Decomposing the Fertility Response to a Mortality-Reducing Intervention in Matlab, Bangladesh

Julia Driessen*

University of Pittsburgh

August 2011

Abstract

This paper examines how individuals alter their fertility decisions in response to a decline in child mortality risk due to the introduction of a measles vaccination program. Past research on the fertility-mortality link looks at 'totals': net fertility outcomes (pregnancies, birth intervals, etc.) in light of net child mortality losses. I use panel data from Matlab, Bangladesh, and examine fertility on a sequential basis, allowing me to decompose the total effect into the sequential decisions. I examine how mothers with varying fertility and child mortality histories adjust their fertility differently to the measles vaccination intervention. The use of fixed effects allows me to address unobservable fertility preferences that might otherwise bias my results. The importance of the sequential approach and the examination of heterogeneity is underscored by my identification of several significant and contrasting fertility responses based on fertility characteristics. This variation is governed by factors such as the number of living children, the number of living children eligible for vaccination, and the number of sons. Solely focusing on the total, overall fertility effect thus underestimates the effect of measles vaccination in this community; with increasing emphasis on reaching vulnerable populations with such interventions, this approach offers valuable insight into how such groups will respond. More generally, this analysis details how a child health intervention is absorbed into the fertility decision process, and how this shift in the utility of future children varies within the population.

*Thanks to Stephen Shore, Robert Moffitt, David Bishai, and Tiemen Woutersen for their comments. The usual disclaimer applies. Comments are welcome. Address: Julia Driessen, A614 Crabtree Hall, 130 De Soto Street, Pittsburgh, PA 15261. Email: driessen@pitt.edu

1 Introduction

Measles continues to be one of the primary causes of child deaths (WHO, 2009); thus, it is a natural target of the United Nations, which has made child mortality reduction one of its Millennium Development Goals. The effectiveness of the measles vaccination in decreasing child mortality is well-established (WHO, 2009), so increasing access to measles vaccination in underdeveloped areas amounts to decreasing the risk of child mortality. This paper looks at the link between vaccination availability and fertility in Matlab, Bangladesh. Ultimately, understanding how individuals react to both the risk and the realization of child mortality allows these health interventions to operate more effectively. Most of the literature focuses on the link between prior experienced mortalities and fertility, but analyzing this health intervention gives some insight instead into how the *risk* of child mortality affects fertility decisions.

Child mortality shocks are predicated on the same basic premises as the shocks more commonly examined in the risk and consumption literatures, so an economic framework adds useful structure from which to analyze the behavioral response to this loss. An individual can devote resources to market or home production, the latter of which includes investing in children. Implicit in the decision of family size is a preference for quantity or quality; resources can either be sparingly invested in many children, or heavily invested in a few. This classic dilemma is compounded by the uncertainty of child survival. This uncertainty could tip the balance either way: an individual may prefer quality because she intends to heavily invest to raise her children's probability of survival, or she may choose quantity to minimize the impact of any one mortality shock.

Measles has both morbidity and mortality consequences. However, child morbidity increases the risk of child mortality (measles can lead to longer-term complications that result in death), so in this paper I will generally refer to the mortality implications of measles. By extension, then, the introduction of measles vaccination to an area can be thought of as a decline in child mortality risk. Note that there are significant indirect

herd immunity benefits of the vaccination, so even just living in a treatment area confers some protection.

This paper examines the fertility response to a measles vaccination program in the upazila, or subdistrict, of Matlab, Bangladesh. Since the early 1980s, overall child mortality has declined dramatically in this area, mostly due to health interventions reducing the prevalence of common childhood diseases (Fauveau 1994, ICDDR,B 2004). Matlab is a good barometer for child health trends in the less developed world, and our understanding of prevention and response to child mortality is important as child health, especially child mortality reduction, becomes an important public health issue.

This research contributes to the existing literature by dissecting the general treatment effect to identify how this intervention actually operates in the population; it is likely that the decline in mortality risk is absorbed heterogeneously within the population. This process emphasizes the importance of analyzing fertility with a sequential, rather than total, approach, looking at fertility decisions individually (yearly) rather than in the aggregate. The sequential fertility results differ significantly from those found using a total fertility approach. Most importantly, the absence of an overall fertility effect is explained by the identification of several strong and conflicting fertility effects of the measles vaccination intervention. This variation in fertility responses is based on fertility characteristics.

The work uses two unique features of the Matlab, Bangladesh surveillance site. First, I take advantage of the panel nature of the data to examine the fertility-mortality link in a sequential fertility setup. I circumvent the endogeneity concern, a standard problem in the fertility-child mortality literature, with fixed effects, recognizing the importance of preferences in the fertility decision. This approach also allows me to explicitly control for the availability of other health interventions over time.

Second, I exploit the staggered introduction of the measles vaccination into the area to examine how individuals adjust their fertility to this intervention, and how this re-

action varies by fertility characteristics. The results have two useful interpretations, depending on how one chooses to interpret the vaccination program: there is the standard program evaluation angle, which can then also be generalized to reflect the response to an exogenous improvement in child health.

In Section 2 the importance of sequential fertility is explained, followed by a review of the relevant literature in section 3. Section 4 describes the data, followed by the empirical strategy in section 5. The results are presented and discussed in sections 6 and 7.

2 The Importance of Sequential Fertility Analysis

While the dynamic aspect of the fertility process is often overlooked for simplicity, its essential role has been recognized: "An important aspect of fertility decisions is that they are dynamic and stochastic. Past fertility choices stochastically influence, through mortality, the number and the age composition of children currently alive. This, in turn, influences current and future fertility decisions" (Sah, 1991).

The standard total fertility approach relies on a two-period model: in the first period, the individual chooses to have k children (all at once!), and then in the ensuing period the survival/mortality uncertainty is realized. This model is too simplistic, and in no way resembles the actual fertility decisions. Note the use of the plural form here, since there is not one fertility decision, but many. Each decision is an investment, and each choice is made under different conditions and different information. The soundness of any investment is *relative*; the time and resources required to raise a child could be used elsewhere, and the relative availability and attractiveness of these options are time-sensitive. For example, a shigella outbreak one year may reduce the expected return of having a child, since it lowers the probability of a child's survival. In that case, an individual may forgo having a child, and invest her resources in additional work or the quality of her existing children. The literature acknowledges that perceptions are formed by contemporaneous

events both within and outside of the family unit (Montgomery 1998, Grether 1980); a total fertility model overlooks this source of information-updating. Sequential fertility allows for a mother's information set to evolve, including a discount factor of sorts and recognizing the importance of timing.

Empirically, total fertility analysis often muddles past, present, and future, as in a simple regression of total children born on total child mortalities. In reality, each individual fertility decision considers only the mortalities up to that point, and lumping them all together dilutes the true link. It is particularly important when considering the fertility response to child mortality; prior research has shown that the replacement effect (accelerating up fertility after the death of a child) is strongest immediately after a loss and wanes within a few years.

In short, models of sequential and total fertility rely on different assumptions about what is important in the fertility decision. This paper is predicated on the belief that individuals determine early on an 'ideal' number of children, k, and then decide on the number of births, b, necessary to have those k surviving children. The fact that b is usually greater than k is termed 'hoarding' against potential losses. The hoarding effect is contrasted with the 'replacement' effect. As a mother ages and experiences child health shocks, she adjusts the number and spacing of births to improve her chances of attaining her ideal family size. These adjustments in fertility are overlooked in the total fertility model, which does not allow for what amounts to second derivative changes later in a woman's life.

Large-scale interventions typically aim to effect change within the community as a whole, and it is this change that is often the focus of program evaluation studies. However, often these efforts also emphasize promoting change among vulnerable subgroups. For example, the family planning program that was unveiled in Matlab in the late 1970s attempted to improve maternal and child health throughout the region, but it particularly sought to increase awareness and access to these products and services among less

affluent and educated adults, since those who were wealthier and better-educated often already had these options.

Thus this research recognizes that an intervention has not just an end goal, but also a means to achieve it. Knowing the general response to a decline in risk or gain in child health does not necessarily indicate how at-risk groups are responding. Armed with sequential fertility I identify how the measles vaccination intervention impacts the individual fertility decision process, and how this influence varies among women with different fertility experiences.

3 Literature Review

As in many underdeveloped rural areas, in Matlab, Bangladesh the practical benefits of children are numerous; the more obvious roles include child as protector, worker, and future provider. Prior research documented that children in rural Bangladesh begin helping to support their families as early as five years of age (Salahuddin, 1981), and male children are the primary earners by their teens (Cain 1977, Khuda 1991). The higher return associated with males has contributed to 'son preference' throughout South Asian countries; of those, Bangladesh exhibits the largest preference for sons over daughters (Khan & Khanum, 2000).

Empirical treatment of the fertility-mortality link often assumes away the possible endogeneity of child mortality. Ben-Porath (1977) confines its examination to the fertility response to child mortality, without addressing the potential endogeneity of the fertility decision. The standard demographic language of child mortality is used to examine this question. Namely, this paper is interested in the impact of child mortality on birth intervals (time spacing between births) and stopping probability (probability that a given birth is the woman's last). These outcomes are looked at in terms of the total number of child deaths to a mother. Portner (2008) considers the impact of income shocks due to hurricanes on fertility and educational investment. The author used

ordinary least squares to examine how hurricane risk and experienced hurricanes impact fertility; since hurricanes are perceived to be random, there is no concern about endogeneity. The results suggest an insurance effect, as fertility increases with risk; there is also evidence of a replacement effect, since experienced hurricanes are associated with increased child mortality. This paper discusses the desirability of sequential fertility analysis, though data limitations prevent it in this setting.

When the endogeneity issue cannot be conveniently assumed away, empirical analyses of the fertility-mortality link have used instrumental variables estimation. Joshi and Schultz (2007) evaluates the family planning and maternal health program in Matlab that began in the late 1970s. Rather than evaluating the overall treatment effect, it interacts being in a treatment area with age group dummies, and the results show significant declines in fertility and improvements in maternal and child health. In this case there is not strong evidence that fertility is endogenous in a model of child mortality. Schultz (1996) analyzes the fertility-mortality link in the Ivory Coast and Ghana, using access to water and other sanitation measures as instruments for mortality. No significant mortality-fertility link is found when mortality is assumed to be endogenous; a positive link is found when mortality is assumed to be exogenous. In both cases, a total fertility approach is taken, so the outcome is total number of children born to a woman.

Theoretical treatments of the mortality-fertility link differ in their conclusions. Wolpin (1984) allows for time-varying variables and area-specific survival rates in a finite horizon, dynamic discrete-choice model of the fertility decision. The taste parameter in this model is mean zero with finite variance, and there is no updating or influence of past mortality and fertility experiences. The results suggest that mortality and fertility are inversely related, or women bear more children overall in higher-survival areas. The opposite relationship is identified in Sah (1991), a result which is argued to match up better with empirical findings. The exogeneity-endogeneity dilemma is analyzed in

Cigno (1998), finding that fertility and mortality move in opposite directions if mortality is exogenous, and they move together if mortality is endogenous. This dichotomy is interpreted as a statement about whether or not parents can influence their risk of experiencing a child mortality loss. These findings conflict with those in Kalemli-Ozcan (2003), which finds comovement of fertility and mortality with an exogenous survival rate.

4 Data

4.1 Matlab

My analysis of the fertility response to the measles vaccination program uses data from Matlab, Bangladesh. Matlab is a surveillance site, with a population of about 200,000; data collection began in 1967. It is divided into a treatment area (blocks A-D) and a control area (block E) (see figure 1). A variety of health interventions have been introduced into the treatment area, beginning with a maternal health and family planning program in 1977. It is important to note that family planning and consultations were also available in the control area, but fortnightly home visits were only made in the treatment area. Childhood vaccination and nutrition programs were introduced in the treatment area in the 1980s.

Data on the households are collected monthly, with more extensive surveys conducted every 10-12 years. In keeping with my hypothesis that time-varying factors influence an individual's fertility decision, I constructed a panel data set using the 1996 Matlab Health and Socioeconomic Survey (MHSS). This survey includes detailed fertility histories for each of the approximately 5000 ever-wed women in the sample. Using this information I constructed a data set that looks at the fertility of each woman on a yearly basis- namely information on children born and lost each year. Thus, an observation in my data set is a snapshot of one year of a mother in the sample between the ages of 15

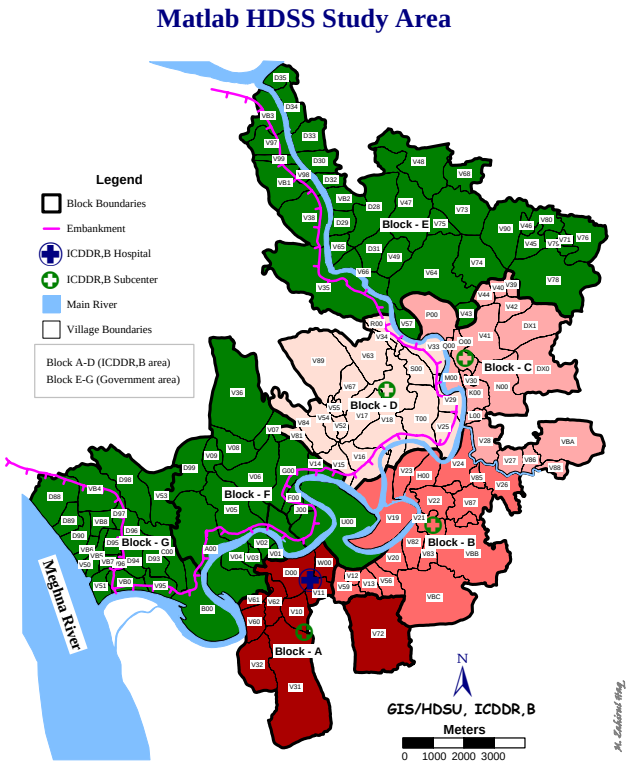

Figure 1: Matlab, Bangladesh

and 50 years. Each observation includes data on how many children were born to that mother in that year, the number of surviving children she had previously, the number of children who died that year, and the number of children who died in prior years. I also know the gender of each child. The distribution of births by year is shown in table 1.

The historical context is also addressed; this reflects that the individual is making a fertility decision based on more than just individual and familial variables. For example, riskier periods such as times of conflict and disease outbreak are noted. There is also a measure of the role of disease in child mortality in each year. Most importantly, the

data set indicates which interventions are available to the mother and her children each period; these interventions included maternal health and family planning, vaccinations, and antenatal care. An indicator variable for each intervention indicates whether the individual had access to that intervention, and allows me to isolate the effect of the measles vaccination program.

Finally, the data set includes information on socioeconomic status. There is data for both parents about educational attainment, age, occupation, and religion. There are also indicator variables for village, and information on whether the mother uses any type of family planning.

The panel data set allows me to look at child mortality in multiple ways- for example, the number of shocks in the last year, or the number of children under five who died in the past five years. These measures are specific to the time period t and mother i. In a total fertility context, I would be limited to the total number of mortality shocks, with no sense of their timing.

The statistics in table 2 summarize the distribution of some of the important explanatory variables in the data. The prevalence of child mortality losses in this area is underscored by the fact that, on average, each mother experiences almost one child mortality. Note that this data set includes only women who have at least one child at the time of the 1996 census; this decision was made because some women are unable to have children, so this decision is not applicable to them, and those who choose not to have children are possibly unobservably different from their child-bearing counterparts.

4.2 Intervention

Measles is a highly contagious disease that is particularly potent among young children with poor nutrition; in this population, as many as 10% of cases result in death (WHO, 2009). Research on the mortality impact of the measles vaccine in Matlab found that it reduced the short- and long-term mortality of children under the age of 5 by 40%

(Koenig, 1990).

The introduction of the measles vaccination was staggered throughout the treatment area (blocks A-D). Blocks A and C received the intervention beginning in 1982, and it was extended to blocks B and D in 1985. At the time, the recommended age range for vaccination was 9-12 months, but children up to 5 years of age were eligible for the vaccination. Prior to the intervention, vaccination rates were well less than 1%, and

The staggered nature of this intervention means that there are several layers to the identification; I am looking at variation at the individual level (before and after the intervention is introduced), within the treatment area (blocks A and C had the intervention before blocks B and D) and between the treatment and comparison areas. The accumulated nature of these interventions since the mid-1970s means that block E, the designated control area, may be quite different from the treatment blocks. Prior research has shown that any difference in fertility between the treatment and comparison areas in the early 1980s is due to the heightened availability of birth control in the treatment blocks (Joshi & Schultz, 2007). I know the timing of birth control usage for each woman, so I can control for the potential for pre-existing fertility differences. To identify any differences in observables within the treatment area, table 3 compares blocks A and C with blocks B and D in 1981. The only significant difference between the two areas is educational attainment, which is observable and thus can be controlled for.

It is also important to note that this study does not use data on vaccination status; the focus is on exposure, so the analysis merely differentiates between those who are living in an area with the vaccine and those who are not. This choice was made because looking at vaccination status would introduce additional endogeneity concerns, and the herd immunity benefits of living in a highly-vaccinated area are significant. Measles has a particularly high rate of reproduction, with each infected individual spreading the disease to 12-18 other individuals in un-immunized areas (Centers for Disease Control

and Prevention). Given the high rates of uptake after the measles vaccination program was introduced, just living in an area with the vaccination provides significant protection. Therefore, the public good nature of measles vaccination makes vaccine availability a cleaner way to analyze how a decline in mortality risk affects fertility.

5 Empirical Strategy

How do individuals adjust their fertility in response to an exogenous decline in child mortality such as through the introduction of measles vaccination? Furthermore, how does this response vary based on fertility history?

5.1 Hoarding vs. Added Value

The premise of the set-up is that each period (year) a woman chooses whether or not to have a child. That decision, B_{it}, is observable; let B_{it}= 1 if the individual i has a child in year t, and B_{it}= 0 if the individual does not have a child in that period. The underlying decision process is captured by the latent variable B^*_{it}. Suppose that B^*_{it} follows:

$$B^*_{it} = B(F_{it}, H(\bar{K}, p), R(K_{it}, p), X_{it}, a_i) \tag{1}$$

$$B_{it} = 1[B^*_{it} > 0], \tag{2}$$

where B^*_{it} can be thought of as the net benefit to mother i of having a child in year t; F_{it} is a vector of fertility history variables; $H(\bar{K}, p)$ is the hoarding impulse as a function of $\bar{K}$, the ideal number of children, and p, the perceived survival probability of an additional child; R represents the expected return on an additional child as a function again of the survival probability and also the number of living children K_{it}; X_{it} is a vector of individual socioeconomic characteristics; and a_i represents a mother-specific fertility preference. Individuals choose to have a child if the expected net benefits of

this addition are positive ((1)>0).

In the context of the model the measles vaccination intervention can be interpreted as an increase in p. The data set observes mothers both before and after the introduction of the intervention, and the randomized nature of the intervention allows for it to be treated as an exogenous change in p in the model. The question is then how the competing tensions of child value and hoarding play out to affect fertility when the measles vaccination increases p. This model suggests the following about a change in survival probability:

$$\frac{\delta B_{it}^{*}}{\delta p} = B_H \frac{\delta H}{\delta p} + B_R \frac{\delta R}{\delta p}. \tag{3}$$

The expected return on an additional child is necessarily increased as the expectation of survival rises. On the other hand, the need for hoarding declines in the presence of improved survival probability. This implies that the impact of a decline in mortality risk (an increase in p) is ambiguous, and depends on the trade-off between the value of an additional child and the decline in the hoarding effect. If the value of the additional child is large enough, the individual will expand her family. The decline in risk makes hoarding less necessary, so if the initial impulse to have a child is more due to hoarding (a version of quantity over quality), then the individual will no longer feel compelled to have an additional child. This conflict also reinforces the need for examining heterogeneity in the fertility response- it is very likely that the relative importance of expected return and hoarding vary over a mother's fertility lifespan.

5.2 Econometric Specification

A straightforward treatment-control design can be used to identify how the measles vaccination program impacts fertility by increasing the survival probability p. The staggered, randomized nature of the intervention means that program availability can be treated as exogenous, and a variable $MeaslesVacc_{it}$ indicates whether mother i is

in an area with the vaccination in period t. Joshi and Schultz (2007) confirms the similarity of the treatment and comparison areas prior to 1977, when the maternal health and family planning program was introduced. To account for the existence of this program, I will condition on the use of birth control, BC_{it}, since the 1977 family planning program is the primary source of any fertility differences between the blocks A-D and block E pre-intervention. The inclusion of a fixed effect a_i will also absorb any pre-existing differences in fertility preferences across individuals and between areas.

Appropriate treatment of F_{it}, individual i's fertility history prior to period t, demands more than just K_{it}, her current number of living children. The use of children for labor coupled with the emphasis on hoarding suggests that the stock of children has a non-uniform effect on the desire for additional births. Those with no children are likely to feel more urgency, while those with a higher-than-average child stock are likely experiencing a stronger hoarding impulse than their peers with smaller families. Thus, rather than including K_{it}, it seems more appropriate to include indicators for no living children and five or more living children.

Similarly, recent fertility events are also likely to disproportionately affect the fertility decision. Women who have recently given birth are less likely to do so in the current period, so it seems prudent to include indicators for whether births occurred in prior periods (B_{t-j}). Similarly, child mortalities are known to speed up fertility through the "replacement impulse." This impulse is rather short-term, so it again makes sense to identify whether a mother has lost a child in the past few years (M_{t-j}). The last five prior periods (years) are considered, which also restricts the minimum age of mothers included in the analysis.

It is also important to control for other factors that may be influencing p. For this reason, a vector of dummy variables T indicating availability of other treatments is also included. For example, this would disentangle the effect of measles vaccination from that associated with the introduction of a trio of vaccinations that occurred in blocks

A-D in 1986.

Without the individual fertility preference factor a_i, the decision model would assume exogeneity of the fertility decision. If this is not the case- if, for example, the number of child mortality losses an individual has recently experienced is correlated with the error term- then the estimation would be biased and inconsistent. Namely, this could be due to an individual's unobserved preference for child quantity or quality. This could very well have a bearing on the individual's number of recent child losses, and also likely influences her fertility decisions. Furthermore, while there is evidence that the introduction of the intervention was random in observables, the use of fixed effects will control for any nonrandomness associated with these unobservable fertility preferences.

The panel nature of the data allows me to deal with this potential endogeneity with fixed effects estimation- I have enough observations of each individual to estimate a mother-specific preference term a_i. Instrumental variables estimation is a common solution in the mortality-fertility literature for addressing the potential endogeneity of mortality shocks, but with fixed effects estimation I do not need to make assumptions about relationships among the variables. Fixed effects allows for a mother-specific component in the fertility decision, which thereby incorporates any unobserved, mother-specific link between fertility and child mortality. As mentioned previously, this measure of preferences also helps ensure the exogeneity of the vaccine program. The trade-off for the absorption of so many unobservables in the preference term is that I must assume that fertility preferences are constant over the period of the analysis. This rigidity is relaxed somewhat by the fertility history indicators, which allow for fluctuations due to fertility events.

Given a mother i of age A in year t, the equation to be estimated takes the following form:

$$B_{it} = \alpha_1 + \alpha_2 \mathbf{1}\{MeaslesVacc_{it}\} + \sum_{j=1}^{5} \alpha_{3j} B_{i,t-j} + \sum_{j=1}^{5} \alpha_{4j} M_{i,t-j} + \alpha_5 \mathbf{1}\{NoKids_{it}\}$$
$$+\alpha_6 \mathbf{1}\{FivePlusKids_{it}\} + \alpha_7 A_{it} + \alpha_8 BC_{it} + \sum_{j=1}^{m} \alpha_{9j} T_{itj} + a_i + \epsilon_{it} \quad (4)$$

5.3 Heterogeneity in the Fertility Response

The sequential fertility approach allows not only for measurement of the general effect of the measles intervention on the individual fertility decision (α_2), but also for identification of variation in that response based on fertility history.

The source of this variation is the natural conflict of factors brought about by the change in p. The effect of a risk-changing tool, such as measles vaccination, on fertility was shown to depend on the relative strengths of the improved return on an additional birth and the decline in the need for hoarding. Thus, in families for whom an additional birth fills an immediate need, such as the need for additional labor on family land, the prevailing effect is likely to be positive and fertility would be expected to increase. On the other hand, those who are continuing their fertility primarily for 'hoarding' purposes are predicted to lower their fertility in response to the vaccination program, since the lessened mortality risk makes hoarding less urgent.

The relative strength of the return and hoarding impulses would be expected to vary over the fertility cycle. Thus, family size is a natural factor in determining the fertility response, since it directly determines hoarding- as the number of living children increases, the immediate utility of each additional birth declines. It follows that (4) should be re-estimated with the addition of an interaction of family size with treatment exposure.

More specifically, within family size the number of children eligible for the vaccine is also likely to affect the fertility response, since those are the only children actually

receiving the health benefit (whether directly or through herd immunity). In this way, the intervention may affect current fertility by improving an individual's future fertility history; the vaccination has the potential to improve both quantity (by reducing child mortality) and quality (by reducing child morbidity). Thus, equation (4) should again be re-estimated with the addition of a variable interacting the number of children under five years of age with treatment availability.

The literature on the fertility-mortality link would suggest that lower input costs would affect the replacement impulse, so it is worth looking at how those with recent child mortalities respond differently to the intervention; this is just a matter of adding a variable interacting the number of recent child mortalities with an indicator of treatment exposure. Another possible mechanism by which the vaccination could affect replacement is if the child mortality response itself has a quantity-quality shift. Surviving children are healthier in areas with the vaccine, which may dampen the need to for additional births after the loss of a child.

Finally, one can also draw on the extensive literature on son preference in Bangladesh in identifying potential sources of variation in the treatment response. The literature suggests that families with a lower proportion of boys have accelerated fertility; one could think of this as a hoarding effect solely for boys. Similarly, families with one or two boys experience sharp declines in their fertility. Thus, to tease out the expected value-hoarding contrast based on gender composition, another version of this equation to estimate would interact the number of sons with treatment availability.

6 Results

6.1 The Sequential Fertility Response and Heterogeneity

The effect of the intervention on fertility was analyzed using fixed effects logit estimation. The results support both sequential analysis and the decomposition of the treatment

effect, finding significant responses to the measles vaccination that the general, total treatment analysis could not detect. These responses work in strong and opposing directions, motivated by recent fertility events, and thus 'net out' to give the appearance that fertility is unaffected by the measles vaccination program when examined from the perspective of total outcomes.

For comparison, table 4 gives examples of total fertility estimation of the impact of the measles vaccination program, and also shows the limitations of detailing the treatment effect in this setting. The results suggest that the intervention does not impact fertility decisions, both overall (column 1) and based on child mortality history (2). Note, I am limited to using just 'total' child mortalities and 'total' births, without any sense of their relative timing; furthermore, total fertility means that the outcome is total children born as of 1984, rather than whether an individual has a child in 1984. These results are similar to those referenced in Joshi and Schultz (2007)- using this method there does not appear to be a fertility effect of the measles intervention.

Table 5, by contrast, examines the intervention from a sequential fertility vantage; the corresponding percentage change in odds is given in table 6. The first column in these tables looks at the overall treatment effect, without any parsing by fertility history. Just as with the total fertility results, no overall fertility effect is found; as is evident in the later columns, this is because there are numerous strong and contradictory fertility responses at work within the population. The other significant findings in column 1 are not surprising: women who have experienced a child death in the last year are more likely to have another birth ("replacement effect"), and there is a 'cascading' influence of past births on the likelihood of a current birth; past births decrease the probability of giving birth in the current year, but the negative effect wanes over time. These results include fixed effects; a Hausman test of the need for fixed effects in this logit model generates a convincing test statistic of 1479.32.

Column 2 examines how response to the measles vaccination varies by number of

children in the household, and is the first indication that the availability of the vaccine is absorbed significantly into the fertility decision-making process. With the help of sequential fertility I identify this nuance in the fertility response to the vaccine program. On average, women in the sample have 3.5 living children, so this version looks at the extremes- those with no living children and those with five or more living children. Women living in a treatment area with no living children are more likely to have a child. This can be interpreted as the vaccine increasing births among women who otherwise are disinclined to have children, or would delay having children; the vaccine reduces the risk and increases the expected benefit of a child relative to the parent's other investment options.

In contrast, the results pick up a general decline in the probability of birth due to having five or more children, but there is an additional decline associated with having five or more children and residing in a treatment area. This result again supports the theory that a decline in mortality risk dampens the 'hoarding' effect. Mothers with more children (i.e., mothers closer to their 'ideal' number) do not overshoot as much because the intervention gives their current children a higher survival probability. There is no evidence of a fertility response among those with 1-4 living children; these families are likely to have the highest marginal benefit of additional births, suggesting that vaccination availability reinforces those who want to slow down or stop their fertility but does not significantly affect those who ceteris paribus desire additional children.

Column 3 presents a twist on the prior results- parsing number of living children to examine how directly benefiting from the treatment influences fertility. The vaccine was given to children under the age of 5 years, so the treatment variable is interacted with the number of children under the age of 5 years in the family. The results again support opposing and statistically significant fertility responses depending on receipt of these direct benefits. Those in the treatment area who do not have any children eligible for the vaccination experience a slight increase in their fertility, again suggesting that

they are responding to the increased expected return of additional vaccinated children. On the other hand, individuals in the treatment area with vaccination-age children are less likely to have another child, and see an overall decline in their fertility if they have more than one child eligible for the vaccination. This result speaks to how a decline in mortality risk is absorbed in the fertility process. The effects on both current and future children are recognized, and in different ways. Those who are already directly benefiting from the vaccine curb their fertility- if a mother has a child who can receive the vaccination, then she appears to revise down the number of births needed to attain her 'target' number of children; this is done because the vaccination-eligible children are more likely to survive. So there appears to be a decline in the 'hoarding' effect; another way of saying this is that there is a shift from quantity to quality as the mortality risk to current children declines and the benefits of the vaccine are realized.

Column 4 takes this decomposition of the treatment effect in a different direction, looking at how response varies based on prior child mortalities. The availability of the vaccination does not appear to affect the strength of the replacement effect. This absence of influence suggests that healthier surviving children do not substitute for the loss of a child, implying that the quantity-quality tradeoff is not realized in the context of child mortality. If this concept was present in this measure, then the death of a child (decrease in quantity) would represent less of a shock because existing children in treatment area families are healthier.

Finally, in column 5 the fertility response to the intervention is evaluated based on the gender of living children. Parents in the treatment area who do not have any sons increase their fertility in response to the vaccination. The findings suggest that son preference does indeed affect the fertility response to the intervention, as each male child in an intervention area decreases the probability of having another child, with the vaccination availability having an overall negative effect on fertility for families with more than two more boys. Any living sons are either protected against measles by herd

immunity or direct vaccination, so there is less need to have additional children to guard against any future mortality losses. This is in addition to the general fertility decline associated with additional male children that is observed in the results.

It is worth noting that while the son preference effect has been described as culturally-rooted, there is also a biological component. Birth ratios naturally favor males, who are more susceptible to illness early in life. Thus, one can also say that proportionally, boys benefit more from the vaccination than do girls.

6.2 Treatment Response with Impulse Response Consideration

Now that fertility effects have been established, it is important to control explicitly for the effect of recent births in areas with the vaccination. The prior tables had general lagged birth dummies that reflected the recent birth histories of mothers in both the treatment and control areas. In addition to these recent birth indicators, this extension also allows for an effect of recent births in the treatment area only, and makes the analysis more dynamic by allowing for the response to change after a mother has realized the benefits of the intervention by having (healthier) children exposed to the intervention. Table 7 presents the results when birth history within the treatment area is controlled for, representing an impulse response function of sorts. As seen in column 1, the overall effect, while still statistically insignificant, is now negative. Consistent with the prior analysis, recent births in both the treatment and comparison areas decrease the likelihood of additional births with a 'cascading' effect. In addition, this specification suggests that births in the vaccination area in the last two to five years further depress fertility.

This extension was also done with even more detailed treatment area-birth history indicators, which included variables for each of the 32 possible recent birth histories (for example, births one year ago and four years ago would be the combination 10010) in the treatment area. The results presented in Table 7 were robust to this extension, but

the results were presented with the more limited impulse response application because these treatment indicators were wholly insignificant in the estimation.

Columns 2-5 in table 7 replicate the heterogeneity analyses controlling for recent treatment area birth patterns, and table 8 gives the associated percentage change in odds. For the most part, adding a set of variables to control for recent treatment area birth history does not significantly change the results from those presented in table 5. However, while these variables provide additional insight into the fertility response by controlling for realized fertility changes, there are a few complications when treatment area birth history is controlled for in this way.

In column 2 it is evident that the effect of family size is preserved under this specification. There is still an increased likelihood of birth for those with no living children, and those considered to be in the 'hoarding' stage see a fertility decline.

However, the effect of eligible children, attempted in column 3, is now highly collinear with the new treatment-birth history dummies, and the treatment-birth history dummies control for eligible children in a more specific, time-sensitive way. Thus, based on this set of indicators, in both column 1 and column 3 it is still true that fertility declines among women with vaccine-eligible children.

There no longer appears to be a fertility increase among those without any children eligible for the vaccination. Similarly, there is still no observed effect on the replacement rate (column 4). The son preference effect on the fertility response to the vaccination program is preserved under this more detailed approach.

Thus, the additional insight gained by controlling for realized births in the treatment area is that there is a recent birth effect associated with the intervention itself, and the trends observed earlier in table 5 persist even after explicitly capturing realized exposure due to treatment-area births (as opposed to just potential benefits).

7 Concluding Remarks

The presented results capitalize on a unique data set to present new information on how a randomized intervention, acting as an exogenous change in child mortality risk, impacts fertility. I attempt to frame the impact of mortality risk on fertility in a more specific way, by examining how the change in risk affects fertility as a function of fertility history. Two steps were taken to develop a more layered understanding of how the intervention works: first, the use of sequential fertility to look at the individual fertility decisions; second, the examination of how variations in fertility and child mortality experiences affect the fertility response.

Total fertility, due to its relative bluntness and inability to consider time-varying variables, did not identify a treatment effect. Sequential fertility gave a more detailed analysis, showing that the lack of an overall effect masks several significant fertility effects that 'net out'. The intervention deters fertility among those who directly benefit (i.e., those with children under 5 years of age), those who already have larger-than-average families, and those with several sons. On the other hand, the measles vaccination encouraged fertility among those without children, and those with no vaccine-eligible children. Thus, this intervention operates in both directions, depressing fertility for some even as it increases fertility in others.

These results describe an intervention in Matlab, but can be generalized to other developing areas. As of 2008, 17% of children worldwide were not vaccinated against measles (World Health Organization, 2009). This relevance is particularly strong given the time period in which this research focused- beginning in the early 1980s, before many other interventions were introduced. Even later in the 1980s, with the introduction of several other health interventions, sequential fertility allows me to isolate the effect of the measles vaccination program.

This type of decomposition is important for evaluating a program of this nature, which attempts to effect change across the board; identifying those individuals whose

response is different from the norm allows for better targeting. These results also highlight the importance of using a sequential fertility approach when trying to analyze how fertility decisions react to various shocks; the fertility effect was not identified in the total approach, leading to underestimation of the effects of this intervention. In terms of program evaluation consequences, the results show that current techniques may not be completely capturing the costs and benefits, possibly leading to biased decision-making.

References

[1] Baqui, A. et al., 2001,"Causes of childhood deaths in Bangladesh: an update," *Acta Paediatr*, 90: 682-690.

[2] Benefo, K., and T.P. Schultz, 1996, "Fertility and Child Mortality in Cote d'Ivoire and Ghana," *The World Bank Economic Review*, 10(1): 123-158.

[3] Bertrand, M., Duflo, E., and S. Mullainathan, 2004, 'How much should we trust differences-in-differences estimates?," *Quarterly Journal of Economics*, 119(1):249-275.

[4] Black, S., 1999,"Do Better Schools Matter? Parental Valuation of Elementary Education," *Quarterly Journal of Economics*, 114(2):577-99.

[5] Bloom, D., Canning, D., Gunther, I., and S. Linnemayr, 2008, "Social interactions and fertility in developing countries," Program on the Global Demography of Aging Working Paper.

[6] Ben-Porath, Y., 1976, "Fertility response to child mortality: micro data from Israel," *Journal of Political Economy*, 84(4): 163-78.

[7] Bongaarts, J., and S. Watkins, 1996, "Social interactions and contemporary fertility transitions," *Population and Development Review*, 22(4): 639-682.

[8] Cain, M., 1977, "The Economic Activities of Children in a Village in Bangladesh.*Population and Development Review*, 3(3): 201-227.

[9] Centers for Disease Control and Prevention, "History and Epidemiology of Global Smallpox Eradication," http://www.bt.cdc.gov/agent/smallpox/training/overview/pdf/eradicationhistory.pdf, slide 18.

[10] Cigno, A., 1998, "Fertility decisions when infant survival is endogenous," *Journal of Population Economics*, 11:21-28.

[11] Clemens, J. et al., 1988, "Measles Vaccination and Childhood Mortality in Rural Bangladesh," *American Journal of Epidemiology*, 128:1330-1339.

[12] De Weerdt, J. and S. Dercon, 2008, "Risk-sharing networks and insurance against illness," *Journal of Development Economics*, 81: 337-356.

[13] Emch, M., 1999, "Diarrheal disease risk in Matlab, Bangladesh", *Social Science & Medicine*, 49: 519-530.

[14] Fauveau, Vincent (ed.), 1994, Women, Children and Health, ICDDR ,B Special Publication No. 35, International Centre for Diarrhoeal Disease Research, Bangladesh, Dhaka.

[15] Grether, D., 1980, "Bayes Rule as a Descriptive Model: The Representativeness Heuristic," *Quarterly Journal of Economics*, 95: 537-57.

[16] Imbens, G., and J. Wooldridge, 2007,"Difference in Difference Estimation," Lecture 10 What's New in Econometrics? NBER, Summer 2007.

[17] ICDDR,B, Centre for Health and Population Research, 2004, Health and Demographic Surveillance SystemMatlab, vol. 35, Registration of Health and Demographic Events, 2002, Dhaka, Bangladesh.

[18] Joshi, S., and T.P. Schultz, 2007, "Family Planning as an Investment in Development: Evaluation of a Program's Consequences in Matlab, Bangladesh", Economic Growth Center Discussion Paper.

[19] Kalemli-Ozcan, S., 2003, "A stochastic model of mortality, fertility, and human capital investment," *Journal of Development Economics*, 70(1): 103-118.

[20] Khan, M. and P. Khanum, 2000, "Influence of Son Preference on Contraceptive Use in Bangladesh," *Asia-Pacific Population Journal*, 15(3): 43-56.

[21] Khuda, B., 1991, "Child Labour in Rural Bangladesh: Some Findings from a Relatively Developed Village," *Child Labour in the Indian Subcontinent: Dimensions and Implications*. K. R. New Delhi, Sage Publication: 46-62.

[22] Koenig, M., Khan, M., B. Wojtyniak, 1990, " Impact of measles vaccination on childhood mortality in rural Bangladesh," *Bulletin of World Health Organization*, 68:441-47.

[23] Montgomery, M.R, 1998, "Learning and lags in mortality perceptions," in: M.R. Montgomery and B. Cohen (eds.), *From Death to Birth: Mortality Decline and Reproductive Change*. Washington, D.C.: National Academy Press.

[24] Portner, C., 2008, "Gone With the Wind? Hurricane Risk, Fertility and Education," Working paper.

[25] Rahman, M., and J. DaVanzo, 1993, "Gender Preferences and Birth Spacing in Matlab, Bangladesh," *Demography*, 30:3, 315-332.

[26] Rosenzweig, M., 1982, "Child Mortality and Fertility in Colombia: Individual and Community Effects," *Health Policy and Education*, 2, 305-348.

[27] Sah, R.J., 1991, "The effects of child mortality on fertility choice and parental welfare", *Journal of Political Economy*, 99: 582- 606.

[28] Salahuddin, K., 1981, "Aspects of Child Labour in Bangladesh." In "Disadvantage Children in Bangladesh: Some Reflections." *Women for Women*, Dhaka, Bangladesh.

[29] Joshi, S., and T.P. Schultz, 2007, "Family Planning as an Investment in Development: Evaluation of a Program's Consequences in Matlab, Bangladesh, Economic Growth Center Discussion Paper.

[30] Wolpin, K., 1984, "An Estimable Dynamic Stochastic Model of Fertility and Child Mortality," *The Journal of Political Economy*, 92(5): 852-874.

[31] World Health Organization, 2009, "Measles Fact Sheet," http://www.who.int/mediacentre/factsheets/fs286/en/index.html, March 28 2010.

Table 1: Birth rate by year

Year	Birth rate	Number of women
80	0.207	3733
81	0.210	3840
82	0.193	3926
83	0.203	4047
84	0.180	4129
85	0.203	4196
86	0.180	4218
87	0.175	4202
88	0.190	4244
89	0.167	4229
90	0.172	4212
91	0.146	4147
92	0.146	4106
93	0.133	4087
94	0.124	3990
95	0.160	3917
Total	0.174	65223

Table 2: Summary Statistics

Variable	Mean	Std. Dev.	Min	Max
Gives birth (in a given year)	0.167	–	0	1
Total number of births	5.399	2.912	1	17
Number of mortality shocks	0.948	1.350	0	10
Number of mortality shocks in the last 5 years	0.254	0.652	0	10
Number of mortality shocks in the last year	0.037	0.188	0	1
Mother's age	31.304	9.735	15	50
Mothers age at marriage	15.640	3.631	0	50
Children in family	3.173	2.333	0	13
Mother's education (years)	1.702	2.688	0	14
Muslim	0.893	–	0	1
Mother uses family planning	0.566	–	0	1

Notes: 5,061 mothers, 68,998 time period x mother observations

Table 3: Comparing Blocks A & C with Blocks B & D Pre-intervention (1981)

Variable	**Blocks A & C**	**Blocks B & D**	**Magnitude**
Gave birth in 1981	0.190	0.194	0.004
Number of child deaths in last three years	0.145	0.158	0.013
Age	29.395	28.715	0.680
Number of children	2.981	2.992	0.011
Number of children under 5	0.952	0.990	0.038
Years of education	1.288	1.877	0.589***

Notes: 893 observations in blocks A & C, 1037 observations in blocks B & D.

Table 4: Total fertility model (1984)

Variable	**(1)**	**(2)**
Vaccine available	-0.188	0.003
	(0.569)	(0.702)
Vaccine available & number of child deaths		-0.225
		(0.430)
Number of child deaths	-0.091	-0.061
	(0.160)	(0.179)
Mother's years of education	0.134	0.132
	(0.119)	(0.119)
Mother's age	0.157**	0.156**
	(0.055)	(0.055)
Intercept	-0.929	-0.940
	(1.597)	(1.597)
Area dummies	YES	YES
Log-likelihood	-110.131	-110.006
χ^2	13.296	13.54

Table 5: Sequential fertility model with fixed effects (1980-1995)

Variable	(1)	(2)	(3)	(4)	(5)
Measles vaccination intervention available (MV)	0.031	0.227	0.292*	0.078	0.865***
	(0.133)	(0.142)	(0.158)	(0.136)	(0.177)
MV X No living children		1.173***			
		(0.358)			
MV X Five or more living children		-0.623***			
		(0.135)			
MV X Number of children under 5 years of age			-0.177***		
			(0.059)		
MV X Number of losses in last 5 years				-0.147	
				(0.090)	
MV X Number of sons					-0.423***
					(0.056)
Child death 1 year ago	0.366***	0.376***	0.365***	0.408***	0.274***
	(0.084)	(0.084)	(0.084)	(0.088)	(0.085)
Child death 2 years ago	-0.038	-0.031	-0.043	0.001	-0.119
	(0.085)	(0.085)	(0.085)	(0.088)	(0.086)
Child death 3 years ago	-0.124	-0.116	-0.128	-0.084	-0.177**
	(0.084)	(0.084)	(0.084)	(0.087)	(0.085)
Child death 4 years ago	-0.030	-0.022	-0.028	0.016	-0.068
	(0.081)	(0.081)	(0.081)	(0.086)	(0.082)
Child death 5 years ago	-0.187**	-0.180**	-0.182**	-0.140*	-0.195**
	(0.080)	(0.080)	(0.080)	(0.085)	(0.080)
Birth 1 year ago	-3.095***	-3.113***	-3.043***	-3.097***	-2.985***
	(0.069)	(0.069)	(0.071)	(0.069)	(0.070)
Birth 2 years ago	-1.393***	-1.410***	-1.330***	-1.393***	-1.268***
	(0.054)	(0.055)	(0.058)	(0.054)	(0.056)
Birth 3 years ago	-0.718***	-0.729***	-0.651***	-0.718***	-0.606***
	(0.053)	(0.054)	(0.058)	(0.053)	(0.055)
Birth 4 years ago	-0.550***	-0.557***	-0.481***	-0.550***	-0.465***
	(0.052)	(0.052)	(0.056)	(0.052)	(0.053)
Birth 5 years ago	-0.303***	-0.307***	-0.236***	-0.303***	-0.250***
	(0.044)	(0.045)	(0.050)	(0.044)	(0.045)
No living children	-1.059***	-1.458***	-1.058***	-1.068***	-0.995***
	(0.194)	(0.225)	(0.194)	(0.194)	(0.195)
Five or more living children	-0.672***	-0.468***	-0.666***	-0.671***	-0.545***
	(0.081)	(0.091)	(0.081)	(0.081)	(0.083)
Number of sons					-0.442***
					(0.053)
Mother's age	-0.217***	-0.224***	-0.217***	-0.218***	-0.161***
	(0.011)	(0.011)	(0.011)	(0.011)	(0.013)
Year dummies	YES	YES	YES	YES	YES
Intervention dummies	YES	YES	YES	YES	YES
N	25,620	25,620	25,620	25,620	25,620
Clusters	2,590	2,590	2,590	2,590	2,590
Log-likelihood	-5520.477	-5500.095	-5507.064	-5517.764	-5409.442
$\chi^2_{(34)}$	6612.157	6652.920	6638.980	6617.580	6834.230
LR test		40.77**	26.83**	5.43*	222.07**

Table 6: Sequential fertility model with fixed effects: Percentage change in odds (1980-1995)

Variable	(1)	(2)	(3)	(4)	(5)
Measles vaccination intervention available (MV)	3.1	25.5	33.9*	8.2	137.6***
MV X No living children		223.2***			
MV X Five or more living children		-46.4***			
MV X Number of children under 5 years of age			-16.2***		
MV X Number of losses in last 5 years				-13.7	
MV X Number of sons					-34.5***
Child death 1 year ago	44.3***	45.6***	44.0***	50.4***	31.6***
Child death 2 years ago	-3.8	-3.1	-4.2	0.1	-11.3
Child death 3 years ago	-11.7	-11.0	-12.0	-8.0	-16.2**
Child death 4 years ago	-2.9	-2.2	-2.7	1.6	-6.6
Child death 5 years ago	-17.0**	-16.4**	-16.6**	-13.1*	-17.7**
Birth 1 year ago	-95.5***	-95.6***	-95.2***	-95.5***	-94.9***
Birth 2 years ago	-75.2***	-75.6***	-73.6***	-75.2***	-71.8***
Birth 3 years ago	-51.2***	-51.8***	-47.9***	-51.2***	-45.4***
Birth 4 years ago	-42.3***	-42.7***	-38.2***	-42.3***	-37.2***
Birth 5 years ago	-26.1***	-26.5***	-21.0***	-26.1***	-22.1***
No living children	-65.3***	-76.7***	-65.3***	-65.6***	-63.0***
Five or more living children	-48.9***	-37.4***	-48.6***	-48.9***	-42.***
Number of sons					-35.7***
Mother's age	-19.5***	-20.1***	-19.5***	-19.6***	-14.9***
Year dummies	YES	YES	YES	YES	YES
Intervention dummies	YES	YES	YES	YES	YES

Interpretation: Column 1 indicates that being in the treatment area increases the probability of birth by 3.1%. Column 2 shows that having an additional child eligible for vaccination decreases the probability of giving birth by 16.2%.

Table 7: Sequential fertility model with fixed effects and recent birth-treatment area controls (1980-1995)

Variable	(1)	(2)	(3)	(4)	(5)
Measles vaccination intervention available (MV)	-0.044	0.150	0.016	-0.029	0.766***
	(0.134)	(0.143)	(0.193)	(0.138)	(0.179)
MV X No living children		0.979***			
		(0.360)			
MV X Five or more living children		-0.585***			
		(0.135)			
MV X Number of children under 5 years of age			-0.037		
			(0.086)		
MV X Number of losses in last 5 years				-0.043	
				(0.092)	
MV X Number of sons					-0.406***
					(0.056)
MV X Birth 1 year ago	0.195	0.223	0.232	0.197	0.238*
	(0.138)	(0.139)	(0.162)	(0.138)	(0.141)
MV X Birth 2 years ago	-0.296***	-0.259**	-0.263**	-0.290***	-0.261**
	(0.101)	(0.102)	(0.128)	(0.102)	(0.103)
MV X Birth 3 years ago	-0.480***	-0.435***	-0.451***	-0.473***	-0.429***
	(0.100)	(0.101)	(0.120)	(0.101)	(0.101)
MV X Birth 4 years ago	-0.418***	-0.370***	-0.392***	-0.411***	-0.368***
	(0.100)	(0.101)	(0.117)	(0.101)	(0.101)
MV X Birth 5 years ago	-0.184**	-0.145	-0.158	-0.179*	-0.136
	(0.093)	(0.094)	(0.112)	(0.094)	(0.095)
Child death 1 year ago	0.353***	0.364***	0.354***	0.366***	0.264***
	(0.084)	(0.085)	(0.084)	(0.088)	(0.085)
Child death 2 years ago	-0.056	-0.047	-0.056	-0.045	-0.134
	(0.085)	(0.085)	(0.085)	(0.089)	(0.086)
Child death 3 years ago	-0.136	-0.127	-0.136	-0.125	-0.187**
	(0.084)	(0.084)	(0.084)	(0.088)	(0.085)
Child death 4 years ago	-0.033	-0.026	-0.032	-0.020	-0.070
	(0.082)	(0.082)	(0.082)	(0.086)	(0.082)
Child death 5 years ago	-0.184**	-0.179**	-0.184**	-0.171**	-0.195**
	(0.080)	(0.080)	(0.080)	(0.085)	(0.080)
Birth 1 year ago	-3.143***	-3.166***	-3.142***	-3.144***	-3.044***
	(0.079)	(0.079)	(0.079)	(0.079)	(0.080)
Birth 2 years ago	-1.302***	-1.331***	-1.299***	-1.304***	-1.190***
	(0.063)	(0.063)	(0.063)	(0.063)	(0.064)
Birth 3 years ago	-0.566***	-0.592***	-0.561***	-0.568***	-0.472***
	(0.062)	(0.062)	(0.063)	(0.062)	(0.063)
Birth 4 years ago	-0.426***	-0.448***	-0.420***	-0.428***	-0.358***
	(0.059)	(0.059)	(0.061)	(0.059)	(0.060)
Birth 5 years ago	-0.253***	-0.268***	-0.246***	-0.254***	-0.214***
	(0.051)	(0.051)	(0.053)	(0.051)	(0.051)
No living children	-1.062***	-1.393***	-1.062***	-1.065***	-1.001***
	(0.193)	(0.226)	(0.193)	(0.193)	(0.194)
Five or more living children	-0.662***	-0.472***	-0.662***	-0.662***	-0.538***
	(0.082)	(0.091)	(0.082)	(0.082)	(0.083)
Number of sons					-0.446***
					(0.053)
Mother's age	-0.217***	-0.223***	-0.218***	-0.218***	-0.161***
	(0.011)	(0.011)	(0.011)	(0.011)	(0.013)
Year dummies	YES	YES	YES	YES	YES
Intervention dummies	YES	YES	YES	YES	YES
N	25,620	25,620	25,620	25,620	25,620
Clusters	2,590	2,590	2,590	2,590	2,590
Log-likelihood	-5455.1818	-5453.2624	-5443.9741	-5455.0442	-5342.6389
χ^2	6742.05	6746.59	6765.16	6743.02	6967.83
LR test		3.84†	22.42**	0.28	225.09**

Table 8: Sequential fertility model with fixed effects and recent birth-treatment area controls: Percentage change in odds (1980-1995)

Variable	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**
Measles vaccination intervention available (MV)	-4.3	16.2	1.6	-2.9	115.1***
MV X No living children		166.2***			
MV X Five or more living children		-44.3***			
MV X Number of children under 5 years of age			-3.7		
MV X Number of losses in last 5 years				-4.2	
MV X Number of sons					-33.4***
MV X Birth 1 year ago	21.6	25.0	26.1	21.8	26.9***
MV X Birth 2 years ago	-25.7***	-22.8**	-23.1**	-25.2***	-23.0**
MV X Birth 3 years ago	-38.1***	-35.3***	-36.3***	-37.7***	-34.9***
MV X Birth 4 years ago	-34.2***	-31.0***	-32.4***	-33.7***	-30.8***
MV X Birth 5 years ago	-16.8**	-13.5	-14.6	-16.4***	-12.7
Child death 1 year ago	42.4***	43.9***	42.5***	44.1***	30.3***
Child death 2 years ago	-5.5	-4.6	-5.4	-4.4	-12.5
Child death 3 years ago	-12.7	-12.0	-12.7	-11.7	-17.1**
Child death 4 years ago	-3.2	-2.5	-3.2	-2.0	-6.8
Child death 5 years ago	-16.8**	-16.4**	-16.8**	-15.7**	-17.7**
Birth 1 year ago	-95.7***	-95.8***	-95.7***	-95.7***	-95.2***
Birth 2 years ago	-72.8***	-73.6***	-72.7***	-72.9***	-69.6***
Birth 3 years ago	-43.2***	-44.7***	-42.9***	-43.3***	-37.6***
Birth 4 years ago	-34.7***	-36.1***	-34.3***	-34.8***	-30.1***
Birth 5 years ago	-22.3***	-23.5***	-21.8***	-22.4***	-19.2***
No living children	-65.4***	-75.2***	-65.4***	-65.5***	-63.2 ***
Five or more living children	-48.4***	-37.6***	-48.4***	-48.4***	-41.6***
Number of sons					-36.0***
Mother's age	-19.5***	-20.0***	-19.5***	-19.6***	-14.8***

Interpretation: Column 1 indicates that being in the treatment area increases the probability of birth by 4.3%. Column 2 shows that having an additional child eligible for vaccination increases the probability of giving birth by 3.7%.