<td>0.0285</td>
</tr>
<tr>
<td>(0.213)</td>
<td>(0.012)</td>
<td>(0.030)</td>
<td>(0.021)</td>
<td></td>
</tr>
<tr>
<td>DDP</td>
<td>-0.0339</td>
<td>0.0016</td>
<td>0.0299</td>
<td>0.0162</td>
</tr>
<tr>
<td>(0.230)</td>
<td>(0.008)</td>
<td>(0.035)</td>
<td>(0.025)</td>
<td></td>
</tr>
<tr>
<td>DID</td>
<td>0.0035</td>
<td>-0.0013</td>
<td>-0.0004</td>
<td>0.0016</td>
</tr>
<tr>
<td>(0.011)</td>
<td>(0.001)</td>
<td>(0.001)</td>
<td>(0.001)</td>
<td></td>
</tr>
<tr>
<td>DDIM</td>
<td>-0.0046</td>
<td>-0.0001</td>
<td>0.0003</td>
<td>0.0029</td>
</tr>
<tr>
<td>(0.022)</td>
<td>(0.002)</td>
<td>(0.003)</td>
<td>(0.003)</td>
<td></td>
</tr>
<tr>
<td>DIDI</td>
<td>0.0093</td>
<td>-0.0022**</td>
<td>-0.0009</td>
<td>0.0008</td>
</tr>
<tr>
<td>(0.018)</td>
<td>(0.001)</td>
<td>(0.002)</td>
<td>(0.002)</td>
<td></td>
</tr>
<tr>
<td>Treated</td>
<td>-0.1697</td>
<td>-0.2177</td>
<td>-0.2170</td>
<td>0.0041</td>
</tr>
<tr>
<td>(0.363)</td>
<td>(0.312)</td>
<td>(0.313)</td>
<td>(0.313)</td>
<td></td>
</tr>
<tr>
<td>QOB effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Baseline</td>
<td>29.576</td>
<td>29.576</td>
<td>29.576</td>
<td>0.0041</td>
</tr>
<tr>
<td>r2</td>
<td>0.002</td>
<td>0.002</td>
<td>0.002</td>
<td>0.002</td>
</tr>
<tr>
<td>N</td>
<td>25,029</td>
<td>25,029</td>
<td>25,029</td>
<td>25,029</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PANEL B</th>
<th>Has Younger Sibling</th>
<th>Birth Rate</th>
<th>Midwife Share</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
</tr>
<tr>
<td>DIDS</td>
<td>-0.0121</td>
<td>-0.0113</td>
<td>-0.1042</td>
</tr>
<tr>
<td>(0.024)</td>
<td>(0.024)</td>
<td>(0.161)</td>
<td>(3.064)</td>
</tr>
<tr>
<td>DDP</td>
<td>-0.0227</td>
<td>-0.0223</td>
<td>-0.0619</td>
</tr>
<tr>
<td>(0.049)</td>
<td>(0.049)</td>
<td>(0.187)</td>
<td>(3.320)</td>
</tr>
<tr>
<td>DID</td>
<td>0.0005</td>
<td>0.0006</td>
<td>-0.0007</td>
</tr>
<tr>
<td>(0.001)</td>
<td>(0.001)</td>
<td>(0.010)</td>
<td>(0.151)</td>
</tr>
<tr>
<td>DDI</td>
<td>0.0009</td>
<td>0.0111</td>
<td>0.0162</td>
</tr>
<tr>
<td>(0.002)</td>
<td>(0.002)</td>
<td>(0.016)</td>
<td>(0.258)</td>
</tr>
<tr>
<td>DDM</td>
<td>-0.0000</td>
<td>-0.0001</td>
<td>-0.0264</td>
</tr>
<tr>
<td>(0.002)</td>
<td>(0.002)</td>
<td>(0.020)</td>
<td>(0.345)</td>
</tr>
<tr>
<td>Treated</td>
<td>0.0237</td>
<td>0.0110</td>
<td>0.0113</td>
</tr>
<tr>
<td>(0.049)</td>
<td>(0.028)</td>
<td>(0.028)</td>
<td></td>
</tr>
<tr>
<td>Twin</td>
<td>-0.0526**</td>
<td>-0.0541**</td>
<td>-0.0530**</td>
</tr>
<tr>
<td>(0.022)</td>
<td>(0.022)</td>
<td>(0.022)</td>
<td></td>
</tr>
<tr>
<td>Wedlock</td>
<td>0.1043***</td>
<td>0.1048***</td>
<td>0.1047***</td>
</tr>
<tr>
<td>(0.019)</td>
<td>(0.020)</td>
<td>(0.020)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>0.0006</td>
<td>0.0003</td>
<td>0.0001</td>
</tr>
<tr>
<td>(0.006)</td>
<td>(0.006)</td>
<td>(0.006)</td>
<td></td>
</tr>
<tr>
<td>MYYoung</td>
<td>0.0328**</td>
<td>0.0324**</td>
<td>0.0326**</td>
</tr>
<tr>
<td>(0.015)</td>
<td>(0.016)</td>
<td>(0.016)</td>
<td></td>
</tr>
<tr>
<td>MOld</td>
<td>-0.0992***</td>
<td>-0.0991***</td>
<td>-0.0991***</td>
</tr>
<tr>
<td>(0.009)</td>
<td>(0.009)</td>
<td>(0.009)</td>
<td></td>
</tr>
<tr>
<td>Maternal IMR</td>
<td>0.2235</td>
<td>0.2259</td>
<td>0.2248</td>
</tr>
<tr>
<td>(0.180)</td>
<td>(0.180)</td>
<td>(0.181)</td>
<td></td>
</tr>
<tr>
<td>Crisis</td>
<td>0.0264</td>
<td>0.0292</td>
<td>0.0292</td>
</tr>
<tr>
<td>(0.025)</td>
<td>(0.000)</td>
<td>(0.000)</td>
<td></td>
</tr>
<tr>
<td>SES effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>QOB effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>YOB effects</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>HealthDistrict FE</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Baseline</td>
<td>0.432</td>
<td>0.432</td>
<td>0.432</td>
</tr>
<tr>
<td>r2</td>
<td>0.147</td>
<td>0.202</td>
<td>0.147</td>
</tr>
<tr>
<td>N</td>
<td>25,039</td>
<td>25,039</td>
<td>25,039</td>
</tr>
</tbody>
</table>

*DIDS* captures possible selection into the programme: it is an interaction between the treatment dummy and a dummy representing birth dates for which self-selection can be suspected. *DIDP* represents persistent effects: it is an interaction between treatment and post-intervention birth. Exact definitions are provided in Section 5.4.3. *DID, DIDM* and *DIDI* are the difference-in-differences variables defined as an interaction between treatment status and duration of eligibility. Each specification also includes all the terms used to build these interactions. *QOB Effects* are fixed effects for each of the 20 birth quarters. *SES Effects* are fixed effects for the professional group of the household head, and *HealthDistrict FE* are fixed effects for the health district the individual lived in at the time of the birth. *Baseline* presents the average of the dependent variable before the intervention. Variable definitions and sources are provided in Appendix A.
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