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BIRTH SPACING, CHILD SURVIVAL AND FERTILITY DECISIONS: ANALYSIS OF CAUSAL MECHANISMS

By

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Birth Spacing, Child Survival and Fertility Decisions: Analysis of Causal Mechanisms[a]

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Birth Spacing, Child Survival and Fertility Decision: Analysis of Causal Mechanisms

Abstract

We jointly analyze infant mortality, birth spacing, and total fertility of children in a rural area in Bangladesh, using longitudinal data from the Health and Demographic Surveillance System (HDSS) in Matlab. To distinguish causal mechanisms from unobserved heterogeneity and reverse causality, we use dynamic panel data techniques. We compare the results in a treatment area with extensive health services and a comparison area with standard health services. Simulations using the estimated models show how fertility and mortality can be reduced by, for example, breaking the causal link that leads to a short interval after a child has died. Eliminating this effect would reduce fertility and increase birth intervals, resulting in a fall in mortality by 0.14 and 2.45 per 1000 live births in treatment and comparison area, respectively. The effects of the numbers of (surviving) boys and girls on birth spacing provide evidence of son preference: having more boys has a stronger effect on the birth interval than having more girls, though both effects are significantly positive. A simulation suggests that if families would behave as if their all children were sons, fertility levels would be reduced by 3.5% and 5.7% in the ICDDR,B and comparison areas, respectively.

Key words: child mortality, birth spacing, fertility, dynamic panel data models, Bangladesh

JEL codes: I15, J13, C33

1. Introduction

According to the demographic transition theory, there is a strong correlation between childhood mortality and fertility. Understanding the nature of the links between mortality, birth spacing, and family planning is important in order to design effective policies in order to achieve the United Nations Millennium Development Goals 4 and 5 (UNDP 2003) of reducing child mortality and improving access to reproductive health. Empirical evidence has shown that a decline in childhood mortality is often a prerequisite for fertility decline (Chowdhury et al. 1976; Pritchett 1994; Wolpin 1997). Other studies have emphasized the reverse direction of this causation: high fertility and the close birth-spacing associated with it cause an increase in child mortality (Cleland and Sathar 1984; Curtis et al. 1993). Yet another set of studies emphasized that the analysis of the direction of causality is hampered by the close interrelations between child mortality, birth intervals, and fertility (Zimmer 1979; Santow and Bracher 1984).

The observed associations between child mortality, birth spacing, and fertility may not only be due to various causal mechanisms but can also be explained by common unobserved factors that drive the various processes. From the point of view of policies aimed at optimal birth spacing, reducing mortality, and reducing fertility, it is crucial to identify the importance of the various causal mechanisms and alternative explanations. If associations reflect spurious correlation or reverse causation instead of the presumed causal effect, then the implications for policy design can be dramatically altered (Moffitt 2005). Ben-Porath (1976, p. S168) already argued that associations may not reflect causal effects but may be spurious and reflect omitted variables operating simultaneously on fertility and mortality. More recently, DaVanzo et al. (2008) emphasized the importance of joint analysis including interval lengths and mortality, allowing for correlated risks among different births to the same mother.

To achieve this latter goal, we use a panel data model similar to the one introduced by Bhalotra and van Soest (2008). This model incorporates various causal mechanisms as well as several potentially correlated unobserved heterogeneity terms, and exploits the sequence of all births and deaths to a mother for identification. It has equations for mortality (neonatal mortality in Bhalotra and van Soest; infant mortality in our study), for the birth interval, and for the ("fertility") decision to have another birth.

Mortality depends on, among other things, the length of the preceding birth interval (for birth orders higher than one), age of the mother, gender of the child, socio-economic status of the family, religion, and an unobserved mother specific effect. The decision whether to have another child or not and the birth interval after a given birth until the next birth in turn depend on gender and survival status of previously born children, age of the mother, socio-economic status, religion, and unobserved mother-specific effects. The three mother-specific unobserved effects are allowed to be correlated to capture the possibility of common unobserved factors driving the various processes. The model is estimated with maximum likelihood, accounting for all the correlations and for censoring in the birth spacing equation (fertility may be incomplete at the end of the observation window). The estimates therefore remain consistent in spite of the endogeneity of some of the explanatory variables.

While Bhalotra and van Soest (2008) used retrospective data from the Indian Demographic and Health Survey, we use prospective data from the Demographic and Health Surveillance System, Matlab, Bangladesh, following mothers residing in the study area over time. This has the advantage that several covariates, such as indicators of socio-economic status and environmental factors such as availability of drinking water) are observed at the relevant points in time when children are born (rather than at survey time in retrospective data). Moreover, it

avoids recall error in, for example, the dates when children were born. A second specific feature of our data is that the study area is randomly split into villages with standard government provided health services (the "comparison area") and villages with additional extensive health services, such as more health clinics and regular visits of health officers (the "ICDDR,B area" or "treatment area"); see Bhatia (1983) or Van Ginneken et al. (1998). Comparing the model estimates for the two areas gives insight in how the extensive health services affect birth spacing, mortality, fertility, and the various relations between these processes.

2. Background and existing studies

Many studies have found a strong positive relationship between child mortality and subsequent fertility, especially in developing countries. For Bangladesh, Chowdhury et al. (1976) find that infant death shortened median birth interval from 37.2 to 24.1 months. Bhalotra and van Soest (2008) conclude that for every neonatal death in India, 0.37 extra children are born.

According to the classical demographic transition theory, child mortality affects fertility in two ways: physiological/biological changes and behavioral/replacement effects. The physiological effect can be explained by the fact that breastfeeding is interrupted with a child death, and consequently, the postpartum infecundable period is shortened (e.g., Van Ginneken 1974). As a result, under ineffective use or non-use of contraception, the mother is able to conceive the next child sooner, leading to a shorter birth interval and, possibly, higher fertility. The association between the death of a child and birth intervals or fertility decisions has been attributed to two strategies of reproductive behavior: replacement and hoarding (Ben-Porath 1976; Wolpin 1998). Hoarding refers to the fertility response to *expected* mortality of offspring,

while replacement is the response to an *actual* child death. Both are closely related to the total number of surviving children that parents ultimately wish to have.

On the other hand, many studies found an association between a short birth interval and neonatal or infant death of the next child, particularly when the preceding sibling survived (Zenger 1993; Koenig et al. 1990; Alam and David 1998). An explanation for this is that the mother has not recuperated physiologically from the previous birth (DaVanzo and Pebley 1993; Scrimshaw 1996). Hence vulnerable families can be caught in a death-trap that leads to clustering of child deaths within families: the death of a child reduces the interval to the next birth and thus increases in the risk of death of the subsequent sibling in the family (Arulampalam and Bhalotra 2006). An alternative explanation is that a child death leaves the mother depressed. This may affect the mother's behaviour, compromising the health of her subsequent child in the womb and in early infancy (Steer et al. 1992; Rahman et al. 2004).

Sibling competition may explain why short birth intervals and high fertility increase death risk: sources of food and care per head diminish as the number of dependent members of a family increases (Cleland and Sathar 1984). This is expected to induce a negative effect of child death on the mortality risk of the next child, since the next child competes with fewer siblings (Alam and David 1998). A similar negative effect could be induced by learning: If the older sibling died due to, for example, diarrhoea or acute respiratory illness (ARI) – two leading causes of child death explaining almost half of all deaths in Bangladesh (NIPORT et al. 2005) - the mother will want to learn how to prevent a death caused by diarrhoea or ARI.

There is evidence that son preference exists in societies like Bangladesh, with a strong patrilineal family system (Chowdhury et al. 1976). It is therefore likely that a couple wants to have another child soon after the death of a son until the desired number of sons is achieved.

Sufian and Johnson (1989) show that the median birth interval in Bangladesh is shorter when the dead child was a boy or when it was survived by fewer than two brothers. Nyarko et al. (2003) show for Ghana that the probability of having a next birth within a given time period is one third higher if a male child died than if a female child died.

Observed clustering in infant or child mortality of successive children may also be due to unobserved confounding factors instead of causal mechanisms. Older studies of birth spacing and childhood mortality usually do not control for both. More recent studies of Arulampalam and Bhalotra (2006) for India and Omariba et al. (2008) for Kenya reveal that the causal effect of previous mortality is overestimated when unobserved heterogeneity is not accounted for.

3. Data

Since 1966 ICDDR,B maintained a Health and Demographic Surveillance System (HDSS) in Matlab, aiming to support the Bangladesh Health and Family Planning programme. In Matlab, an area located in 60 km southeast of Dhaka, all births, deaths, causes of deaths, pregnancy histories, migrations in and out of the area, marriages, divorces, and several indicators of socioeconomic status are recorded for the complete population of about 220,000 people. The HDSS data on the timing of pregnancy outcomes and deaths are considered to be of very high quality because they are collected during regular visits (every two weeks until the late 1990s and every month since then) by well-trained female community health workers (see, e.g., D'Souza 1981 or van Ginneken et al. 1998). We combined the health and demographic surveillance system data from 70 villages in the ICDDR,B area and 79 villages in the comparison area obtained from 1 July, 1982 until 31 December, 2005 (the study period). Data from before 1 July 1982 have not (yet) been made available for research.

The complete data set has records on about 63,000 mothers, with more than 165,000 child births – including live singleton births, multiple births, and still births. For our purposes, we selected a subsample of mothers without multiple births [d] and with complete [e] live birth information who were continuously living in the Matlab area since the birth of their first child. This implies that we deleted mothers who migrated out of Matlab during the period under study. Moreover, we discarded stillbirths.[f] Finally, we have excluded the children born in three villages that shifted from the ICDDR,B area to the comparison area in 2000. This leads to working samples of 31,968 children and 13,232 mothers in the ICDDR,B area and 32,366 children and 11,856 mothers in the comparison area.

Table 1 presents sample means (percentages of outcome 1 for dummy variables) by area. In the ICDDR,B area, 5.09 percent of all live births resulted in infant death; 10.66 percent of all

[d] We eliminated multiple births as children of a multiple birth face much higher odds of dying. This requires a separate analysis, as has been documented in the demographic literature.

[e] To have a mother's complete birth information during the study period we have calculated parity (total number of live births) from the pregnancy history variables. For example, if a mother has parity four, this means she has had four live births, so she will appear four times as giving birth, with four recorded birth dates. In all other cases (e.g., if a child was born outside of the Matlab area or before study period or deleting multiple births may caused incomplete birth information of a mother), we have deleted all children's records of this mother.

[f] One reason why we eliminated stillbirths is that gender, an important covariate in our analysis, is missing for stillbirths. We define birth intervals as intervals between reported dates of live births, ignoring stillbirths in between live births.

families experienced at least one infant death and 0.79 percent lost all their children in infancy. The percent of infant death among first born is 6.70, substantially higher than among children of higher birth order (3.95 percent). In the comparison area, infant death was more common: 6.82 percent of all children - 8.90 percent among first born and 5.62 percent among higher order births. Of all families, 15.66 per cent experienced at least one infant death and 1.08 per cent lost all their children. About 20.6% birth intervals are shorter than or equal to 24 months in the comparison area, compared to about 12.9 % in the ICDDR,B area.

The average number of children born per mother is 2.42 in the ICDDR,B area and 2.73 in the comparison area; 19 percent of families had more than three children in the ICDDR,B area, compared to 29 percent in the comparison area (not reported in the table). No differences between areas are observed in the mother's average age at birth. In the comparison area, mothers less often have access to the more hygienic source of drinking water (tubewell/filter) and live much farther away from the nearest health facility (7.1 kilometres on average, compared to 1.9 km in the ICDDR,B area).

The non-parametric regressions of infant mortality on the preceding birth interval in Figure 1 show a sharp decline in infant mortality rates when birth intervals increase in both areas. The probability of infant death falls with birth interval length until an interval length of about 4.5 years (exp(4)=54 months). Particularly in the ICDDR,B area, the survival chances stabilize or even increase somewhat when birth intervals increase beyond 4.5 years. This pattern is in line with the extensive literature on this issue; see, e.g., Bhalotra and van Soest (2008, Figure 1).

Figures 2 and 3 show the distributions of the log birth interval by survival status of the previous child and by gender in the two areas. In both areas, there is a large difference between the distributions after infant death and infant survival. This difference is much larger than the

difference in Uttar Pradesh (India) found by Bhalotra and van Soest (2008, Figure 2). In the ICDDR,B area, the median birth intervals are 20 months after an infant death and 48 months otherwise (averages are 23 and 51 months). The medians are 17 and 37 months (averages are 22 and 42 months) in the comparison area. No significant difference by gender is observed.

4. Model Specification

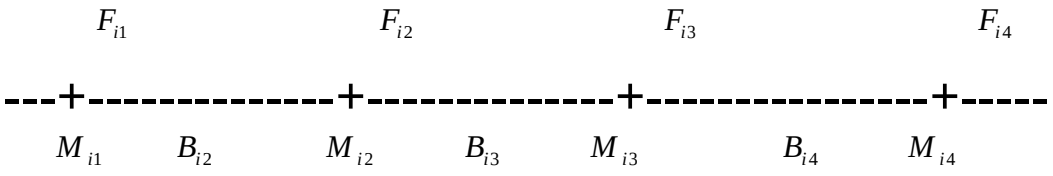
In this section we present the econometric model. This is similar to the model in Bhalotra and van Soest (2008; see also their online appendix for details), though we do not incorporate local community effects. The sensitivity analysis of Bhalotra and van Soest suggests that this has no effect on the point estimates though it may mean that our standard errors are somewhat underestimated. The endogenous variables in the model are the following, with *i* denoting a mother and $t=1,..,T_i$ denoting her consecutive live births:

M_{it} : Infant mortality dummy: 1 if child *t* dies; 0 if it survives the first twelve months after birth.

F_{it} : Decision to have another child (1) or not (0).

B_{it} : Log birth interval preceding birth of child *t* (*t>1* only)

The sequence of events is illustrated in the following time line:



We do not explain the timing of the first birth; it is taken as given. The first event we explain is infant survival of the first born child M_{i1}. The second is the decision to have more children ($F_{i1}=1$) or not ($F_{i1}=0$). This is never observed directly, but if a second birth is observed we know that $F_{i1}=1$. If not, this can be because $F_{i1}=0$ or because the next birth

interval is too long in the sense that it exceeds the observation window or the woman's fertile age (set to 45 years).

If $F_{i1} = 1$ and if the birth interval is not too long, we observe the birth interval B_{i2}. The second born child can die during infancy or survive, etc.: the sequence of events continues until the mother decides not to have more children ($F_{iT} = 0$) or at the end of her fertile period (age 45) or the observation window (December 2005).

The model is recursive in the sense that each dependent variable may depend on outcomes realized earlier in the sequence of events, but not on future outcomes. Moreover, each outcome may depend on unobserved factors common to all children of a given mother, treated as unobserved individual (mother specific) effects. We use probit equations for the binary outcomes (infant mortality of each child; fertility decision after each birth) and a regression equation for the continuous outcomes (log birth intervals). Below we discuss the equations for the various outcomes in detail.

Infant mortality

For higher birth orders, a dynamic probit equation with (random) mother specific effects is used. The explanatory variables include the preceding birth interval and variables related to the mother's age at birth, which is a function of previous birth intervals: For child t ($t=2,\ldots,T_i$) of mother i, the equation is

$$M_{it}^{*} = \mathbf{X_{it}}\beta_m + Z_{it}\gamma_m + \alpha_{mi} + u_{mit} \qquad (1)$$

$$M_{it}=1 \text{ if } M_{it}^{*}>0 \text{ and } M_{it}=0 \text{ if } M_{it}^{*}\leqslant 0$$

Here X_{it} contains (functions of) the strictly exogenous variables, such as gender of the child, socio-economic status indicators of the household (mother's and father's education, etc.) and characteristics of the village where the household resides. Z_{it} is the vector of explanatory variables that are functions of previous outcomes (and are therefore not strictly exogenous), including the preceding log birth interval B_{it}, (functions of) age of the mother at birth t and, following the literature on scarring (see, for example, Arulampalam and Bhalotra 2006), survival status of the previous child M_{it-1}. The mother specific unobserved heterogeneity term α_{mi} captures unobservable time invariant characteristics influencing the infant mortality risk of all children in the family. The error term u_{mit} captures idiosyncratic health shocks specific to child t. We assume that the u_{mit} follow a standard normal distribution, independent of each other and of all covariates, and that α_{mi} is normally distributed with mean 0 and variance $\sigma^2_{\alpha m}$ independent of all u_{mit} and X_{it} (but not of Z_{it}).

For mortality of the first child, a separate equation is needed, since there is no preceding birth interval or preceding mortality outcome. Age at first birth is assumed to be exogenous and included in X_{i1}. The equation for the first child's infant mortality is then given by:

$$M_{i1}^{*} = X_{i1}\beta^{1} + \theta\alpha_{mi} + u_{mi1} \qquad (2)$$

$$M_{i1}=1 \text{ if } M_{i1}^{*}>0 \text{ and } M_{i1}=0 \text{ if } M_{i1}^{*}\leqslant 0$$

Here β^1 and θ are (auxiliary) parameters to be estimated and the error term u_{mi1} is assumed to satisfy the same assumptions as the other u_{mit}.

Birth-spacing

For a mother who has given births to T_i children, we observe the exact log durations in between two consecutive births $b_{2i},\ldots,b_{T_i i}$ preceding births $2,.....,T_i$. We model these intervals using the following equation:

$$b_{it} = X_{it}\beta_b + Z_{it}^b\gamma_b + \alpha_{bi} + u_{bit} \qquad (3)$$

Here X_{it} denotes the vector of strictly explanatory variables, as before.[g] Z_{it}^b includes survival status of the preceding sibling and family composition variables (functions of the numbers of surviving girls and boys). The unobserved heterogeneity term α_{bi} captures unobserved time invariant characteristics of the mother (or her household or village) influencing the birth interval. The error term u_{bit} captures idiosyncratic errors. We assume that the u_{bit} follow a normal distribution, independent of each other and of all covariates, and that α_{bi} is normally distributed independent of all u_{bit} and X_{it} (but not of Z_{it}^b).

Fertility decisions and right censoring

There is right-censoring in the data since some mothers will not have completed their fertility at the time of the survey. After the end of the observation window (ultimo 2005), some mothers will still have another birth, and others will not. In principle, this could be captured by the model as it is described until now, with a birth interval after the last observed birth that lasts longer than until end 2005. Following Bhalotra and van Soest (2008), however, the model fit can be improved substantially by adding a separate equation reflecting the possible decisions to stop

[g] Another determinant of birth spacing would be the use of contraceptives. We do not include this in X_{it} since it is not observed in the comparison area and may be endogenous due to correlation with unobservables in the model.

having children after each birth. This improves the fit since it can explain why some mothers who are still of reproductive age have no more births long before the end of the observation window. (We assume that women older than 45 years are no longer of reproductive age - an age beyond which very few births are observed in our data.) Without the additional equation, this would have to be explained by a very long birth interval.

The equation determining whether the woman continues to have children after birth t ($F_{it}=1$) or not ($F_{it}=0$) is specified as follows:

$$F_{it}^{*} = X_{it}\beta_f + Z_{it}^{f}\gamma_f + \alpha_{fi} + u_{fit} \qquad (4)$$

$$F_{it} = 1 \text{ if } F_{it}^{*} > 0 \text{ and } F_{it} = 0 \text{ if } F_{it}^{*} \leq 0$$

As before, X_{it} denotes the vector of strictly exogenous explanatory variables. The vector Z_{it}^{f} includes survival status of the preceding sibling and family composition variables (based upon the number of surviving girls and boys). The mother specific unobserved heterogeneity term α_{fi} captures unobservable time invariant characteristics influencing the fertility decision after each child birth and the term u_{fit} captures idiosyncratic errors. We assume that the errors u_{fit} are standard normally distributed, independent of each other and of the X_{it}. The mother specific unobserved heterogeneity terms α_{fi} are normally distributed with mean 0 and variance $\sigma_{\alpha f}^{2}$, independent of all u_{fit} and X_{it}.

The outcome F_{it} is observed only partially. If birth t is not the last birth ($t<T_i$) then we know that the mother has decided not to stop having children, so that $F_{it} = 1$. But if $t=T_i$, she may have decided to stop having children ($F_{it} = 0$), but it may also be the case that the next birth

interval extends beyond reproductive age or the end of the observation window (F_{it} = 1 and right censoring).

Confounding unobserved factors are controlled for by allowing arbitrary correlations amongst $\alpha_{fi,}$ α_{mi}, and α_{bi}. We will assume they are drawn from a three-dimensional normal distribution with zero mean and an arbitrary covariance matrix, independent of the X_{it} and of the idiosyncratic error terms $u_{fit,}$ u_{mit}, and u_{bit}.

Estimation

The equations of this model (equations (1)-(4)) are estimated jointly using simulated maximum likelihood, similarly as in Bhalotra and van Soest (2008); see also their online appendix for details. Conditional on the random mother specific effects, the likelihood contribution of a given mother can be written as a product of univariate normal probabilities and densities over all births following the order of observed events (see the time line) and accounting for the right censoring. The actual likelihood contribution is the expected value of the conditional likelihood contribution, with the expectation taken over $\alpha_{fi,}$ α_{mi}, and α_{bi}. This three-dimensional integral is approximated using (smooth) simulated ML: Independent standard normal errors are drawn, and transformed into draws of the random effects using the parameters of the random effects distribution; the conditional likelihood contribution is then computed for each set of draws and the mean across R independent draws is taken. If $R \to \infty$ with the number of mothers N, this gives a consistent estimator; if draws are independent across households and $R \to \infty$ faster than $\sqrt{N}$, the estimator is asymptotically equivalent to exact ML (see, for example, Hajivassiliou and Ruud 1994). To reduce the sampling variance in the simulations, we used Halton draws (see Train 2003). The results we present are based on $R=100$; for larger R, we got very similar results. We have

checked the sensitivity of our parameter estimates for the number of the draws (comparing with different values of *R*) and the nature of the draws (using Halton draws with different seeds) and always got very similar results.

5. Estimation results

Mortality equation

The estimates of the mortality equation are given in Table 2.[h] Figure 4 helps to interpret the parameters on lagged mortality, log birth interval and its square, and the interaction of lagged mortality with the log birth interval. It presents, for both areas, the estimated mortality risk as a function of the birth interval separately for when the previous child died and did not die during infancy, with other covariates set to their means. In the ICDDR,B area, the interaction term and lagged mortality are both significant. The significantly positive interaction term is in contrast with Bhalotra and van Soest (2008), but consistent with other studies (Conde-Agudelo et al. 2006; Whitworth and Stephenson 2002). For a given length of the birth interval, the "state dependence" effect of lagged mortality depends on the magnitude of the interval. For short birth intervals, the mortality risk is larger if the previous sibling survived than if it died (negative state dependence), but for long birth intervals the difference changes sign (positive state dependence). This result is consistent with sibling competition for scarce family resources which are particularly needed when children are still very young. For long birth intervals, sibling competition plays is less important while other mechanisms such as depression due to the previous infant's death may still matter.

[h] Results of the equation for mortality of the first child are available upon request of authors; they are very similar to those reported in Saha and van Soest (2011).

In the ICDDR,B area, for the case where the previous sibling did not die, the mortality risk falls with the birth interval until about the mean interval length and then remains constant. Surprisingly, a quite different pattern is found when the previous sibling died – in this case the mortality risk seems to increase with the birth interval length. Perhaps this is due to the relatively small number of observations and the rather small mortality risk in this case.

In the comparison area, the difference between the two curves is smaller and insignificant (see Figure 4). The mortality risk is consistently larger if the previous sibling survived than if it died (keeping the birth interval and other covariates and unobserved mother specific factors constant), consistent with a learning effect. Both mortality risks in the comparison area are essentially falling with birth interval length, flattening out only after more than 50 months, much beyond the median birth interval length.

The estimated coefficients on the other covariates are in line with those in Saha and van Soest (2011). The mother's age at birth has a significantly U-shaped effect in the ICDDR,B area with a minimum at about 30 years, whereas it is insignificant in the comparison area. Mortality risk is U-shaped in birth order, but this is significant in the comparison area only. The gender of the child is insignificant in both areas, implying that there is no evidence of an effect of son preference on infant mortality.

Mother's schooling is insignificant once the father's schooling is controlled for (it is significant for the mortality risk of the first born child). On the other hand, secondary schooling of the father significantly reduces infant mortality of higher birth orders in both areas. The dummy indicating whether the father is a day labourer, an index of lower occupational and socio-economic status, has a significant positive effect on mortality in both areas. The distance to the nearest health facility has a significant positive effect on infant mortality in the comparison area

for higher order births, and the effect is even stronger for the first born child. The fact that distance plays no significant role in the ICDDR,B area is probably due to the fact that almost all families live rather close to a health facility in that area (see Saha and van Soest 2011).

Those who used tube well or pipe water as a source of drinking water are less likely to see their children die in infancy, but this is significant in the ICDDR,B area only. Over the various birth cohorts (the reference mother is born before 1966), mortality decreases sharply in the comparison area, while in the ICDDR,B area, the decreasing trend seems to level off for the younger cohorts.

Birth interval equation

Table 3 reports the estimates of the birth spacing equation. Since the dependent variable is log birth interval, parameters must be interpreted in terms of percentage changes in the expected length of the birth interval. Death at infancy of the previous child shortens the subsequent birth interval by about 49% (exp(-0.6741)-1) in the ICDDR,B area and 46% in the comparison area, consistent with the replacement hypothesis and existing findings (e.g. Chowdhury et al. 1976; Bhalotra and van Soest 2008). The size of the effect is much larger than in Bhalotra and van Soest. The effects of the surviving boys and girls variables are consistent with son preference: In both areas, having at least one boy has a stronger positive effect on the birth interval than having a girl. The same applies to each additional boy. For example, in the ICDDR,B area, the ceteris paribus difference between the next birth interval of families with one boy and families with one girl is 6.5% (exp(0.1726-0.1099)). Comparing families with two boys and one girl and with one boy and two girls, it is 6.7% (exp(0.0978-0.0325)).

Birth intervals shorten with birth order (as in, for example, Miller et al. 1992). They are longer for the younger birth cohorts of mothers, which may explain part of the reduction in fertility over time. In the ICDDR,B area, birth-spacing is hump-shaped in maternal age at previous birth with a maximum at about 35 years. In the comparison area, birth interval length essentially increases with the mother's age at the previous birth over the whole reproductive age range. This is in line with the negative effect of maternal age on the hazard rate of a new conception found by Rahman and DaVanzo (1993, Table 2). Birth intervals increase with the mother's education level, in line with the positive relation between birth intervals and socioeconomic status. Mothers in more developed villages with drinking water from a tube well or pipe water also tend to have longer birth intervals.

Fertility equation

Table 4 presents the estimates of Equation (4), determining the probability of having another child after each birth. In both areas, the most important variables in this equation concern family composition. Having at least one son or at least one daughter substantially and significantly reduces the probability to have further children, and the size of the effect is much larger in the comparison area than in the ICDDR,B area. There is no son preference in this respect. On the other hand, if we consider the number of sons and daughters given there is at least one of each, we do find evidence of son preference: Additional sons substantially reduces the desire to have more children, but additional girls have a much smaller effect (significant in the comparison area but insignificant and of the wrong sign in the ICDDR,B area).

Fertility falls with the level of education of both parents, with a larger effect of mother's education. In both areas, Muslim families show a higher tendency to continue fertility than non-

Muslims. The desire for continued fertility falls with birth order in both areas and surprisingly, younger mothers are less likely to continue fertility than older mothers (keeping family composition and other variables constant). There are strong cohort differences in the comparison area where the younger cohorts less often want more children, but not in the ICDDR,B area. Mothers in villages with access to tube well or pipe water as a source of drinking water are less likely to continue their fertility. In the comparison area, families living farther away from a health centre have a larger probability to have another child. These results are in line with a negative relation between socio-economic status and fertility. This is not the case for the father's occupational status: in both areas day labourers have smaller chances to have more children.

Unobserved heterogeneity

The estimates of the covariance matrix of the three unobserved heterogeneity terms are given in Table 5. The heterogeneity terms in all three equations are statistically significant but smaller than the idiosyncratic errors. Mother specific effects in the mortality equation explain about 23% (0.3014/(1+0.3014)) in the ICDDR,B area and about 6% (0.0625/(1+0.0625)) in the comparison area of the total unsystematic variation in infant mortality. For the birth spacing equation the idiosyncratic noise terms have estimated standard deviation 0.442 in the ICDDR,B area and 0.436 in the comparison area, and the unobserved heterogeneity terms explain 8.1% of the unsystematic variation in birth intervals in the ICDDR,B area and only 3.6% in the comparison area. The small correlations between unobserved heterogeneity in the mortality and birth interval equations suggest that hoarding does not play much of a role – hoarding would predict that women who know their children have high mortality risk tend to have shorter birth intervals, in

order to attain their desired family size even if some children die; to the extent this is not captured by observed covariates, this would imply a negative correlation between α_{mi}, and α_{bi}.

The heterogeneity terms in the fertility equation explain about 70% (comparison area) and 44% (ICDDR,B area) of total unsystematic variation. In both areas, a large negative correlation is observed between unobserved heterogeneity in birth interval and fertility equations, suggesting that mothers who desire many children also tend to use shorter birth intervals. This is consistent with the target fertility model of Wolpin (1997) and in line with the finding of Bhalotra and van Soest (2008). The correlation between the individual effects in the mortality equation and the fertility equation is positive but not significant in the ICDDR,B area but significantly negative in the comparison area. We do not have a good explanation for this.

6. Simulations

To illustrate the importance of the various causal mechanisms between birth spacing, fertility, and infant mortality, we performed some simulations, in a similar way as Bhalotra and van Soest (2008, Table 3). They illustrate the main feature of our joint model: the fact that it incorporates various mechanisms that lead to associations between planning, birth spacing, fertility, and mortality outcomes, accounting for the effects of endogeneity in the timing of births (and therefore also age at birth etc.), birth intervals, and mortality risks.

The simulations start from the observed covariates (including, for example, date of first birth) for the actual sample of mothers. For each mother, we generated unobserved heterogeneity terms, error terms, and new outcomes (the dependent variables in our model) using the estimated parameters of each equation. The outcomes were generated recursively, using the timing of the

events as sketched in Section 4. For example, for a given mother, we take the date of first birth as given and first generate the mortality outcome of the first child (using equation (2)). Given simulated mortality, we then generated the fertility decision after the first birth (equation (4)). If the fertility decision is positive, we then generate a birth interval, and update calendar time and age of the mother at her second birth. Given these variables, other covariates, and the previous mortality outcome, we then generate the mortality outcome of the second born child, etc. In this way we generate complete birth spacing, mortality, and fertility patterns for all mothers in the sample. To reduce simulation variance, this is repeated 25 times for each mother.

Table 6 shows the results of several simulations. Column 1 summarizes the outcomes according to the benchmark simulation where all mechanisms that are incorporated in the model are active. As expected (unless the model would fit the data quite poorly), this column reproduces several features of the raw data, such as the differentials in infant mortality rates and median birth intervals between the two areas.

The other columns present percent deviations from the benchmark for scenarios in which some behavioural or non-behavioural mechanisms are "switched off." Column 2 switches off the replacement effects of infant mortality on both birth intervals and the probability of having another child. The estimates imply that families respond to infant mortality by shortening the next birth interval and increasing the number of births, and this is incorporated in the benchmark simulation in column 1. The simulation in column 2 produces the counterfactual outcomes that would arise if families would space their births and plan the number of births as if every child survived its infancy. The results show that this increases median birth interval length by 5.9% and 6.3% in the two areas, consistent with other studies (see for example Chowdhury et al. 1976,

pp. 259; Bhalotra and van Soest 2008, p.286). In other words, the replacement effect on the birth intervals reduces birth interval lengths by 5.9% and 6.3% in the two areas.

In the comparison area, the total replacement effect as a result of the infant mortality rate of 68.5 per 1000 live births is an increase in the number of births by 3.72%, that is, 0.54 births for every infant that died (37.2/68.5). In the ICDDR,B area, the replacement effect is an increase of the total number of births by 2.20%, or 0.42 births for every infant that died. The larger effect in the comparison area is mainly due to the larger response of fertility decisions to the family composition variables in that area (Table 4). Because of the longer birth intervals and the reduction in fertility, eliminating the replacement effects also has an indirect effect on infant mortality: it falls by 0.27% (0.14 per 1000 live births) in the ICDDR,B and by 3.6% (2.45 per 1000 live births) in the comparison area. In other words, replacement effects are responsible for a very small fraction of all infant deaths only, particularly in the ICDDR,B area.

Column 3 shows what happens if the direct effect of mortality of the previous child on survival chances is eliminated. (It does not eliminate replacement effects.) Since this direct effect was negative in both areas (Table 2), eliminating it increases infant mortality: by 4.85% (2.51 infant deaths per 1000 live births) in the ICDDR,B area and by 1.56% (1.07 per 1000 live births) in the comparison area. The difference between the two areas is in line with the larger state dependence effect in the ICDDR,B area. As discussed in Section 5, learning effects or sibling competition can explain this negative state dependence mechanism: Eliminating such a learning effect and eliminating the benefits of reduced sibling competition increases infant mortality among children whose previous sibling died. Because of replacement behaviour, the larger infant mortality rates indirectly also shorten birth intervals and increase total fertility, but Table 6 shows that these indirect effects are quite small.

Hoarding implies that families anticipate a large risk of child mortality by adjusting birth spacing and family planning behaviour. In our model this leads to a correlation between unobserved heterogeneity terms in the mortality equation on the one hand and the birth spacing and fertility equations on the other hand. In the simulation presented in column 4, we eliminate these correlations, taking out the part of mother specific unobserved heterogeneity in birth intervals and fertility decisions that is correlated with the unobserved heterogeneity term in the mortality equation (so that the variance of the unobserved heterogeneity terms α_{fi}, and α_{bi} is also reduced). Since this does not change the *average* values of α_{mi}, and α_{bi}, the direct effects on birth intervals and total fertility are very small. In the ICDDR,B area, the estimated correlation between α_{fi}, and α_{mi} was positive, implying that mothers with high risk births tend to have higher fertility, in line with the theory of hoarding. Eliminating this correlation therefore reduces the number of high risk births (and increases the number of low risk births), so that infant mortality falls. Column 4 shows that the estimated reduction is 2.26%, or 0.18 infant deaths per 1000 live births. In the comparison area, the estimated covariance structure is very different with a negative correlation between α_{mi} and α_{fi} that is not in line with hoarding and the effect on mortality has the opposite sign. The increased infant mortality rate also leads to a modest increase in total fertility, due to replacement (cf. column 2).

The final simulation (column 5) illustrates the importance of son preference in family planning. We suppress son preference by simulating counterfactual birth spacing and fertility decisions assuming that families behave as if all their children were boys. This would lengthen the median birth interval by 3.9% in the ICDDR,B area and by 3.1% in the comparison area, and it would reduce total fertility by 3.4% in the ICDDR,B area and by 5.7% in the comparison area.

Although these behavioural changes would reduce the infant mortality rates for higher order births, the ultimate effect on the infant mortality rate is positive. This is due to a composition effect: since the number of higher order births is reduced, the weight of relatively risky first births in the total infant mortality rate is increased. Our results are in the line of son preference of earlier work by Chowdhury and Bairagi (1990) who estimated that in the absence of sex preference fertility will fall by 8% in the ICDDR,B area and by 4% in the comparison area.

7. Discussion

We analyzed birth spacing, infant mortality, and family planning, distinguishing causal mechanisms from unobserved heterogeneity and reverse causality by using dynamic panel data techniques, building on recent work by Bhalotra and van Soest (2008). We used prospective data covering two rural areas in Matlab, Bangladesh: a treatment area with extensive health services and a comparison area with the standard health services provided by the government.

The main goal was to explore the causal mechanisms between infant deaths and total fertility, and how birth spacing shapes this relationship. We compared the pattern of this relationship in two areas and found several significant different differences, suggesting that one model for both areas would be too restrictive.[i] The extensive maternal and health interventions in the ICDDR,B area help to explain these differences (see Hale et al., 2009). We also tried using

[i] We could also combine the two areas and allow for interactions where necessary (according to tests). In Saha & van Soest (2011) we did this but found hardly any efficiency gain. Since the interactions also make interpretation less easy, we did not pursue this here.

dummies for whether specific interventions were introduced at the time of birth, but these were not significant.

Controlling for birth spacing, unobserved heterogeneity, and a large set of socio-economic and cultural covariates, we found negative state dependence in both areas and this relationship is significant in the treatment area. This finding is unique among studies of infant mortality. For example Alam and David (1998) found higher risks in sibling's death in Matlab if the previous sibling died at the same age (either the neonatal or the post-neonatal period). DaVanzo et al. (2008) found positive state dependence in the neonatal as well as the post-neonatal period. In India, Arulampalam and Bhalotra found that infant death of the previous sibling increases the likelihood of infant death by between 2.2 and 9.2 percent points. Similarly, Omariba et al. (2008) found a positive scarring effect of 4.8 percent points for Kenya. These studies do not control for birth intervals. In Saha and van Soest (2011, Table 5), we also found negative state dependence when keeping preceding birth intervals constant, but the negative effect is about two to three times larger in the current study, which emphasizes the importance of allowing for the endogeneity of birth-spacing in the model.

Even though they have shorter birth intervals and higher fertility, Muslims exhibit lower mortality in both the ICDDR,B and the comparison area, similar to what was found for India (Bhalotra et al. 2010). Cultural beliefs and practices might be a leading cause of the higher mortality risks among Hindus. For example, around the time of giving birth, Hindu women in rural Bangladesh often reside in poorly constructed (mainly thatches) houses, and are not given warm clothes for baby and mother (personal observation).

We find evidence of causal effects in two directions: a short preceding birth interval reduces survival chances of infants, and an infant death increases the probability of a next birth

and shortens the time until the next birth (replacement behaviour). We estimate that, as a result of replacement, 0.54 children are born for each infant death (and 0.51 births survive the first 12 months) in the comparison area and 0.42 children in the ICDDR,B area.

We find that the birth intervals minimizing the mortality risk are about 50 months in ICDDR,B area and 60 months in the comparison area for the majority of cases where the previous child did not die during infancy. In both areas, higher mortality risks are observed after long birth intervals after an infant death, suggesting that after an infant death and a long interval the mother may behave as the mother who gives birth to her first child (see Conde-Agudelo et al. 2006).

Estimates of fertility behaviour are consistent with son-preference: having more surviving boys significantly reduces the probability of having a next child and this effect is strongest in the comparison area. The latter is somewhat surprising since according to literature, son preference in fertility is associated with better access to contraception and higher levels of contraceptive use (see Chowdhury and Bairagi 1990; Rahman and DaVanzo 1993). On the other hand it has also been suggested that those who already have more daughters may terminate childbearing earlier because of the concern that the next birth, if female, will worsen the existing sex composition (Chowdhury and Bairagi 1990).

Those who used tube well or pipe water as a source of drinking water are less likely to see their children die in infancy, and this in turn decreases fertility and increases birth spacing. This finding is unique in this study and guides policies to enhance safe drinking water. We find evidence that mortality risks change with reproductive behaviour and by socio-economic indicators, which has implications for the advice that should given about pregnancy spacing. Indeed, this advice is more important for women with low socio-economic status.

Concerning policies targeted at achieving the fourth millennium development goal to reduce under-five mortality, our findings highlight the important role of extensive maternal and child health interventions: comprehensive health infrastructure, providing extensive health services and health information in the ICDDR,B area, strengthens learning effects that can reduce mortality risk.

Table 1. Descriptive statistics, Matlab, 1982-2005.

Variables	ICDDR,B area	Comparison area
Infant deaths (all live-births) (%)	5.09	6.82
Infant deaths excluding first-borns (%)	3.95	5.62
Infant deaths among first borns (%)	6.70	8.90
Families with no infant deaths (%)	89.34	84.34
Families in which all births die in infancy (%)	0.79	1.08
Preceding birth interval in months (%)		
<=24 months	12.93	20.65
25-36 months	19.92	32.73
>=37 months	67.14	46.63
Age of mother at first birth*	21.16 (3.23)	21.08 (3.21)
Age of mother at birth*	24.70 (5.03)	24.58 (4.85)
Mother's education level (%):		
No education	48.48	50.50
Some primary education	24.86	25.51
At least some secondary education	26.66	23.99
Mother Muslim (%)	82.71	89.85
Child male (%)	50.97	51.12
Birth order (%)		
1	41.39	36.63
2	28.93	26.74
3	17.62	18.26
4+	12.06	18.36
Father's education level (%):		
No education	55.67	56.28
Some primary education	22.65	24.15
At least some secondary education	21.68	19.57
Father day labourer (%)	19.61	20.96
Drinking water tubewell/piped water (%)	87.76	76.91
Distance to health facility (km) *	1.87 (0.98)	7.07(4.04)
Number of mothers in sample	13,232	11,856
Number of children in sample	31,968	32,366

*: Means and standard deviations (in parentheses).

Table 2. Estimation Results Mortality Equation, Birth Order > 1 (Equation (1)).

Covariates	ICDDR,B area		Comparison area	
	estimates	s.e	estimates	s.e
Previous sibling died	-1.9904**	0.4637	-0.2703	0.3712
Preceding birth interval (log)	-2.7871**	0.4772	-1.7239**	0.4191
Preceding birth interval square (log)	0.3565**	0.0644	0.2094**	0.0571
Log birth interval_lagged mortality	0.5471**	0.1384	0.0648	0.1157
Male	0.0352	0.0399	0.0111	0.0309
Muslim	-0.0275	0.0604	-0.0503	0.0516
Birth order	0.0494	0.1091	-0.1512*	0.0583
Birth order square	-0.01327	0.0152	0.0199*	0.0069
Mother's birth cohort:				
1966-1970	-0.0213	0.0548	-0.1516**	0.0400
1971-1975	-0.1513*	0.0674	-0.3055**	0.0486
1976+	-0.1878*	0.0807	-0.5461**	0.0619
Mother's age at birth	-0.1260**	0.0371	-0.0321	0.0333
Mother's age at birth square	0.0020**	0.0006	0.0004	0.0006
Mother's education some primary	-0.0616	0.0537	0.0096	0.0400
Mother's education at least some secondary	-0.2305**	0.0697	0.0896	0.0543
Father's education some primary	0.0604	0.0506	0.0286	0.0393
Father's education at least some secondary	-0.2305**	0.0684	0.1312*	0.0500
Father's occupation is day labourer	0.1271*	0.0636	0.1239*	0.0452
Source of drinking water: tubewell /piped	-0.1767*	0.0633	0.0194	0.0395
Distance to health facility (km)	-0.0002	0.0227	0.0064	0.0039
Constant	5.4656**	0.9774	2.8594**	0.8554

* 2<t-value<3; ** t-value>3

Notes: Reference categories of categorical variables used in the model: female, non-Muslim, no schooling years, no access to piped water, not day labourer, mother born before 1966.

Table 3. Estimation Results Log Birth Interval Equation, Birth Order > 1 (Equation (3)).

Covariates	ICDDR,B area		Comparison area	
	estimates	**s.e**	**estimates**	**s.e**
Previous sibling died	-0.6741**	0.0178	-0.6107**	0.0147
First boy surviving	0.1726**	0.0203	0.1226**	0.0160
First girl surviving	0.1099**	0.0198	0.0723**	0.0161
After first boy, number of boys surviving	0.0978**	0.0191	0.0764**	0.0143
After first girl, number of girls surviving	0.0325	0.0186	0.0197	0.0136
Male	-0.0104	0.0103	-0.0306	0.0092
Muslim	-0.0145	0.0105	0.0090	0.0111
Birth order	0.1136**	0.0219	0.0746**	0.0160
Birth order square	-0.0228**	0.0026	-0.0136**	0.0018
Mother's birth cohort:				
1966-1970	0.0659**	0.0098	0.0461**	0.0090
1971-1975	0.1556**	0.0116	0.1072**	0.0109
1976+	0.2320**	0.0130	0.1554**	0.0131
Mother's age at birth	0.0262**	0.0065	0.0207*	0.0082
Mother's age at birth square	-0.0004*	0.0001	-0.0002	0.0002
Mother's education some primary	0.0372**	0.0091	0.0565**	0.0083
Mother's education at least some secondary	0.0035**	0.0107	0.1247**	0.0101
Father's education some primary	-0.0054	0.0088	-0.0171**	0.0081
Father's education at least some secondary	0.0372	0.0098	0.0066	0.0089
Father's occupation is day labourer	-0.0046	0.0121	-0.0440**	0.0104
Source of drinking water: tubewell /piped	0.0414**	0.0101	0.0243**	0.0080
Distance to health facility (km)	0.0042	0.0037	-0.0009	0.0008
Constant	3.0807**	0.0801	3.0370**	0.0982
Sigma error in birth interval equation	0.4422**	0.0029	0.4356**	0.0027

* 2<t-value<3; ** t-value>3;

Notes: Reference categories of categorical variables used in the model: female, non-Muslim, no schooling years, no access to piped water, not day labourer, mother born before 1966.

Table 4. Estimation Results Decision to Have Next Child (Equation (4)).

Covariates	ICDDR,B area		Comparison area	
	estimates	s.e	estimates	s.e
Previous sibling died	-0.15572	0.1004	-0.2092*	0.0991
First boy surviving	-0.5969**	0.1568	-1.2778**	0.1699
First girl surviving	-0.5211**	0.1537	-1.2930**	0.1641
After first boy, number of boys surviving	-0.3367*	0.1443	-1.1801**	0.1503
After first girl, number of girls surviving	0.0307	0.1403	-0.6347**	0.1104
Male	-0.0462	0.0462	-0.0197	0.0485
Muslim	0.6076**	0.0787	0.3869**	0.1001
Birth order	-0.3857*	0.1640	0.3148**	0.1015
Birth order square	0.0100	0.0089	-0.0173*	0.0069
Mother's birth cohort:				
1966-1970	0.0418	0.0473	-0.1730*	0.0672
1971-1975	0.1051	0.0720	-0.5095**	0.0991
1976+	1.2814	0.6862	-0.9052**	0.1573
Mother's age at birth	0.0008	0.0333	-0.0613	-0.0613
Mother's age at birth square	-0.0026	0.0007	-0.0028**	-0.0028
Mother's education some primary	0.0331	0.0539	-0.1940*	0.0711
Mother's education at least some secondary	0.3843**	0.0790	-0.5045**	0.1017
Father's education some primary	0.0229	0.0522	0.1156	0.0664
Father's education at least some secondary	-0.1248*	0.0603	-0.0957	0.0770
Father's occupation is day labourer	-0.5451**	0.0790	-0.4155**	0.0862
Source of drinking water: tubewell /piped	-0.1205*	0.0554	-0.1453*	0.0606
Distance to health facility (km)	-0.0216	0.0181	0.0245**	0.0068
Constant	4.4781**	0.5415	6.9565**	0.9225

*2< t-value<3; ** t-value>3;

Notes: Reference categories of categorical variables used in the model: female, non-Muslim, no schooling years, no access to piped water, not day labourer, mother born before 1966.

Table 5: Mother specific unobserved heterogeneity.

	Mortality	Birth interval	Fertility
ICDDR,B area			
Covariance matrix			
Mortality	0.301**		
Birth interval	-0.012	0.017**	
Fertility	0.189	-0.099**	0.793**
Correlation matrix			
Mortality	1		
Birth interval	-0.167	1	
Fertility	0.386	-0.856**	1
Comparison area			
Covariance matrix			
Mortality	0.063**		
Birth interval	-0.0002	0.007**	
Fertility	-0.188**	-0.088**	2.306**
Correlation matrix			
Mortality	1		
Birth interval	-0.012	1	
Fertility	-0.495**	-0.698**	1

** t-value>3

Table 6. Simulations.

ICDDR,B area	**1**	**2**	**3**	**4**	**5**
Infant mortality	51.8/1000	-0.27	4.85	-2.26	1.63
Birth interval (months) a	43.12	5.87	-0.20	0.70	3.87
Number of births (fertility)	2.43	-2.20	0.01	-0.20	-3.32
Number of survivors	2.31	-2.18	-0.26	-0.08	-3.40
Comparison area					
Infant mortality	68.5/1000	-3.57	1.560	2.208	0.43
Birth interval (months) a	35.95	6.30	-0.20	-0.10	3.15
Number of births (fertility)	2.75	-3.72	-0.35	0.73	-5.68
Number of survivors	2.56	-3.47	-0.46	0.57	-5.71

Notes: Column 1 presents simulated outcomes for the benchmark model. Columns 2-5 show percentage deviations from the benchmark outcomes that arise when selected mechanisms are "switched off" as follows:

Column 2: no effect of infant mortality on birth interval or probability of having another child
Column 3: no direct effect of lagged mortality on mortality
Column 4: no correlation between unobserved heterogeneity in mortality equation and other equations (no hoarding)
Column 5: birth spacing and family planning as if all children are boys (no gender preference in birth intervals or probability of having another child)

Figure 1: Infant mortality and preceding birth interval

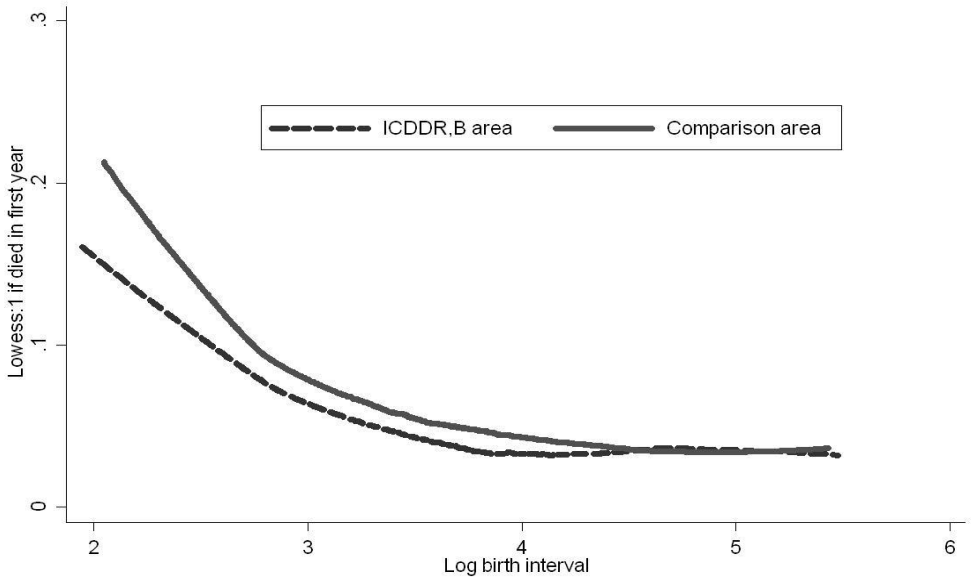


Figure 2: Birth intervals by survival status and gender of previous child, ICDDR,B area

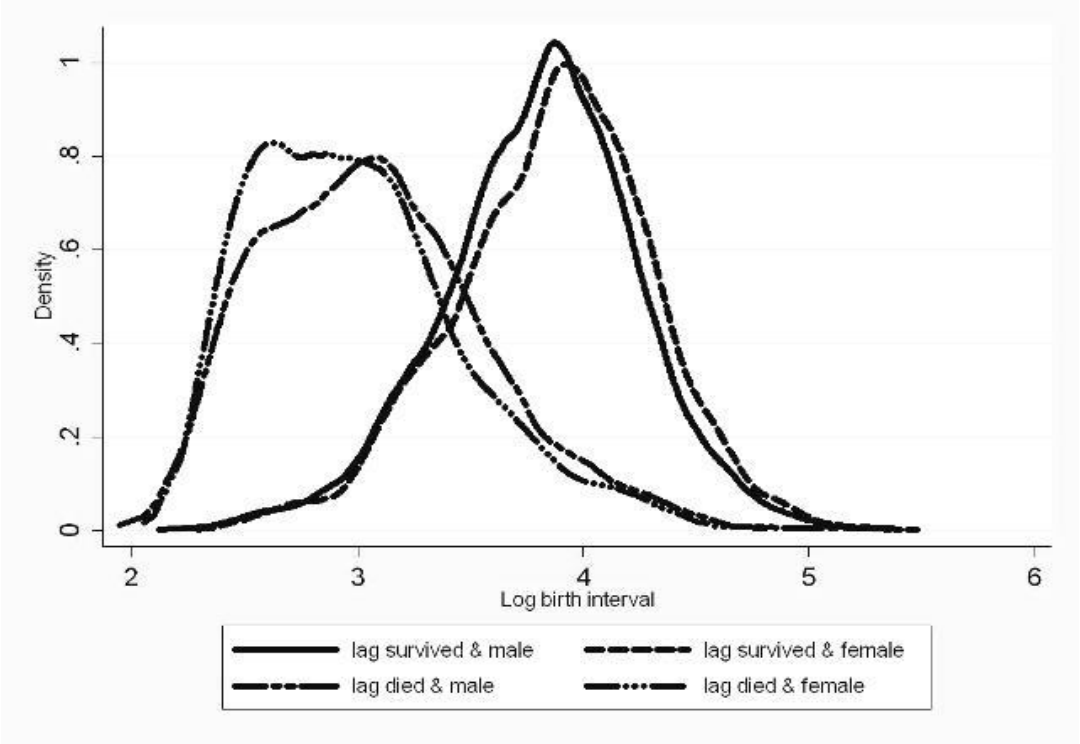


Figure 3: Birth intervals by survival status and gender of previous child, comparison area.

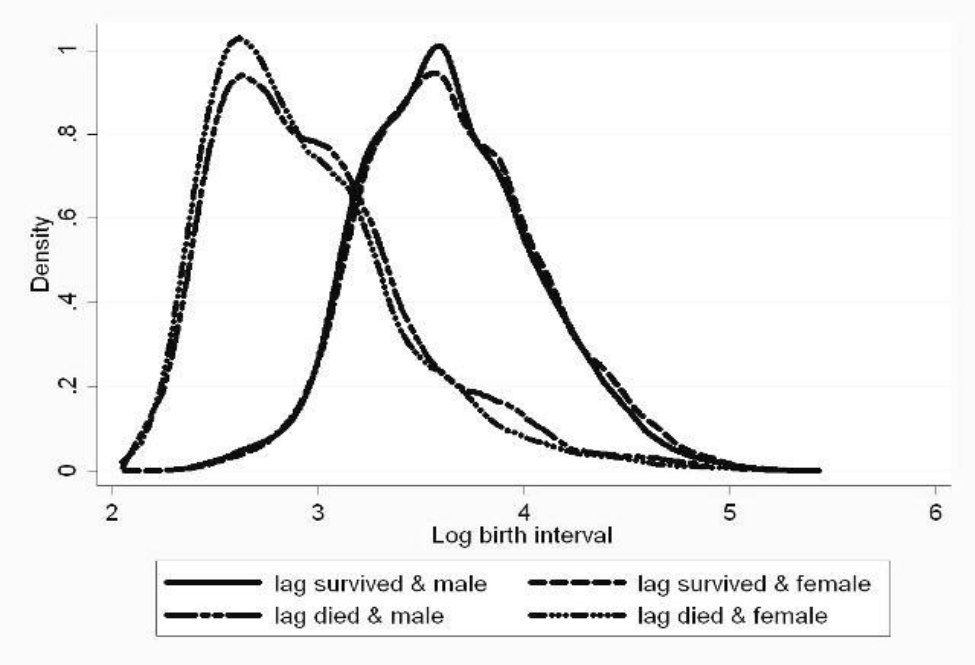
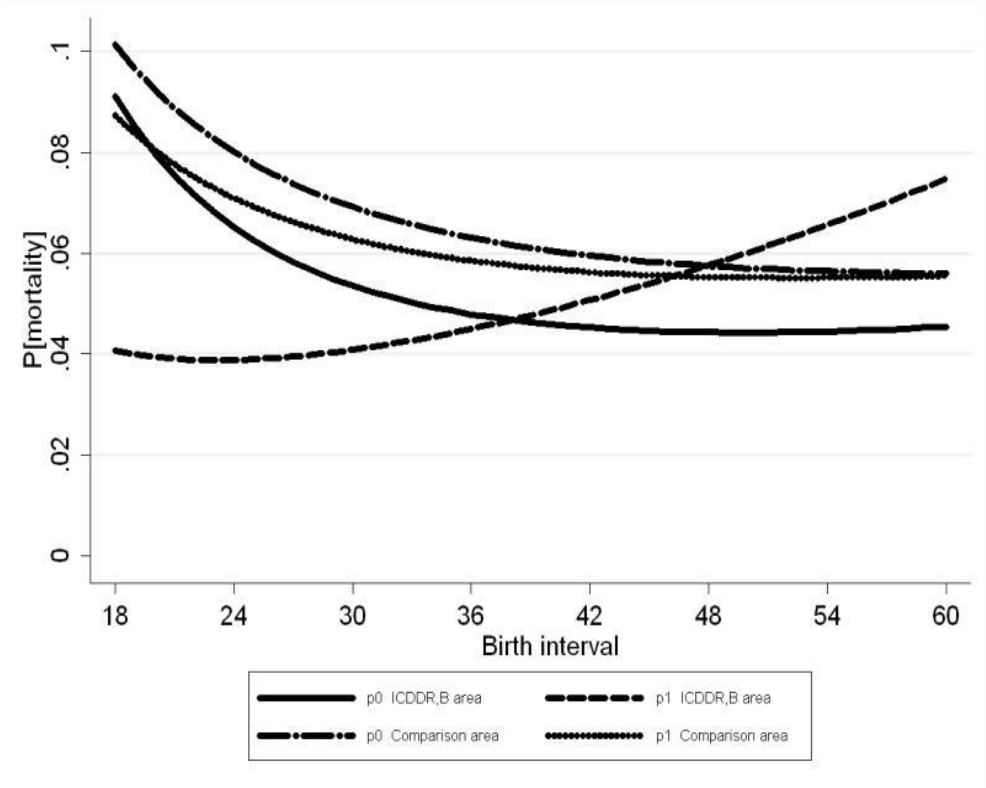


Figure 4: Predicted mortality of index child by survival status of previous child at infancy and log birth interval in both areas ICDDR,B and Comparison.



Notes: p_0 = fraction of infant deaths among those whose previous sibling survived at infancy.

P_1 = fraction of infant deaths among those whose previous sibling died at infancy.

References

Alam, N. & David, P.H. (1998). Infant and child mortality in Bangladesh: Age-specific effects of previous child's death. *Journal of Biosocial Science*, 30: 333-348.

Arulampalam, W. & Bhalotra, S. (2006). Sibling death clustering in India: State dependence versus unobserved heterogeneity. *Journal of the Royal Statistical Society A*, 169: 829-848.

Ben-Porath, Y. (1976). Fertility response to child mortality: Micro data from Israel. *The Journal of Political Economy*, l84: S163-S178.

Bhalotra, S. & van Soest, A. (2008). Birth spacing, fertility and neonatal mortality in India: Dynamics, frailty and fecundity. *Journal of Econometrics*, 143: 274-290.

Bhalotra, S., Valente, C., & van Soest, A. (2010). The puzzle of Muslim advantage in child survival in India. *Journal of Health Economics*, 29: 191-204.

Bhatia, S. (1983). Contraceptive users in rural Bangladesh - a time trend analysis. *Studies in Family Planning*, 14: 20-28.

Chowdhury, A.K.M., Khan, A.R. & Chen, L. (1976). The effect of child mortality experience on subsequent fertility: In Pakistan and Bangladesh. *Population Studies*, 30: 263-278.

Chowdhury, M.K. & Bairagi, R. (1990). Son preference and fertility in Bangladesh. *Population and Development Review*, 6: 749-757.

Cleland, J. & Sathar, Z.A. (1984). The effect of birth spacing on childhood mortality in Pakistan. *Population Studies*, 38: 401-418.

Conde-Agudelo, A., Rosas-Bermudez, A., & Kafury-Goeta, A.C. (2006). Birth spacing and risk of adverse perinatal outcomes: a meta-analysis. *Journal of the American Medical Association*, 295: 1809-1823.

Curtis, S.L., Diamond, I. & McDonald, J.W. (1993). Birth interval and family effects on postneonatal mortality in Brazil. *Demography*, 30: 33-43.

DaVanzo, J. & Pebley, A.R. (1993). Maternal depletion and child survival in Guatemala and Malayasia. Santa Monica: Rand Corporation.

DaVanzo, J., Hale, L., Razzaque, A., & Rahman, M. (2008). The effects of pregnancy spacing on infant and child mortality in Matlab, Bangladesh: How they vary by the type of pregnancy outcome that began the interval. *Population Studies*, 62: 131-154.

D'Souza, S. (1981). A population laboratory for studying disease processes and mortality- the demographic surveillance system, Matlab Comilla, Bangladesh. *Rural Demography*, 8: 29-51.

Hale, L., DaVanzo, J., Razzaque, A., & Mizanur, R. (2009). Which factors explain the decline in infant and child mortality in Matlab, Bangladesh? *Journal of Population Research*, 26: 3-20.

Hajivassiliou, V.A., & Ruud, P.A.(1994). Classical estimation methods for LDV models using simulation. In: Robert F. Engle and Daniel L. McFadden (eds.). *Handbook of Econometrics.* Volume IV, Amsterdam: North-Holland: 2384-2438.

Koenig, M.A., Phillips, J.F., Campbell, O.M., & D'Souza, S. (1990). Birth intervals and childhood mortality in rural Bangladesh. *Demography*, 27: 251-265.

Miller, J.E., Trussell, J., Pebley, R.A., & Vaughan, B. (1992). Birth spacing and child mortality in Bangladesh and Philippines. *Demography*, 29: 305-318.

Moffitt, R. (2005). Remarks on the analysis of causal relationships in population research. *Demography*, 42: 91-108.

National Institute of Population Research and Training (NIPORT), Mitra and Associates, and ORC Macro. (2005). Bangladesh Demographic Health Survey 2004. Dhaka, Bangladesh and Calverton, Maryland [USA].

Nyarko, P., Madise, N.J., & Diamond, I. (2003). Child loss and fertility behaviour in Ghana. Southampton, UK: Social Statistics Research Centre, S3RI Application and Policy Working Paper, A03/08.

Omariba, D.W.R., Fernando R., & Roderic, B. (2008). Correlated mortality of siblings in Kenya: The role of state dependence. *Demographic Research,* 18: 311-336.

Pritchett, H.L. (1994). Desired fertility and the impact of population policies. *Population and Development Review*, 20: 1-55.

Rahman, M. & DaVanzo J. (1993). Gender preference and birth spacing in Matlab, Bangladesh. *Demography*, 30: 315-332.

Rahman, A., Iqbal, Z., Bunn, J., Lovel, H., & Harrington, R. (2004). Impact of maternal depression on infant nutritional status and illness: A cohort study. *Archives of General Psychiatry*, 61: 946-952.

Saha, U.R. & van Soest, A. (2011). Infant death clustering in families: Magnitude, causes, and the influence of better health services, Bangladesh 1982-2005. *Population Studies*, 65: 273-287.

Santow, G., & Bracher, M.D. (1984). Child death and time to the next birth in Central Java. *Population Studies*, 38: 241-53.

Scrimshaw, N. (1996). Nutrition and health from womb to tomb. *Nutrition Today*, 31: 55-67.

Steer, R. A., Scholl, T.O., Hediger, M.L., Fischer, R.L. (1992). Self-reported depression and negative pregnancy outcomes. *Journal of Clinical Epidemiology*, 45: 1093-1099.

Sufian, A. J. M., & Johnson, N.E. (1989). Son preference and child replacement in Bangladesh: A new look at the child survival hypothesis. *Journal of Biosocial Science*, 21: 207-16.

Train, K.E. (2003). Discrete choice methods with simulation. Cambridge: Cambridge University Press.

UNDP (2003). Millennium development goals: A compact among nations to end human poverty. Human development report 2003.

Van Ginneken, J.K. (1974). Breastfeeding as a birth spacing method. *Studies in Family Planning*, 5: 201-206.

Van Ginneken, J.K., Bairagi, R., De Francisco, A., Sarder, A.M., & Vaughan, P. (1998). Health and demographic surveillance in Matlab: Past, present, and future. Scientific Publication Number 72.

Whitworth, A. & Stepheson, R. (2002). Birth spacing, sibling rivalry and child mortality in India. *Social Science & Medicine*, 55: 2107-2119.

Wolpin, K.I. (1997). Determinants and consequences of the mortality and health of infants and children. In: M. Rosenzweig and O. Stark (eds.). *Handbook of Population and Family Economics.* Vol. 1A, Amsterdam: North Holland: 483-557.

Wolpin, K.I. (1998). The impact of infant and child mortality risk on fertility. In: Committee on Population, National Research Council (eds.). *From Death to Birth*. Washington, D.C.: National Academy Press: 74-111.

Zenger, E. (1993). Siblings' neonatal mortality risks and birth spacing in Bangladesh. *Demography*, 30: 477-488.

Zimmer, B.G. (1979). Consequences of the number and spacing of pregnancies on outcome. and of pregnancy outcome on spacing. *Social Biology*, 26: 161-78.