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Birth Spacing, Fertility and Neonatal Mortality in India: Dynamics, Frailty and Fecundity

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ABSTRACT

Birth Spacing, Fertility and Neonatal Mortality in India: Dynamics, Frailty and Fecundity*

A dynamic panel data model of neonatal mortality and birth spacing is analyzed, accounting for causal effects of birth spacing on subsequent mortality and of mortality on the length of the next birth interval, while controlling for unobserved heterogeneity in mortality (frailty) and birth spacing (fecundity). The model is estimated using micro data on almost 30,000 children of 7,300 Indian mothers, for whom a complete retrospective record of fertility and child mortality is available. Information on sterilization is used to identify an equation for completion of family formation that is needed to account for right-censoring in the data. We find clear evidence of frailty, fecundity, and causal effects of birth spacing on mortality and vice versa, but find that birth interval effects can explain only a limited share of the correlation between neonatal mortality of successive children in a family. We also predict the impact of mortality on total fertility. Model simulations suggest that, for every neonatal death, an additional 0.37 children are born, of whom 0.3 survive.

JEL Classification: I12, J13, C33

Keywords: fertility, birth spacing, neonatal mortality, health, dynamic panel data models, siblings

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1 Introduction

In developing countries, 30% of deaths are amongst children, compared with less than 1% in rich countries, and most of these deaths are avoidable (e.g. Cutler et al. 2005). High levels of childhood mortality are closely tied to high fertility. Understanding the way in which biological and behavioural factors shape this relation at the family level is crucial to understanding the demographic transition that has historically preceded economic growth. In particular, declining rates of birth and child death are associated with higher levels of human capital investment, and economic growth (e.g. Kalemli-Ozcan 2002). By these endogenous processes, economies evolve past the Malthusian spectre; see, for example, Galor and Weil (2000).

Time series analyses of historical data for today's industrialized countries suggest that a decline in childhood mortality preceded the decline in fertility (see Ben-Porath 1976, Mattheisen and McCann 1978, Eckstein et al. 1999), and a similar tendency has been observed in recent data for developing countries (e.g. Pritchett 1994, Nyarko et al. 2003). At the same time, cross-sectional studies using household survey data have argued the reverse direction of causation, namely that high fertility, associated with close birth spacing or an early start to childbearing, causes an increase in childhood mortality (e.g. Cleland and Sathar 1984, Curtis et al. 1993).

In families with multiple children, the raw data suggest that there is in fact a recursive bi-causal relation of these variables. The death of a child has been found to be often followed by a shorter interval to the next birth, which may be explained in terms of a behavioural choice, that is, volitional replacement (see Preston 1985) or else by a biological process, the fact that the mother stops breastfeeding and, thereby, is able to conceive the next child sooner than otherwise (e.g. Bongaarts and Potter 1983, Chen *et al.* 1974). A short birth interval, in turn, seems to increase the mortality risk of the *next* child in the family, for example because the mother has not recuperated physiologically from the previous birth (e.g. DaVanzo and Pebley 1993, Scrimshaw 1996). In this way, vulnerable families are caught in a *death trap*, an endogenous process that creates persistence in death risk within families. The causal mechanisms make this a case of genuine state dependence, in the sense that the death of a child causes an increase in the risk of death of the subsequent sibling in the family. The mechanisms described here

operate by the (endogenous) shortening of intervening birth intervals. Of course a birth interval is only observed if the mother decides to have another birth. This fertility decision is also influenced by whether her previous birth survived or not. While these relationships have each been studied in the literature, it is uncommon that their interactions have been jointly studied, and unobserved heterogeneity, another potential source of correlation of death risks within a family, has often been ignored.

This paper makes a contribution by jointly estimating equations for mortality, birth spacing and the probability of having another birth, allowing unobserved heterogeneity in each equation, and correlations between the unobservables. Despite a long-standing interest amongst economists and demographers in the relationship of reproductive behaviour and childhood mortality, the literature is scarce in a complete micro-data analysis of the inter-relations of these variables (see section 2). This paper estimates the causal effects of birth interval length on subsequent mortality risk *and* of mortality on subsequent birth interval length, after controlling for unobserved heterogeneity in both processes (referred to as *frailty* and *fecundity*, respectively). It also provides estimates of the effect of expected mortality (hoarding) and realized mortality (replacement) on fertility. It offers further methodological innovations, relating to the way in which we deal with right-censoring of birth intervals and the initial conditions problem that arises in dynamic models with unobserved heterogeneity.

Analysis of the joint model makes clear that policy interventions (e.g. vaccination) targeted at a particular outcome (e.g. childhood mortality) may (a) impact on other outcomes as well (e.g. total fertility) and (b) have a long run impact that is bigger than the short run impact, through impacting on siblings of the index child. By neglecting to account for all inter-related mechanisms, the single-equation models that are more common in the literature can yield misleading predictions. Our framework is useful for analyzing the full impact of any specific intervention on the target and related variables.

Separation of state dependence from unobserved heterogeneity and identification of the causal mechanisms underlying state dependence (in particular, assessing the role of birth spacing in relation to other possible mechanisms) are of direct importance to understanding the widespread phenomenon of death clustering amongst siblings (e.g. Zenger 1993). The stronger is state dependence, the greater are the multiplier effects of

policy interventions that reduce infant mortality. The larger is the role of birth spacing in state dependence, the stronger is the case for interventions that focus on encouraging the use of contraception in achieving longer birth intervals.

For reasons elaborated in section 2, we focus on neonatal death, which is death in the first month of life. The model is estimated on longitudinal data constructed from retrospective fertility histories provided by a sample of about 7300 Indian mothers. The rest of this section summarizes our main findings.

Raw persistence, a measure of the clustering of sibling deaths in the data, is 14 percentage points (section 3.2). Genuine state dependence, or causal effects, account for 37% of this, the remaining 63% being on account of (observed and unobserved) heterogeneity between families (and communities). Endogenously determined birth spacing explains about a fourth of total state dependence (sections 5.1, 5.2).

A neonatal death shortens the subsequent birth interval by about 20 per cent. This, in turn, raises the neonatal mortality risk of the next child in the family by almost 1 percentage-point. With birth interval length and unobserved heterogeneity held constant, there is an additional risk-raising effect of the preceding sibling's mortality of about 4.3 percentage-points. In this way, birth spacing explains only about one quarter (1.0 of the 4.3 percentage-points) of state dependence in mortality. This suggests a role for other factors, identification of which is an important avenue for further research. One possibility is maternal depression, a mechanism that has been shown in an earlier study to affect child health, but has not previously been recognized as a potential mechanism for state dependence and, thereby, sibling death clustering.

We find direct evidence of replacement behaviour: a child death not only results in a shortening of the interval to the next birth, but also increases the probability of a next birth. By both mechanisms, it results in an increase in the total number of births. Our model simulations suggest that, accounting for direct and indirect effects, 37 in 100 children who die during the neonatal mortality are replaced by new births. Of these, about 30 survive the neonatal period.

There is clear evidence of unobserved heterogeneity in the mortality, birth-spacing and fertility equations. There is no evidence that frailty is correlated with fecundity. This suggests that couples do not practice hoarding, i.e., we find little evidence

that women who know that their children are at relatively large risk of neonatal death anticipate this by reducing the length of their birth intervals *ex ante.* There is a significantly negative correlation between the unobserved heterogeneity terms in the equations for birth spacing and continued fertility. This is unsurprising, implying that mothers who tend to have shorter birth intervals also tend to have more children, even if age, calendar year, and other characteristics are held constant. We find that neglecting to allow for frailty and fecundity biases upwards the effect of lagged mortality on mortality of the index child, but that it has no significant effect on the estimated impact of birth interval length on mortality risk, or on the impact of mortality on birth interval length. Geographic cluster effects (accounting for sampling design) are significant. Incorporating these effects increases standard errors, but does not change the main findings.

As a measure of the importance of allowing for the joint determination of death risk and reproductive behaviour, we estimated the effects on the main outcomes of eliminating all behavioural and non-behavioural relations between the mortality process on the one hand and the birth interval and fertility processes on the other (section 5.6). The predicted reduction in neonatal mortality is 10.9%, most of which is due to eliminating state dependence. On average, birth intervals are 2.6% longer, mainly because the replacement effect is eliminated. The total number of children born falls by 3.3%, while the total number of children surviving the neonatal period falls by 2.5% (because of the reduced mortality probability).

The next section summarizes related research. Section 3 describes the data and the endogenous variables. The econometric model is detailed in section 4. The main results are reported in section 5, where we also report specification checks and simulation results. Section 6 concludes.

2 Related Literature and Contributions

Previous demographic research provides estimates of some of the main effects analyzed in this paper, although not in a unified framework: for example, see Curtis *et al.* (1993), Madise and Diamond (1995), Hobcraft et al. (1985) for analysis of the effects of birth-spacing on mortality, and Zenger (1993) or Frankenberg (1998) for analysis of the

effects of mortality on birth-spacing. The limitation of these studies is that their estimates cannot be given a causal interpretation (also see Moffitt 2003).

In previous research in economics, equations for childhood mortality (or other indicators of child health) have been estimated in which endogeneity of birth spacing has been allowed. For example, Bhargava (2003) argues that the endogeneity of birth spacing is taken care of by controlling for the survival status of older siblings. This, in turn, is instrumented using household possessions and number of previous births. Maitra and Pal (2004) estimate a simultaneous hazards model of birth-spacing and child mortality, relying upon similarly strong identifying assumptions. Rosenzweig and Schultz (1983b) estimate a model of infant mortality in which birth-spacing is instrumented using household incomes and local prices. However, as discussed in the more recent papers of Rosenzweig and Wolpin (1988, 1995), the implied exclusion restrictions typically do not hold. Rosenzweig and Wolpin (1988, 1995) instead use sibling differences to eliminate the mother-specific endowment. In order to further allow for differences across siblings in frailty, they instrument inputs in a differenced equation using "lagged" inputs from older siblings, and parental characteristics. The econometric strategy in this paper is similar insofar as it relies upon information restrictions associated with the sequencing of births. Rather than use lagged inputs as instruments, we allow lagged mortality to affect the endogenous variables.

A contribution of this paper is that it simultaneously provides estimates of the causal effect that runs in the opposite direction, namely from previous mortality to birth spacing and fertility. Most previous estimates of these effects have been obtained under the implausible assumption that parents have no influence on the survival chances of their offspring (e.g., Ben-Porath 1976; see Wolpin 1997, Cigno 1998). Exceptions are Olsen (1980) and Olsen and Wolpin (1983), both of which analyze the response of the *number of births* to child mortality, allowing for endogeneity. The approach in the current analysis is different, in that we use a dynamic panel data framework and provide estimates of the response of birth spacing as well as total fertility to mortality (together with estimates on the same data of the reverse relation, described above).

A further contribution of this paper is that it introduces lagged mortality (i.e. the survival status of the previous child) in the mortality model, in addition to the preceding

birth interval. There is relatively little previous research on state-dependence effects in analysis of sibling data, although sibling correlations in outcomes have been widely studied (e.g. Solon *et al.* 1991).[1] A recent demographic literature has highlighted the widespread phenomenon of sibling death clustering, emphasizing the role of unobserved heterogeneity, estimated using multi-level models that incorporate a random effect at the mother-level (e.g. Guo 1993, Zenger 1993, Curtis *et al.* 1993, Sastry 1997). Arulampalam and Bhalotra (2004a,b) contribute to this discussion by distinguishing unobserved heterogeneity from causal mechanisms that produce genuine state dependence. By jointly modelling birth-spacing with mortality, this paper estimates the extent to which birth-spacing drives state dependence, as opposed to other possible causal processes.

In a broader scope, this paper relates to a larger literature in economics on how allocation decisions of parents affect the quality of their children. For example, numerous studies have attempted to estimate the effect of child schooling, a parental input, on child quality, allowing for unobserved (inheritable) endowments or ability (e.g. Card 1999). This is similar to the present analysis, insofar as it allows endogeneity in inputs to health, given endowment heterogeneity. This paper also relates to research on the dynamics of family formation, focusing on birth and death (which is more appropriate in a developing country context where these rates are high) in contrast to recent research on marriage and divorce in industrialized countries (e.g. Akerlof 1998).

3 Data & Descriptive Statistics

3.1 The Data

The data are from the second round of the National Family Health Survey of India (NFHS-II) which recorded complete fertility histories for ever-married women aged 15-49 in 1998-99, including the time and incidence of child deaths.[2] Mothers constitute the

[1] Identification of the (endogenous) effect of an outcome for one individual on the outcome of a "proximate" individual has, in research on neighbourhood effects, proved challenging (e.g. Manski 1993). Here, this problem is resolved by the natural sequencing of siblings and the fact that, after controlling for heterogeneity, the effects of predetermined variables can be interpreted as causal.

[2] For details on sampling strategy and context, see IIPS and ORC Macro (2000).

cross-sectional dimension of the data. As mothers are observed repeatedly, in relation to every birth, birth-order creates the time dimension of the panel.

We use data for Uttar Pradesh (UP), the largest Indian state, which, in the year 2000, contained 17.1% of the country's population (approximately 165 million people). It has social and demographic indicators that put it well below the Indian average (see Drèze and Sen 1997). After dropping mothers with at least one multiple birth, the sample contains 29,747 live births of 7286 mothers, that occurred between 1963 and 1999.[3]

The incidence of neonatal death over the sample period in UP was 7.39%, compared with an all-India average of 5.21%.[4] Previous research on developing country data suggests that birth intervals less than 24, and especially 18, months have deleterious effects. The percent of birth intervals in the sample that are shorter than 18 months is 17.5, and the percent that are 18-23 months long is 18.3. The mean number of births per mother is 4.04, the median number is 4, and the maximum is 14. The mean age of mothers at first birth is 18.4, and the median is 18. The mean age of mothers at birth is 22.2, and the median is 22. As many as 28.3% of all live births are to teenage mothers (age range 12-19) and 14.3% are to mothers under 18 (i.e. 12-18).

Although contraceptive prevalence is increasing in India, and this has contributed to fertility decline since the mid-1980s, it seems to have had a limited impact on neonatal mortality (e.g. James *et al.* 2000). This is because contraception in India is used primarily to limit fertility rather than to control early childbearing and lengthen birth intervals. At the time of the survey, women were asked what their current contraceptive method was. In the state of UP, 65.8% were using no method, 5.9% were using a modern method (pill, IUD or condom), 5.9% were practicing periodic abstinence or withdrawal, 1.1% reported sterilization of their (male) partners, and 19.6% reported female sterilization. Thus, female sterilization is the predominant form of contraception, as in other parts of India.

[3] Elimination of multiple births is in line with the demographic literature on mortality. Children of a multiple birth face hugely higher odds of dying, other things equal. Including multiple births would complicate the relation of mortality and birth intervals that is of interest in this paper.

[4] These figures are averages over the data sample. As this contains retrospective data, it includes children born across almost four decades. Although we do not have recent figures for neonatal death (death in the first month of life), the infant death rate (death in the first year of life) in India is estimated to have been 6.7% in 2001 (UNDP 2003), while the all-India average of the infant mortality rate in our sample is 8.2%. In our all-India sample, 63.4% of infant deaths occurred in the neonatal period.

Of the women who report sterilization, 41% were sterilized at a parity of less than 5.

The Indian survey used in this paper is one of a family of about 70 Demographic and Health Surveys (see www.measuredhs.com). The methods used in this paper are therefore immediately applicable to a vast array of countries with different profiles of the structural processes. For instance, persistently high fertility and childhood mortality are a greater problem in many African countries than in India. The analysis in this paper could fruitfully be applied to analyze the extent to which the African problem reflects a "demographic trap", described by the inter-dependence of mortality and fertility.

3.2 The Endogenous Variables

Means and standard deviations of all variables used in the analysis are in Appendix Table 1. The focus in this paper is on *neonatal mortality*, or death in the first month of life.[5] This assists the statistical modelling since it means that we can be sure that if the preceding sibling died, then this event occurred before the birth of the index child. In other words, lagged mortality is always a predetermined variable in the birth interval equation. There are also substantive reasons for this choice. The association of birth spacing and mortality tends to be strongest in the neonatal period (e.g. Cleland and Sathar 1984, Zenger 1993, Frankenberg 1998, Nyarko *et al.* 2003). It is estimated that, in 1995, about 5 million neonates died each year, 98% of whom were in developing countries, and 1.2 million of whom were in India (Hyder *et al.* 2003, pp.894-898). About two-thirds of infant (under the age of one) and almost a half of under-5 deaths occur in the neonatal period, and this proportion tends to increase as over time, as the level of under-5 mortality declines (e.g. World Bank 2004). This is partly because common interventions like immunization, oral rehydration and control of acute respiratory infection tend to have more of an impact on post-neonatal death. Increasing attention to such programmes in India has contributed to raising the ratio of neonatal to under-5 deaths (James *et al.* 2000). The incidence of neonatal death in the sample we analyse averages at about 7%. This remarkable loss of life soon after birth is tied to maternal health and reproductive behaviour.

[5] Strictly, neonatal death refers to death in the first four weeks or 28 days of life. We include deaths occurring up to a month in order to allow for age-heaping at 7-day intervals.

The birth interval is the interval between reported dates of birth, rather than the inter-conception interval. As a result, measured birth intervals will be shorter on account of premature births (e.g., Gribble 1993). This is investigated in section 5. A further potential problem is that birth intervals, as measured, will be longer on account of miscarriage or stillbirth (e.g. Madise and Diamond 1995). We do not have estimates of the extent of miscarriage or stillbirth in the data and are therefore unable to directly assess this problem. Ignoring miscarriage and stillbirth may lead to under-estimation of the mortality-raising effect of short birth intervals in equation (1) below if women who have these problems also tend to produce weaker live births, since then falsely long intervals will be associated with higher mortality. However, this bias may be expected to be small once we control for mother-specific frailty and fecundity.

Before introducing any structure, let us describe the main relationships of interest. Figure 1 is a non-parametric regression of the (unconditional) predicted probability of neonatal death on the logarithm of the preceding birth interval. This is seen to decline monotonically over most of the range of the data. At short birth intervals, the probability of neonatal death is highest, and the gains from an additional month's spacing are largest. Figure 2 plots the kernel density functions of the birth interval for two sub-samples of the data, selected according to whether or not the previous child in the family survived the neonatal period. It shows that the birth interval distribution for the case where the preceding child has died lies to the left of the other. The median birth interval is 23 months after a neonatal death and 27 months when the previous sibling has survived the neonatal period (the corresponding means are 24.3 and 31.2 months). The raw data thus exhibit the patterns that, as argued in section 1, contribute to "death traps" at the family level: Figure 1 shows that short birth intervals raise subsequent mortality risk, and Figure 2 shows that previous mortality in the family results in shorter birth intervals.

In order to describe the degree of persistence in the data, that is, to see how strongly correlated the mortality risks of successive siblings are, let us exclude first-born children for the moment, as lagged mortality (i.e. mortality of the preceding sibling) is undefined for them. In the sample of second and higher-order children, the average probability of neonatal death is 6.28%. Consider how the probabilities of neonatal death compare conditional upon the neonatal survival status of the preceding sibling. In the

sub-sample in which the previous sibling survived, this probability is 5.20%, and amongst those whose previous sibling died, the probability is a remarkable 18.80%. Thus the death of a preceding sibling is associated with an increase in mortality risk of 13.6 percentage points. This clustering of sibling deaths can be explained by both unobserved heterogeneity and genuine state dependence, and state dependence can, in turn, be explained by short birth-spacing or other mechanisms. The analysis to follow will (a) disentangle the causal effects from correlations amongst siblings, and (b) consider the contribution of causal effects working through birth-spacing.

4 The Model

The model has a recursive dynamic structure: the propensity of neonatal mortality risk depends upon previous mortality amongst siblings (and, thereby, on lagged inputs to child health) and on the preceding birth interval, while the birth interval, in turn, depends upon the mortality status of the preceding sibling. Similarly, the probability of continuing fertility also depends on previous mortality. Identification of the main causal effects rests on exploiting the natural sequencing of the birth spacing (or birth) and mortality processes. An important advantage of this approach is that it avoids the need to impose exclusion restrictions. Amongst other covariates in the model are maternal age at birth of the child, and the year of birth of the child. Together with birth order, these are endogenous because they depend upon the entire history of birth intervals (and maternal age at first birth). This is allowed for (see section 4.1).

The mortality equation can be regarded as a health production function in which the birth interval is an endogenous input (as in Rosenzweig and Schultz 1983a,b). The birth spacing equation is an input equation, but it also describes an outcome that depends upon tastes and technology. These two equations are estimated jointly with an equation for continued fertility to account for right-censoring of the birth interval, and an equation for mortality risk of the first-born child, that addresses the initial conditions problem.

The estimation allows for endowments (persistent mother-specific traits), unobservable by the econometrician but potentially known to the mother, and for the agency of the parent in influencing outcomes. The health endowment is referred to as *frailty*. By modelling this term we allow for the fact that children of the same mother

have correlated mortality risks, because of shared genetic or environmental factors. We also incorporate inter-family unobserved heterogeneity in the birth spacing and fertility equations (for convenience both of these heterogeneity terms are henceforth referred to as *fecundity*, although they are not restricted to be the same), and allow this to be correlated with frailty. This allows, for example, that women who are more careful about contraception may also be more careful in maintaining the health of their children. Ignoring unobserved heterogeneity would bias estimates of the dynamics of each process (see Heckman 1981, Heckman *et al.* 1985, Hyslop 1999) and might also bias estimates of the causal effect of each of these variables on the other (e.g. Alessie *et al.* 2004).

The econometric model is an extension of the univariate model of Heckman (1981) and Hyslop (1999), and is broadly similar to the bivariate model of Alessie *et al.* (2004) although, here, the second equation (for birth interval length) is continuous rather than discrete, and subject to right-censoring. The approach we take to dealing with right-censoring is new, exploiting data on sterilization (section 4.3). The way in which the initial conditions problem is addressed is also novel (section 4.4). To take account of the sampling design, we use random effects at the community (cluster) level (section 4.5). The model is estimated by simulated maximum likelihood (section 4.6).

Let there be n_i children in family i (which implies an unbalanced panel). M_{ij} denotes an indicator variable with value 1 if child j in family i suffers neonatal death, and 0 otherwise. B_{ij} is the log of the length of the interval between the birth of child j-1 and child j in family i. In other words, B_{ij} refers to the interval closed by the birth of child j. As it is the preceding birth interval for child j, it is, by definition, predetermined with respect to M_{ij}. The rest of this section describes each of the four equations in the model, and explains the estimation procedure.

4.1 Neonatal Mortality

For child j ($j=2,\ldots,n_i$) in family i ($i=1,2,\ldots, N$), the equation for neonatal mortality is

$$(1) \qquad M_{ij}^{*} = g(\mathbf{x_i}, \mathbf{x_{i1}}, \mathbf{x_{ij}}, M_{i1},\ldots M_{i,j-1}, B_{i2},\ldots, B_{ij}; q_m) + a_{mi} + u_{mij};$$
$$M_{ij}=1 \text{ if } M_{ij}^{*}>0 \text{ and } M_{ij}=0 \text{ if } M_{ij}^{*}<0$$

In order to explain the assumptions needed for consistent estimation, it is initially written in a general form. Here a_{mi} is family (or mother)[6] specific unobserved heterogeneity. This reflects the child's health endowment or "*frailty*", which may derive from genetic sources (e.g maternal propensities to low birth weight and prematurity), environmental factors, or child-care behaviours (e.g., Sastry 1997, Curtis *et al.* 1993, and Rosenzweig and Schultz 1983a,b). As emphasized in Rosenzweig and Wolpin (1988), the fact that endogenous inputs like breastfeeding are not explicitly incorporated implies that the estimated family-effect will reflect not only inter-family heterogeneity in endowments but also any inter-family heterogeneity in preferences. Although a_{mi} is, by definition, unobservable to the econometrician, it may or may not be known to the family. The error term u_{mij} varies over mothers as well as children. It is revealed at the birth of child j and we assume that it does not influence parental inputs to child j in the one month of life during which parental choices can influence neonatal mortality risk. However, we allow u_{mij-1} to influence parental inputs into child j through past mortality in the family, M_{ij-1}.

The vectors $\boldsymbol{x_i}$, $\boldsymbol{x_{i1}}$, and $\boldsymbol{x_{ij}}$ are exogenous explanatory variables, partitioned into variables that vary over children ($\boldsymbol{x_{ij}}$, $j=2,\ldots,n$), are specific to the first child ($\boldsymbol{x_{i1}}$), or do not vary over children ($\boldsymbol{x_i}$). The vector of unknown parameters is denoted by q_m. The variables $M_{i1},\ldots M_{i,j-1}$, $B_{i2},\ldots, B_{ij}$ are realized at or before the birth of child j.

For the function g, we will use a linear specification in $\boldsymbol{x_i}$, $\boldsymbol{x_{ij}}$, $M_{i,j-1}$, B_{ij}, and also include quadratic terms in the year of birth of the child, and in the age of the mother at birth of the index child, both of which are functions of $\boldsymbol{x_{i1}}$ and $B_{i2},\ldots, B_{ij}$.[7] Since the age of the mother at birth of child j depends upon her age at birth of child j-1 and the length of the intervening birth interval, B_{ij}, it is clear from recursivity of the model that maternal age at birth of j can be expressed as a function of maternal age at first birth (in $\boldsymbol{x}_{i1}$) and the history of birth intervals up until that date ($B_{i2},.. B_{ij}$). Thus, by allowing for endogeneity of birth intervals and conditioning on x_{i1}, we are allowing for the endogeneity of maternal age. Since the data used include births that occurred across a span of about 30 years, a quadratic in the year of birth of the child is included to capture

[6] Re-marriage (and re-partnering) amongst Indian women is rare enough that it is reasonable to use "mother" interchangeably with "family".

any technological change. This, like maternal age, is a function of the birth year of child 1 (assumed exogenous, and in $\boldsymbol{x}_{i1}$), and of all previous birth intervals of the mother.

We expect a negative effect of B_{ij} on M_{ij}, consistent with the hypothesis of maternal depletion indicated in section 1, and also with competition amongst closely spaced siblings (e.g. Cleland and Sathar 1984, Zenger 1993). The effect of lagged mortality, $M_{i,j-1}$ on M_{ij} may be negative if learning effects dominate, or positive if there is a strong role for factors like maternal depression (see section 1). The first-order Markov assumption implicit in our specification of g is justified by the nature of the mechanisms driving state dependence (that is, a causal effect of M_{ij-1} on M_{ij}): see Zenger (1993).

We assume that $\boldsymbol{x}_i$, $\boldsymbol{x}_{i1}$, and $\boldsymbol{x}_{ij}$ are independent of a_{mi} and u_{mij}. Mean independence of ($\boldsymbol{x}_i$, $\boldsymbol{x}_{i1}$) and a_{mi} is the usual assumption in a random effects model, needed for identification; the conditional mean of a_{mi} given $\boldsymbol{x}_i$ and $\boldsymbol{x}_{i1}$ is subsumed in g. In $\boldsymbol{x}_i$, we include variables reflecting education levels of the mother and father, and caste and religion dummies. In $\boldsymbol{x}_{i1}$ we also include calendar year and age of mother at first birth.

A potential drawback of random effects models as compared with fixed effects models is the assumption that the "*time-varying*" (in this context, varying across siblings and, thereby, implicitly over time) regressors $\boldsymbol{x}_{ij}$ are assumed to be independent of the individual effects a_{mi}. In our case, however, the only variables included in $\boldsymbol{x}_{ij}$ are child gender and birth-order. Since it is plausible that these are uncorrelated with mother-level frailty, the independence assumption seems unproblematic in this model.

4.2 Birth Spacing

The log length of the birth interval is modeled in a similar way as mortality:

$$(2) \qquad B_{ij} = h(\boldsymbol{x}_i, \boldsymbol{x}_{i1}, \boldsymbol{x}_{i,j-1}, M_{i1},\ldots M_{i,j-1}, B_{i2},\ldots, B_{ij-1}; q_b) + a_{bi} + u_{bij};$$

The family-specific effect in the birth spacing equation, a_{bi}, is referred to as "*fecundity*" though it will include not only biological fecundity but also any other unobserved sources of persistent inter-family heterogeneity, e.g. variation in preferences for family planning

[7] We experimented with interactions and squares of other terms but found no significant improvement.

or desired fertility. A causal effect of mortality of child *j-1* on the birth interval to child *j* is allowed through $M_{i,j-1}$. Past death shocks are, in this way, allowed to influence current behaviour. We include $\mathbf{x}_{i,j-1}$ since the gender of the previous child *(j-1)* may have an effect on the interval to the birth of child *j*. The function *h* is specified as a linear combination of $\mathbf{x}_i$, $\mathbf{x}_{i,j-1}$, $M_{i,j-1}$, and the calendar year and age of the mother at the time of the birth of child *j-1* and their squares. As discussed in section 4.1, the year of birth of the child, and maternal age at birth of the child are functions of $\mathbf{x}_{i1}$ and $B_{i2},\ldots, B_{ij-1}$. Biomedical and demographic research provide no clear argument for a causal effect of B_{ij-1} on B_{ij}, conditional on a_{bi}, so we do not allow for this.[8] The assumptions concerning family-specific effects and errors u_{bij} are similar to those for equation (1). We assume that $\mathbf{x}_i$, $\mathbf{x}_{i1}$, and $\mathbf{x}_{ij}$ are independent of a_{bi} and u_{bij} and that u_{bij} is independent of the past.

We allow for correlation between the unobserved heterogeneity terms a_{bi} and a_{mi} in equations (1) and (2). This allows an alternative, non-causal explanation for the correlation between birth interval lengths and mortality in the raw data. It also accounts for the potential endogeneity of the preceding birth interval in equation (1), which, although predetermined, may be correlated with frailty, a_{mi}. For example, parents with weak endowments may choose shorter birth intervals in order to meet their target number of children in a given time. Similarly, our model allows M_{ij-1} in equation (2) to be correlated with family-level fecundity, a_{bi}.

The distribution of the family effects (a_{mi}, a_{bi}) is assumed to be bivariate normal with mean zero, variances s_m^2, s_b^2, and covariance $s_m s_b r_a$. The child-specific error terms u_{mij} and u_{bij} are assumed to be independent of a_{mi} and a_{fi} and normally distributed with mean zero. Without loss of generality, the variance of u_{mij} is set to 1.

4.3 Right-Censoring

Inclusion of the birth spacing equation, (2), in the model demands a correction for right-censoring because some mothers will not have completed their fertility at the time

[8] Heckman *et al.* (1985) show, for a sample of (married) Swedish mothers, that there is no state dependence in the birth spacing process once controls for unobserved heterogeneity are introduced.

of the survey.[9] To account for this, we model the probability that mother i will have another child after the birth of child j, as follows:

$$(3) \qquad F_{ij}^{*} = f(\boldsymbol{x_i}, \boldsymbol{x_{i1}}, \boldsymbol{x_{ij}}, M_{i1},\ldots M_{i,j-1}, B_{i2},\ldots, B_{ij}; q_f) + a_{fi} + u_{fij};$$
$$F_{ij}=1 \text{ if } F_{ij}^{*}>0 \text{ and } F_{ij}=0 \text{ if } F_{ij}^{*}<0$$

We specify f as a linear combination of $\boldsymbol{x_i}$, the calendar year and age of the mother at the time of the birth of child j-1 and their squares (functions of $\boldsymbol{x_{i1}}$ and $B_{i2},\ldots, B_{ij-1}$), dummies for the presence of boys and the presence of girls in the family (that did not suffer neonatal death), and the total numbers of boys and girls in the family who survived the neonatal period (functions of j, $M_{i1},\ldots M_{i,j}$, and $B_{i2},\ldots, B_{ij-1}$). The variables are gender specific to allow for son-preference, of which there is considerable evidence for the state of UP (e.g. Drèze and Gazdar 1997). Endogeneity of the gender-specific sibship variables is taken care of in the same way as in the other equations – these variables are a function of lagged dependent variables. Moreover, confounding unobserved factors are controlled for by allowing arbitrary correlations of a_{fi} with a_{mi} and a_{bi}, assuming joint trivariate normality with arbitrary covariance matrix and independence of exogenous variables. We make similar assumptions on u_{fij} as on the other error terms: normality, independence of individual effects and error terms for other birth-orders or other equations, and independence of exogenous variables. Equation (3) is estimated jointly with equations (1), (2) and (4) (below).

The data contain information on whether a mother is sterilized at the time of the survey, which helps to estimate the parameters of the model more efficiently. For these mothers, who constitute 19.6% of the sample, it is safe to assume that the complete birth process is observed. Of the remaining mothers, some will have another child after the survey date, and others will not. Sterilization is an incomplete indicator of whether the mother will have another child; it is an implicit dependent variable in our model but not a

[9] It may be useful to think in terms of the fertility equation being to the birth-spacing equation, what the participation equation is to the hours of work equation in the more familiar context of selection into wage work (e.g. Heckman 1974). The hours equation gives hours for those who have decided to participate; hours are automatically zero for those who do not. The birth interval

variable of interest as such, as it is the decision to have another child or not that is modelled here. To identify equation (3) using data on sterilization, we assume that women who have decided to have no more children get sterilized with a fixed probability *?*, a nuisance parameter to be estimated.

Let us be more precise. If mother *i* has more than *j* children, then we know she has given birth to another child after child *j*, and the likelihood will incorporate the probability that $F_{ij}=1$. If the mother reports that she has had exactly *j* children and was sterilized after the birth of the *j*-th child, then the likelihood will incorporate the probability that $F_{ij}=0$ and the probability *?*. If at the time of the survey, the mother had *j* children but was not (yet) sterilized, then it is unclear whether child *j* is the last child or not; it could be that the birth interval after the birth of child *j* extends beyond the time of the survey. The probability that this will happen, given that there will be another birth and given unobserved heterogeneity components, follows from (2) and is given by $F([T-\{h(\mathbf{x_i}, \mathbf{x_{i1}}, \mathbf{x_{i,j-1}}, M_{i1},\ldots M_{i,j-1}, B_{i2},\ldots, B_{ij-1}; q_b)+ a_{bi}\}]/s)$, where T is the length of the time interval elapsed between the birth of child *j* and the time of the survey, and *s* is the standard deviation of the error term in (2). In this case, the likelihood (conditional on unobserved heterogeneity terms) will contain a factor that accounts for the fact that we do not observe whether or not there will be another birth after birth *j*.[10]

The usual approach to right-censoring is to assume that the same process continues but that we simply stop observing it at the time of the survey (e.g. Wooldridge, 2002, Chapter 20). This approach does not work well in the current application since the fertility process is necessarily finite (though at different points for different women) and ended well before the time of the survey for many women in the sample.[11] In the absence of information on sterilization, natural but less promising alternatives would be to assume

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equation gives the interval length if the woman decides to have another child; the interval length is infinity for others.

[10] This will be the sum of the probability that the mother will still have children but the birth interval extends beyond the date of the survey, and the probability that the mother has decided to have no more children but does not use sterilization. Detailed likelihood contributions are available in an online Appendix, at http://www.ecn.bris.ac.uk/ecsrb/bhalotra.

[11] Initial experimentation with our data showed that the usual procedure produces a poor fit, being unable to explain why so many women suddenly completely stop having children. This is because it merges the birth interval with the fertility decision, when in fact we need two separate equations for these two processes.

that fertility stops at a given age (e.g. 40) for all mothers, or to estimate equation (3), but without the sterilization information. In this case, the fertility equation would only be indirectly identified in the sense that we would observe many women with very long birth intervals, and the model estimates would attribute this to cessation of fertility. These estimates are likely to be much less precise than those exploiting the sterilization information.

4.4 The Initial Conditions Problem

"Lagged" mortality, M_{ij-1}, is endogenous in equation (1) by virtue of being correlated with frailty, a_{mi}. This creates the initial conditions problem commonly encountered in analysis of dynamic models with unobserved heterogeneity (e.g. Heckman 1981). This problem is addressed by formulating a separate equation for the mortality risk of the first-born child of every mother, which can be estimated jointly with the other equations in the model:

$$(4) \qquad M_{i1}^{*} = g_1(\mathbf{x_i}, \mathbf{x_{i1}}; q_{m,1}) + l_m a_{mi} + l_b a_{bi} + l_f a_{fi} + u_{mi1};$$
$$M_{i1}=1 \text{ if } M_{i1}^{*}>0 \text{ and } M_{i1}=0 \text{ if } M_{i1}^{*}<0$$

In most existing applications of these sorts of models, described by Heckman (1981), Hyslop (1999) and Wooldridge (2000), the true process is ongoing and the first observation is generated in the same way as later observations, the only difference being that it is the first observation in the sampling window. Heckman *et al.* (1985) is an exception. They model birth spacing and observe the process from its natural start, the start of menarche. Here, similarly, we observe the birth and mortality processes from their beginning for each mother in the sample, and the first child is a genuine starting point of that process (as already shown in Arulampalam and Bhalotra 2004a,b). This makes Heckman's approach quite natural compared to, for example, the alternative approach to addressing initial conditions recently proposed by Wooldridge (2000).

We will work with a linear specification of g_1, in line with the specification of (1). It seems likely that M_{i1} will be correlated with a_{mi} but since the equation for M_{i1} contains no lagged dependent variable, the coefficient on a_{mi} is allowed to be different from 1 (by

l_m). M_{i1} is also allowed to be correlated with a_{bi} or a_{fi}, the family-specific effects in the birth-spacing equation, (2), and the fertility equation, (3). The error term u_{mi1} is assumed to be standard normal and independent of the other error terms in the model, of the individual effects, and of the exogenous regressors $\boldsymbol{x_{ij}}$ and $\boldsymbol{x_i}$. $?_{m1}$, l_m, l_b and l_f are auxiliary parameters. Equation (4) is a flexible function of the exogenous variables. We do not impose restrictions on the relation of the parameters in (4) (risk for first born child) to those in (1) (risk for other children in the family).

4.5 Geographical Cluster Effects

The data are collected in 333 geographical clusters ("communities") with, on average, 24.4 mothers per cluster. To allow for the possibility that mothers (and children) within a cluster share unobservable traits (for example, sanitation or social norms), we need to include a cluster-level term in the equation error.[12] As the large number of clusters makes it infeasible to use cluster dummies, we incorporate random cluster effects in equations (1) and (2) and (3) in the same way as the mother-specific effects, with similar assumptions.[13] A linear combination of the cluster effects in (1), (2) and (3) is added to equation (4), with three additional auxiliary parameters as coefficients. For identification, it is assumed that the cluster effects are independent of mother-specific effects. Thus common characteristics of all mothers in a given community will be picked up by the cluster effects rather than by the mother-specific effects.

4.6 Estimation

The complete model can be estimated by maximum likelihood, including the nuisance parameters of the initial conditions equation, and the fertility equation.[14] Conditional on the random (mother and cluster level) effects, the likelihood contribution of a given mother can be written as a product of univariate normal probabilities and densities over all births of a mother, and the likelihood for a given cluster can be written as the product over all mothers in that cluster. Since random effects are unobserved, the

[12] The data has some information on community characteristics at the time of the survey. We did not use this since it may not reflect community characteristics at time of birth.

[13] That is, trivariate normal with arbitrary covariance structure to be estimated, independent of exogenous variables and error terms.

actual likelihood contribution is the expected value of the conditional likelihood contribution, with the expected value taken over all random effects (three in the model without cluster effects, six in the model with cluster effects). This is a three or six-dimensional integral, which could in principle be approximated numerically using, for example, the Gauss-Hermite-quadrature.

In this paper, we instead use (smooth) simulated ML, drawing multivariate errors from $N(0, I_3)$. These are then transformed into draws of the random effects using the parameters of the random effects distribution. The conditional likelihood contribution is then computed for each draw and the mean across R independent draws is taken. If $R \to \infty$ with the number of observations (i.e., in this case, clusters, since mothers are no longer independent observations), this gives a consistent estimator; if draws are independent across households and $R \to \infty$ faster than $\sqrt{N}$, then the estimator is asymptotically equivalent to exact ML (see, for example, Hajivassiliou and Ruud 1994). We use Halton draws, which have been shown to give more accurate results for smaller values of R than independent random draws (see Train 2003). The results we present are based on $R=100$. Using $R=50$ gives very similar results (see section 5.5).

5 Results

This section presents the results of the complete "benchmark" model (Tables 1-3) and a sensitivity analysis to some changes in specification (section 5.5). Some simulation results for the benchmark model are presented in Section 5.6. Table 4 presents the estimated covariance structure of the mother and community level random effects.

5.1 Neonatal Mortality

Table 1 reports the parameter estimates of the equation for neonatal mortality. It also reports marginal effects for the second child, assuming that the first child survived the first month of life, and setting all family characteristics to their benchmark values when categorical (boy, Hindu, not of a backward caste, maternal and paternal education zero), and to their average values for second children when not (birth year 1986.8, age of

[14] An explicit specification of the likelihood function can be found in the online Appendix.

the mother at birth 21.3 years, previous log birth interval 3.32).[15] The estimated probability of neonatal mortality for this benchmark child is 4.84%.

The preceding birth interval has the expected negative effect on the probability of neonatal death. A ten percent increase in the length of the birth interval reduces the probability of death by about 0.45 percentage-points in the benchmark case, and the marginal effect is similar for higher birth-orders. In view of the finding, in previous research, that the deleterious effects of short birth intervals are enhanced if the previous sibling has survived (e.g. Zenger 1993, Cleland and Sathar 1984), we also included an interaction of "lagged" neonatal mortality and the log of the preceding birth interval but this was insignificant. This suggests that maternal depletion rather than sibling competition explains the mortality-increasing effects of short birth intervals. Maternal depletion is likely to be especially pronounced amongst poor women who need longer to replenish stocks of nutrients like calcium and iron that are needed to support a healthy pregnancy.

Neonatal mortality of the previous sibling makes neonatal death significantly more likely for the index child, even with the birth interval held constant. For the benchmark second child, the estimated difference is 4.16 percentage-points. Similar effects are found for the third, fourth, and later children. This suggests that any learning effects, whereby a mother is better able to avoid a further child death once she has experienced one, are dominated by state dependence mechanisms that create a positive association of sibling deaths and that do not operate *via* the shortening of birth intervals. As indicated in section 1, we hypothesize that the loss of a child may create psychological effects that the mother may not have recovered from by the time she conceives her next child, as a result of which there may be physiological effects that make this child more vulnerable both in the womb and after birth.[16] This is in line with a recent literature that finds a negative effect of depression on child outcomes. Steer *et al.* (1992), e.g., show that depression can cause adverse pregnancy outcomes; Rahman *et al.*

[15] The marginal effects are birth-order-specific. A full set of marginal effects by birth-order is available on request; not shown for parsimony.

[16] If M_{ij-1} were capturing a depression effect and if depressed mothers systematically had shorter or longer birth intervals, then we would expect the interaction term between preceding birth interval (B_{ij}) and M_{ij-1} to be significant but, as discussed above, it is not.

(2004) find that maternal depression in the prenatal and postnatal periods is a risk factor for malnutrition and illness in infants in Pakistan. While depression is one plausible causal mechanism there may, of course, be other processes at work too.[17]

Conditional on the other covariates, neonatal mortality of boys and girls is not significantly different. Neonatal mortality is also not sensitive to birth-order.[18] For the benchmark child, there is a trend reduction of 0.16 percentage-points per year (3.3% of the benchmark probability) in the risk of death. Neonatal mortality risk is U-shaped in mother's age at birth of the index child, a pattern familiar from other studies using developing country data. The minimum occurs at about 29 years of age. On average, mothers are much younger than this when giving birth to their second child (21.3 years old). This explains the significantly negative marginal effect obtained for the benchmark second child: if the mother's age increases by one year, the mortality probability falls by 0.21 percentage-points. At higher birth-orders, the average age of the mother increases and the U-shape implies that for birth-orders above six, the marginal effect turns positive. For example, it is 0.09 percentage-points per year for the benchmark seventh child. Mortality risk tends to be decreasing in both maternal and paternal education, larger and more significant marginal effects being associated with maternal education. For example, secondary or higher education of the mother (which 3.8% of mothers in the sample have) is associated with a 2.3 percentage point reduction in mortality, relative to the case of mothers having no education (75% of mothers). A striking result, that deserves further investigation, is that children of Muslim families are significantly less likely to die in the first month than Hindu children, with an estimated difference of about 1.7 percentage-points.[19] We find no significant differences between castes.

[17] If the periods in which child j-1 and child j were exposed to the risk of death overlapped then the transmission of infection amongst siblings might contribute a further source of genuine state dependence. In our model, this is less likely since, if child j-1 suffered neonatal death on account of an infection, child j was, by definition, not yet born. Nevertheless, if the infection were persistent and stayed around until j was born, this could be a mechanism for state-dependence. Of course if infection were just more prevalent in certain households or communities all the time, then this would be picked up by the random effects in the model.

[18] Note that these results are for the sample of children of birth-order two or higher. There may well be a birth-order effect that is significant for first-borns. Similarly, son-preference effects may show up at a later age, or in interaction with birth-order.

[19] The raw data probability of neonatal and infant death is also lower amongst Muslims. Since, compared with Hindus, Muslims exhibit shorter birth intervals, higher fertility and a greater

Estimates of the "reduced form" probit equation for mortality of *first-born* children (equation 4) are in the online Appendix (see footnote 10). The female dummy is now negative and significant at the two-sided 10% level, consistent with the fact that girls are born with a survival advantage, and with previous research that shows that discrimination against girls is increasing in birth-order (e.g. DasGupta 1990). Other effects are broadly similar.

5.2 Birth Spacing

Estimates of the birth spacing equation are in Table 2. Since the dependent variable is in logs, the interpretation of the parameters is in terms of percentage changes in the expected length of the birth interval. Note that all covariates in this model refer to the preceding child (i.e. the child born at the start of the birth interval).

There is a strong negative effect of neonatal death of the previous child on the subsequent birth interval, reducing its expected length by about 21%. This is consistent with replacement behaviour (e.g. Ben-Porath 1976). Feeding this into equation (1), we can conclude that the effect of M_{ij-1} that operates via B_{ij} results in an increase in M_{ij} of about 1.06%-points. Since the direct effect of M_{ij-1} on B_{ij} in equation (1) was found to be 4.16%-points, total state dependence increases the risk of death by 5.2%-points. Thus genuine state dependence accounts for a sizeable 37% of the clustering of sibling deaths or raw persistence, which was 14%-points on average (as was shown in section 2). The residual 63% can be attributed to (observed and unobserved) heterogeneity.

The gender of the last-born child is significant, consistent with son-preference. If the last birth was a girl, the expected birth interval is about 3% shorter than if it was a boy. The quadratic trend is hump shaped, with a maximum at about 1980. Thus birth intervals have tended to get shorter in recent decades (1980-1998). This may be explained by rising living standards. In particular, since better-nourished mothers will tend to suffer less deleterious effects from a short birth interval, they can "afford" shorter birth intervals. There is some indication that spatial (inter-state) patterns in India resemble the

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proportion of mothers and fathers with no education, our finding suggests that the mortality-reducing intercept effect of religion identified here dominates the mortality-increasing effects flowing from these explanatory variables. In research in progress, we are examining this seeming paradox.

inter-temporal pattern detected here, with the wealthier states (like Punjab) having a greater proportion of births with short intervals while, at the same time, having lower neonatal mortality (see Arulampalam and Bhalotra 2004b). Birth spacing is hump-shaped in the age of the mother at birth, with a maximum at about 29 years of age. This means that, for the average mother, birth intervals increase until the sixth child is born. Parental education has no significant effect on birth spacing. Birth intervals are shorter amongst Muslim families by 7.6%, compared with Hindu families. There are no significant differences in birth spacing by caste-group. Other things equal, birth-order exhibits a non-monotonic pattern, with the shortest birth intervals preceding the birth of the fourth child.

5.3 Fertility Equation

Table 3 presents estimates of the probability of having another child after each birth, as a function of calendar time and family characteristics such as family composition and maternal age. Of particular interest are the family composition variables. The results indicate son-preference, of which there is considerable evidence from Northern India, especially in UP (e.g. Drèze and Sen 1997). The probability of continued fertility is decreasing in the number of surviving children, but more than three times as rapidly in the number of surviving boys than in the number of girls. Also, if the family has no surviving boys, the probability of having another child is larger than if there are no surviving girls. Similar results have been reported for other countries in Asia and North Africa (e.g. Rahman and DaVanzo 1993, Nyarko *et al.* 2003).[20]

The quadratic in the child's year of birth is hump-shaped, with a maximum at about 1981. The quadratic in mother's age is decreasing over the whole relevant age range (until age 47). Fertility falls with the level of education of both mother and father, with mother's education having larger effects. Muslims show a higher tendency to continue fertility. Mothers in backward castes other than scheduled castes and tribes have lower fertility than others.

5.4 UnobservedHeterogeneity

Table 4 presents the estimated covariance structure of the mother and community level random effects. A sensitivity analysis shows that the covariance structure of the mother and community-specific effects is not robust to specification choices (such as the number of draws in the simulated ML procedure), but the covariance structure of the sum of the two terms is robust to most specification details. We therefore focus on the latter, referred to as *total* unobserved heterogeneity. There is significant evidence of both effects in the mortality and birth interval equations, but only community-specific effects are significant in the fertility equation. Compared to the idiosyncratic noise term (with variance 1), the two heterogeneity terms in the mortality equation make a modest contribution, capturing about 15% of the total unsystematic variation in M_{ij}^* (0.173/(1+0.173)). More than half of this is heterogeneity across communities; the remainder is across mothers within communities. Previous research in rich and poor countries has found evidence of mother-level frailty, with varying estimates of its contribution to the overall variation in mortality risk (e.g. Rosenzweig and Schultz 1983a,b, Rosenzweig and Wolpin 1988, 1995, Curtis *et al.* 1993, Guo 1993, Zenger 1993) but since these studies typically do not allow for clustering at the community level, they will tend to over-estimate the mother effects (also see Bolstad and Manda 2001).

In the equation for the log birth interval, the idiosyncratic noise term has estimated variance 0.206 (0.454^2), and the heterogeneity terms together pick up only about 11% of the total unsystematic variation. We find significant negative correlation between the mother-specific, and positive correlation between the community-specific heterogeneity terms in the birth spacing and neonatal mortality equations, but the estimated covariances are of opposite sign and almost cancel out against each other, giving a correlation coefficient of –0.004 for the total unobserved heterogeneity terms.

The heterogeneity terms in the fertility equation explain about 15% of the unsystematic variation in F_{ij}^*, but this estimate is not very accurate. We find a large negative correlation between total heterogeneity terms in the fertility and birth interval equations of –0.73. This suggests that, as expected, mothers who desire many children tend to use shorter birth intervals to achieve this, other (observed) explanatory variables

[20] Angrist and Evans (1998) find no such asymmetry for the US; they do find that the probability of a third child is larger if the first two children are of the same sex than if they are of different

constant. This is consistent with replacement behaviour in, for example, the target fertility model (see Wolpin 1997). On the other hand, the small correlations between total unobserved heterogeneity in the mortality equation and both the birth interval and the fertility equations suggest that hoarding does not play much of a role: there is hardly any evidence that mothers who perceive their children to have relatively high mortality risk react *ex ante* by having persistently shorter birth intervals.

Overall, the heterogeneity terms are statistically significant but relatively small compared to the idiosyncratic errors. This raises the question of whether neglecting to allow for unobserved heterogeneity would lead to biased estimates of the parameters of interest. This question is explored in the following subsection.

Table 4 also shows how the unobserved heterogeneity terms enter the equation for neonatal mortality of the first child. As expected, mothers with a relatively large probability of neonatal mortality of higher birth-order children are also more likely to experience higher mortality risk for the first child, although this is only significant at the 6% level. Somewhat surprisingly, we do not find the same for the community effects - these are small and insignificant in the mortality equation for the first child.

5.5 Sensitivity Analysis

Table 5 presents estimates of the coefficients on the (lagged) endogenous variables for alternative specifications. The effects of the other variables are not shown since they do not change much compared to the estimates of the benchmark model (*Model 1*) in Tables 1-3. Consider the consequences of omitting the birth interval from the mortality equation (*Model 2*). This increases the estimated effect of lagged mortality in the mortality equation, consistent with the mechanisms described in section 1. It also biases the effect of lagged mortality on the birth interval in equation (2). This is because omission of the birth interval induces a significant negative correlation (of –0.43) between the (total) unobserved heterogeneity terms in equations (1) and (2), which creates an upward simultaneity adjustment on the coefficient of lagged mortality. In Model 1, this correlation was small and insignificant (at –0.004).

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sex

Model 3 excludes the community effects. There is now a positive correlation of 0.21 between the unobserved heterogeneity terms in equations (1) and (2). This explains why the point estimates indicate somewhat larger negative effects of the birth interval on the mortality probability and *vice versa* than in the benchmark model. The main difference is that this model underestimates the standard errors on account of its ignoring correlations across observations.

Model 4 does not allow for unobserved heterogeneity at the community or mother level. This creates some significant changes. The most salient is the effect of lagged on current mortality, which is about 80% larger than in the benchmark model (and 67% larger than in the model that allows mother-specific but not community-specific unobserved heterogeneity, i.e. Model 3). This is consistent with the traditional argument that ignoring heterogeneity leads to overestimation of state-dependence effects (Heckman 1981). There is little change in the effect of mortality on the next birth interval, probably because the correlation between the total unobserved heterogeneity terms is very close to zero in the benchmark model.

Model 5 combines the restrictions imposed in arriving at Models 2 and 4. The two positive biases on the effect of lagged mortality on mortality together lead to an estimate that is twice as large as in the benchmark model. There is hardly any bias on the coefficient of mortality in the birth interval equation, for the same reason as in Model 4.

A challenging finding is that the effect of lagged mortality on mortality in equation (1) is strong even when the length of the preceding birth interval is controlled for. We now consider if this might reflect a specification error. For example, the family may have suffered a temporary shock (a poor harvest, maternal illness) that spans two or more births, resulting in greater vulnerability of two successive children. This was investigated by including the second lag of the neonatal mortality dummy in equation (1) (*Model 6*). The coefficient on the second lag is positive and statistically significant (0.191 with standard error 0.075). Instead of reducing the effect of the first lag (as would be expected if M_{j-2} were in fact an omitted variable),[21] it limits the role of unobserved heterogeneity: the standard deviation of the total unobserved heterogeneity term in the

mortality equation falls from 0.416 to 0.274. Thus it seems that the results suggested by our benchmark model cannot be attributed to misspecification of the lag structure.[22,23]

We performed some additional sensitivity checks, results for which are not presented in Table 5 since they were virtually identical to those of the benchmark model. The results presented are based on 100 random draws for each observation (*R=100)*. Reducing this to 50 draws hardly changes the results.[24] We found higher neonatal mortality amongst children with a shorter preceding birth interval. In order to ensure that this is not simply the result of a selective over-representation of premature births (Cleland and Sathar 1984, p406), we re-estimated the model after removing from the sample all mothers with at least one birth interval under 9 months. This resulted in a loss of 0.6% of all mothers. The estimates of the main parameters are virtually the same as when the short birth intervals are included. Adding an interaction term of the log birth interval and lagged mortality in the mortality equation does not lead to a significant improvement. Similar minimal deviations compared to the benchmark model are found when the square of the log birth interval is added to the mortality equation. We also investigated a specification that is piecewise linear in the log birth interval but, again, were unable to reject the reported specification against this more general specification. This seems in line with the simple association shown in Figure 1.

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[21] It may be better to compare the state dependence estimate of 0.431 to the estimate of the same coefficient in a model without second lag but with a separate equation for mortality of the second child. Such a model gives a coefficient of 0.374 (with standard error 0.095).

[22] An alternative would be to allow for autocorrelation between the error terms in the mortality equation. We experimented with this in a single equation framework (using the GHK algorithm to obtain the simulated likelihood) but found an insignificant (negative) autocorrelation coefficient rather than the positive coefficient that would be expected under the hypothesis that the significance of M_{ij-1} reflects a temporary shock. Also, there is, again, an increase in the coefficient on the lagged dependent variable.

[23] For computational convenience and given the similarity of the results for models 1 and 3, we did not incorporate community clusters in this variant of the model. To do this would require specification of a separate equation not only for the first but also for the second child (for whom the second lag cannot be included). See Heckman (1981); details available upon request.

[24] The only thing that changes somewhat is the decomposition of total unobserved heterogeneity terms into a mother and a community specific effect.

5.6 Simulations

Some simulations are performed with the benchmark model, to investigate the effects of neonatal mortality on birth intervals and fertility, and to analyze the importance of scarring and hoarding.

The first step is the benchmark simulation where all mechanisms at work in the estimated model are active. More detail on this is in the online Appendix. The first column of Table 6 presents these results. The simulated average of 4.12 births is somewhat larger than the average number in the sample, because the numbers in the sample are truncated at the time of the interview.[25] The average number of children surviving the neonatal period is 3.82. Thus the simulated neonatal mortality rate is 7.4%. Finally, the overall mean of simulated birth intervals is 30.59 months, somewhat smaller than the observed mean of 30.78 in the data, the difference being due to a small number of large outliers. The other columns in Table 6 present percentage deviations relative to the benchmark for scenarios in which selected behavioural or non-behavioural mechanisms in the model are "switched off."

The first alternative simulation switches off the effect of mortality on the next birth interval. This increases the average length of birth intervals by 1.7%.[26] Since longer birth intervals depress mortality, overall neonatal mortality falls by almost 3.2% (from 7.40 to 7.17 %-points). The longer birth intervals imply that women have their children later, and since mother's age has a negative effect on the probability of having another child, this reduces the number of births by 0.72%. Due to the fall in mortality, the reduction in the number of surviving children is smaller (0.47%).

The next simulation (col. 3) shows what happens if deaths have no effect on the probability of having another child. Total births fall by almost 2%. Because this works mainly by reducing later births that have relatively large probabilities of neonatal mortality, it has a negative effect on the mortality rate (0.94%). It has hardly any effect on birth interval lengths.

[25] If we apply the same truncation in the simulation, the average number in the simulations is virtually very similar to the observed number.

[26] The 1.7% is a "first order effect". It is the product of the neonatal death rate (7.4%) and the effect of neonatal death on the length of the subsequent birth interval (21%; see section 5.2). The other effects in Table 6 bear a similar relation to the marginal effects reported in section 5.

Column 4 combines the changes in columns 2 and 3. This increases birth intervals and reduces total fertility. It reduces neonatal mortality by 4.4%, mainly because of the longer birth intervals. This simulation can be used to estimate the total size of the replacement effects as follows. Total mortality in the column 4 simulation is 7.08% of all births. The number of births as a result of replacement is about 2.6% of all births, that is, 0.37 births for every neonatal death. Because replacement increases mortality, the replacement effect on the number of surviving children is smaller – about 0.30 surviving replacement children for every neonatal death. These effects, although they refer to replacements for neonatal deaths only, are well in line with the existing literature, which puts estimates between 0.2 and 0.5 (see Schultz 1997, pp.384-385).

In column 5, the only change compared to the benchmark simulation is that the effect of "lagged" mortality (i.e. mortality of the previous child) on the probability of mortality of the index child is suppressed. Mortality is lower by 0.44 and the number of surviving children higher by 0.33 percentage-points. There is little impact on birth spacing or fertility.

Column 6 combines the "experiments" conducted in columns 4 and 5, suppressing all causal effects of previous mortality. Mainly because of suppressing the replacement effects, the average length of birth intervals increases by 0.50 months and the total number of children born falls by 2.6%. Neonatal mortality falls by about 0.66 %-points, because scarring is suppressed and also because of the longer birth intervals. Since the fertility effect dominates the mortality effect, the total number of surviving children falls.

In column 7, the only deviation compared to the benchmark is that hoarding is eliminated: the correlations of the unobserved heterogeneity term in the mortality equation with those in the birth spacing and fertility equations are set to zero. Thus "high frailty" mothers are now not expected, ex ante, to have shorter birth intervals or higher probabilities of having another child (and vice versa). The net result is a small increase in the length of the average birth interval (0.9%) and an accordingly small reduction of neonatal mortality (1.54%). The total number of children born and surviving falls slightly.

Finally, column 8 combines "no hoarding" (col. 7) with "no causal effects of neonatal mortality" (col. 6) and thus essentially eliminates all behavioural and non-behavioural relations between the mortality process on the one hand and the birth interval and fertility processes on the other. The total reduction in neonatal mortality compared to the benchmark is 10.9% (0.805 percentage-points), most of which is due to eliminating scarring (col. 5). On average, birth intervals are 2.6% longer, mainly because the replacement effect is eliminated. The total number of children born falls by 3.3%, the total number of children surviving the first 30 days of their lives falls by less (2.5%), because of the reduced mortality probability.

6 Conclusions

Interest in the determinants of child mortality has recently surged, with the inclusion of targets for child mortality amongst the Millennium Development Goals (see Lancet 2003, UNDP 2003), and short birth-spacing is widely regarded as one of the most important causes of early childhood death. A number of international organizations have programmes that encourage longer birth-spacing. For instance, USAID is currently supporting the Optimal Birth Spacing Initiative.

Using retrospective fertility histories from a large sample of Indian mothers, a dynamic panel data model is estimated that describes the complete process of child survival and birth spacing (and thus also fertility), allowing for endowment heterogeneity, input endogeneity, right-censoring and the initial conditions problem.

We find evidence that childhood mortality risk is influenced by the pattern of childbearing, that is, by the timing and spacing of births, and that birth-spacing and fertility are, in turn, a function of realized mortality. Together, these recursive causal effects suggest multiplier effects of policies that reduce mortality or lengthen birth intervals. They also suggest that the full impact of family planning interventions extends to reducing mortality and, similarly, that mortality-reducing interventions like provision of piped water will tend to impact also on birth spacing and fertility.

Our results show that unobserved heterogeneity in the form of mother or community specific effects explains part of the correlation between neonatal mortality of successive children observed in the data. Another part is explained through birth spacing.

The largest part of the correlation, however, is explained by neither the birth interval mechanism nor unobserved heterogeneity and could, for example, be due to a mental health shock induced by the death of a child, leading to maternal behavior that increases the chances of subsequent mortality. This is a striking result, especially as previous demographic research has restricted attention to the birth spacing mechanism.

Using data on sterilization to estimate an equation for the decision to have another child at each birth, we find that women who have many children also tend to choose shorter birth intervals, a result that has some intuitive appeal. We find evidence consistent with son-preference. The probability of having another birth is much larger if there are no surviving boys as compared with girls, and it decreases more quickly in the number of surviving boys. Furthermore, birth intervals are shorter following the death of a boy rather than a girl.

Mortality and fertility are U-shaped in maternal age at birth, although most of the sample points lie in the region with a negative slope. Birth spacing is hump-shaped in maternal age, with most sample points lying in the region with a positive slope. Maternal education decreases mortality and fertility but has no effect on birth spacing. Paternal education depresses the probability of another birth but has no significant effect on the other endogenous variables. Being Muslim lowers mortality and, at the same time, reduces birth spacing. Mothers from backward castes other than scheduled castes and tribes have lower fertility, but there are no other significant effects of caste on the outcomes analyzed. Conditional upon the other covariates, we estimate a trend reduction in mortality of 0.16%-points p.a., which is about 3.3% of the benchmark probability. We find that birth intervals have got shorter in the last two decades (1978-98), even as fertility has been declining.

Future work could extend the framework to analyze infant or child (under-5) mortality. This creates the additional complication that mortality events and births can take place in overlapping time periods, requiring a different modelling approach. Other extensions could make explicit use of data on breastfeeding, although this would mean restricting the analysis to recent births as these data are not available in most DHS surveys for children born more than five years before the survey. Finally, these results are for one Indian state, albeit a state with a population estimated at more than 166 million in

2001. Extension of the analysis to consider other Indian states or other developing countries will lend important insight into the extent to which the key relationships analysed here are altered by socio-economic development.

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Table 1: Neonatal Mortality (equation 1)

	Parameter	Std error	ME	Std. err. (ME)
lagged mortality	0.320*	0.068	4.254	1.248
log birth interval	-0.447*	0.050	-4.472	0.685
Religion:				
Muslim	-0.197*	0.065	-1.647	0.562
Other	-0.043	0.338	0.252	3.546
Caste:				
scheduled caste	0.098	0.136	1.273	1.594
scheduled tribe	0.100	0.054	1.118	0.585
other backward caste	-0.061	0.052	-0.574	0.513
caste missing	0.063	0.099	0.555	0.888
Maternal education:				
incomplete primary	-0.029	0.094	-0.184	0.932
complete primary	-0.197*	0.093	-1.580	0.724
incomplete secondary	-0.093	0.099	-0.880	0.872
secondary & higher	-0.297*	0.144	-2.243	0.875
Paternal education:				
incomplete primary	-0.004	0.083	-0.022	0.883
complete primary	-0.109	0.076	-0.980	0.686
incomplete secondary	-0.103	0.059	-0.963	0.538
complete secondary	-0.134*	0.067	-1.197	0.571
higher than secondary	-0.031	0.066	-0.286	0.647
Gender:				
Female	-0.043	0.038	-0.391	0.374
Birth year of child			-0.161	0.037
year of birth of child/10	-0.028	0.620		
(year/10) squared	-0.008	0.036		
Age mother at birth of child			-0.205	0.097
maternal age at birth/10	-0.817*	0.341		
(age/10) squared	0.143*	0.063		
Child birth-order			0.444	0.555
birth-order	0.044	0.049		
square of birth-order	-0.001	0.004		
Constant	1.633	2.706		

Notes: *: parameter (and marginal effect) significant at the two-sided 5% level. ME denotes marginal effects. These are computed for a benchmark child, defined in section 5.1. The (omitted) reference cases for the categorical variables (religion, caste, maternal and paternal education, gender) are defined in Appendix Table 1.

Table 2. Log Birth Interval (equation 2)

	Parameter	Std error	t-value
lagged mortality	-0.237*	0.017	-14.34
Religion:			
Muslim	-0.076*	0.014	-5.55
Other	-0.098	0.070	-1.41
Caste:			
scheduled caste	0.025	0.034	0.73
scheduled tribe	0.001	0.013	0.11
other backward caste	-0.005	0.012	-0.40
caste missing	0.001	0.024	
Maternal education:			
Incomplete primary	0.011	0.024	0.43
complete primary	0.034	0.021	1.61
Incomplete secondary	0.013	0.024	0.56
secondary & higher	0.034	0.022	1.53
Paternal education:			
Incomplete primary	0.013	0.020	0.66
complete primary	0.002	0.015	0.14
Incomplete secondary	0.004	0.015	0.25
complete secondary	-0.001	0.016	-0.09
higher than secondary	0.010	0.016	0.62
Gender:			
Female	-0.028*	0.008	-3.52
Trend effects:			
year of birth of child/10	0.415*	0.121	3.43
(year/10) squared	-0.026*	0.007	-3.68
Maternal age:			
maternal age at birth/10	0.308*	0.072	4.29
(age/10) squared	-0.054*	0.015	-3.68
Child birth-order			
birth-order	-0.025*	0.008	-2.95
square of birth-order	0.003*	0.001	3.49
Constant	1.402*	0.509	2.75
sigma error	0.454*	0.002	188.22

Notes: See Notes to Table 1.

Table 3. Fertility: Probability of another birth (equation 4)

	Parameter	Std error	M E	Std.err(ME)
Religion:				
Muslim	0.379*	0.045	11.497	1.300
Other	-0.404	0.242	-14.442	9.537
Caste:				
scheduled caste	0.101	0.088	3.456	2.721
scheduled tribe	-0.044	0.039	1.529	1.321
other backward caste	-0.158*	0.038	5.597	1.360
caste missing	-0.038	0.074	0.111	0.222
Maternal education:				
incomplete primary	-0.115	0.070	-4.152	2.561
complete primary	-0.136*	0.059	-4.956	2.257
incomplete secondary	-0.266*	0.067	-9.743	2.580
secondary & higher	-0.544*	0.062	-20.782	2.438
Paternal education:				
incomplete primary	0.116*	0.057	3.806	1.853
complete primary	-0.035	0.049	-1.344	1.724
incomplete secondary	-0.098*	0.042	-3.486	1.434
complete secondary	-0.198*	0.048	-7.297	1.702
higher than secondary	-0.228*	0.047	-8.315	1.767
Year of birth child:			-5.172	0.301
year of birth of child/10	14.462*	0.850		
(year/10) squared	-0.895*	0.051		
Maternal age:			-1.715	0.167
maternal age at birth/10	-1.195*	0.190		
(age/10) squared	0.126*	0.033		
Surviving children				
1 if no boys	0.206*	0.049	7.060	1.645
1 if no girls	0.147*	0.041	5.042	1.440
number of boys	-0.249*	0.025	-8.638	0.975
number of girls	-0.072*	0.020	-2.477	0.726
Constant	-53.872*	3.420		

Notes: See Notes to Table 1. The marginal effects are given for the benchmark case after the birth of the fifth child, with probability 70.3% of having another child. The reason for taking the fifth child is that probabilities of having another child after an earlier birth are larger, giving rather small marginal effects.

Table 4. Unobserved Heterogeneity
Mother plus community level effects

Covariance matrix			
	Mortality	Birth interval	Fertility
Mortality	0.173		
Birth interval	-0.000	0.025	
Fertility	0.003	-0.049	0.181
Correlation matrix			
	Mortality	Birth interval	Fertility
Mortality	1.000		
Birth interval	-0.004	1.000	
Fertility	0.015	-0.725	1.000

Table 5. Sensitivity Analysis:
Endogenous coefficients in alternative specifications

	Mortality equation		**Birth interval eq.**
	$M_{i,j-1}$	**Ln $B_{i,j}$**	**$M_{i,j-1}$**
Model 1			
Benchmark model	0.320	-0.447	-0.237
	(0.068)	(0.050)	(0.017)
Model 2			
No lagged birth interval	0.390		-0.211
	(0.065)		(0.016)
Model 3			
No cluster effects	0.369	-0.482	-0.246
	(0.055)	(0.046)	(0.016)
Model 4			
No unobserved heterogeneity	0.585	-0.411	-0.243
	(0.042)	(0.039)	(0.013)
Model 5			
No lagged birth interval &	0.643		-0.243
no unobserved heterogeneity	(0.042)		(0.013)
Model 6			
2^{nd} lag in mortality equation	0.431	-0.458	-0.221
	(0.097)	(0.066)	(0.025)

Notes: See section 5.5 of the text for explanation. Figures are parameter values, with standard errors in parentheses.

Table 6. Simulations

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Neonatal mortality (%)	7.404	-3.17	-0.94	-4.40	-5.97	-8.92	-1.54	-10.86
Birth interval (months)	30.586	1.70	-0.05	1.66	0.08	1.66	0.91	2.63
Number of births (fertility)	4.125	-0.72	-1.96	-2.59	-0.15	-2.59	-0.72	-3.34
Number of survivors	3.819	-0.47	-1.88	-2.25	0.33	-1.90	-0.60	-2.50

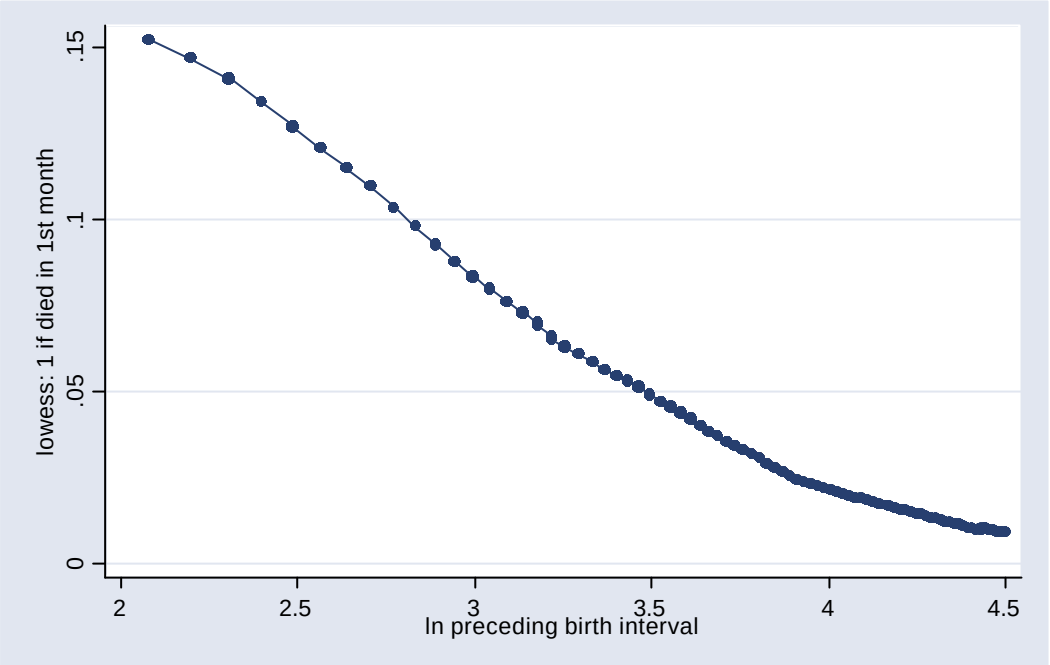
Notes: Column 1 presents sample averages of the simulated outcomes for the benchmark model. Columns 2-8 show percentage deviations from the benchmark that arise when selected mechanisms are "switched off" as follows:
2: No effect of mortality on birth interval.
3: No effect of mortality on probability of having another child.
4: No effect of mortality on birth interval or probability of having another child (2+3).
5: No effect of lagged mortality on mortality.
6: (4+5).
7: No hoarding (i.e. unobserved heterogeneity in the mortality equation is not correlated with unobserved heterogeneity in the equations for birth spacing and fertility).
8: (6+7).

Appendix Table 1
Variable Definitions and Summary Statistics

Variable	Mean	Std. Dev.	Min	Max
neonatal mortality	0.069		0.0	1.0
lagged neonatal mortality	0.060		0.0	1.0
log birth interval*	3.306	0.486	2.1	5.7
Hindu	0.824		0.0	1.0
Muslim	0.168		0.0	1.0
other religions	0.007		0.0	1.0
not backward caste	0.454		0.0	1.0
scheduled caste	0.196		0.0	1.0
scheduled tribe	0.022		0.0	1.0
other backward caste	0.276		0.0	1.0
mother has no education	0.753		0.0	1.0
ma has incomplete primary	0.045		0.0	1.0
ma has completed primary	0.075		0.0	1.0
ma has incomplete secondary	0.061		0.0	1.0
ma has secondary or higher	0.064		0.0	1.0
father has no education	0.334		0.0	1.0
pa has incomplete primary	0.068		0.0	1.0
pa has completed primary	0.110		0.0	1.0
pa has incomplete secondary	0.195		0.0	1.0
pa has completed secondary	0.125		0.0	1.0
pa has higher than secondary	0.164		0.0	1.0
Female	0.475		0.0	1.0
year of birth of child*	86.992	7.394	630	99.0
maternal age at birth*	23.224	5.539	12.0	47.0
birth-order*	3.179	2.051	1.0	14.0
no surviving boys	0.122		0.0	1.0
no surviving girls	0.188		0.0	1.0
number of surviving boys*	1.962	1.386	0.0	8.0
number of surviving girls*	1.782	1.461	0.0	10.0

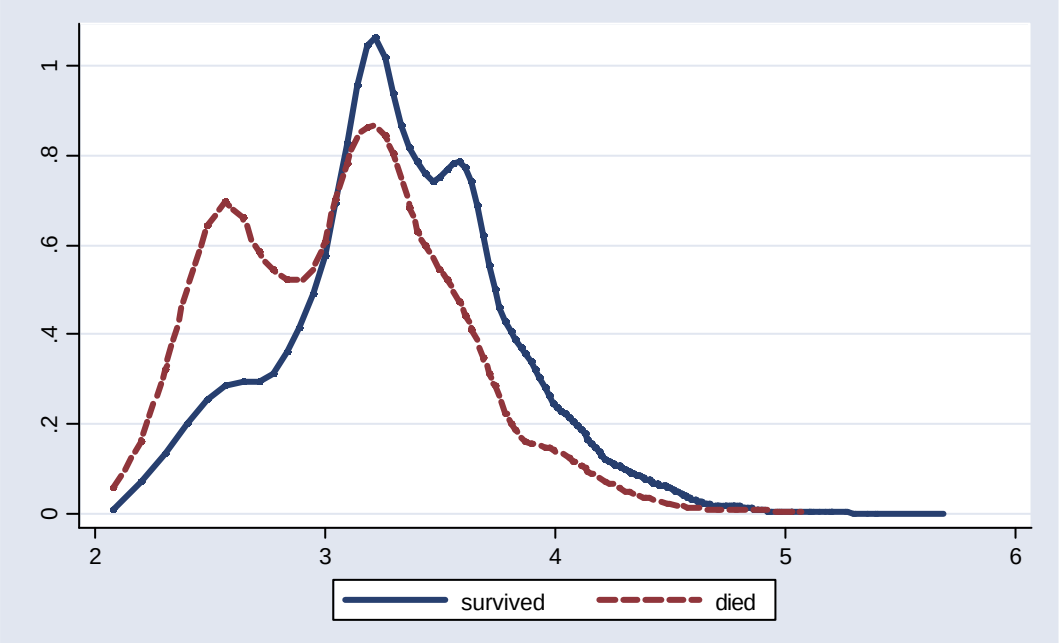
Notes: All variables other than those with a * are dummies. Lagged mortality refers to the mortality status of the preceding sibling. Italics indicate reference category omitted in the regressions.

Figure 1: Nonparametric (lowess) relation of (predicted) neonatal mortality and the preceding birth interval



Notes: The top 1% of observations were deleted.

Figure 2: Density of log birth interval by survival status of preceding sibling



Appendix 1: Likelihood Contributions

In this appendix we present the likelihoods of the models with and without community specific effects. The model is presented in Section 4, but now we explicitly incorporate the community specific effects. Let $c = c(i)$ denote the community where mother i lives.[1] Let z_{ij}^M denote the vector of explanatory variables affecting the probability of neonatal mortality of child j of mother i in equation (1) (and, for $j = 1$, equation (4)), i.e., z_{ij}^M contains (functions of) the exogenous variables x_i, $x_{i1}, \ldots, x_{i,j}$, and the "lagged dependent variables" $M_{i1}, \ldots, M_{i,j-1}$ and $B_{i2}, \ldots, B_{ij}$. Similarly, let z_{ij}^B and z_{ij}^F denote the vectors of explanatory variables in the birth spacing and fertility equations, (2) and (3), respectively. Let $\alpha_i^m = (\alpha_{mi}^m, \alpha_{bi}^m, \alpha_{fi}^m)$ be the vector of mother specific effects, let $\alpha_i^c = (\alpha_{mi}^c, \alpha_{bi}^c, \alpha_{fi}^c)$ be the vector of community specific effects in the mother's local community $c(i)$, and let $\alpha_i = \alpha_i^m + \alpha_i^c$.

Conditional on the individual effects $\alpha_i = (\alpha_{mi}, \alpha_{bi}, \alpha_{fi})$, the likelihood can be written as a product of conditional probabilities of consecutive events given the birth and mortality process until then, and given the individual effects. For neonatal mortality of child $j > 1$ with observed outcome M_{ij} we get, using equation (1) in Section 4.1:

$$P_{ij}^M(M_{ij}|z_{ij}^M, \alpha_i^m, \alpha_i^c) = [\Phi(g(z_{ij}^M; \theta_m) + \alpha_{mi})]^{M_{ij}} + [1 - \Phi(g(z_{ij}^M; \theta_m) + \alpha_{mi})]^{1-M_{ij}}$$

For the first child, we get a similar expression using equation (4) in Section 4.4 and with $\lambda^m = (\lambda_m^m, \lambda_b^m, \lambda_f^m)'$ and $\lambda^c = (\lambda_m^c, \lambda_b^c, \lambda_f^c)'$:

$$\begin{aligned} P_{i1}^M(M_{i1}|z_{i1}^M, \alpha_i^m, \alpha_i^c) &= [\Phi(g(z_{i1}^M; \theta_{m,1}) + \lambda^{m\prime}\alpha_i^m + \lambda^{c\prime}\alpha_i^c)]^{M_{i1}} \\ &\quad + [1 - \Phi(g(z_{i1}^M; \theta_{m,1}) + \lambda^{m\prime}\alpha_i^m + \lambda^{c\prime}\alpha_i^c)]^{1-M_{i1}} \end{aligned}$$

For a woman who has given birth to n children, we observe the exact log durations $B_{i2}, \ldots, B_{in}$ of birth intervals preceding births $2, \ldots, n$. The conditional likelihood contributions for these observations are densities of the normal distribution that immediately follow from equation (2) in Section 4.2:

$$p_{ij}^B(B_{ij}|z_{ij}^B, \alpha_i^m, \alpha_i^c) = \frac{1}{\sigma(u_b)}\phi\left(\frac{B_{ij} - h(z_{ij}^B; \theta_b) - \alpha_{bi}}{\sigma(u_b)}\right)$$

[1] Since the survey does not provide retrospective information on moving, we have to make the assumption that the mother has not moved since she first gave birth.

where ϕ is the density of the standard normal distribution and $\sigma(u_b)$ is the standard deviation of the error term u_{bij} in equation (2).

Since births $1, \ldots, n-1$ are followed by another birth, we also know that fertility is not yet completed after each of these births. Using equation (3) in Section 4.3, this adds the following contributions to the conditional likelihood for $j = 1, \ldots, n-1$:

$$P_{ij}^F(1|z_{ij}^F, \alpha_i^m, \alpha_i^c) = \Phi(f(z_{ij}^F; \theta_f) + \alpha_{fi})$$

Finally, the information about what happens after birth n must be incorporated. We assume that a woman who has decided not to have more children gets a sterilization with a fixed probability κ. With probability $1-\kappa$ she uses another way to make sure she has no other children. The parameter κ is a nuisance parameter to be estimated. If the mother was sterilized after birth n, we know she has decided not to have more children, and equation (3) leads to the contribution:

$$L_{in}^F(\alpha_i^m, \alpha_i^c) = P_{in}^F(0|z_{in}^F, \alpha_i^m, \alpha_i^c) = [1 - \Phi(f(z_{in}^F; \theta_f) + \alpha_{fi})]\kappa$$

If the mother was not sterilized after the birth of child n, we do not observe whether the duration between the birth of child n and the time of the survey is a right censored birth interval ($F_{in} = 1$ but child $n+1$ will be born after the time of the survey) or the mother will not have more children ($F_{in} = 0$) but has decided not to get a sterilization. Let $B_{i,n+1}$ be the log of the duration between birth n and the time of the survey. Then this implies a conditional likelihood contribution given by:

$$\begin{aligned} L_{in}^F(\alpha_i^m, \alpha_i^c) &= \Phi(f(z_{in}^F; \theta_f) + \alpha_{fi})[1 - \Phi(\frac{B_{i,n+1} - h(z_{i,n+1}^B; \theta_b) - \alpha_{bi}}{\sigma(u_b)})] \\ &\quad + [1 - \Phi(f(z_{in}^F; \theta_f) + \alpha_{fi})][1 - \kappa] \end{aligned}$$

Combining these contributions gives the total conditional likelihood of mother i, given the mother specific and community specific effects:

$$\begin{aligned} L_i(\alpha_i^m, \alpha_i^c) &= \prod_{j=1}^{n} P_{ij}^M(M_{ij}|z_{ij}^M, \alpha_i^m, \alpha_i^c) \prod_{j=2}^{n} p_{ij}^B(B_{ij}|z_{ij}^B, \alpha_i^m, \alpha_i^c) \\ &\quad \prod_{j=1}^{n-1} P_{ij}^F(1|z_{ij}^F, \alpha_i^m, \alpha_i^c) \quad L_{in}^F(\alpha_i^m, \alpha_i^c) \end{aligned}$$

To obtain the actual, unconditional, likelihood contributions, the expectation needs to be taken over the unobserved mother and community specific effects.

Model without community specific effects

In the model without community specific effects ($\alpha_i^c = 0$ for all i), observations on all N mothers are independent. The likelihood can be written as:

$$L = \prod_{i=1}^{N} L_i$$

with

$$L_i = \int_{\infty}^{\infty} \int_{\infty}^{\infty} \int_{\infty}^{\infty} L_i(\alpha^m, 0) f_{\alpha^m}(\alpha^m) d\alpha^m$$

The three-dimensional integral is approximated using R simulated draws α_r^m from the three variate normal distribution of α^m:

$$L_i^R = \sum_{r=1}^{R} L_i(\alpha_r^m, 0)$$

To be precise, the α_r^m are obtained by transforming Halton draws from the uniform distribution on the $(0, 1)$ interval to draws from the three variate normal distribution, see Train (2003).[2] Maximizing $L^R = \prod_{i=1}^{N} L_i^R$ over all the parameters gives a smooth simulated maximum likelihood estimator. If $R \to \infty$ faster than $\sqrt{N}$, this estimator is asymptotically equivalent to exact maximum likelihood (see, e.g., Hajivassiliou and Ruud, 1994).

Model with community specific effects

In the model with community specific effects, each community is treated as one observation. Let N^c be the number of communities. The likelihood can be written as:

$$L = \prod_{c=1}^{N^c} L_c$$

with

[2] See main document for references.

$$L_c = \int_\infty^\infty \int_\infty^\infty \int_\infty^\infty L_c(\alpha^c) f_{\alpha^c}(\alpha^c) d\alpha^c$$

where $L_c(\alpha^c)$ is the product of the likelihood contributions of all mothers in community c, conditional on the community specific effect α^c (but not conditional on α^m):

$$L_c(\alpha^c) = \prod_{i \text{ with } c(i)=c} \int_\infty^\infty \int_\infty^\infty \int_\infty^\infty L_i(\alpha^m, \alpha^c) f_{\alpha^m}(\alpha^m) d\alpha^m$$

Thus the exact likelihood is a product of three-dimensional integrals of functions which are by themselves products of three-dimensional integrals. Our smooth simulated maximum likelihood estimator deals with all three-dimensional integrals for a given community at once, and replaces L_c by the approximation

$$L_c^R = \sum_{r=1}^R L_c^s(\alpha_r^c)$$

with

$$L_c^s(\alpha_r^c) = \prod_{i \text{ with } c(i)=c} L_i^s(\alpha_r^c)$$

and

$$L_i^s(\alpha_r^c) = \sum_{r=1}^R L_i(\alpha_{ri}^m, \alpha_r^c)$$

The draws of α_{ri}^m are taken (quasi-) independent across mothers; the draws of α_r^c are taken (quasi-) independent across communities. Draws of α_{ri}^m and α_r^c are (quasi-) independent. In each case, we use Halton draws to reduce the variance of the simulator.

If $R \to \infty$ faster than $\sqrt{N^c}$, then maximizing $L^R = \prod^{N^c} L_c^R$ over all the parameters gives a smooth simulated maximum likelihood estimator. This estimator is asymptotically equivalent to exact maximum likelihood (see, e.g., Hajivassiliou and Ruud, 1994). Note that in this case, the asymptotics are asymptotics for $N^c \to \infty$, while the number of mothers per cluster does not need to become large. With about 330 communities and only about 30 mothers per community, this seems the most relevant asymptotic approximation for our data.

Appendix 2: Benchmark Simulation

This appendix describes the benchmark simulation of Section 5.6 and Table 6. The benchmark simulation has all mechanisms at work as specified in equations (1)-(4). Observations for the exogenous variables are taken from the data (7286 mothers in 333 clusters, with their ethnicity, their and their partner's education levels, their date of birth, and the date of birth of their first child; all the explanatory variables in the equation for mortality of the first child). The mother specific effects, the neighborhood specific effects, and all error terms in the equations for mortality, birth interval, and the decision to have another child are drawn from their estimated joint distribution.[1]

Dependent variables are then simulated recursively for each mother: the mortality outcome of the firstborn child; the decision to have a second birth or not; if this decision is positive, the birth interval until the second birth; the mortality outcome of the second birth; the decision to have a third birth or not; and so on until the mother decides to have no more births, or until the maximum number of births (14) is reached.

Table A3 presents the benchmark simulation results by birth order. The second and third columns present the frequency distributions of births and neonatal-survivors.. By design, the sample consists of women who gave birth at least once. About 12% have only one birth, 17.2% have two births, etc. About 0.6% of all women have no surviving children – they have one or two births, but all their children die within 30 days. The average number of surviving children (3.82) is 7.4% smaller than the average number of births. Thus the average neonatal mortality rate in this simulation is 7.4%. Column 4 presents simulated mortality by birth order. Neonatal mortality is largest among firstborn children and among children of birth order higher than six. The estimates for high birth orders, however, become rather inaccurate due to small numbers of mothers with so many children; this is also why the Table only presents results up to birth order 10.

[1] The maximum number of births in the data is 14; we thus draw 14 error terms for each equation, 42 in total, although not all of them will actually play a role. (E.g., if the mother decides not to have more children after her fifth birth, errors in birth intervals and mortality equations for the sixth and further children play no role.)

Column 5 presents the average length of the simulated preceding birth interval by birth order. On average, intervals are shorter for higher birth orders. This is primarily a selection effect. In other words, it reflects that fact that women who have high-order births (i.e. high fertility) tend to have shorter birth intervals.

Table A1. Benchmark Simulation

Birth order	Births (frequency, %)	Survivors (frequency, %)	Mortality risk (%)	Av.birth interval (months)
0		0.62		
1	12.01	13.50	9.17	
2	17.22	17.90	6.89	30.90
3	17.17	18.24	5.93	31.04
4	15.26	15.87	6.79	30.73
5	12.63	12.31	6.34	30.42
6	9.44	9.50	5.98	29.89
7	6.60	5.60	8.61	29.17
8	4.63	3.60	10.37	29.65
9	2.24	1.59	10.35	28.07
10	1.54	0.70	8.33	30.24

Appendix 3: Auxiliary Tables

Table A2: Neonatal Mortality of the first-born child (equation 3)

	Parameter	Std error	t-value
Religion:			
Muslim	-0.132	0.103	-1.28
Other	-0.070	0.423	-0.17
Caste:			
scheduled caste	0.139	0.225	0.62
scheduled tribe	0.121	0.094	1.30
other backward caste	-0.031	0.092	-0.34
caste missing	0.056	0.165	0.34
Maternal education:			
incomplete primary	0.108	0.148	0.73
complete primary	-0.119	0.139	-0.86
incomplete secondary	0.063	0.130	0.48
secondary & higher	-0.298	0.160	-1.87
Paternal education:			
incomplete primary	-0.245	0.164	-1.49
complete primary	-0.008	0.114	-0.07
incomplete secondary	-0.093	0.099	-0.94
complete secondary	-0.107	0.113	-0.94
higher than secondary	-0.163	0.116	-1.41
Gender:			
Female	-0.143	0.076	-1.88
Trend effects:			
year of birth of child/10	-0.722	0.814	-0.89
(year/10) squared	0.036	0.048	0.76
Maternal age:			
maternal age at birth/10	-1.340	0.963	-1.39
(age/10) squared	0.250	0.236	1.06
Constant	3.645	3.517	1.04

See Notes to Table 1 in the text.

Table A3: Unobserved Heterogeneity: Underlying parameter estimates

	Parameter	Std error	t-value
p_{mm}	0.283*	0.061	4.67
p_{bm}	0.084*	0.023	3.67
p_{bb}	0.041	0.027	1.49
p_{fm}	-0.091	0.072	-1.27
p_{fb}	0.086	0.103	0.83
p_{ff}	0.110	0.139	0.79
p_{0m}	0.480	0.266	1.80
p_{0b}	-0.345	0.261	-1.32
t_{mm}	0.305*	0.069	4.41
t_{bm}	-0.079*	0.027	-2.95
t_{bb}	0.099*	0.025	3.96
t_{fm}	0.094	0.071	1.31
t_{fb}	-0.372	0.106	-3.51
t_{ff}	0.072	0.150	-0.48
t_{0m}	0.037	0.173	0.21
t_{0b}	-0.235	0.215	-1.09

Notes: Refer section 5.4 of the text.

*: parameter (and marginal effect) significant at the two-sided 5% level

Mother-specific effects are parameterized as follows:

Mortality: $a_{mi} = p_{mm}u_{mi}$;

Birth interval: $a_{bi} = p_{bm}u_{mi} + p_{bb}u_{bi}$;

Fertility: $a_{fi} = p_{fm}u_{mi} + p_{fb}u_{bi} + p_{ff}u_{fi}$;

u_{mi}, u_{bi}, u_{fi} independent standard normal, independent of exogenous variables and error terms. The parameters p_{0m} and p_{0b} are the coefficients of u_{mi} and u_{bi} in the equation for neonatal mortality of the first child.

Community-specific effects are parameterized as follows:

Mortality: $(_{mi} = t_{mm}v_{mi}$;

Birth interval: $(_{bi} = t_{bm}v_{mi} + t_{bb}v_{bi}$;

Fertility: $(_{fi} = t_{fm}v_{mi} + t_{fb}v_{bi} + t_{ff}v_{fi}$;

v_{mi}, v_{bi}, v_{fi} independent standard normal, independent of u_{mi}, u_{bi}, u_{fi}, exogenous variables, and error terms. The parameters t_{0m} and t_{0b} are the coefficients of v_{mi} and v_{bi} in the equation for neonatal mortality of the first child.