Infant Mortality in Rural Bangladesh: State Dependence vs. Unobserved Heterogeneity

Unnati Rani Saha, Ph.D student, Tilburg University, The Netherlands
Arthur van Soest, Professor, Tilburg University, The Netherlands

Abstract:

In view of the millennium development goal to reduce child mortality by two-thirds between 1990 and 2015 (UN, 2003), this paper investigates the dynamic of siblings' death at infancy. Data come from the Health and Demographic Surveillance System (HDSS) in Matlab, Bangladesh in which all demographic events including births, deaths, are recorded monthly. Probit models incorporating survival status of the previous child of the same mother and unobserved mother specific heterogeneity are estimated. The likelihood of infant death is about 32% more if the older sibling died at infancy and the estimates suggest that, in the absence of this "scarring" effect, the infant mortality rate would fall by 7.13% among the second and higher order births in the comparison area. The evidence of scarring is weak (negative) in the treatment area, where health care facilities are better; perhaps learning effects play a role with the available extensive health interventions.

Introduction

By 1993-1994, according to the first Demographic and Health Survey (DHS) in Bangladesh the under-five mortality (mortality before reaching five years old) rate was 133 per thousand live births and infant mortality (mortality before reaching one year old) rate was 87 per thousand live births (reference period 1989-1993). Infant deaths thereby account for 65 percent of all under-five deaths. However, since then Bangladesh recorded a sharp decline in under-five deaths where it is observed 65 deaths per thousand live births in the period 2002-2006 accompanied by five percentage points decline each year. In contrast, infant deaths declined by 2 percentage points (from 87 to 52 deaths per thousands live births) over the same period. Thus, in view of the millennium development goal to reduce under-five mortality in Bangladesh by two thirds between 1990 and 2015 it is an added emphasis for further decline in these rates. There is a considerable research on the determinants of infant mortality for example; mothers with younger, and with less schooling years are at higher risk of their child deaths. Recent demographic data from a wide range revealed that child deaths are clustered in some families and in addition, along with observed characteristics there is substantial unobserved heterogeneity between families (Das Gupta, 1990; Guo 1993; Zenger 1993; Sastry 1997; Arulampalam and

Bhalotra, 2006). There is limited research in developing countries including Bangladesh in this area. Thus, following the recent work on sibling's death-clustering by Arulampalam and Bhalotra 2006 which is a methodological improvement over the previous studies, this paper investigates interfamily heterogeneity in infant deaths and in addition whether there is a causal process at work, whereby the actual event of death of a infant results in a higher risk of death for the next child at infancy (state dependence) in the family.

Health and Demographic Surveillance System (HDSS) in Matlab, Bangladesh with regular collection of prospective data for birth, death and other relevant information eliminates biases that would occur if surveillance was done retrospectively, making the dataset ideal for assessment of the potential effects of covariates on child deaths. HDSS halved into two areas: ICDDR,B and comparison. A general decline in under-five mortality took place until 1990, both areas apart from 1984 the year Shigella epidemic peaked. Since then an impressive decline in under five mortality is observed in 2005 where 45.3 per thousand live births in the ICDDR,B and 60.2 per thousand live births in the comparison area.

After controlling for all observed and unobserved between mother-differences there is evidence of scarring in comparison area. Negative scarring is observed in the ICDDR,B area, where health care facilities are better; perhaps learning effects play a role with the available extensive health interventions. Mother's education plays a bigger role on less likelihood of dying of first-borns if the mother attained secondary or higher levels of education. Conditional on other covariates, we find that boys are more likely to die in the ICDDR,B area whereas no difference reveal in the comparison area. Mother specific unobserved heterogeneity is found significantly high 19 percent in the ICDDR,B area and 8 percent in the comparison area. The second contribution of this paper is to investigates the attributed mother specific variation in infant deaths in the ICDDR,B area which is unique of this paper over other studies on infant deaths.

Background

Related literature

Demographers using data on siblings' death-clustering have long been interested in knowing whether unobserved factors at the family level, such as genetic factors, lead to biased parameter estimates (estimates without accounting for the correlation among deaths of siblings), and spurious correlation (reverse causality), which may have important implications for conclusions concerning policy design. The conventional statistical tools

which the researchers, including DaVanzo et al. 1983, Hobcraft et al. 1985, Pebly et al. 1987 and Koenig et al. 1990, used for studying child mortality, often made the assumption that unobservables in the death risk of consecutive children are independent, and this is incorrect if unobserved heterogeneity plays a role (Guo 1993).

Zenger (1993) discussed different statistical methods for accommodating the correlation structure of the death of siblings. The first approach is to estimate a marginal logistic model, and this approach avoids the problem of misspecification because no assumptions are made about the form of correlation. The second approach is to accommodate the correlation by including the survival outcomes of elder siblings as explanatory variables in the regression model. The extended model is then called a transitional model or Markov model. The third approach is known as random intercept model to allow for correlation assuming that mortality risks vary among families, and to assume the random intercepts follow some probability distribution. However, the models she estimated included either the previous child's survival status or unobserved heterogeneity but, in no case, both.

Guo (1993) and several other studies (Curtis et al. 1993; Sastry 1997a,b; Bostlad and Manda 2001 have included survival status of preceding sibling in the model allowing for unobserved heterogeneity. They did not, however, interpret these effects in terms of causality and correlation. The estimated coefficient on the survival status of the previous sibling was biased in all these studies (Arulampalam and Bhalotra, 2006). Additional studies, including studies based upon data from Matlab, on siblings' death-clustering, discarded the first-born child in the family (known as initial conditions problem (Heckman, a,b,c)) which can result in biased estimates. Consequently, in issues of methodological development, Arulampalam and Bhalotra 2006 estimated models incorporating both previous sibling survival status (as lagged dependent variable) and unobserved heterogeneity and, in addition, interpreted the former in terms of the causal process. They also addressed the issue of initial conditions that arises in dynamics model with unobserved heterogeneity and showed that the null hypothesis of an exogenous initial condition (no correlation between family level unobservable and survival status of the first child) is rejected.

Concept of Death-clustering/State dependence

Death-clustering of siblings is widely noticed in the demographic literature of developing countries, including Bangladesh (Hobcraft et al. 1985, Koenig et al. 1990, Das Gupta 1990,

Sastry 1990, Guo and Rodriguez 1991, Miller et al. 1992, Curtis et al. 1993, Zenger 1993, Guo 1993, Majumder et al.1997, Alam and David 1998, Arulampalam and Bhalotra 2006, Bhalotra and van Soest 2008). It is apparent from the data that sibling deaths are likely to be clustered (positive association) within families. Possible factors explaining this are that siblings share same genetic traits, the mother has similar problems such as premature delivery or intrauterine growth retardation, or maternal inability, and environmental factors, such as poor water supply. Maternal inability relates to the mother's capacity in child care and running household management. For example, Das Gupta 1990 argues that some women are less resourceful in caring for their children and managing household activities.

Death clustering of sibling can also be due to a causal process called state dependence or scarring (Arulampalam and Bhalotra 2006; Bhalotra and van Soest 2008). They conceive the idea of state dependence that the death of one child results in a higher risk of death for the next child and refer to this as *positive scarring*. A further explanation of state dependence is that a child's death leaves the mother depressed as a result of which her subsequent child's health is compromised in both womb and infancy (Steer et al.1992 and Rahman et al. 2004). This is referred as *depression hypothesis*. Another explanation of maternal depression is that women with closely-spaced pregnancies may suffer from nutritional depletion which affects the health of her subsequent children (Gyimah and Rajulton 2004). Short preceding birth interval might be also one underlined causal mechanisms that elevates the risk of the child's mortality in the family through *fecundity hypotheses* or *replacement hypotheses* (Hobcraft et al. 1983, Cleland and Sattar 1984, Koenig et al.1990, Zenger 1993, Miller et al.1992, Da Vanzo and Pebley 1993).

Alternatively, one might also expect negative scarring, in the case of competition for the use of family resources – if the previous child had died, the next child competes with fewer siblings, potentially improving its survival chances. Learning effects may also lead to *negative scarring*. For example, if the older sibling dies because of diarrhoea or acute respiratory illness (ARI), the mother may want to learn how to prevent a death of her next child caused by diarrhoea or ARI.

Data and Methodology

Health and Demographic Surveillance System, Matlab

Since 1966, the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B) has maintained a Health and Demographic Surveillance System (HDSS) in Matlab, a typical rural area located 60 km southeast of Dhaka in which all births, deaths, causes of deaths (through verbal autopsy), pregnancy histories, migrations in and out, marriages, divorces, and socioeconomic status are recorded within 220,000 population.

The ICDDR,B started a Maternal Child Health and Family Planning Programme (MCH-FP) Project in October 1977 in half of the HDSS area, formerly known as MCH-FP area and currently as ICDDR,B area, which enhanced government health services and collected additional data on a range of health indicators – immunization status with specific date, breast-feeding, morbidity status (e.g., Diarrhoea, ARI), causes of death (based on verbal autopsy), MUAC measures for nutrition status. The other half of the area, known as Comparison area, remained under the usual programme of the Government of Bangladesh. Health and Demographic data have been collected systematically through regular household visits (every 2 weeks until January 1998, and once every month since then).

At each birth, the child is registered and the mother is asked about her previous pregnancy histories, including livebirths, gender, deaths, stillbirths which again help to calculate the total number of livebirths including the current birth that the mother has. Further, causes of death which extend to validate the death information, are matched with the pregnancy history of a mother. Pregnancy history variables provides us with all information on the children of a woman if all the births the woman gave took place in the HDSS area and were registered at birth. Alternatively, if a woman migrated out and gave birth outside of the HDSS area and again migrated in with the child at age below five years, this child also is registered (birth date, survival status, etc.) in HDSS. Otherwise, the child's records are not registered in HDSS which sometimes creates incomplete records of a mother in the HDSS.

Study Sample

Among the 142 villages, about half (70) of the villages are within the ICDDR,B and the rest (72) of the villages are in the Comparison area. During the 1990s, two villages were permanently inundated in the ICDDR,B area due to river erosion, and another was shifted from the ICDDR,B area to the Comparison area. We combined both health and demographic surveillance system data from the 70 villages within ICDDR,B and 72

villages within Comparison area obtained from 1 July, 1982 through 31 December, 2005 (the study period).

Excluding still[a] and multiple[b] births, data on 67,696 children from 31,361 mothers and on 74,214 children from 30,264 mothers were recorded during the study period in the ICDDR,B and Comparison areas, respectively.

ICDDR,B area:

To have complete birth information[c], we have deleted 11,930 mothers with 24,508 children's records from a total of 67,696. Finally, deleting 11,930 mothers from the initial 31,361 mothers we have retained 19,431 mothers in our sample with complete information about their all children, giving 43,186 children records in our file.

Due to out-migration[d] of their mothers about 8,224 child records were deleted from 43,186 children's records which gave 34,962 child records of 14,631 mothers. Further, due to incompleteness of birth records (as we have deleted some children as their mothers migrated out with them and some did not which made again incomplete birth histories of some women), 578 mothers were dropped, leaving 14,053 mothers and 33,994 children records in our sample of mothers with complete birth history and who never migrated out.

In addition, 24 children were deleted as they migrated out before completing the first year of life. Thus, again their siblings have been deleted from the file, reducing the number of children's records from 33,994 to 33,931 retained in our sample. As these 24 children came from 24 mothers these mothers again deleted from 14,053, so that the remaining

[a] We eliminated stillbirths as gender is one important covariate in our analysis and data on gender is missing for stillbirths.

[b] We eliminated multiple births as children of a multiple birth face higher odds of dying which has been documented in demographic literature on mortality.

[c] To have complete birth information of a woman (during study period) we have calculated parity (total livebirths given) from the pregnancy history variables. We have checked parity and birth dates of all children. For example, if a mother has parity four this means this mother has had four live births, so the birth dates of four children will be available in the file and this mother will appear four times as giving birth. If this was not the case, we have deleted all children's records of this mother from our file. As study modeled the sequence of children of a mother, we did not include a mother with incomplete birth information.

[d] In aiming to have complete follow up of all children after their born and to avoid the effect of migration we also took the sample who gave all births in MCH-FP area and continued to live in the same area till 2005 at the end of study period, we have excluded the mothers who have migrated out in some point of study period and their children from our study sample. Finally, we will work on this sample for analyzing death clustering in Bangladesh.

sample has 14,029 mothers. No still births were observed among these 24 mothers. Further, due to variation over the period, we have excluded the children of these three villages of ICDDR,B area, and finally worked with a sample of 31,968 children and 13,232 mothers.

Comparison area

To have complete information, we have deleted 12,485 mothers which enforced us to delete about 30,701 children's records from a total of 74,214 and deleting these 12,485 mothers from the initial 30,264 mothers we have retained 17,779 mothers in our sample with complete information on all their 43,5131 children.

Due to out-migration of their mothers, 10,142 were deleted from 43,513 children's records which gave 33,371 records in our file. Thus, by reshaping wide command in Stata, 33,371 children belonged to 12,419 mothers. Furthermore, due to incompleteness of birth records (as we have deleted some children as their mothers migrated out with them and some did not, which led to incomplete birth histories for some women), 546 mothers were dropped from 12,419 which gave about 11,873 mothers and 32,422 children's records with complete birth history and who never migrated out.

In addition, 31 children were deleted as they migrated out before completing the first year of life. Their siblings have also been deleted from the file, which reduces the number of children's records from 32,422 to 32,391 retained in our final sample. As these 31 children came from 17 mothers, these mothers were also deleted from the sample of 11,873 mothers, so that the final sample has 11,856 mothers. No still births were observed among these 17 mothers.

Model Specification

This paper models the propensity of death in infants among Bangladeshi families, allowing for the identification of state dependence (scarring) taking account of the potentially confounding effects of unobserved intrafamily heterogeneity. State dependence refers to the issue whether the survival status of the previous child (t-1) of a family (i) has an influence on the death of their next child (t) at infancy.

Let there be T_i children in family i where i=1, 2,…,N; t=1, 2,…,T_i denotes birth order (the panel will be unbalanced), the unobserved propensity to experience an infant death, y^*_{it} is specified as

$$y^*_{it} = x_{it}\beta + \gamma y_{it-1} + \alpha_i + u_{it} \quad \text{.....(1)}$$

Where, y^*_{it} is the unobserved propensity of infant death. The observed infant death outcome $y_{it} = 1$ if the child's propensity for death crosses a threshold that is $y^*_{it} > 0$; otherwise, if $y^*_{it} < 0$, $y_{it} = 0$. x is a vector of strictly exogenous observed explanatory variables and β is the vector of coefficients associated with x. The term α_i captures unobserved heterogeneity at the family level, which accounts for all unobservable family characteristics including genetic characteristics and variables such as innate maternal ability which influence the index child's propensity to die. The coefficient γ is associated with state dependence – the effect of death in infancy of the previous child on the next child's survival chances - and the null hypothesis of no state dependence implies that $\gamma=0$.

The model assumes that the history of infant deaths among older children other than the immediately preceding child has no effect on $y^*_{it.}$ For example, if child t-2 died in infancy then in our model this will affect the risk of death of child t-1 and, thereby, affect the risk of death of child t, but there is no direct effect on death of child t. This is the first order Marcov assumption (Zenger 1993, Arulapalam and Bhalotra 2006). The risk factors both observed and unobserved all are common to all children and are captured in our model by x_{it} and α_i.[e]

According to the theoretical aspects of econometrics of dynamic (causal) models with unobserved heterogeneity (Hsiao 1986, Wooldridge 2002), the literature on economics with unemployment scarring (Heckman 1981a,b,c, Stewart 2007) and the recent literature on death clustering (state dependence/scarring) of infant deaths in India (Arulapalam and Bhalotra 2006), this paper extends the proposed model in terms of the covariates used in this model and using data from HDSS which are collected prospectively. The covariates entered in the model except access to piped water are collected at each time the mother gave a birth. In this way, this data have an advantage compared to retrospective survey

[e] If the previous child dies would die in infancy after the index child, then the model assumptions would be violated but no such case was observed in our data.

data used by, e.g., Aulampalam and Bhalotra (2006), in terms of time consistency and measurement error.

With these above specifications the conditional probability model of the observed binary outcome of death for an infant t of mother i is given by

$$P[y_{it}/y_{it-1,}\, x_{it},\, \alpha_i\,] = \Phi\,[(x'_{it}\beta + \gamma y_{it-1} + \alpha_i)(2y_{it} - 1)] \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (2)$$

Where, Φ denotes the cumulative distribution function of the standard normal distribution. And, dropping x and the index i for convenience, the joint conditional probability of the observed sequence of binary outcomes given α is

$$P(y_{T,\ldots\ldots\ldots,}\, y_{2,}\, y_1/\,\alpha) = P(y_T/y_{T-1,}\,\alpha)\; P(y_{T-1}/y_{T-2,}\,\alpha)\ldots\ldots\; P(y_2/y_{1,}\,\alpha)\; P(y1/\,\alpha)\ldots \quad (3)$$

It is clear from the sequence above that it is necessary to give a specification for P (y1/ α) (the "initial condition problem" in dynamic models with unobserved heterogeneity (e.g. Heckman 1981)). Modeling the outcome for the first child is especially relevant because the first child shares unobservable traits α_i with its younger siblings. If there were no unobserved heterogeneity α_i, then the initial observation could be treated as exogenous, and the model that is given in equation (1) could be estimated by using the sample of children t (t= 2,…..,T). Alternatively, Hsiao suggested that it the initial condition problem can be ignored even with unobservable heterogeneity if the time dimension of panel (Ti) is large but in our case Ti is the total number of children born in family i, and this is typically small, so that large T asymptotics will not apply. Since correlation between α_i and y_{it-1} makes y_{it-1} endogenous in equation (1), it would probably lead to overestimation of γ (Fatouhi 2005), and thus overstate the state dependence. This is why we specify a separate equation for the risk of mortality of the first-born child (first observation) of each mother. The equation for the process of generating first observations will have the same form as for equation (1) (as explained in Arulapalam 2007) and is given by

$$y^*_{i1} = z'_{i1}\pi + \theta\alpha_i + u_{i1} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (4)$$

Where z_i is a vector of exogenous covariates associated with first the observation. Exogeneity of first child survival corresponds to θ=0 which can be tested in a standard way. The distribution function is same as for equation (1). The probability corresponding to equation (4) is

$$P(y_{i1}/\alpha_i) = \Phi\,[(z'_i\lambda + \theta\alpha_i)(2y_{i1} - 1)] \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (5)$$

Assuming u_{i1} and u_{it} for t>2,…., T are independently distributed following standard normal distributions , and combining the equations (1) and (4) gives a complete dynamic

model for all observations with observed and unobserved heterogeneity at family level i. The conditional probability of the observed sequence of binary outcomes for deaths of infants for family i can be written as

$$P(y_{it} / y_{it-1}, x_{it}, z_i, \alpha_i) = \Phi\{(z'_i\lambda + \theta\alpha_i)(2y_{i1} - 1)\} \prod \{ \Phi(x'_{it}\beta + \gamma y_{it-1} + \alpha_i)(2y_{it} - 1)\} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots(6)$$

and marginalizing the likelihood with respect to the unobserved heterogeneity component α_i gives the following likelihood contribution for family i:

$$L_i = \int \left[\Phi\{(z'_i\lambda + \theta\alpha_i)(2y_{i1} - 1)\} \prod \{ \Phi(x'_{it}\beta + \gamma y_{it-1} + \alpha_i)(2y_{it} - 1)\} \right] f(\alpha_i)\, d\alpha_i \quad (7)$$

Where, $f(\alpha_i)$ is the probability density function of α, which is taken to be normally distributed $(0,\sigma^2_\alpha)$, iid & independent of all family level unobservables. The integral in (7) can be computed using Gaussian-Hermite quadrature (Butler and Moffitt 1982).

The joint random-effects dynamic probit model taking account of initial conditions is non-standard and cannot be estimated using the routines available in standard statistical software. Stewart (2006, 2007) has written stata code for fitting the random-effects dynamic probit model, and we have fitted this model in our data. Our results (presented in Table3) are based on specifying 32 quadrature points.

Empirical Analysis and Results

Description of Variables Used in the Empirical Model

The dependent variable is infant death (y_{it}) and is defined as 1 if the child is observed to die before the age of 12 months and as 0 otherwise. The main interest is in the effect of the lagged dependent variable y_{it-1}, the infant survival status of the preceding sibling, similarly defined as the dependent variable y_{it} . The other explanatory are x_{it} (t>=2) which are the same variables as in z_{i1} in the first child equation.

All child specific covariates are measured at the time of birth: child birth order, gender, and the age of mother at birth of index child; education of the mother is denoted by a set of dummy variables for the years of school attained such as 0, 1-5, 6+ schooling years which might reflect the ability of mother. Similarly, education and occupation of the father are also included with a set of dummy variables, reflecting the family's socioeconomic status.

Birth intervals can be calculated as the time intervals between consecutive birth dates. However, we will not enter birth interval in our model as it is argued that through fecundity and replacement hypothesis, the birth interval as proximate variable weakens the effect of scarring and, thus, the degree of scarring will tend to be underestimated (Arulampalam and Bhalotra, 2006). There are also measurement error (this is perhaps not the case in our data as birth date is recorded at each birth) and endogeneity problem in entering birth interval in the model which needs separate specification for joint-modeling.

The mother's birth cohort also enters the model, giving insight in the trend of scarring over the period. Other family level observable variables included in the model are religion and relationship with household head. These allow for controlling for *sociological* and *intergenerational* relationship influences on the risk of infant death of a family. Source of drinking water piped/tube well also enters the model, to control for environmental factors.

Descriptive Statistics

A profile of both areas ICDDR,B and Comparison is given in Table1. Some figures in Table 1 are sample averages, others are percentages.

The average number of children born per family/mother is 2.42 in the ICDDR,B area and is 2.73 in the Comparison area (not shown in Table). 16 percent of families had more than four children in the ICDDR,B area, compared to 28 percent in the Comparison area. 82.7 percent of all women in the ICDDR,B area and 89.8 percent of the Comparison sample are Muslims. A. higher percentage (Table 1) of women in the Comparison area attended Maktab/Madrasa, academic institutions where religious education is given. However, those who attended Maktab/Madrasa are entered as *no schooling years* against the variable 'schooling years'. Comparison area mothers have less access to environmental factors like hygienic latrine use and source of drinking water tubewell/filter. No differences are observed in average schooling years or mothers' age at birth between the two samples.

A total of 1,694 (4.99 of all births) infant deaths in the sample occurred to 1,471 (10.49) families, and 12,558 (89.51) families had no experience of infant deaths in the ICDDR,B area. Moreover, 0.01 percent of family had lost their all children in infancy. The percent of first born children is 41.4 and the percent of infant death of first born is 6.61, which is higher than the infant death rate of all children (4.99).

In the Comparison sample, 2,180 (6.74% of all births) infant deaths occurred to 1,834 families (15.47% of all families); the remaining 84.53 percent of all families had no experience of infant deaths. Like in the ICDDR,B area, 0.01 percent of all families had lost their all children at infancy, and the percentage of infant death is high among the first born children (8.78% for first born children, 5.55% for other children).

Among families experiencing infant deaths, about 13.19 percentages had more than one death in ICDDR,B area, while this percentage is 26.17 in the Comparison area (not shown in the table).

Table 2 shows the raw probabilities of infant death conditional on the survival status of the preceding sibling. Explaining this is the primary goal of this paper. The probability of infant death is higher by 0.0442 (i.e. it is 0.0796 rather than 0.0354) if the preceding sibling died as an infant in the ICDDR,B area, and by 0.0507 in the Comparison area (0.107 rather than 0.0510). In other words, the likelihood of infant death is 2.25 times higher in the ICDDR,B area and 2 times higher in the Comparison area if the preceding sibling died than if it survived.

Estimation Results

Several dynamic random effects probit models that incorporate the lagged dependent variable (survival status of the previous sibling) and unobserved heterogeneity are estimated. The first (Model 1) includes only the status of previous siblings' death; the second (Model 2) adds both child-level and mother-level factors, and the third also includes father-level factors (Model 3). In Models 2 and 3, the covariates in the equation for the first-borns include family-level characteristics and the gender of the first child.

The results are presented in Table 3a (equation for children of birth order larger than one) and Table 3b (equation for the first born child). The results of Model 1 with only the lagged dependent variable (with parameter γ) show that the death of an immediate older sibling had a positive and significant effect (p=0.001) on the conditional probability of infant death in the comparison area whereas a positive but insignificant effect (p=0.401) is observed in the ICDDR,B area (Table 3a).

The partial effect of y_{it-1} on $P(y_{it}=1)$ can be derived from the estimates by constructing counterfactual outcome probabilities $p_{0,}$ $p_{1,}$ taking y_{it-1} as fixed at 0 and 1, and evaluated at

the overall mean of the exogenous variables ($x_{it}=x..$). The difference between p_0 and p_1 can be interpreted as average partial effect (APE); the ratio of the two is the predicted probability ratio (PPR) (Stewart 2007:522). The average partial effect is then called state dependence or scarring which is about 2.16% in the comparison area whereas it is less than 1% in the ICDDR,B area (see Table 3c). An alternative interpretation of state dependence (from PPR) is that the likelihood of infant death is about 42% more if the older sibling died at infancy in the comparison area and about 14% in the ICDDR,B area.

Model 2, including child and mother-level variables, reduces the parameter estimate of γ and its significance level ($p=0.04$) in the comparison area. However, model 3 shows an increase compared to model 2 both in the estimate of γ and its significance level ($p=0.01$). Thus, it is revealed that covariates used in the models explain the effect of previous sibling's death differently in the comparison area (Table 3a). Whereas, in the ICDDR,B area, adding the regressors in models 2 and 3 leads to small negative and insignificant estimates of the effect of previous sibling's death. All the three models in the ICDDR,B area find an insignificant effect of previous sibling's death (Table 3a).

Furthermore, the predicted probability ratios (PPR) in Table 3c show that according to model 3, the likelihood of infant death in the comparison area is higher by 31% if the previous child died at infancy than if it was alive. This rate is less than the estimate of Model 1, due to including the covariates. In the ICDDR,B area, negative scarring was observed which means a mother's experience with child death reduces the risk of death of her next child, and this association is not significant.

The part-b of Table 2 explains the estimation results of dynamic random effect model 3. Comparing the estimated average partial effect (APE) probability p_0 reported in part-b of Table 2 with the average partial effect (APE) raw data probability p_0 reported in part-a of Table 2 provides an estimate of the percentage of raw persistence (clustering) which explains about 32% of the clustering observed in the comparison area. Similarly, in the ICDDR,B area about 4% negative scarring effect was observed (row 11, Table 2).

Further, comparing the predicted probability of infant's death (excluding first-borns) with that of the predicted probability of infant's death when previous sibling was alive (setting $\gamma=0$) offers an estimate of the reduction in mortality. This reduction would be achievable if only scarring were eliminated ($\gamma=0$). The estimates suggest that, in the absence of scarring,

the infant mortality rate among children who are born after the first would fall by 7.13% in the comparison area (row 13, Table 2).

We can use the estimates of this dynamic model to test whether the initial period outcome (survival of the first child) within a family can be treated as exogenous. If $\theta=0$ in equation (4), then unobservables in the equation for the first observation are uncorrelated with unobservables in the (dynamic) equation for subsequent observations, and in this case, no need for the specification of separate equation for the first observations (Stewrat, 2006; Arulampalam and Bhalotra, 2006). In our case, the null hypothesis $\theta=0$ is rejected significantly ($p<0.001$) across all the models in both areas of ICDDR,B and comparison (Table 3b). This test thus confirms the importance of specifying a distinct reduced form equation (4) for the first child that is estimated jointly with the dynamic equations for other children.

The proportion of variance that is attributable to family-level unobservables α_i is estimated to be 8% in the comparison area and 19% in the ICDDR,B area. The estimates decisively reject the null hypothesis of no family-level unobservables in both areas (Table 3b). Further, in the pooled probit model which ignores family-level correlation we found that the effect of previous sibling's death was overestimated: the estimate of γ parameter was higher (0.2917) than the estimate of γ parameter in the dynamic model 3 (0.1442), in the comparison area. In the ICDDR,B area, this difference was even larger (0.3332 vs -0.0242). However, the results from the random effects probit model cannot be directly compared with the results from the pooled probit model because they use different normalizations: while the former uses a normalization of $\sigma^2_u=1$, the pooled probit model uses $\sigma^2_v=1$. For comparisons, the random effects probit model estimates should be rescaled by an estimate of $\sigma_u/\sigma_v=\sqrt{1-\lambda}$ (Stewart 2007; Walter 2008).

For example, the rescaled estimate of γ for model 3 in the comparison area was 0.138. The effect of the lagged dependent variable was more than 50% higher (overestimation) in the pooled probit model compared to the dynamic model, showing the importance of controlling for α_i. However, in the ICDDR,B area, the rescaled estimate of γ for model 3 was -0.0216 which is lower than the γ estimate of pooled probit model (changes from positive effect to negative effect) (0.3332 vs -0.0216), and again this finding hints on the importance of controlling of α_i in the analysis.

Some of the covariates x_{it} used in the dynamic model are estimated to be significant determinants of infant deaths in both areas (Table 3a). For example, the likelihood of death for the children after first born-child falls with the schooling years of father. In this case, the mother's education plays no significant role keeping the father's education constant, (model 2 vs model 3). However, in model 3, the mother's education plays a significant role in reducing the infant's death for the first-born child over the father's education. Father's occupation also makes different in the likelihood of dying for the children after first-born. A day labourer more likely to experienced with infant deaths. Significantly boys are more likely to die among first-born child in ICDDR,B area where no gender difference is observed in the comparison area. The reference category of birth cohort is the cohort of mothers who were born before 1966. Compared to the reference category, later birth cohorts have significantly smaller likelihood of infant death for all children, both areas. Those who used tube well or pipe water as a source of drinking water are less likely to see their children die in infancy die but this finding is significant in the ICDDR,B area only. It is important to note here that the information on drinking water is collected periodically.

Discussion

Policymakers and planners in Bangladesh are concerned about further decline in under-five mortality by two thirds between 1990s and 2015. Although an impressive decline with a wide variation by administrative areas is observed in under-five mortality, Bangladesh runs the risk of failing in its effort to achieve the Millennium Development Goal 4. Evidence of recent demographic literature some families experience multiple child deaths while some experience none. Thus with an ideal demographic data laboratory in Matlab, this is a renewed interest in research and policy design to explain this phenomenon in this area. This paper contributes new insight into the determinants of infant mortality that the event of child death creates a dynamic that makes further children of the same mother more vulnerable to early death. Separating causality from correlation in this area has important implications for policy and for research on the inter-relations of family behaviour and mortality.

We find a significant *positive scarring* effect in the comparison area. The likelihood of infant death is about 32% more if the older sibling died at infancy and, further, the estimates suggest that, in the absence of scarring, the infant mortality rate would fall by 7.13% among the second and higher order births in the comparison area implies social multipliers activated in this area. Thus, policies targeted at reducing childhood mortality

are important to avoid the death of subsequent siblings. The evidence of scarring is weak (negative) in treatment area; perhaps learning effect plays role with available extensive health interventions. Arulampalam and Bhalotra also found a weak scarring in few states for example Punjab which is richest, and Kerala which is socially the most advanced (Arulampalam and Bhalotra, 2008). Another explanation of weak scarring might be due to the settings of low fertility and low mortality in ICDDR,B area because the ability to measure the clustering of mortality risks is much greater in settings with high fertility and high mortality (Sastry, 1997).

Learning effects activated in the ICDDR,B area might be another explanation is that it has extensive maternal child health and family planning (MCH&FP) interventions where mothers experiencing infant deaths are more skilled to handle how to prevent a death with available resources, such as health interventions, health workers, and health clinics. However, mothers of the comparison area have no access to improved health services as they are under the usual government health systems and, thus, are less resourceful with health information. Mothers of the ICDDR,B area are routinely visited by the community health workers which helped them to be resourceful with knowledge and health information. Again, a positive and significant scarring in neonatal deaths in the ICDDR,B area confirms learning effect because neonatal deaths perhaps due to genetic traits as discussed before.

The mother-level variation in infant deaths is revealed high 19 percent in the ICDDR,B area compared to the comparison area 8 percent. This variation can be explained thus: as ICDDR,B interventions are phased out at different times in different blocks, mothers receive health services differently which might make a difference in the expected outcome of child health. Another explanation is that some mothers receive health information and can retain it more compared to others. Education might be a causal process, involved to keep them well-informed with updated health information. Besides these two points, distance from the health centres (health clinics) might also play a role as the ICDDR,B health facilities are located in different locations. Detail investigations need for further clarification of such variation. However, in absence of available health services in the comparison area education might play a role for this variation in infant deaths as evident from the results of this study.

Comparison of other covariate effects between areas offers some interesting new insights. The likelihood of death for the children after first born-child falls with the schooling years of father. In this case, the mother's education plays no significant role keeping the father's education constant. However, the mother's education plays a significant role in reducing the infant's death for the first-born child over the father's education. A day labourer more likely to experience with infant deaths, and a similar finding is revealed from other study (D'Souza et al., 1982). This finding might reflect the association high mortality and poor socioeconomic condition with insecure household income. While it diminishes the gender differentials in child mortality this paper gives a new insight where boys are more likely to die in ICDDR,B area. In contrast, no difference by gender is observed in comparison area.

In context of methodological improvement the test of hypothesis of no family level unobservables that is $\theta=0$ is rejected significantly ($p<0.001$) across all the models in both areas of ICDDR,B and comparison which confirms the importance of specifying a distinct reduced form equation (4) for the first child that is estimated jointly with the dynamic equations for other children. Alternatively, the effect of the lagged dependent variable overestimated in the pooled probit model compared to the dynamic model, showing for further importance of controlling for unobserved heterogeneity α_i in the family level.

Table 1. Descriptive Statistics

Variables	Area	
	Treatment (ICDDR,B)	Comparison
% of infant deaths (all live-births)	4.99	6.74
% of infant deaths excluding first-borns	3.85	5.55
% of infant deaths among first borns	6.63	8.78
Mean age of mother at first birth	21.16 (3.23)	21.08 (3.21)
Mean age of mother at birth	24.70 (5.03)	24.58 (4.89)
% women who never used any method of contraception	2.55	13.37
Mean years of schooling	3.31	3.05
% women no schooling	48.40	50.50
% women attending maktab/madrasa education‖	21.0	36.82
% women Muslim	82.71	89.85
Total children per mother		
% women with 1-2 children	35.02	26.84
% women with 3-4 children	49.50	45.51
% women with 5 or more children	15.48	27.65
% families with no infant deaths	89.51	84.53
% families in which all births die in infancy	0.009124	0.012989

% first-born children	41.35	36.63
% family drinking water source: tubewell/filter	87.12	76.91
Number of mothers in sample ++	13,232	11,856
Number of children in sample +++	31,968	32,366

+ According to events in 2002 published in Scientific Report No. 91 – September 2004, HDSS

++ Sample mothers are who continue living in Matlab (never migrated out) since 1982 June to 2005 December after given first birth

+++ All births given in Matlab MCH-FP area during 1982 June to 2005 December and all births are observed upto one year age of their born for calculating infant deaths. Single births are accounted in our sampel

⇊ These women included with % of women with no schooling

⫩ Still births are excluded from the children sample in both areas which might increase birth interval.

In MCH-FP area, 3,039 women on sample women had still births and 79.47% of these had one still birth and the rest had more than 1 still births ranging 2 to 6 in their birth history. Altogether, these women constituted 3,824 still births which have been excluded from the birth sample.

In comparison area, 3,334 women on sample women had still births and 75.14% of these had one still birth and the rest had more than 1 still births ranging 2 to 7 in their birth history. Altogether, these women constituted 4,437 still births which have been excluded from the birth sample.

Table 2. Clustering and scarring in sibling infants deaths

Estimates	**Area**	
	Treatment (ICDDR,B)	Comparison
(a) **Raw data**		
1 Incidence of infant death	0.0500	0.0674
2 Incidence of infant death excluding first-borns	0.0385	0.0555
3 Probability ($y_{ij}=1/y_{ij-1}=1$), $p_1^{\bullet}$	0.0796	0.1017
4 Probability ($y_{ij}=1/y_{ij-1}=0$), $p_0^{\bullet}$	0.0354	0.0510
5 Persistence due to y_{ij-1} (difference measure) (row 3- row 4), APR	0.0442	0.0507
6 Persistence due to y_{ij-1} (ratio measure) (row 3/row 4), PPR	2.2486	1.9941
(b) **Model estimates (Model 3)**		
7 Probability ($y_{ij}=1/y_{ij-1}=1$), $p_1^{\bullet}$	0.0345	0.0685
8 Probability ($y_{ij}=1/y_{ij-1}=0$), $p_0^{\bullet}$	0.0362	0.0521
9 Persistence due to y_{ij-1} (difference measure) (row 7- row8), APE	-0.0017	0.0164
10 Persistence due to y_{ij-1} (ratio measure) (row 10/row 11), PPR	0.9533	1.3148
11 % raw persistence explained (row 9/row 5)	-3.85	32.35
12 Predicted probability of infant death excluding first-borns	0.0386	0.0561

- p_1 is computed using the estimated marginal predicted probably of y_{it} for each observation under the condition previous sibling died at infancy ($y_{it-1}=1$) and then averaging over all observations excluding the first borns. (part b, Table 2)
- Similarly, p_0 is obtained as setting $y_{it-1}=0$ (part b, table 2)
- p_1 is the observe probability of infant deaths conditional on previous sibling died at infancy. (part 1, table 2)
- Similarly, p_0 is obtained as the observed probability of infants death conditional on previous sibling survived at infancy (part a, table 2)

13 % reduction in mortality if $\gamma = 0$ (with respect of row 12) 1-(row8*/row 12)*100	-	7.1301
14 Variance of family level heterogeneity (standard error)	0.1966 (0.0412)	0.0839 (0.0278)
15 % variance explained by family level heterogeneity	19.78	8.37
Number of mothers in sample	13,232	11,856
Number of children in sample	31,968	32,366

Table 3a. Results of Dynamic Random Effects Probit models for siblings deaths at infancy in Matlab, Bangladesh (child>1)

Covariates	Treatment (ICDDR,B) area			Comparison area		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Previous sibling died (γ)	0.0646 (0.0770)	-0.0194 (0.0832)	-0.0242 (0.0833)	0.1875 (0.0577)	0.1270 (0.0621)	0.1442 (0.0613)
Male		0.0475 (0.0378)	0.0452 (0.0378)		0.0144 (0.0304)	0.0139 (0.0303)
Birth order		-0.0158 (0.0947)	-0.0043 (0.0950)		-0.1275 (0.0541)	-0.1348 (0.0524)
Birth order square		-0.0003 (0.0127)	-0.0010 (0.0127)		0.0166 (0.0064)	0.0181 (0.0061)
Mother's age at birth		-0.0111 (0.0059)	-0.0098 (0.0059)		-0.0671 (0.0338)	-0.0217 (0.0050)
Muslim		-0.0261 (0.0527)	-0.01610 (0.0532)		-0.0818 (0.0510)	-0.0718 (0.0507)
Schooling years (Mother) 1-5 years		-0.0893 (0.0493)	-0.0593 (0.0508)		-0.0282 (0.0390)	0.0063 (0.0402)
Schooling years (Mother) 6+ years		-0.1605 (0.0601)	-0.5302 (0.0658)		-0.1597 (0.0517)	-0.0891 (0.0552)
Mother's birth cohort						
1966-1970		-0.0175 (0.0517)	-0.0114 (0.0519)		-0.1631 (0.0406)	-0.1535 ((0.0406)
1971-1975		-0.1589 (0.0614)	-0.1695 (0.0614)		-0.2970 (0.0491)	-.3014 (0.0488)
1976+		-0.1422 (0.0716)	-0.1571 (0.0716)		-0.5457 (0.0623)	-0.5411 (0.0622)

Source of drinking water: pipe water		-0.1918 (0.0588)	-0.1799 (0.0589)		-0.0279 (0.0398)	-0.0280 (0.0395)
Schooling years (Father) 1-5 years			0.0652 (0.0482)			-0.0416 (0.0388)
Schooling years (Father) 6+ years			-0.1761 (0.0636)			-0.1347 (0.0491)
Father's occupation Day labourer			0.1454 (0.0509)			0.0871 (0.0393)
Constant	-1.9807 (0.0467)	-1.3802 (0.1931)	-1.4898 (0.1995)	-1.7209 (0.0278)	0.0275 (0.4508)	-0.6037 (0.1469)

*** Standard errors are in parenthesis

*** reference category of categorical variables used in the model: female, non-muslim, no schooling years, not pipe water, not day labourer, mother born before 1966.

Table 3b. Results of Dynamic Random Effects Probit models for siblings deaths at infancy in Matlab, Bangladesh (child=1)

Covariates	**Treatment (ICDDR,B) area**			**Comparison area**		
	Model 1	**Model 2**	**Model 3**	**Model 1**	**Model 2**	**Model 3**
Male		0.1226 (0.0376)	0.1224 (0.0375)		0.0654 (0.0342)	0.0655 (0.0342)
Mother's age at birth		-0.1009 (0.0387)	-0.0995 (0.0386)		-0.1546 (0.0356)	-0.1514 (0.0357)
Mother's age at birth square		0.0017 (0.0008)	0.0016 (0.0008)		0.0029 (0.0008)	0.0028 (0.0008)
Muslim		-0.0115 (0.0489)	-0.0106 (0.0492)		-0.0162 (0.0569)	-0.0117 (0.0572)
Schooling years (Mother) 1-5 years		-0.2023 (0.0486)	-0.1951 (0.0501)		-0.1515 (0.0443)	-0.1396 (0.0457)
Schooling years (Mother) 6+ years		-0.3433 (0.0519)	-0.2998 (0.0570)		-0.3294 (0.0485)	-0.3153 (0.0528)
Mother's birth cohort						
1966-1970		-0.1457 (0.0578)	-0.1429 (0.0580)		0.01588 (0.0568)	0.0295 (0.0572)
1971-1975		-0.1827 (0.0605)	-0.1809 (0.0616)		0.0257 (0.0598)	0.0538 (0.0615)

1976+		-0.4152	-0.4162		-0.1430	-0.1102
		(0.0631)	(0.0640)		(0.0619)	(0.0641)
Source of drinking water:		-0.0336	-0.0313		-0.0812	-0.0785
Pipe water		(0.0521)	(0.0522)		(0.0422)	(0.0423)
Schooling years (Father)			0.0643			-0.0195
1-5 years			(0.0465)			(0.0427)
Schooling years (Father)			-0.0783			0.0127
6+ years			(0.0539)			(0.0485)
Father's occupation			0.0328			0.0818
Day labourer			(0.0452)			(0.0421)
Constant	-1.6433	0.0953	0.0497	-1.4011	0.7771	0.6666
	(0.0550)	(0.4607)	(0.4643)	(0.0321)	(0.4240)	(0.4290)
Log-likelihood	-6238	-6101	-6087	-7879	-7702	-7694
Rho	0.1684	0.2018	0.1966	0.0977	0.0935	0.0839
	(0.0369)	(0.0411)	(0.0412)	(0.0241)	(0.0280)	(0.0278)
Theta	0.9779	0.8504	0.8564	0.8054	0.7909	0.8407
	(0.2576)	(0.2060)	(0.2125)	(0.2748)	(0.3045)	(0.3458)

Table 3c. Probability outcomes calculated from the estimated results of dynamics random-effects models 1, 2 and 3

Probabilities	Treatment (ICDDR,B) area			Comparison area		
	Model 1	Model 2	Model3	Model 1	Model 2	Model 3
P_0	0.0354	0.0369	0.0362	0.0511	0.0532	0.0521
P_1	0.0403	0.0355	0.0345	0.0726	0.0676	0.0685
APE: $p_1 - p_0$	0.0049	-0.0014	-0.0017	0.0216	0.0144	0.0164
PPR: p_1/p_0	1.136	0.9628	0.9533	1.422	1.271	1.315

Annex:

Table 4. Pooled results of Dynamic Random Effects Probit models for siblings' deaths at infancy in Matlab, Bangladesh (child>1)

Covariates	**Treatment (ICDDR,B) area**			**Comparison area**		
	Model 1	**Model 2**	**Model 3**	**Model 1**	**Model 2**	**Model 3**
Previous sibling died (γ)			0.3332			0.2917
			(0.0550)			(0.0440)

Male			0.0380 (0.0341)			0.0140 (0.0290)
Birth order			0.0377 (0.0866)			-0.1267 (0.0497)
Birth order square			0.0006 (0.0115)			0.0197 (0.0058)
Mother's age at birth			-0.0130 (0.0052)			-0.0223 (0.0048)
Muslim			-0.0189 (0.0462)			-0.6755 (0.0472)
Schooling years (Mother) 1-5 years			-0.0456 (0.0439)			0.0125 (0.0375)
Schooling years (Mother) 6+ years			-0.0138 (0.0569)			-0.0692 (0.0518)
Mother's birth cohort						
1966-1970			-0.0096 (0.0443)			-0.1480 (0.0376)
1971-1975			-0.1355 (0.0528)			-0.2824 (0.0452)
1976+			-0.1198 (0.0624)			-0.5078 (0.0578)
Source of drinking water: pipe water			-0.1562 (0.0517)			-0.0298 (0.0373)
Schooling years (Father) 1-5 years			0.0629 (0.0416)			-0.0452 (0.0363)
Schooling years (Father) 6+ years			-0.1499 (0.0551)			-0.1310 (0.0461)
Father's occupation Day labourer			0.1342 (0.0449)			0.0877 (0.0371)
Constant			-1.3801 (.1766)			-0.5796 (0.1389)
Log likelihood			-2995			-4269

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