Fertility and Birth Spacing Consequences of Childhood Immunization Program: Evidence from India

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Abstract

What are the effects of childhood immunization program (UIP) on women's fertility and birth spacing? I examine the effect of this immunization program on women's subsequent fertility and birth spacing by exploiting district-by-cohort variation in exposure to the program. The results indicate that exposure of the first-born child to the immunization program reduces the likelihood of subsequent and cumulative fertility of women and increases the birth intervals between first and second births. The effects are more pronounced in urban areas. The significant program effect on fertility and birth intervals can be explained in terms of reduction in child mortality due to the immunization program. Kumar (2009) finds that UIP has a significant and negative effect on infant and under-five mortality.

JEL classification: I1, I2, J13, J18, O15.
Keywords: Immunization, Mortality, Fertility, India.

1   Introduction

One of the classical and most intriguing questions in demographic economics: What is the impact of child mortality decline on fertility? The relationship between infant and child mortality and fertility occupies a central if somewhat unsettled place in demographic research (Palloni and Rafamanani, 1999). It is difficult to establish a causal relationship between child mortality and fertility because of reverse causality and omitted variable bias. On one hand, fertility affects child mortality through length of birth interval and birth order and other indirect mechanisms. On the other hand, child mortality affects fertility levels and patterns through three different mechanisms - Physiological effect, Replacement effect, and Insurance or Hoarding effect. In addition, there are common unobserved factors that affect both fertility and child mortality.

There are credible causal evidence of impact of fertility on child mortality in demographic research. High level of fertility leads to high level of child mortality. A large empirical literature documents that short birth intervals, birth order, and mother's age at birth have strong effects on child mortality (Hobcraft 1992; Hobcraft, McDonald, and Rutstein 1983; LeGrand and Phillips 1996; Miller 1991). In contrast, the empirical evidence of the impact of child mortality on fertility has been elusive and far from being settled.

In this paper, we examine empirical evidence for the relationship between child mortality and fertility patterns in India. We investigate this causal link between child mortality and fertility by exploiting the phase-in roll out of a large-scale government-sponsored childhood immunization program in India called "Universal Immunization Program" (hereafter, UIP). In particular, we examine how the exposure of first born child in each household to UIP affects the likelihood of subsequent fertility, total fertility[1], and birth intervals. To identify the effects of exposure of the first-born child to UIP on subsequent fertility and total fertility, we exploit information on the timing of roll-out of UIP into different

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[1]Total fertility is defined as total number of children ever born.

districts of India. In 1985-86 the Government of India launched UIP in 31 districts. Each year additional districts were phased into the program and by 1990 all 443 districts of India were covered by UIP. The program delivered free immunization shots for children under one year of age to protect them from six Vaccine Preventable Diseases (hereafter, VPDs).[2]

UIP has features that allows me to identify the causal effect of the program on women's fertility and birth intervals. On one hand, there is a district variation in UIP exposure: UIP was implemented gradually across districts in India, with the timing apparently determined by fixed district characteristics. On the other hand, there is cohort variation in UIP exposure: only children who were twelve months old or younger at the time the program began would have been eligible to receive free immunization shots. I utilized these two sources of UIP-exposure variation on the first-born child of each women and use a difference-in-differences-type estimation strategy to identify the effect of UIP. The identifying assumption is that without UIP, the cohort difference in fertility and birth intervals outcomes would have been the same between the districts that implemented UIP sooner and the districts that implemented UIP later. I apply this identification strategy using the "Reproductive and Child Health Survey" (hereafter, RCH), a large nationally representative individual-level data-set.

The key independent variable is a dummy variable that captures whether the first born child in the family is exposed to UIP (hereafter, UIP child), and is constructed from the information on age of children and timing of UIP roll-out across districts. The key dependent variables are different measures of fertility: likelihood of subsequent births in the next 2 years, 3 years, and 5 years after the first birth; total fertility; birth intervals. The main finding of this paper is that exposure of the first born child to UIP reduced probability of subsequent births and it also reduced the total fertility of women. The negative effects are more pronounced in urban areas than rural areas. The program also

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[2]The six VPDs are Diphtheria, Pertussis, Tetanus, Poliomyelitis, Measles and Tuberculosis.

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had significant and positive effect on birth intervals

This paper is related to two strands of literature. The first is the existing literature on the determinants of fertility and birth intervals, with particular reference to the role of childhood immunization program in India. I am not aware of any previous studies that rigorously quantify the effects of childhood immunization programs on women's outcomes. This is the first paper that explores the unintended effects of an child immunization program.

The second strand of literature related to this paper is that on the effects of child mortality on women's fertility and birth spacing. Most studies linking child mortality and fertility are unable to distinguish causality from mere correlation. Existing literature on the effect of child mortality on fertility is inconclusive and inconsistent (Preston 1978). This paper adds to the literature on the effects of own children mortality on fertility. Another relevant question can be how women respond to changes in general mortality risk/changes in the expected mortality. In this paper, we explore the effect of own child mortality rather than changes in mortality risk on women's fertility. Though , in theory, it is clear that child mortality affects fertility through three different mechanisms: Physiological, replacement,and insurance effect. Bhalotra and Soest (2008) finds that neonatal death of a child shortens the interval until birth of next child. Palloni and Rafalimanana (1999) suggest very small positive effects of infant mortality on fertility. Since immunization program affects mortality and morbidity of children, it may have an effects on reproductive behavior and attitudes of women.

The rest of the paper is structured as follows: in Section 2, I discuss the related literature and provide an overview of UIP. Section 3 presents the empirical framework and Section 4 describes the data. Section 5 presents the results on fertility outcomes and Finally, Section 7 concludes.

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2 Background

2.1 The Universal Immunization Program

Approximately 3 million children die each year of VPDs with a disproportionate number of these children residing in developing countries (Kane and Lasher, 2002). Vaccines remain one of the most cost-effective public health initiatives, yet the cover against VPDs remains far from complete; recent estimates suggest that approximately 34 million children are not completely immunized with almost 98 percent of them residing in developing countries (Frenkel and Nielsen, 2003). Reducing child mortality by two-thirds between 1990 and 2015 is the fourth of eight Millennium Development Goals endorsed by world leaders in the Millennium Declaration in 2000.

In India, immunization of children against VPDs has been a central goal of the health care system from the 1970s. The Expanded Program on Immunization (hereafter, EPI) was initiated in 1978 to make six childhood vaccines (BCG, DPT, TT, DT, Polio and Typhoid) available to all eligible children. The main objective of EPI was to reduce mortality and morbidity by controlling six target diseases- Tuberculosis, Diphtheria, Tetanus, Pertussis, Polio and Typhoid. EPI failed to achieve the objective of immunizing children; because the program was limited primarily to major hospitals in urban areas and coverage levels were very low. Failure of EPI led the Government of India to make childhood immunization a top priority. In 1985, the Government of India made childhood immunization a Technology Mission and launched UIP with much dynamism to attain the goal of achieving 85 percent coverage for Tuberculosis, Diphtheria, Tetanus, Pertussis, Polio and Measles for all children by 1990.

Under UIP, each child had to be vaccinated before he or she turned one year of age with three doses of DPT vaccine, three doses of polio vaccine and one dose each of measles and BCG vaccine. Table 1 in the appendix lists some symptoms associated with the diseases that these shots protect against. The symptoms range from mild to severe, with

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serious sickness and death more likely among infants (whose immune systems are not yet mature) and poor children (whose immune systems are weakened due to malnutrition). It is worth noting that immunization protects individuals not only from illness per se, but also from the long-term effects of that illness on their physical, emotional, and cognitive development (Bloom et al., 2005). Additionally these diseases are communicable, so there are significant positive externalities from being vaccinated. That is, the vaccines reduce the risk of disease not only for the children vaccinated but also for people around them by reducing the transmission rate of the diseases.

There were not sufficient resources to implement the program all over the country at the same time. Thus, UIP had a phased roll-out, beginning with 31 districts in 1985-86 and covering all districts by 1990. The program was implemented through the existing network of primary health care infrastructure which consists of a referral center called "community health center" for every 80 to 120 thousand people, a primary health center for 20 to 30 thousand people, and a sub-center for every 3 to 5 thousand people. The program made provision for additional inputs in the form of additional staff, vaccines, and equipment for storage and transportation of vaccines such as walk-in-coolers, refrigerators and vaccine carriers.

Below I take advantage of the staggered implementation of UIP across districts to help identify UIP's effect on women's fertility, therefore it is essential to understand what determined the timing. Toward this end, I had numerous conversations with officials in the UIP division of the Ministry of Health and Family Welfare. The timing was not completely random. It seems that the capacity of the district to achieve the immunization coverage rates targeted by UIP and to maintain this level in subsequent years was a major factor in the selection of the district. In addition, infrastructure and other health facilities to deliver the UIP services were also taken into account while selecting the districts. In other words, selection of districts was based on fixed characteristics of the districts. For example, early-adopting districts may have more primary health centers, more nurses, or

have better health care infrastructure. Selection on fixed district characteristics does not cause problems for the interpretation of my estimated treatment effects because they rely on within-district variation in exposure to UIP only; that is, I always control for district fixed effects. A more serious problem would be if the timing of implementation depended on underlying district-specific trends in the outcome variables. It must be emphasized that UIP officials never indicated that district trends in fertility were part of the criteria for earlier implementation.

The extent to which a childhood immunization program affects fertility behavior of women remains an open questions. Answering these questions is of great interest for India because of the complementarity between child health and women health program (family planning program). Many developing countries have child health programs which are separate from women health or family planning program. Given the possibility that immunization program may have an impact on women's fertility, it is essential to understand the extent of immunization program on women's fertility. Child health programs and women health programs compete for limited funding for public health initiatives and other welfare programs, with budget constraints especially tight in poor developing countries. Do immunization programs affects women's fertility? I present my strategy for examining UIP's effect on fertility after briefly reviewing the related literature.

2.2   Related Literature

This section delineates the previous research related to the theme of this paper. This paper contributes to the related research in economics and demography. I am not aware of any previous study that rigorously quantify the effect of a childhood immunization program on fertility in a developing country setting. Kumar (2009) examines the effect of immunization program on children's outcomes and finds significant reduction in child mortality and mixed evidence on educational outcomes. This is one of the few papers that examines effects of childhood immunization program on women's outcomes.

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There is a large body of literature that examines the impact of child mortality on fertility. The existing literature addresses two type of questions: how women respond to own child mortality and another question that has been extensively examined is how women respond to expected mortality risk. This paper contributes to the literature on effect of own child mortality on fertility. Surprisingly, there is little empirical evidence of an effect of infant and child mortality on women's fertility. Different data, models, and methods yield inconsistent results.[3]. Existing literature documents that the death of a child leads to higher number of births for three different reasons: (a) Physiological effect- the death of an infant leads to sudden termination of breastfeeding, which, in turn, increases the period of exposure to a new conception. The magnitude of physiological effect is quite large and remarkably consistent across different demographic contexts (Jones and Palloni 1990). Fertility responses associated with this mechanism follow changes in infant mortality with a lag of one or two years, (b) Replacement effect- this effect induces families to have additional children to replace any child who dies in order to attain a desired number of children at the end of the reproductive lives. Unlike physiological effect, replacement effects dictates that fertility will be affected not only by infant mortality but also by child mortality, and (c) Insurance/Hoarding effect- this refers to the practice of bearing children beyond the desired family size to insure that the target number of surviving children is eventually attained. Hoarding effect works through changes in perceived mortality risk unlike the previous two channels which work through own child mortality. This paper does not attempt to explore the any of these mechanisms. Nevertheless, this paper examines the effect of own child mortality on fertility.

Empirical studies that looks at effect of own child mortality on fertility conclude that child mortality reduction modestly decreases the number of births, increases the number of surviving children, and thereby stimulates population growth.[4] Schultz (1969) shows that

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[3]Palloni and Rafalimanana (1999)

[4]See, for example, Preston (1978) for a collection of demographic essays that come to such conclusion and Palloni and Rafalimanana (1999) for a broad survey of literature; see also Rutstein (1974), Chowdhury et al. (1976), Balakrishnan (1978), Olsen (1980), and Olsen and Wolpin (1983).

a decline in child death rate is associated with fully compensating decline in birth rate. It is an exceptional results and is suspect because he used crude death rate as a proxy for child death rate. Hossain et. al. (2005) examines the effect of child mortality on fertility in six rural thanas in Bangladesh and find that child mortality increases subsequent fertility. Bhalotra and Soest (2008) finds that neonatal mortality results in increase in subsequent fertility in India.

Other studies (e.g. Barro and Becker 1988; Barro and Becker 1989; Dahan and Tsiddon 1998) suggest that exogenous decline in child mortality may increase fertility. In the Barro-Becker model (1989), infant and child mortality rates affect choices only to the degree that they influence the overall cost of a surviving child. Falling mortality rates lower the cost of having a surviving child, hence net fertility actually increases, not decreases, as mortality declines (this is discussed in Boldrin and Jones 2002 and Fernt'andez-Villaverde 2001). Dyson and Murphy (1985) present an excellent survey of a pre-decline increase in fertility that many countries experienced.

There is also a substantial amount of studies on the effects of expected mortality risk on fertility. These studies allow for uncertainty, and argues that when mortality is stochastic and parents want to avoid the possibility of ending up with very few (or zero) surviving children, a precautionary demand for children arises. This type of an increase in fertility in response to expected future child mortality is the "Hoarding Effect". A number of models have shown that under specific assumptions, fertility declines as a consequence of exogenous mortality reduction (O'Hara 1975; Sah 1991; Cigno 1998). Kalemli-Ozcan (2003) argues that as the child mortality falls, uncertainty about the number of surviving children falls, and this results in decrease in demand for precautionary children causing fertility to fall. Most recently, Doepke (2005) examined the relationship between child mortality and fertility in a Barro and Becker framework and finds that total fertility rate falls as child mortality declines.

Zenger (1993) and Frankenberg (1998) analyse the effect of child mortality on birth-

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spacing, but these studies have limited relevance as their estimates cannot be given causal interpretation (see, Moffitt, 2003). This paper overcomes this limitation and estimating a causal effect of the program on women's outcomes is the main contribution of this paper to the demographic research.

This paper takes advantage of a natural experiment to identify the effect of child mortality on fertility. I use district-by-cohort variation in exposure to UIP of the first-born child to obtain estimates of the effect of child mortality on fertility. It is one of only a handful of studies that addresses the issue of endogeneity in child mortality when estimating its effect on fertility.

3   Empirical Framework

The objective of this study is to estimate causal impact of a childhood immunization program on fertility and birth spacing. I provide empirical evidence for this causal relationship by combining information on fertility with information on the timing of implementation of UIP in different districts of India. I use variation provided by India's implementation of UIP in the 1980s. In particular, I estimate the program effect by utilizing the following two sources of variation in exposure to UIP: variation across districts and variation across cohorts. First, variation across districts comes from the fact that districts got the program in different years. Figure 2 in the Appendix shows the number of districts added on to UIP each year. UIP was implemented in 48 districts (31 according to old district definitions) in the first year, 92 additional ones in the second year and so on until all 563 districts (443 according to old district definitions) were covered in 1990.[5] Second, variation across cohorts comes from the fact that only children who are twelve months or younger when UIP was implemented would have been eligible to receive the shots. Table 2 in the appendix shows the schedule for the vaccines that UIP provided; the

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[5]The number of districts increased from 443 to 593 between the UIP period (1985-1990) and the RCH survey year (2002-04). The data section has more details.

shots are administered on a strict schedule in the first year of a child's life for maximal efficacy. Children older than one year were not treated by UIP. Table 3 of the Appendix shows the birth cohorts that were eligible for UIP by district's year of UIP inception. For example, a child born in 1985 would have been exposed to UIP if he lived in one of the 48 districts that implemented UIP first (in 1986), but not if he lived in a district that implemented UIP later.

The difference-in-differences approach used in this paper exploits variation in program's exposure for first-born child in the family. It uses only the within-district cross-cohort variation in exposure to UIP for first-born child to identify the effect of UIP. This is elaborated next.

3.1 Difference-in-Differences Strategy

Consider the following equation:

$$Y_{icd} = \beta_0 + \beta_1 UIP_{icd} + \delta X_{icd} + e_{icd} \tag{1}$$

where Y_{icd} is the outcome variables for an individual i, in cohort c residing in district d. UIP_{icd} indicates if her first born child is exposed to UIP. γ is district fixed effects and X is individual and household controls. We cluster the standard errors by district in all the regressions. UIP exposure variable is a dummy variable and is equal to 1 if (UIPyear - Birthyear) $\leq$ 1, and 0 otherwise.

The parameter β_1 in equation (1) can be interpreted as the causal effect of UIP under the assumption that the difference in outcomes between the women with the first-born as a UIP child and the women with the first-born as non-UIP child would have been the same between earlier-implementing districts and later-implementing districts in the absence of UIP. [6]

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[6] If UIP exposure were a simple interaction between two binary variables, say being in an earlier-implementing district and being in a younger birth cohort, then β_1 would be a differences-in-differences estimate, i.e., the cohort difference in outcome in earlier-implementing states that is in excess of the cohort difference in later-implementing states. In fact I use more variation in UIP exposure but the intuition is similar to the simple binary case and so I term my approach a difference-in-differences-type strategy.

One potential source of endogeneity can be the timing of of UIP's implementation across different districts. It may be possible that the implementing authority, Ministry of Health and Family Welfare (MOHFW) have used some selection criteria. As explained in the previous section, it was never indicated by the government official that selection decisions were linked to district level fertility levels or patterns. Moreover, the main equation includes district fixed effects, which take care of time-invariant unobserved characteristics that may affect fertility and may also be correlated with the timing of of UIP's implementation.

I also consider if length of exposure to program during period of women's childbearing age leads to different outcomes in terms of fertility behavior. To estimate the effect of duration of program's exposure, I estimate the following equation:

$$Y_{icd} = \beta_0 + \beta_1 N_{icd}^{20-30} + \gamma_d + \delta X_{icd} + e_{icd} \tag{2}$$

where N_{icd}^{20-30} is the number of years woman i, in cohort c living in district d has been exposed to the program during age 20-30 years.

3.2 Allowing for Heterogeneity in Program Effects

It may be possible that UIP has heterogeneous program impact. The impact may differ based on women's education, gender of the first child, socioeconomic status, rural/urban, caste, etc. For example, an educated mother would have a different fertility behavior than an uneducated mother. As another example, the program may have different impacts in rural areas from urban areas due to differences in the availability of health care infrastructure to deliver the services.

To test to examine whether there is a differential program impact by type of residence (Rural vs Urban), I estimate equation (1) separately for rural areas and for urban areas:

$$Y_{icd} = \beta_0 + \beta_1 UIP_{icd} + \delta X_{icd} + e_{icd} \qquad (3)$$

4 Data

My empirical analysis uses data from two sources: individual-level data from the Reproductive and Child Health (RCH) Survey and administrative data about UIP from the Ministry of Health and Family Welfare, Government of India.

The RCH survey is a large, nationally representative survey. RCH survey is a District Level Household Survey (DLHS) which has two waves. The first wave was conducted in 1998-99 in 504 districts and second wave was conducted in 2002-04 in 593 districts. Due to the timing of UIP, it is appropriate to use the second wave of the RCH survey. The survey contains information about 6,20,107 households and 3.2 million individuals. From these households, 5,07,622 eligible women, currently married aged 15-44 years whose marriage was consummated were interviewed. The survey is designed to collect data on marriage, fertility, family planning, reproductive health, maternal and child health, and HIV/AIDS. I use the "fertility module" to construct the sample for the analysis. Fertility history is collected for one woman who is aged 15 to 44 from each surveyed household. Women are asked about their birth history, including children ever born, dates of birth, if the children are alive, and, if not, when they died. The fertility file contains information on control variables as district, rural/urban, child sex, age of women,age at the consummation of marriage, women's age at each child's birth etc., and household social group, religion and socio-economic condition.

The Ministry of Health and Family Welfare provided administrative information about UIP. First, I talked to several UIP officials to find out the details of how UIP was implemented. It was these conversations that led me to believe that the timing of UIP could be considered conditional on district fixed effects. Second, I obtained from them a list of

new districts that implemented UIP each year, from year 1 (1985-86) to year 5 when all districts were covered (1989-90).

I mapped the year of UIP implementation from the district-level administrative data back to the individual-level RCH survey data using the district codes. One complication was that the number of districts increased from 443 to 593 between the UIP period (1985-1990) and the survey period (2002-04). Either an existing district split into two or more new districts or a new district was formed by taking areas from two or more districts. I successfully match 563 districts by looking at district census handbooks, district websites and other government sources (a success rate of 95 percent).

The women sample is reduced to 480,210 after matching the RCH data with the UIP roll out data. In our analysis, we only consider women gave birth at least once at the time of survey (we lose 54,457 women from the sample). We also restricted the sample to women who were of age between 13 and 35 at the time of birth of their first child (a loss of 274 women). This reduces the sample to 425,028. We consider women who are aged 30 to 44 at the time of survey. This age restriction is imposed in order to align the childbearing age of women with the roll out of the program. Women aged less than 30 years at the time of survey are too young to be exposed to the program when the program is rolling out. Finally, our main sample comprises of 202,167 women.

The main outcome variables for my fertility analysis are the probability of subsequent birth within 12 months of first birth, 24 months, 36 months, and 60 months conditional on the fact that first-born child is exposed to UIP. We also look at total fertility i.e. total number of children ever born and birth interval (time in months between first and second birth). We use log length of birth interval in order to have a normal distribution of this variable. The birth interval is the interval between reported dates of birth, rather than the inter-conception interval. As a result, measured birth intervals will be shorter on account of premature births (e.g., Gribble 1993). This issue is surmounted in this paper by dropping mothers with at least one birth interval less than 9 months.

The following control variables at women and household level are included in all the regressions. Since many previous studies find that better educated women tend to have fewer children, holding other resources constant (Mincer, 1963; Schultz 1981, 2002), I include mother's literacy as a dummy variable in all regressions. Years of schooling would have been a better variable to capture the education effect, but due to data limitation I do not use years of schooling.[7] I also include dummies for different religion based on the fact that Muslim fertility tends to be higher than Hindus, possibly due to unobserved cultural factors. Controls are also included for the husband's education as a measure of household income/wealth, which are not expected to reduce fertility as much as their wife's education because children occupy primarily women's time (Schultz, 1981). Finally, mother's age, household socioeconomic status, mother's age at first birth, gender of first-born are also included in all regressions.

Table 1 shows the descriptive statistics of the variables used in the fertility analysis. The paper uses retrospective fertility history of women born between 1958 and 1972. The total number of observations for fertility analysis is 202,167. In the sample, 67 percent of the women lives in rural areas and 42 percent belongs to low socio-economic status household. Majority of the women are Hindu (76 percent) and disadvantaged minority group ST and SC forms 32 percent of the sample. The mean mother's age is 36.80 years and 46 percent of the mother's are literate, whereas 71 percent of father's are literate. The mean mother's age is higher than the mother's age of the average child because the survey was done in 2002-04 and the paper uses the women who are between 30 to 44 years of age at the time of survey.

The mean age of mother at first birth is 19.24 years and mean age of mother's at marriage is 17.69 years. The average number of children ever born is 3.98 and the average number of surviving children in the sample is 3.54. Figure 1 shows the average number of births by different categories of mother: the average number of births for women in

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[7]In the survey, years of schooling are asked only if the individual is a literate.

the 30-34 age group is 3.65, for women in the age group of 35-39 is 4, and for women in the age group of 40-44 is 4.32 children. The monotonic relationship between age group and average number is birth is due to incomplete fertility of women in lower age category. Illiterate mother has higher average number of births than the literate mothers and also if the first born child is exposed to UIP, the average number of births is lower than if the first born is a non-UIP child. The mean birth interval (i.e. number of months between first birth and second birth) is 33.95 months (approximately 3 years). In sample, 37 percent of the women gave birth within 2 years after the first birth, about 68 percent of the women gave second birth within 3 years, and about 91 percent of the women gave second birth in next 5 years. on average, 41 percent of first born children are exposed to UIP and about 52 percent of these first born children are male.

5 Effect of UIP on Fertility

5.1 Basic Results

Table 2 reports the results of estimating equation (1) for different measures of fertility.[8] The main coefficient of interest is the coefficient for the variable "First-born is a UIP Child", which gives the effect of having the first-born exposed to UIP on different measures of fertility. Estimates from Column (1)- Column (3) suggest a significant negative impact of the program on Pr(subsequent birth within 2 years), Pr(subsequent birth within 3 years), and Pr(subsequent birth within 5 years). Estimates from Column (4) suggest a significant and negative program effect on total number of children ever born (another measure of fertility).

Results from column (1) and Column (2) suggest that the program decreases the probability of subsequent birth within 2 years by 1.4 percentage points and the probability of subsequent birth within 3 years by 2.3 percentage points. The results from Column (3)

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[8]I estimate these models using OLS, i.e., using the linear probability model with standard errors clustered at the district level. I also estimated these models using logit and find qualitatively similar results; these results are available upon request.

indicates that the program decreases the probability of subsequent birth within 5 years by 1.5 percentage points. It should be noted that dependent variables in Column (1)- (3) are subsequent births within next 2 years, 3 years, and 5 years after the first-born, and are dummy variables. Finally, Column (4) results suggest that program decreases the total number of children ever born by 0.03.

In all the regression models in Table 2, the signs of the control variables are as expected. Mother's education have negative and significant effect on all the measures of fertility. Poor and disadvantaged minority women (ST and SC) are more likely to have higher number of children. For Other Backward Caste and Muslim women, the estimates are positive and significant, meaning that women belonging to these categories have higher probability of giving births very soon after the first birth and also they have higher total fertility rate.

Estimates of the birth-interval equation are in Table 3. Since the dependent variable is in logs, the interpretation of the parameters is in terms of percentage changes of the expected length of the birth-interval. There is a strong positive effect of program exposure of the first-born on the length of birth-interval between first birth and second birth. The program increases the expected length of birth-intervals between first two children by about 2.5 percent. The gender of the first-born is also significant and consistent with son-preference. If the first-born is a boy, the expected birth interval is about 3 percent greater than if it the first-born is a girl. Parental education has significant effect on birth spacing as well. Mother's literacy has positive effect on birth intervals. Birth intervals are shorter amongst Muslim families by 6 percent, which is consistent with the findings in the literature (Bhalotra and Soest, 2008). There are no significant differences in birth spacing by socio-economic status, but there are significant differences in birth spacing by caste-groups. As expected, disadvantaged minorities (ST, SC, and OBC) have shorter birth intervals.

In Table 4, we estimate the effect of length of exposure of women to the program.

We also allow for a differential effect of exposure at different ages. The variable years exposed 20-30 are defined as the number of years women has been exposed to the program during childbearing age between 20-30 (a 10 year bracket). Age bracket of 20-30 years are chosen because this age bracket is the most fertile childbearing age group. We do not find significant effect of length of program exposure on total fertility (children ever born) among women aged 30-44 (full sample), among women aged 35-39, and among women aged 35-39. Results from Column (2) suggest that among women aged 30-34, one more year of exposure during childbearing age of 20-30 decreases total fertility by 0.04.

5.2 Heterogeneity in Program Effects

Table 4 and Table 5 explores the presence of heterogeneous effects along the dimension of type of residence i.e. rural vs urban. Table 4 reports the results for rural areas and Table 5 reports the results for urban areas. Results from Table 4 and Table 5 suggest that program did have a different effect in rural areas and urban areas. Subsequent fertilities are significantly reduced in both the areas but the negative effect of program is stronger in urban areas, which is expected. Urban women adjusted their short term fertility behavior quickly than the rural women. Column (4) in Table 4 suggests that the program significantly reduced the total fertility by 0.06 in rural areas but surprisingly, there is no such impact on total fertility in urban areas (Column 4, Table 5).

In all the regression models in Table 4 and Table 5, the signs of the control variables are as expected. Mother's education have negative and significant effect on all the measures of fertility. Poor and disadvantaged minority women (ST and SC) are more likely to have higher number of children. For Other Backward Caste and Muslim women, the estimates are positive and significant for majority of fertility measures, meaning that women belonging to these categories have higher probability of giving births very soon after the first birth and also they have higher total fertility rate.

6   Conclusion and Policy Implications

Using retrospective fertility histories from a large sample of Indian mothers, and the phase-in feature and eligibility rules of India's Universal Immunization Program immunization program, I estimate the causal effect of having a UIP exposed first-born on subsequent and total fertilities of women in India. This paper is the first rigorous estimates of the causal effect of UIP on fertility and subsequent birth spacing.

I find that the program significantly reduced subsequent fertility and total fertility. I also find that the program significantly increases birth intervals between first and second births. In addition, program has a stronger effects on subsequent fertilities in urban areas than rural areas. These results can be explained in terms of reduction of mortality and morbidity among children. Kumar (2009) finds that UIP had a negative impact on infant mortality and under-five mortality and this reduction in child mortality is believed to be the driving factor for negative impact of UIP on women's fertilities. Contrary to the popular belief that India is plagued with inefficient program implementation capacity and poor public health service delivery system, this paper establishes that UIP was successful in achieving its unintended objective of reducing fertility and increasing the length of birth intervals between first two births..

The results of this paper have important policy implications for the design of optimal health policy in developing countries. While the program had the intended benefit of increasing the survival probability of young children (Kumar, 2009), it also had an unintended benefit of reducing fertility and increasing birth intervals between successive births. A lesson may be that child health and women health policies have to be considered jointly and should be implemented in a integrated manner in order to reap the maximum benefits of these polices and interventions. A good example is implementation of Child Survival and Safe Motherhood (CSSM) program in India, which is a more integrated program and it targets both children and the mothers, unlike UIP which targeted children

only.

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Table 1: Descriptive Statistics

Variables	Mean	S.D.
First Born Child Level Variables:		
First Born	0.41	(0.49)
is a UIP Child		
Male	0.52	0.50
Mother's Level Variables:		
Birth within 2 years	0.37	0.48
Birth within 3 years	0.68	0.47
Birth within 5 years	0.91	0.29
Literate	0.46	0.50
Husband's Literate	0.71	0.45
Age	36.80	4.04
Age at first birth	19.24	3.38
Age at Marriage	17.69	3.37
Number of Children ever Born	3.98	1.82
Number of Surviving Children	3.54	1.52
Birth Interval (in months)	33.95	0.70
Household Level Variables:		
Rural	0.67	(0.47)
Low SES	0.42	(0.49)
Middle SES	0.31	(0.46)
High SES	0.27	(0.41)
ST	0.16	(0.37)
SC	0.16	(0.36)
OBC	0.37	(0.48)
Hindu	0.76	(0.43)
Muslim	0.12	(0.32))
Christian	0.08	(0.27)
Number of States and UTs	35	
Number of Districts	561	
Number of Observations	202,167	

Notes: ST, SC and OBC are Scheduled Tribe, Scheduled Caste and Other Backward Caste respectively. ST and SC are historically disadvantaged group. SES is socio-economic status of the households.

Figure 1: Average number of births by age, mother's literacy and UIP Child categories
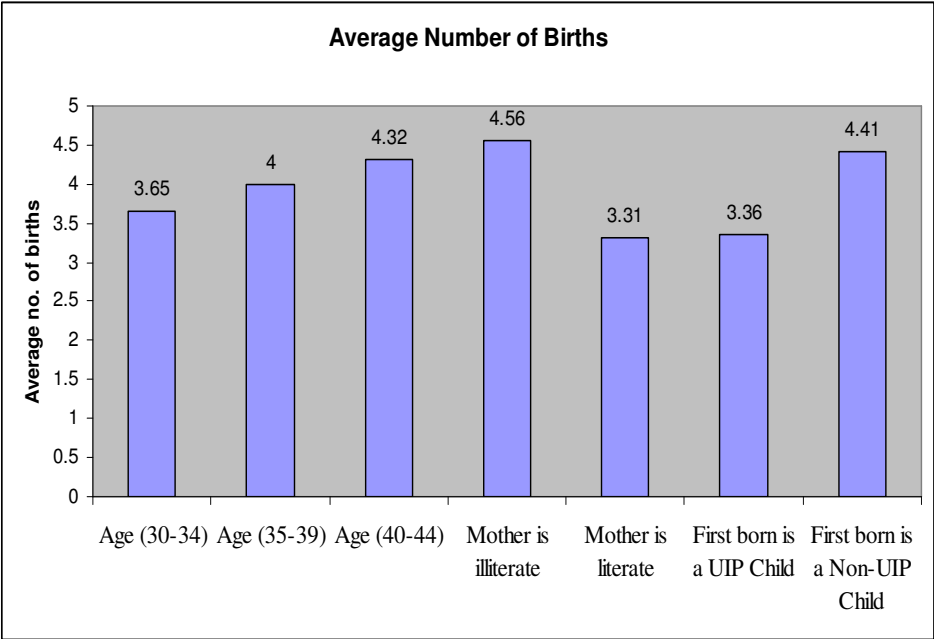
Table 2: Effect of UIP on Fertility

Independent variables	Pr(Subsequent birth within 2 years)	Pr(Subsequent birth within 3 years)	Pr(Subsequent birth within 5 years)	Total Number of children ever born
	(1)	(2)	(3)	(4)
First Born is a UIP child	**-0.014***	**-0.023***	**-0.015***	**-0.034***
	(0.004)	(0.004)	(0.002)	(0.012)
Poor	-0.003	0.005	0.003	0.69***
	(0.004)	(0.005)	(0.003)	(0.018)
ST	0.013**	0.028**	0.005	0.425***
	(0.006)	(0.006)	(0.004)	(0.019)
SC	0.015***	0.019***	0.007**	0.443***
	(0.004)	(0.004)	(0.002)	(0.019)
OBC	0.004	0.014***	0.006***	0.184***
	(0.004)	(0.003)	(0.002)	0.014)
Hindu	-0.001	-0.009	-0.009**	0.024
	(0.007)	(0.009)	(0.004)	(0.032)
Muslim	0.044***	0.047***	0.014***	1.023***
	(0.008)	(0.009)	(0.005)	(0.045)
Christian	0.021*	0.018	0.006	0.25***
	(0.010)	(0.011)	(0.006)	0.053)
Father's Literacy	-0.003	-0.003	0.008***	-0.194***
	(0.003)	(0.003)	(0.002)	(0.013)
Age at Marriage	0.001**	0.002***	0.001***	0.002
	(0.0007)	(0.0007	(0.0004)	0.002)
Mother's Literacy	-008**	-0.004	-0.001	-0.314***
	(0.003)	(0.003)	(0.002)	0.012)
Rural	-0.004	0.011***	0.012***	0.068***
	(0.003)	(0.003)	(0.002)	0.014)
First Child is a Male	-0.017 ***	-0.022***	-0.009***	-0.034***
	(0.002)	(0.002)	(0.002)	0.009)
District Fixed Effects	Y	Y	Y	Y
N	202,167	202,167	202,167	202,167
R Square	0.02	0.03	0.03	0.33

Notes: Each column is from estimating a separate linear probability model. Robust standard errors clustered at district
level are in parentheses. Poor is a dummy indicating household with low socio-economic condition.
Survey year dummy used. Scheduled Caste(SC) and Scheduled Tribe(ST)are traditionally disadvantaged minority
group. OBC is Other Backward Caste. All regressions include whether
first child is a twin, mother's age dummies and dummies for age of mother at first birth. RCH district
sample weights applied . * shows significance at 10-percent level, ** at 5-percent level and *** at 1-percent level.

Table 3: Effect of UIP on Birth Interval

Independent variables	Log Birth Interval
	(1)
First Born is a UIP child	**0.025*****
	(0.004)
Poor	-0.006
	(0.005)
ST	-0.022***
	(0.006)
SC	-0.026***
	(0.005)
OBC	-0.013***
	(0.004)
Hindu	0.007
	(0.008)
Muslim	-0.059***
	(0.009)
Christian	-0.025**
	(0.012)
Father's Literacy	0.002
	(0.003)
Age at Marriage	-0.003***
	(0.0008)
Mother's Literacy	0.010***
	(0.003)
First Child is a Male	0.026 ***
	(0.003)
District Fixed Effects	Y
N	200,608
R Square	0.03

Notes: Each column is from estimating a separate linear probability model. Robust standard errors
clustered at district level are in parentheses. Poor is a dummy indicating household with low
socio-economic condition. Survey year dummy used. Scheduled Caste(SC) and Scheduled Tribe(ST)are
traditionally disadvantaged minority group. OBC is Other Backward Caste.
All regressions include whether first child is a twin, mother's age dummies and dummies for age of mother at first birth.
RCH district sample weights applied . * shows significance at 10-percent level, ** at 5-percent level and *** at 1-percent level.

Table 4: Effect of UIP on Fertility: Exposure effects

	Total Number of Children Ever Born			
	30-44	30-34	35-39	40-44
Independent variables	(1)	(2)	(3)	(4)
Years Exposed 20-30	**-0.010**	**-0.04***	**-0.010**	**-0.011**
	(0.007)	(0.021)	(0.016)	(0.028)
District Fixed Effects	Y	Y	Y	Y
N	222983	71025	78880	63078
R Square	0.43	0.41	0.35	0.35

Notes: Each column is from estimating a separate linear probability model. Robust standard errors
clustered at district level are in parentheses. Poor is a dummy indicating household with low
socio-economic condition. Survey year dummy used. Scheduled Caste(SC) and Scheduled Tribe(ST)are
traditionally disadvantaged minority group. OBC is Other Backward Caste.
All regressions include whether first child is a twin, mother's age dummies, dummies for age of mother at first birth
RCH district sample weights applied . * shows significance at 10-percent level, ** at 5-percent level and *** at 1-percent level.

Table 5: Effect of UIP on Fertility- Rural

Independent variables	Pr(Subsequent birth within 2 years)	Pr(Subsequent birth within 3 years)	Pr(Subsequent birth within 5 years)	Total Number of children ever born
	(1)	(2)	(3)	(4)
First Born is a UIP child	**-0.010***	**-0.016***	**-0.009***	**-0.056***
	(0.005)	(0.005)	(0.003)	(0.016)
Poor	-0.011*	0.009	0.002	0.625***
	(0.006)	(0.006)	(0.004)	(0.021)
ST	0.013**	0.024***	-0.0002	0.443***
	(0.007)	(0.007)	(0.003)	(0.034)
SC	0.011**	0.014***	0.002	0.447***
	(0.005)	(0.005)	(0.002)	(0.024)
OBC	0.004	0.010**	0.002	0.163***
	(0.004)	(0.004)	(0.002)	(0.019)
Hindu	0.003	-0.015	-0.012**	-0.029
	(0.011)	(0.012)	(0.005)	(0.047)
Muslim	0.028**	0.032**	0.004	0.976***
	(0.012)	(0.013)	(0.006)	(0.065)
Christian	0.020	0.015	0.004	0.186**
	(0.014)	(0.015)	(0.007)	(0.074)
Father's Literacy	-0.005	-0.008**	0.005***	-0.195***
	(0.003)	(0.003)	(0.002)	(0.014)
Age at Marriage	0.003***	0.005***	0.003***	0.007**
	(0.0009)	(0.0008)	(0.0005)	(0.003)
Mother's Literacy	-0.006*	0.003	0.003	-0.236***
	(0.005)	(0.004)	(0.002)	(0.013)
First Child is a Male	-0.013 ***	-0.020***	-0.010***	-0.366***
	(0.002)	(0.003)	(0.002)	0.012)
District Fixed Effects	Y	Y	Y	Y
N	134,593	134,593	134,593	134,593
R Square	0.02	0.03	0.03	0.38

Notes: Each column is from estimating a separate linear probability model. Robust standard errors
clustered at district level are in parentheses. Poor is a dummy indicating household with low
socio-economic condition. Survey year dummy used. Scheduled Caste(SC) and Scheduled Tribe(ST)are
traditionally disadvantaged minority group. OBC is Other Backward Caste.
All regressions include whether first child is a twin, mother's age dummies, dummies for age of mother at first birth
RCH district sample weights applied . * shows significance at 10-percent level, ** at 5-percent level and *** at 1-percent level.

Table 6: Effect of UIP on Fertility- Urban

Independent variables	Pr(Subsequent birth within 2 years) (1)	Pr(Subsequent birth within 3 years) (2)	Pr(Subsequent birth within 5 years) (3)	Total Number of children ever born (4)
First Born is a UIP child	**-0.019*** (0.007)	**-0.036*** (0.007)	**-0.028*** (0.004)	**-0.002** (0.018)
Poor	-0.003 (0.008)	-0.002 (0.007)	-0.005 (0.005)	0.663*** (0.027)
ST	-0.0004 (0.011)	0.020* (0.010)	0.011 (0.007)	0.316*** (0.037)
SC	0.020*** (0.007)	0.02*** (0.008)	0.012*** (0.005)	0.417*** (0.023)
OBC	-0.001 (0.006)	0.015*** (0.006)	0.010*** (0.004)	0.199*** (0.017)
Hindu	0.007 (0.010)	0.001 (0.010)	-0.007 (0.007)	0.059** (0.030)
Muslim	0.065*** (0.012)	0.059*** (0.012)	0.016** (0.007)	0.998*** (0.041)
Christian	0.026* (0.015)	0.018 (0.016)	-0.001 (0.011)	0.288*** (0.049)
Father's Literacy	0.001 (0.001)	0.007 (0.006)	0.011*** (0.004)	-0.209*** (0.024)
Age at Marriage	-0.001 (0.001)	-0.004*** (0.001)	-0.001* (0.0007)	-0.01*** (0.003)
Mother's Literacy	-0.007 (0.006)	-0.015** (.006)	-0.010*** 0.003	-0.457*** (0.019)
First Child is a Male	-0.024 *** (0.004)	-0.028*** (0.004)	-0.009*** (0.003)	-0.283*** 0.013)
District Fixed Effects	Y	Y	Y	Y
N	67,574	67,574	67,574	67,574
R Square	0.03	0.04	0.03	0.45

Notes: Each column is from estimating a separate linear probability model. Robust standard errors
clustered at district level are in parentheses. Poor is a dummy indicating household with low
socio-economic condition. Survey year dummy used. Scheduled Caste(SC) and Scheduled Tribe(ST)are
traditionally disadvantaged minority group. OBC is Other Backward Caste.
All regressions include whether first child is a twin, mother's age dummies, dummies for age of mother at first birth
RCH district sample weights applied . * shows significance at 10-percent level, ** at 5-percent level and *** at 1-percent level.