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ABSTRACT

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This paper examines the relationship between early childbearing and child mortality in Bangladesh, a country where adolescent childbearing is of particular concern. We argue that effective use of specific health inputs could however significantly lower child mortality rates even among adolescent women. This offers an attractive policy option particularly when compared to the costly alternative of delaying age at marriage. In particular, we find that women having early childbirth tend to use health inputs differently from all other women. After correcting for this possible selectivity bias, the adverse effects of early childbirth on child mortality are reversed. The favourable effects of use of health inputs however continue remain statistically significant.

JEL Classification: D13, I12, O15

Keywords: family formation, adolescent childbearing, hospital delivery, child vaccination, child mortality, selectivity bias, unobserved heterogeneity, correlated estimates

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Early Childbirth, Health Inputs and Child Mortality:

Recent Evidence from Bangladesh

1. Introduction

Despite the substantial decline in child mortality rates in Bangladesh over the last two decades or so [e.g., see Bairagi, Sutradhar and Alam (1999) among others], child mortality continues to remain a major problem in Bangladesh: infant mortality rates in 1996 – 97 were as high as 100 per thousand births (compared to 79 in India, 31 in China and 18 in Sri Lanka in 1992) and under-five mortality rates were even higher at 130 per thousand births. In recent years adolescent childbearing has also emerged as an issue of increasing concern in Bangladesh. Early marriage combined with low levels of contraceptive use has resulted in adolescent child birth with high risks of both maternal and infant mortality. In fact child mortality rates are more than double for adolescent mothers (see Table 1).

Improving child health is one of the important challenges in the battle against poverty. There is a large literature on child mortality in low-income countries that offers a range of policy options that have the potential to improving child health. These include increased contraceptive use, increased duration between births (birth spacing), parental (especially mother's) literacy, household income and/or use of health inputs. Unfortunately many of these policy options are not particularly relevant to fighting problems of adolescent child bearing as adolescent mothers are more likely to be drop outs from schools, do not have a steady job and often suffer from financial difficulties. Young mothers are more likely to suffer from various reproductive health problems and may not be knowledgeable enough to adequately care for her child. There is a limited literature on teen-age pregnancy in developed countries that focuses on the problems of dropping out from school, increased substance abuse and welfare

dependence [see for example Senderowitz and Paxman (1985) and Geronimus and Korenman (1992)]. Problems of teen-age pregnancy take a new dimension in the context of low-income countries as it is often associated with high child mortality as well though these issues remain unexplored.[1]

In the context of developing countries, therefore policy makers need to devise alternative policies to protect the interests of these younger yet high risks mothers. Possible policies include use of contraception, incentives to girls to complete secondary school or introduction of laws relating to minimum age at marriage. Policies of this nature have been introduced in a number of developing countries but have so far been met with limited success. For example, scholarships for secondary education among girls from poor background has been introduced in selected localities in Bangladesh in 1994 and found some immediate effect on the timing of marriage among girls though its long-term effects will only be realised when benefits of girls' education become more evident to society. Moreover these incentive schemes will be more effective when problems of frequent teachers absenteeism, lack of education materials or discriminatory behaviours of teachers and peers are tackled with a view to improve quality of existing schools [Chaudhury, Hammer, Kremer and Rogers (2006)]. Furthermore, benefits of delayed marriage in Bangladesh come at a cost in the form of substantially increased dowry [by about 40% for each additional

[1] Previous research has suggested that there is a strong relationship between mother's age at birth and child mortality rates. In particular, the literature predicts a u-shaped relationship between the age at the time of child birth and child mortality. Biologically speaking, early or late childbearing may be detrimental to the health of the fetus because of impaired functioning of a woman's reproductive system. Evidence from the National Family Health Survey 1998-99 data set from neighbouring India [Pandey, Choe, Luther, Sahu and Chand (1998)] shows that mortality rates are lower for children born when their mother was aged 20 – 29, compared to children that were born to adolescent/teenage mothers or children born when their mother was more than 30 years old. Using the 1999-2000 Demographic Health Survey data from Bangladesh we do not find any evidence of a pronounced u-shaped relationship between mother's age at birth and child mortality (see Table 1). Instead we find that child mortality rates are higher when the age of the mother at the time of birth is less than twenty but these rates tend to stabilise beyond the age of 20. Accordingly, in this paper we focus on the effects of adolescent childbirth on child mortality and ignore the effects of late childbirths. In either case, there appears to be a substantial potential for reducing child mortality by designing policies aimed at these high-risk adolescent women

year delay in marriage; Field and Ambrus (2005)].[2] In this paper we instead focus on a simpler and more cost-effective policy option and that is to promote use of available health inputs among adolescent mothers with a view to tackle child mortality among this group of high-risk women. While this is an obvious policy choice, its effectiveness for child mortality among adolescent mothers remains (to the best of our knowledge) unexplored.

Our analysis is based on household-level data from the recent round of Bangladesh Demographic Health Survey (DHS) 1999-2000 (see Section 2 for data description). We find that adolescent women tend to use health inputs differently from all other women and also that the adverse effects of early marriage and early childbearing on child mortality could be reduced, at least to some extent, if these high-risk women made use of the available health inputs.

Underlying explanations of this argument could be quite complex and intertwined. One possibility is that women who become mothers in their adolescence are more likely to be less educated and have a number of intrinsic disadvantages. For example, they might have less information about the advantages of using available health inputs (for example, hospital delivery or a range of vaccinations) and/or might even have little say in aspects of female/child health care, especially if they need to travel some distance to avail of the facilities.

There could be other possibilities as well. It is well documented that there are adverse physical/health consequence of early child bearing for both the mother (for example anaemia, haemorrhage, sepsis, preclampsia, obstructed labour) and the baby (e.g., low birth-weight, malnutrition, early death) many of which are private information to the woman and remain unobserved to the researcher. In consequence

[2] On a more positive note, they also find that each additional year that marriage is delayed is associated with 0.30 additional years of schooling and 6.5% higher probability of literacy. Delayed marriage is also associated with a significant increase in the use of preventative health care services.

there are some important selection issues that need to be addressed. Women who experience adolescent childbirth and the women who choose to deliver their child in a hospital or choose to vaccinate their children might not be a random subset of all women in the sample (women who have had at least one child in the five year period prior to the survey). It is possible that young women experiencing complications in pregnancy are more likely to go to the hospital for delivery and/or vaccinate the child, which in turn, may affect the child health outcomes. Similarly, while a young mother may end up having home delivery, given private health information, she might choose to vaccinate the child. Thus the effects of adolescent child birth on child mortality may be closely correlated with the decisions to use available health inputs. Econometrically this implies that the unobserved error terms in the mortality, early childbirth, hospital delivery and child vaccination equations could be correlated; in other words, conventional single-equation child mortality estimates (from probit or hazard equation), which includes early childbirth, hospital delivery and child vaccination, among other explanatory variables, could suffer from an endogeneity bias.

The standard approach to address this endogeneity bias has been to estimate a mortality equation with instruments for early birth, hospital delivery and child vaccination. It is however difficult to obtain good instruments and in order to avoid the pitfalls associated with poor instruments we estimate child mortality, the mother's age at birth and the use of health inputs as a recursive system of equations with (different) mother-specific fixed-effects (unobserved heterogeneity) in each of these four equations.[3] Identification of the system is ensured by virtue of recursivity as well as inclusion of fixed effects [see Chamberlain and Griliches (1975)]. We allow for

[3] See Brien and Lillard (1994), Lillard and Willis (1994), Panis and Lillard (1994), Brien, Lillard and Waite (1999) and Upchurch, Lillard and Panis (2002), Makepeace and Pal (2007) for more on the methodology. We also discuss the econometric methodology in detail in section 3 below.

correlations between each pair of common mother-specific fixed effects (i.e., unobserved heterogeneity), which in turn enable us to remove the implicit bias resulting from these correlations (see discussion in section 3). In other words, our approach to address the endogeneity bias has been to include the source of endogeneity (i.e., cross-correlations) in the relevant equations.[4]

We also compare the correlated estimates obtained from the recursive system of equations with a whole range of alternative single-equation estimates: fixed effects logit, random effects probit, instrumental variable probit. We also re-estimated the correlated model after excluding the common mother/household specific variables, some of which could be correlated with common mother/household specific unobserved heterogeneity. The single-equation mortality estimates (fixed or random effects) suffer from an endogeneity bias while the correlated estimates are qualitatively similar to the instrumental variable probit estimates that address the possible selection bias. These correlated estimates are also quite robust to the choice of different specifications (e.g., with/without household-specific characteristics) and samples (all women, women with at least two children).

2. Data and Descriptive Statistics

The analysis is based on the Bangladesh DHS 1999-2000 data set. The survey collected information on use of health inputs (e.g., hospital delivery, child vaccination) for children born in the last five years preceding the survey date. We use this sub sample (which includes 6832 children born to 5194 women in this sample) to

[4] Our analysis however does not account for the possible correlation between child-specific unobserved error terms in our system that could also generate some inequality among siblings born to the same mother. To the best of our knowledge, Rosenzweig (1986) is the only paper that attempts to identify this kind of correlation using an instrumental variable method derived from events in the prenatal period. Unfortunately this kind of information is not available in our data-set. However our analysis in section 4.2 highlights the presence of this correlation and an attempt to account for this by including some interaction terms.

analyse the likelihood of a child dying before reaching his/her fifth birthday. This allows us to distinguish between biological and other socio-economic factors affecting child mortality.

Adolescent (teenage) childbearing is widely prevalent in our sample: 76% of the first-born children were born to women before their 20th birthday. Early child bearing is often associated with higher than average mortality rates. In our sample, mortality rate for children born to adolescent mothers is 10.4% (the corresponding number for the full sample is 7.4%). 27% of adolescent mothers (41% of all) had prenatal check-up with a qualified health professional; 93% of adolescent mothers (79% of all) had home delivery; 48% of children born to adolescent mothers (45% of all) had never been vaccinated.

In Bangladesh there is a great deal of variation between the provinces in terms of availability of health services and also expenditure on health services and facilities. The latter could partly explain the inter-regional variation in child mortality in the country that is evident in our sample (see Table 2A). Table 2B further illustrates the extent of inter-regional differences with respect to access to sanitary latrine, safe drinking water, and rate of immunizations as well as government expenditure on health services per capita. In particular Table 2B indicates a bias in the distribution of various health services in favour of Dhaka division as against relatively poorer region of Rajsahi and Sylhet, for example (see further discussion in section 4.2).

Next we identify the socio-economic characteristics of parents experiencing adolescent childbirths from those who did not. Table 3 summarises the differences in religion, literacy and women's say in various family decisions for these two groups of couples. Muslim women are 7% more likely to experience early childbirth compared to Hindu women, which might reflect cultural aversion towards contraceptive use

among Muslims in general. Parental literacy levels, especially mother's literacy levels are lower for couples experiencing early childbirth. Clearly less educated women are more likely to have early childbirth. The latter is reiterated in the women's say in female/child health care decisions. In particular, about 7-8% less women experiencing adolescent childbirth have any say in female/child health care decisions.

3. Estimation Methodology and Explanatory Variables

The main variable of interest in our analysis is child mortality. The unit of analysis is a child i born to a particular woman j. Remember that there may be multiple children born to the same mother during the period under consideration (that enables us to identify the mother/household specific unobserved effects). The estimating equation for child mortality is specified as follows:

$$CHDEAD_{ij}^{*} = \beta X_{cij} + u_{cij} \qquad (1)$$

where $CHDEAD_{ij}^{*}$ is the propensity of child mortality. However, $CHDEAD_{ij}^{*}$ is not observed and what we observe instead is

$$CHDEAD = \begin{cases} 1, \text{ if the child is dead at the time of the survey} \\ 0, \text{ otherwise} \end{cases}$$

Here $\mathbf{X}_{cij} \equiv \left(Z_{cij}, X_{cj}\right)$ is the vector of individual $\left(Z_{cij}\right)$ and parental/household and other characteristics $\left(X_{cj}\right)$ that can potentially affect child mortality. We model child mortality as a probit equation.[5] The unmeasured determinants of child mortality can be divided into two parts:

$$u_{cij} = \eta_{cj} + \varepsilon_{cij}$$

[5] Later we also estimated child mortality using a hazard model. See Section 4.2.

The first part η_{cj} is common to all children born to a particular woman j and captures mother/parents/household level unobserved heterogeneity that affects the health of all children born to the same woman. This could include biological/genetic factors that are unobserved to the researcher: for example, a particular woman/couple might have some biological problem that is transmitted genetically to her children and worsens the health status of her children, thereby increasing the probability of the child dying. The heterogeneity term $\eta_c \sim N\left(0, \sigma_c^2\right)$ is assumed to be uncorrelated with the other covariates. All other residual variation is captured by ε_{cij} specific to the i^{th} child born to the j^{th} woman where $\varepsilon_c \sim IIDN\left(0,1\right)$. Note that while the mother/household level unobserved heterogeneity term $\left(\eta_c\right)$ is assumed to be uncorrelated with the other covariates,[6] it is not the case with the other residual variation $\left(\varepsilon_c\right)$ as explained in footnote 4.

The child-specific characteristics $\left(Z_{cij}\right)$ include binary variables to indicate if the child is male, if s/he is the oldest, youngest or the only child, whether the child was born in a hospital, whether the child received any vaccination and also whether age of the mother at the time of the birth of the child was less than 20.[7] The parental/household level variables $\left(X_{cj}\right)$ include the highest education attained by the mother and the father, a composite index of household assets[8], a dummy for rural

[6] Note that this is a standard assumption in random effects models.

[7] We have also treated the mother's age when the child is born as a continuous variable in alternative specifications, using a quadratic or log quadratic functional form for flexibility. These results are available on request.

[8] This asset index is computed because the DHS do not obtain any information on household income or expenditure. This is a composite asset index and we use principal component analysis to construct this index from household ownership of agricultural land, farm equipment, cycle, scooter, car, radio and television. Note that specification of a pure health production function should not include household assets variable. The non-significance of the assets variable (see Table 5, specification 5) in our sample confirms that the included health inputs in the health production function constitute a complete set; otherwise the asset variable could be significant, suggesting the importance of omitted health inputs.

residence, household religion and a dummy to indicate whether the mother ever received tetanus vaccination. Finally in the absence of data on local availability of health services and facilities, we include a set of region dummies to account for the variation in child mortality across the regions in Bangladesh. These region dummies control for the region-specific infrastructure availability in the country and thus capture the otherwise omitted community level effects. See Table A1 for a list of the explanatory variables used.

3.1 Endogeneity Issues

The set of child-specific explanatory variables above $\left(Z_{cij}\right)$ includes a number of individual (child-specific) characteristics that could give rise to the problems of endogeneity in this context. The first relates to the age of the mother at the time of the birth of the child.[9] Mother's age at childbirth is potentially endogenous in that it is related to parental choices regarding the timing and spacing between successive births and could be regulated by the use of traditional/modern forms of contraception. It is commonly accepted that there are adverse physical/health consequences of early child bearing for both the mother and the baby. However the age below which the physical risks of child bearing are considered to be significant varies depending on general health conditions and on access to good prenatal care. In a country like Bangladesh where anaemia and malnutrition are common and where access to health care are poor (especially in rural areas), child bearing among teenage mothers (whose physical

Given that concerns may be raised about the measurement errors in survey data on these assets variables, we also check the robustness of our results (i) by including the individual assets variables rather than the composite index and also (ii) by dropping the assets variables altogether from all the equations. The mortality results (which are not presented but are available on request) remain very similar in both cases.

[9] One could argue that it is the mother's age at first birth and not the age at birth of each individual child that matters in terms of its effects on child health (and child mortality). Note however that age at first birth is essentially a mother level fixed-effect (same for all the children born to the woman). The latter therefore does not allow us to identify the component of the error due to the mother level unobserved heterogeneity from all unobserved determinants of the age at first birth.

growth is incomplete) is likely to bring disproportionate health risks for the child including low birth weight and death of the child. The age of the mother at the time of childbirth could also be viewed as an indicator of the socio-economic status of the mother. Young mothers may be more likely to be less affluent, less educated and have less control over their environment as compared to older mothers. As with child mortality equation (1), what we estimate is the propensity for early child bearing $\left(EARLY^{*}\right)$. The estimating equation for $EARLY^{*}$ is:

$$EARLY^{*}_{mij} = \xi_1 X_{mj} + \eta_{mj} + \varepsilon_{mij} \qquad (2)$$

Given that $EARLY^{*}$ is unobservable, we instead use a binary variable $EARLY$ as follows:

$$EARLY = \begin{cases} 1 \text{ if the age of the mother at the time of birth of the child was below 20} \\ 0 \text{ otherwise} \end{cases}$$

X_{mj} refers to the set of woman/household-specific explanatory variables that affect the probability of having an early childbirth.[10] The unmeasured residual component is broken up into a mother specific unobserved heterogeneity component $\left(\eta_m\right); \eta_m \sim N\left(0, \sigma_m^2\right)$, which is common to all children born to the j^{th} mother (and is assumed to be uncorrelated with the other co-variates) and a term $\varepsilon_m; \varepsilon_m \sim IIDN\left(0,1\right)$ that captures any other child-specific residual variation. The unobserved heterogeneity component might capture some additional (unobserved to the researcher) information relating to health considerations and/or economic/family/cultural circumstances on part of the woman/couple, which might cause the women to have children early or

[10] While we observe EARLY for each child born to a given woman, there are no child-specific explanatory variables in this equation. Without much loss of generality, we assume that the decision as to when to have another child depends on the preferences and characteristics of the couple rather than those of child.

late. Equation (2) is estimated as a probit.[11] See Table A1 for a list of the explanatory variables used.

The second endogeneity issue relates to the possible endogeneity of the health inputs variables. For example, one way of reducing health risks for the newborn (and also for mothers) is to increase the fraction of babies that are delivered in a proper medical facility (for example a hospital). Previous research using the Bangladesh DHS data sets shows that nearly 95% of all births are at home and in the majority of these cases (57%) assistance is provided by (often untrained) local birth attendants (*dais*), followed by other relatives (25%). Trained doctors/mid-wives attend only 5% of births [Mitra, Al-Sabir, Cross and Jamil (1997)]. Often these children are born in quite unhygienic conditions and hence are susceptible to increased risk of infections and hence child mortality.

Yet another way of reducing child mortality rates is to provide the full set of recommended vaccination to the child.[12] Why is immunization important? According to the World Bank, immunization is one of the most cost effective ways to prevent major illnesses, particularly in environments where children are malnourished and die of preventable diseases [WorldBank (1993)]. The Expanded Programme on Immunization (EPI) was launched by the WHO and the UNICEF in the late 1970s. Overall the programme has been quite a success, with the percentage of children that have been immunized globally increasing from less than 5% in 1977 to 20 – 30% in 1983 and to about 80% coverage with polio, DPT and measles vaccines by 1990.

[11] Preliminary data analysis in section 2 indicated the absence of a u-shaped relationship between mother's age at birth and child mortality rates. To be absolutely sure, we also estimated mother's age at birth as an ordered probit model. The regression results (which are available on request) show that compared to children born to women in their 20's, child mortality rates are higher for children born to adolescent women but not so for children born to women in their 30's. The ordered probit results corroborate the descriptive statistics presented in Table 1.

[12] While tetanus vaccination of the mother is regarded as another cost-effective way of preventing infant mortality, we cannot include it in our analysis as the information is not available for each child We however keep mother ever receiving tetanus vaccination as an explanatory variable in the mortality equation (see further discussion in section 4.2)

Unfortunately the program had a late start in Bangladesh – in 1985, the programme covered only two percent of all children. However, in 1989, the Ministry of Health and Family Planning joined forces with other government bodies and non-governmental organizations to improve the service.

In estimating the effect of health inputs on child mortality, it is important to take into account the issue of self-selection in the use of health inputs. Women who demand health care (choose to deliver the child in a hospital or choose to vaccinate their children) might not necessarily be a random subset of all women in our sample. It is likely that these women are those who anticipate complications at birth or other unobserved factors that might lead to an increased risk of child mortality and hence are more likely to seek health care (remember that health is private information to the woman and unobserved to the researcher). This could be termed as adverse self-selection. Ignoring this adverse self-selection could underestimate the effect of prenatal care on birth outcomes. On the other hand, women who choose increased health inputs could be low risk women, with a strong preference for healthy children. This could be termed as favourable self-selection.[13] Ignoring favourable self-selection actually causes the effects of health inputs on birth outcomes to be overstated. What this implies is that health inputs are endogenous in the child health outcome (child mortality) regression.

We focus on two particular health inputs – the decision to deliver the baby in a hospital and the decision to vaccinate (even partly) their children; the choice of health inputs has essentially been driven by data availability[14]. If we indeed find that increased use of health inputs (like hospital delivery or child vaccination) have

[13] This definition of favourable self-selection is due to Gortmaker (1979).

[14] One can consider other possible health inputs affecting child mortality, e.g., tetanus vaccination during pregnancy or prenatal check-up with a qualified person. However in neither of these cases did we have the relevant data for all children born in the last five years: it was only available for the last child born.

significant positive effects on child health we would have identified an important policy tool. The estimating equations for propensity to deliver the child in a hospital and that of vaccinating the child are as follows:

$$HOSPDEL^{*}_{hij} = \alpha \mathbf{X}_{hij} + \eta_{hj} + \varepsilon_{hij} \tag{3}$$

and

$$VACCN^{*}_{vij} = \psi \mathbf{X}_{vij} + \eta_{vj} + \varepsilon_{vij} \tag{4}$$

Since $HOSPDEL^{*}$ and $VACCN^{*}$ are not observable, we use two variables $HOSPDEL$ and $VACCN$ as follows:

$$HOSPDEL = \begin{cases} 1, \text{ if the child was born in a hospital} \\ 0, \text{ otherwise} \end{cases}$$

$$VACCN = \begin{cases} 1, \text{ if the child has received } \textit{any} \text{ vaccination} \\ 0, \text{ otherwise} \end{cases}$$

Both the decision to deliver the baby in a hospital and the decision to vaccinate the children depend on a set individual/child $\left(Z_{kij}\right)$ and parental/household $\left(X_{kj}\right)$ characteristics, where $k = h, v$ for hospital delivery and child vaccination respectively. The unexplained component of the demand for health input is again divided into two parts: one that captures mother specific unobserved heterogeneity $\eta_{kj}; k = h, v$ and applies to all children born to the j^{th} mother (again assumed to be uncorrelated with other covariates)[15] where $\eta_{kj} \sim N(0, \sigma_k^2); k = h, v$ and a component $\varepsilon_{kij} \sim IIDN(0,1), k = h, v$ that captures all other residual variation. Once again we assume that the unobserved heterogeneity component of the error term $\left(\eta_{kj}; k = h, v\right)$ is uncorrelated with the other co-variates. Here $\mathbf{X}_{kij} \equiv \left(Z_{kij}, X_{kj}\right); k = h, v$ is a vector

[15] These would relate to unobserved woman/household-specific factors like reproductive history, other health and/or cultural considerations or even personal experience, which may encourage/discourage use of these health inputs.

of individual $\left(Z_{kij}\right)$ and parental/household specific $\left(X_{kj}\right)$ characteristics that affect the probability of hospital delivery and immunization. We estimate *HOSPDEL* and *VACCN* as separate probits. See Table A1 for a list of the explanatory variables used.

We allow the mother specific unobserved heterogeneity terms in equations (1) – (4) to be correlated. The argument here is that the unobserved mother-level characteristics that affect child mortality might also affect the choice of health inputs and the decision to have an early child. Women who have children early, women who choose to deliver their children in a hospital and women who choose to vaccinate their children are not necessarily a random subset of all women in the sample. In addition women who have early children might well have some (additional) private information about their own health and might choose to deliver their children in a hospital and/or choose to vaccinate their children. Suppose that a woman chooses to deliver the child in a hospital because she has experienced some specific health scare during pregnancy. Unless the woman experiences the same health scare for all her pregnancies, this type of adverse self-selection will operate at the child level and not at the mother level. Conditional on the η residuals, however, these choices are independent of one another and of child mortality. By modelling this aspect of the data generation as a common fixed effect, we are able to remove the implicit bias resulting from the correlation between each pair of common fixed terms $\left(\eta_m, \eta_h, \eta_v, \eta_c\right)$. Note that the expectations of both η_{kj} and ε_{kij} for given $\mathbf{X} \equiv \left(X, Z\right)$ (the set of explanatory variables) is zero and that each has a constant variance while the covariance between any pair of η's and ε's is zero for given $\mathbf{X}$ (see equations 13 – 19 in Greene (2003), page 294).

3.2 Joint Estimation:

When early childbirth $(EARLY)$, hospital delivery $(HOSPDEL)$ and child vaccination $(VACCN)$ are all treated as endogenous in the child mortality probit regression, the joint marginal likelihood function is written as:

$$\int_{\eta_m}\int_{\eta_h}\int_{\eta_v}\int_{\eta_c}\left[\prod L^m(\eta_m)\prod L^h(\eta_h)\prod L^v(\eta_v)\prod L^c(\eta_c)\right] f(\eta_m,\eta_h,\eta_v,\eta_c)d\eta_m d\eta_h d\eta_v d\eta_c \quad (5)$$

where $f(\eta_m,\eta_h,\eta_v,\eta_c)$ is the joint distribution of the unobserved heterogeneity components. Here $f(\eta_m,\eta_h,\eta_v,\eta_c)$ is a four dimensional normal distribution characterised as follows:

$$\begin{pmatrix}\eta_m\\ \eta_h\\ \eta_v\\ \eta_c\end{pmatrix} \sim N\left(\begin{bmatrix}0\\0\\0\\0\end{bmatrix}, \begin{pmatrix}\sigma_m^2 & & & \\ \rho_{mh}\sigma_m\sigma_h & \sigma_h^2 & & \\ \rho_{mv}\sigma_m\sigma_v & \rho_{hv}\sigma_h\sigma_v & \sigma_v^2 & \\ \rho_{mc}\sigma_m\sigma_c & \rho_{hc}\sigma_h\sigma_c & \rho_{vc}\sigma_v\sigma_c & \sigma_c^2\end{pmatrix}\right) \quad (6)$$

Thus conditional on the η residuals, these choices are independent of one another and of child mortality and the conditional joint likelihood can be obtained by simply multiplying the individual likelihoods. The marginal joint likelihood is obtained by integrating out the heterogeneity terms [see Panis and Lillard (1994)].[16] The model is estimated using Full Information Maximum Likelihood (FIML) Method. Typically the joint estimates give us the lower bounds for the effect of these endogenous variables on child mortality

An analogue to this procedure is the treatment model using Heckman-type selection adjustments to correct for omitted variable bias. Women who have children early, women who choose to deliver their children in a hospital and women who

[16] Many models require that one or more residuals are integrated out. Where a closed form solution to the integral does not exist, the likelihood may be computed by approximating the normal integral by a weighted sum over conditional likelihoods, i.e., likelihoods are conditional on certain well-chosen values of the residual. The software that we use [Lillard and Panis (2003)] makes use of the Gauss-Hermite Quadrature to approximate normal integrals [see for example Abramowitz and Stegun (1972), pp. 890 and 924].

choose to vaccinate their children are not necessarily a random subset of all women in the sample – there is an implicit self-selection issue here. To pursue this analogy, the mortality equation models the outcome of the treatments (early child birth, hospital delivery and/or vaccination in our study) and the early childbirth equation, for example, (very much like other possible selection mechanisms pertaining to the use of health inputs, e.g., hospital delivery or child vaccination) the selection into the treatment.

Thus given the problem of finding appropriate instruments, this correlated model allows us to derive selectivity-corrected estimates of child mortality as long as the equations are identified.

3.3 Identification

Identification is ensured by the recursive structure and the covariance restrictions imposed by the inclusion of a fixed effect in each equation (1) – (4). A recursive structure is ensured by the fact that child mortality equation (1) depends on early birth (equation 2) and use of health inputs (equations 3-4), but not the other way round. This issue is discussed in Chamberlain and Griliches (1975). Therefore strictly speaking we do not need to use instruments for identification purposes.

Nevertheless, there naturally arises a set of identifying variables by the very nature of the decisions pertaining to each of the three potentially endogenous variables. In particular, there are three identifying variables in the *early childbirth equation* $(EARLY)$: the age difference between the mother and the father; whether the father is an unskilled agricultural labourer; and number of children at the *first* use of contraception that are not included in any other equations. These variables are likely to affect the decision to have an early child, but are unlikely to have a direct effect on child mortality. In particular, a smaller age difference between the wife and

the husband is typically indicative of increased balance of power within the marriage. Secondly, father's occupation as unskilled agricultural labourer is indicative of low education and/or low income and it, in all likelihood, would not be directly correlated with child mortality per se. Finally, use of contraception is an obvious way of delaying/spacing child birth; while current use of contraceptives could be a choice variable, number of children at the *first* use of contraception can be treated as exogenous.

We use bargaining power of the wife relative to the husband within the household in the two health input equations. This is because relative bargaining power of the husband and the wife cannot directly affect child health (and child mortality) but can indirectly affect child health through its effects on the use of health inputs (hospital delivery and child vaccination). See Maitra (2004) for a similar assumption in the context of India. In particular, for the *hospital delivery equation* we use the following binary variables "whether the woman has any say on female health care" and "whether the mother can go to hospital without the husband". These variables are likely to have very little direct relevance on the vaccination decisions. For the *child vaccination equation* too we use similar, but somewhat different binary variables which could directly affect the couple's decision whether to vaccinate a child. These are "whether the woman has any say on child health care"; "whether the woman go anywhere without her husband" and "whether there are health facilities nearby".[17] In addition, for the child vaccination regression we also include an indicator dummy for first-born male. In many societies, including those in South Asia firstborn males receive preferential treatment (in terms of inputs, health and educational) and this

[17] Access to nearby health facilities is likely to be more important for the vaccination equation as child vaccination is a recurring event.

kind of parental preferences might be reflected in the fact that vaccination rates are higher for first-born males.

4. Results:

We now turn to the actual regression results. The primary variable of interest in our analysis is child mortality. Five sets of results are presented. *Specification* 1 is the simplest specification where we assume that early childbirth $(EARLY)$, hospital delivery $(HOSPDEL)$ and child vaccination $(VACCN)$ are all exogenous and we also assume that there is no mother level unobserved heterogeneity. In *specification* 2, while $EARLY$, $HOSPDEL$ and $VACCN$ are still assumed to be exogenous, we allow for unobserved mother level heterogeneity in the child mortality equations (we still restrict the cross equation correlations to be zero). In specifications 3, 4 and 5 we successively allow the unobserved heterogeneity terms to be correlated. In *specification* 3, $EARLY$ is assumed to be endogenous in that we allow for the possibility that $\rho_{mc} \neq 0$, but $HOSPDEL$ and $VACCN$ are assumed to be exogenous (i.e., $\rho_{hc} = \rho_{vc} = 0$). In *specification* 4, $EARLY$ is assumed to be exogenous (i.e., $\rho_{mc} = 0$) but $HOSPDEL$ and $VACCN$ are assumed to be endogenous so that we allow for the possibility that $\rho_{hc} \neq 0; \rho_{vc} \neq 0$. Here we also allow for the possibility that $\rho_{hv} \neq 0$. Finally in *specification* 5, $EARLY$, $HOSPDEL$ and $VACCN$ are all assumed to be endogenous so that $\rho_{mc} \neq 0, \rho_{hc} \neq 0, \rho_{vc} \neq 0$. This is the complete correlated model.

Given the space constraints, here we will present and discuss the results only for the child mortality regressions: both the probit[18] and the hazard estimates. The probit estimation results for early child birth, hospital delivery and child vaccination are not presented here but are available on request.

4.1 Unobserved Heterogeneity:

Incorporation of unobserved heterogeneity is a distinctive feature of our analysis that remains much overlooked in the literature. Table 4 presents the estimates for the unobserved heterogeneity components corresponding to *specification* 5, where $EARLY$, $HOSPDEL$ and $VACCN$ are all assumed to be endogenous in the child mortality regressions. The diagonal elements are the standard deviations and the off-diagonal elements are the correlation coefficients. Self-selection in the demand for health inputs (hospital delivery and child vaccination) are captured by the statistically significant correlation coefficients between the unobserved heterogeneity coefficients in the hospital delivery and the child vaccination equations on the one hand and the child mortality equation on the other. Given the strong correlation between the unobserved heterogeneity coefficients in the different equations, one could convincingly argue that ignoring unobserved heterogeneity and the correlation between the unobserved heterogeneity coefficients would result in biased estimates. We will, for the rest of the paper, discuss the results corresponding to the complete model (*specification* 5) and use the results for *specifications* 1 – 4 for comparison purposes, i.e., to highlight the consequences of ignoring the possible sources of endogeneity problems.

4.2 Regression Results on Child Mortality:

[18] These estimates are obtained from the full sample of all children. We also estimated the specification 5 for the correlated model for women with at least 2 births and the correlated estimates are very similar irrespective of whether we consider the full sample or sub-sample of women with at least two births.

We start with a discussion of the probit estimates of child mortality for the full sample, presented in Table 5.

The effect of *EARLY* on child mortality depends on assumptions regarding the endogeneity of early childbirth on child mortality. In particular it is worth noting that both the sign and significance of *EARLY* changes once we account for the potential endogeneity of *EARLY* in the child mortality regressions. For example, early childbirth is associated with significantly higher child mortality in *specifications* 1, 2 and 4. However, the estimate from the complete *specification* 5 implies that early childbirth has a negative effect on the probability of child mortality, though the effect is not statistically significant. This is quite a surprising result, especially in view of the descriptive statistics presented in Table 1 that suggest that the unconditional child mortality rates are higher for early childbirths. One possible explanation of this rather surprising result could be along the following lines. Women having early childbirth tend to use health inputs differently from other women. The latter may be related to the unobservable health (reproductive/child health) or socio-economic problems they face with the particular birth. In order to investigate this further, we jointly estimated (a) *EARLY* [equation (2)] and *HOSPDEL* [equation (3)] and also (b) *EARLY* [equation (2)] and *VACCN* [equation (4)]. As with the complete system (1) – (4), we make each of these two equations system (a) and (b) recursive. So in addition to the set of explanatory variables originally included in $\mathbf{X}_h$ and $\mathbf{X}_v$, we also included *EARLY* as an additional explanatory variable in each of HOSPDEL and VACCN equations. The coefficient estimates for *EARLY* in the two regressions are presented in Table 6A. Note that we estimate (and present) three different specifications: first where *EARLY* is exogenous in the *HOSPDEL* and *VACCN* regressions and we do not account for any unobserved heterogeneity; second where *EARLY* is exogenous in

the *HOSPDEL* and *VACCN* regressions but we allow for unobserved heterogeneity; and third where *EARLY* is endogenous in the *HOSPDEL* and *VACCN* regressions (and we allow for $\rho_{mh} \neq 0$ and $\rho_{mv} \neq 0$); the latter corresponds to the complete specification. When we allow for endogeneity of early childbirth in the health input regressions in the complete correlated model, we find that women who have children early are more likely to vaccinate their children and are less likely to deliver their children in a hospital. This could imply that women who have children early behave quite differently compared to others, at least in terms of use of health inputs.

A possible explanation of this result could be related to the omission of the correlation between the child specific unobserved heterogeneity terms (the only correlation that we allow for is that between the unobserved household/mother-specific heterogeneity components of the error terms). In other words, there may arise some unobserved child specific health factor that may induce an adolescent mother to use health-input differently (as compared to other children born to the same woman). To the best of our knowledge, Rosenzweig (1986) is the only paper that allows for this kind of correlation between the child specific unobserved heterogeneity components of the error term. While Rosenzweig (1986) used a set of pre-natal characteristics to control for this kind of omitted variable bias, we lack information on similar variables in our data-set, which in turn prevents us from replicating this technique.[19]

[19] We attempted to (at least partially) address this issue by including two interaction terms between early birth and health inputs (*EARLY*HOSPDEL* and *EARLY*VACCN*) in the correlated child mortality regression. The coefficient estimate of *EARLY*HOSPDEL* is positive and statistically significant while *EARLY*VACCN* is not. Thus there is some indication that children born to adolescent women are more likely to die despite having hospital delivery. This might reflect the fact that women having early child birth may self select them into hospital delivery because of some unobservable child-specific health problems (not common to other children born to her) and may still face higher risks of mortality. .

The probability of child mortality is significantly lower when the child is born in a hospital and if he/she is vaccinated. However the coefficient estimates of *HOSPDEL* and *VACCN* for the five specifications tell us an even more interesting story. Note that the coefficient estimate of hospital delivery is actually positive (and weakly statistically significant) in *specifications* 1 – 3. So failure to account for the self-selection (and endogeneity) in the choice of hospital delivery not only results in biased estimates, but more importantly the bias is so strong that it changes the sign of the coefficient estimate; the true estimate is given by *specification* 5, which is negative and statistically significant at the 10% level. In other words if we fail to account for this self-selection we erroneously conclude that hospital delivery has a harmful effect on child health while the true effect is just the opposite. The coefficient estimates of child vaccination, on the other hand, are always negative and statistically significant though it is worth noting that the beneficial effect of child vaccination on child mortality is underestimated when we do not take account of the self-selection in the decision to vaccinate the child. Our results are therefore quite similar to results obtained using data from other countries: Panis and Lillard (1994) for Malaysia, Maitra (2004) for India and Ghilagaber (2004) for East Africa.

Other results are generally in agreement with the existing findings. First, mortality risks are also significantly lower if the mother has ever received tetanus vaccination.[20] In South Asia, tetanus has long been a major killer of newborn and very young children and it has been documented that two doses of the tetanus toxoid vaccine given to the mother when she is pregnant prevents nearly all tetanus

[20] Note that the mother ever receiving tetanus vaccination could be subject to the same sort of endogeneity issues that the other health input variables are subject to. However in this case we have only one observation per woman so adding an additional equation would lead to problems because the mother specific unobserved heterogeneity component of the error terms cannot be identified. In addition it is difficult to obtain good instruments. So while we agree that this variable could be potentially endogenous, we ignore this endogeneity in our analysis. We also find that our main results are not sensitive to the inclusion of this variable.

infections in both the mother and the newborn child. Alternatively the woman ever receiving tetanus vaccination could be viewed as a proxy for increased overall awareness of the woman on matters regarding health, which has a significant effect on the health outcome of the child, independent of the effect of the maternal tetanus vaccination per se.

Second, while parental educational attainment does not generally have a particularly strong direct effect on child mortality, (note that the only significant parental educational attainment variable that is statistically significant is that the highest education attained by the mother is more than primary schooling), parental education has significant effects on early childbirth, hospital delivery and child vaccination (results available on request). That even the direct effect of the mother having more than primary school is statistically significant in the child mortality regression, emphasizes the importance of maternal education on child health in general. Compare this to the fact that father's educational attainment does not have a direct effect on child mortality. The results are also indicative of a threshold level of education that must be attained before educational attainment starts having a statistically significant effect on child mortality.

The sign and significance of the birth order variables are interesting. The probability of child mortality is significantly higher for the oldest child and significantly lower for the youngest child. However it is also interesting that the probability of child mortality is significantly lower when s/he is the only child. Our results are therefore indicative of significant life-cycle effects on child mortality. The statistical insignificance of the household wealth variable in the mortality regression actually suggests that the health input controls that we have in the set of explanatory

variables constitute a complete set and the coefficient estimates do not suffer from omitted variable bias at least in this respect.

Finally several of the region dummies are statistically significant indicating that there is significant regional variation in child mortality rates. What is interesting is that all of the regional dummies are positive and statistically significant. These imply that compared to the reference category Khulna, child mortality rates are higher in other regions (this corroborates the descriptive statistics presented in Table 2A). We argue that these region dummies account for the community health facilities in the country. Though Dhaka division is clearly better off in terms of the provision of health services, it is not associated with lower child mortality rates. This is not particularly surprising. Indeed several studies [as summarized in Strauss and Thomas (1998)] have argued that local infrastructure could be endogenous in the child health regressions. This could happen because of two reasons. First, individuals might choose their residence based on the availability of public health services [see Rosenzweig and Wolpin (1988)]. Second, local infrastructure itself might be placed selectively by public policy, perhaps in response to local health conditions [see Rosenzweig and Wolpin (1986)]. The first issue is unlikely to be particularly important for a country like Bangladesh because migration in this case would have to be correlated with the unobserved factors that are correlated with health in a location, such as availability of clinics, over and above other measures included in wage differentials. Selective placement of health services is however potentially a much more important issue in this respect (though beyond the scope of this paper), which is also evident in striking regional variation in health spending (see Table 2B).

We also have information on the number of days the child was alive (before dying) if he/she is dead at the time of the survey or the age of the child, in days, at the

time of the survey. So an alternative way to model mortality would be to use a hazard model represented by a log hazard of duration equation. The coefficient estimates from a proportional hazard model are presented in *specification 6* in Table 5. The baseline hazard model is estimated non-parametrically as a piece-wise constant log hazard model with one node at 6 months i.e., there are two intervals (0, 6) and (6+).[21] The sample is censored if the child is alive at the time of the survey and is uncensored if the child is dead at the time of the survey. The hazard coefficient estimates presented correspond to the case where early childbirth $(EARLY)$, hospital delivery $(HOSPDEL)$ and child vaccination $(VACCN)$ are treated as endogenous in the child mortality hazard regression. Effects of early childbirth and use of health inputs are qualitatively similar to those obtained from the child mortality probit equation (*specification* 5) – early childbirth reduces the hazard of child mortality (significant only at 10% level), as does hospital delivery (though in this case the effect is not statistically significant) and child vaccination, as we take account of the important self-selection effect.

4.3 Robustness Checks: Comparison with Alternative Estimates

Finally, we compare the correlated estimates with various estimates obtained from possible alternative models of child mortality. These are summarised in Table 7. Column 1 presents the coefficient estimates from a single-equation fixed-effects logit, while column 2 presents those for the random effects probit regression for child mortality.[22] These mortality estimates highlight the persistence of endogeneity bias in that estimates of early birth, hospital delivery and vaccination remain statistically

[21] The configuration of signs of Duration Spline 0 – 6 Months and Duration Spline > 6 months indicate that the hazard of child mortality is increasing in the first 6 months following childbirth but is decreasing thereafter.

[22] Fixed effects single equation logit estimates of child mortality are calculated for women with two or more children. We have also computed the corresponding estimates for the sample of women who have had at least one child born in two years preceding the survey. These estimates are available on request.

insignificant in both samples. In column 3 we present the two-step instrumental variable probit regression, where we account for endogeneity of early child bearing, hospital delivery and vaccination[23] and the standard errors are corrected for clustering at the mother level. These two step instrumental variable estimates are qualitatively similar to the correlated estimates presented in Table 5, *specification* 5. This is because in both cases we account for the potential endogeneity of *EARLY*, *HOSPDEL* and *VACCN* (though in the complete set of correlated estimates we allow for mother level unobserved heterogeneity as well and also estimate a structural equation model).

One may also argue that since the vector of explanatory variables $(\mathbf{X})$ in the correlated model includes both child-specific (Z) and mother/household specific variables (X) and it is possible for some of the mother/household specific observables (for example parental education, contraceptive use, household assets, choice of residence, mother's tetanus vaccination) to be correlated with the household-specific error terms $\eta_{kj}; k = c, m, h, v$ and this could bias the estimates. To examine this issue, we re-estimated the correlated model after omitting these mother/household specific observable explanatory variables[24] for women with multiple births.[25] We present two sets of estimates: mortality estimates shown in Table 7 column 4 corresponds to *EARLY* as a function intercept and MUSLIM while those in Table 7 column 5 corresponds to *EARLY* as a function of intercept and the

[23] We could not however compute the corresponding maximum likelihood estimates because of convergence problems.

[24] While we could drop all the mother/household-specific variables from hospital delivery and child vaccination equations, we keep only one of the household-specific variables in the *EARLY* equation. Note that the equation for *EARLY* does not have any child-specific explanatory variables and the likelihood function failed to converge if we had kept just the intercept term in the *EARLY* equation.

[25] Note that in this case, we also drop the only child variable as it takes a value of 0 for all children in the chosen sample.

age difference of the couple (AGEDIFF).[26] While being born in a Muslim household is determined by birth, age difference of the couple in the $EARLY$ equation is purely a cultural variable in a society where arranged marriages are predominant even today and hence is unlikely to be correlated with the household specific unobserved heterogeneity. We continue to obtain similar signs (as presented in Table 5, specification 5) on early pregnancy $(EARLY)$, hospital delivery $(HOSPDEL)$ and child vaccination $(VACCN)$ in the mortality regression irrespective of whether we include the full set of explanatory variables or not. In other words, correlated estimates of mortality are not sensitive to the inclusion of mother/household specific explanatory variables.

Two other issues are worth noting in this context. One possible problem with the vaccination variable is that not all vaccinations are given at birth but when a child reaches a particular age. Accordingly any child who has died at a young age is less likely to have been vaccinated (or did not reach the point of completion of the full dose of vaccination). In a sense this could mean that the vaccination variable is censored and could lead to an overestimation of the effect of vaccination on the child mortality variable. To address this issue we conducted separate regressions with the sample restricted to children aged (i) 3 months and higher, (ii) 6 months and higher and (iii) 12 months and higher. The results remain similar to the complete model specification 5 shown in Table 5: both hospital delivery and vaccination continue to have a very strong beneficial effect on child health while early birth turns out to be insignificant.

Finally, we assess the contribution of use of health inputs towards reducing mortality risks in case of *adolescent* childbirth. This is because the correlated

[26] In each case hospital delivery and child vaccination equations are estimated using the intercept and two binary variables indicating if the child is the oldest and the youngest.

coefficient estimates (corresponding to *specification* 5) presented in Table 5 cannot be treated as the marginal effects. We therefore calculate the conditional likelihood estimates for children born to different categories of women experiencing adolescent child birth: (i) early pregnancy, but no hospital delivery and no vaccination (1, 0, 0); (ii) early pregnancy, hospital delivery, but no vaccination (1, 1, 0); (iii) early pregnancy, no hospital delivery but vaccination (1, 0, 1); and (iv) early pregnancy, hospital delivery and vaccination (1, 1, 1). We stratify the sample into these four categories and then using coefficient estimates obtained from *specification* 5, we calculate the likelihood of mortality for each sub-sample. Finally we calculate the conditional likelihood with respect to the base category (1, 0, 0). The results are summarised in Table 6B. These likelihood estimates do confirm that mortality risks among adolescent mothers could be sufficiently reduced by encouraging the use of hospital delivery, child vaccination or both. In this respect the beneficial role of child vaccination is particularly noteworthy: while mortality risks among adolescent mothers with hospital delivery is 47% (relative to case 1) if no vaccination is used, the risk comes down to about 4% if the child is not only born in a hospital, but is also vaccinated after birth.

5. Conclusion

This paper examines the relationship between early childbearing and child mortality in Bangladesh, a country where adolescent childbearing and high rates of child mortality are of particular concern. We argue that effective use of specific health inputs could however significantly lower child mortality rates even among adolescent women. This offers an attractive policy option particularly when compared to the costly alternative of delaying age at marriage. In particular, we find that women

having early childbirth tend to use health inputs differently from all other women thus establishing direct evidence of self-selection in the use of health inputs. We show that failure to account for this endogeneity results in biased estimates. In an attempt to reduce this bias we jointly estimate child mortality along with mother's age at birth, and use of health inputs (namely, hospital delivery and child vaccination), allowing for the cross-correlation between the mother-specific unobserved components of the residual terms in these equations. Once we correct for the underlying self-selection issue, early child birth in our sample is no longer associated with higher mortality. While uncorrected estimates of child mortality emphasize the adverse effects of early childbirth on child mortality, this effect is reversed, once we take account of the possible endogeneity of early childbirth and use of health inputs on child mortality.

Taken together, our results suggest that use of health inputs is one possible way of mitigating the adverse effects of early childbirth. There is strong evidence that children delivered in hospitals and children vaccinated against major childhood diseases have better chances of survival, even in case of adolescent childbirth. A comparison of these correlated estimates with a range of alternative estimates establishes the robustness of these results. From a policy point of view this is an important finding. Both researchers and policy makers agree that increasing the stock of human capital is essential to increase the rate of growth of any economy. Good health is now regarded as a basic pre-requisite for human capital formation, which in turn help increasing the income levels in a country. Poor child health therefore has long-term implications in the form of poor adult health and low levels of human capital formation. The finding that the adverse effects of adolescent childbirth on child survival is correlated with the use of health inputs like hospital delivery and child vaccination implies that one has in principle identified a convenient policy

instrument of encouraging use of health inputs in the short run; the latter could accompany other long-drawn options of inducing social change to delay age at marriage.

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Table 1: Effect of Age of Mother at Time of Birth on Child Mortality

Age of the Mother at the Time of Birth	Number of children dead	Total number of children born	Probability that the child is dead
Less than 15	17	121	0.14
15 – 19	251	2803	0.09
20 – 24	168	2792	0.06
25 – 29	56	929	0.06
30 or Higher	14	187	0.07
Total	506	6832	0.074

Table 2A: Regional Differences in Child Mortality Rates.

Region	All Children	Early Born and First Born			
		All	Home delivery	No tetanus Injection	No child vaccination
Barisal	7.2	10.2	9.8	16.1	19.1
Chittagong	6.0	8.8	6.4	8.5	20.5
Dhaka	8.1	10.5	10.2	20.8	21.9
Khulna	4.9	9.9	7.9	28.6	19.1
Rajsahi	7.6	10.3	10.4	18.5	20.6
Sylhet	11.3	15.4	14.4	20.5	29.4

Table 2B: Regional differences in the provision of public services

Division	Adult Literacy rate	poverty Head count index	Access to sanitary latrine		Access to safe drinking water		Rate of Immunization DPT 12-23 months		Govt. [1] expenditure on health per capita	Number of Specialized Hospitals
	1995	1995	1995	2000	1995	2000	1995	2000	1996-97	1996-97
Barisal	56.4	59.9	51.7	50.1	93.2	95.4	80.5	71.2	126	1
Chittagong	41.2	44.9	41.1	41.9	93.8	96.3	66.5	78.7	120	4
Dhaka	43.0	52.0	35.0	38.0	99.8	99.6	69.3	71.7	196	10
Khulna	47.2	51.7	41.8	63.2	91.3	91.4	92.1	82.3	113	3
Rajsahi	35.2	62.2	27.0	39.6	99.2	99.9	84.1	74.2	117	7
Sylhet	-	-	-	47.0	-	95.0	-	64.9	117	3
Sector										
Rural	36.6	56.7	36.4	41.3	96.7	97.3	76.0	73.5	-	-
Urban	60.0	35.0	79.1	61.2	99.3	99.5	80.0	82.7	-	-

Source: Sen and Ali (2003); Institute of Policy Studies (2001).[1] This is measured in Bangladeshi Taka.

Table 3: Selected Parental Characteristics (sample proportion/average)

	Adolescent birth	Non-adolescent birth
Muslim	0.9152	0.8590
Hindu	0.0805	0.1267
Mother has primary or higher schooling	0.5162	0.5703
Father has primary or higher schooling	0.5297	0.6310
Mother's education in single years	2.54	3.78
Say in female health care	0.4284	0.5007
Say in child health care	0.4975	0.5828

Table 4: Structure of Unobserved Heterogeneity (corresponding to specification 5)

	Early Childbirth	Hospital Delivery	Child Vaccination	Child Mortality Probit
Early Childbirth (η_m)	1.5634 ***			
	(0.0836)			
Hospital Delivery (η_h)	-0.2163 ***	2.4129 ***		
	(0.0451)	(0.2049)		
Child Vaccination (η_v)	0.0026	-0.1065 *	0.8266 ***	
	(0.0413)	(0.0597)	(0.0558)	
Child Mortality (η_c)	0.2482 **	0.3663 **	0.6890 ***	1.1403 ***
	(0.1169)	(0.1466)	(0.1111)	(0.171)

Notes: Asymptotic standard errors in parentheses. Diagonal Elements are Standard Deviations. Off-diagonal Elements are Correlation Coefficients. Significance: '*'=10%; '**'=5%; '***'=1%. Estimates of the Heterogeneity Structure correspond to the full specification (see specification 5 in Table 5, below).

Table 5: Probit Estimates of Child Mortality: Full sample

	All Exogenous (No Unobserved Heterogeneity) (1)	All Exogenous (With Unobserved Heterogeneity) (2)	Early Child birth Endogenous (3)	Health Inputs Endogenous (4)	All Endogenous (5)	Hazard Specification All Endogenous (6)
Constant	-0.6978 ***	-0.8075 ***	-0.7756 ***	-0.2374	-0.2066	0.5594 ***
	(0.1595)	(0.2848)	(0.2812)	(0.2864)	(0.2866)	(0.0722)
Duration spline 0-6 months						-0.5686 ***
						(0.0648)
Duration spline > 6 months						-0.6133 ***
						(0.0638))
Male Child	0.0529	0.0741	0.0855	0.074	0.0815	0.0489
	(0.0625)	(0.0995)	(0.0994)	(0.1034)	(0.1041)	(0.1465)
Oldest Child	0.2082	0.2534	0.3345 *	0.21	0.2881	0.3278
	(0.1365)	(0.1943)	(0.198)	(0.1968)	(0.2027)	(0.2458)
Youngest Child	-0.6621 ***	-0.8097 ***	-0.7867 ***	-0.9152 ***	-0.8803 ***	-1.2113 ***
	(0.1031)	(0.1482)	(0.1463)	(0.151)	(0.1507)	(0.2153)
Only Child	-0.4868 ***	0.6455	0.7699	1.126	1.2362	1.6138
	(0.1195)	(1.403)	(1.2979)	(1.474)	(1.4014)	(1.5013)
Education of Mother Less than Primary School	-0.0876	-0.2235	-0.2197	-0.2762 *	-0.2679 *	-0.3175
	(0.0812)	(0.1387)	(0.1385)	(0.1457)	(0.1462)	(0.194)
Education of Mother More than Primary School	-0.2071 **	-0.3116 **	-0.3381 **	-0.3104 **	-0.3342 **	-0.4525 **
	(0.0822)	(0.1424)	(0.1429)	(0.1527)	(0.1539)	(0.2032)
Education of Father Less than Primary School	0.0106	0.1151	0.1077	0.1168	0.1089	0.1928
	(0.0739)	(0.1236)	(0.123)	(0.1295)	(0.1295)	(0.1713)
Education of Father More than Primary School	-0.1097	0.0375	0.0061	0.2065	0.1627	0.2453
	(0.0845)	(0.1409)	(0.1401)	(0.1541)	(0.1538)	(0.2045)
Muslim	-0.0671	-0.19	-0.1504	-0.2263	-0.1985	-0.2351
	(0.0898)	(0.1523)	(0.1521)	(0.1602)	(0.1626)	(0.2144)
Asset Index	-0.0521	-0.0917	-0.1119 *	-0.081	-0.0942	-0.1087
	(0.0382)	(0.0646)	(0.0651)	(0.0661)	(0.0671)	(0.0894)
Rural Resident	0.0026	-0.0511	-0.0289	-0.0928	-0.0701	0.0285
	(0.0661)	(0.1132)	(0.1125)	(0.1299)	(0.1294)	(0.1717)
Mother Ever had Tetanus Vaccination	-0.3013 ***	-0.5395 ***	-0.5407 ***	-0.6293 ***	-0.6281 ***	-0.6474 ***
	(0.088)	(0.144)	(0.1435)	(0.1513)	(0.1515)	(0.2156)
Early Child Birth (EARLY)	0.1558 ***	0.2464 **	-0.1763	0.2844 ***	-0.1310 **	-0.3994 **
	(0.0587)	(0.0969)	(0.1987)	(0.1013)	(0.0574)	(0.182)
Hospital Delivery (HOSPDEL)	0.1709 *	0.1497	0.1705	-0.6357 *	-0.6217 *	-0.6967 *
	(0.1006)	(0.1815)	(0.1811)	(0.3455)	(0.3449)	(0.4145)
Child Vaccination	-2.6371 ***	-3.5548 ***	-3.5764 ***	-4.5460 ***	-4.5168 ***	-7.5158 ***

(VACCN)	(0.2076)	(0.3953)	(0.3988)	(0.5308)	(0.5305)	(0.841)
Resident of Barisal	0.3442 ***	0.4849 **	0.5161 **	0.5045 **	0.5434 **	0.5782 *
	(0.1256)	(0.2168)	(0.2167)	(0.2256)	(0.2259)	(0.3042)
Resident of Chittagong	0.2233 **	0.0979	0.1307	0.1201	0.1603	0.2704
	(0.1095)	(0.1875)	(0.185)	(0.1976)	(0.1968)	(0.2613)
Resident of Dhaka	0.4275 ***	0.5751 ***	0.5954 ***	0.6216 ***	0.6462 ***	0.9214 ***
	(0.1047)	(0.1851)	(0.1837)	(0.1921)	(0.1924)	(0.2461)
Resident of Rajsahi	0.3603 ***	0.4878 **	0.5276 ***	0.5647 ***	0.6034 ***	0.7231 ***
	(0.1099)	(0.1953)	(0.1944)	(0.204)	(0.2052)	(0.2593)
Resident of Sylhet	0.4532 ***	0.5071 ***	0.5411 ***	0.4663 **	0.4980 **	0.6504 **
	(0.1106)	(0.1923)	(0.1915)	(0.1996)	(0.2003)	(0.2561)
Log Likelihood	-12097.79	-8794.24	-4138.23	-5573.17	-8757.92	-9347.23

NOTE: Asymptotic standard errors in parentheses; significance: '*'=10%; '**'=5%; '***'=1%.

Table 6A: Effect of Early Child birth on Use of Health Inputs

	Early Child birth Exogenous (No Unobserved Heterogeneity)	Early Childbirth Exogenous (With Unobserved Heterogeneity)	Early Childbirth Endogenous
Hospital Delivery			
Early Child Birth	-0.3925 ***	-0.7314 ***	-0.1321
	(0.0550)	(0.1245)	(0.2512)
Child Vaccination			
Early Child Birth	0.0465	0.0661	0.2268 **
	(0.0325)	(0.0444)	(0.0995)

NOTE: Asymptotic standard errors in parentheses; significance: '*'=10%; '**'=5%; '***'=1%.

Table 6B: Conditional likelihood estimates of child mortality from the correlated model (specification 5)

Case	Early child birth	Hospital Delivery	Child Vaccination	Likelihood	Conditional likelihood (with respect to Case 1)
1: Early birth, no health input	1	0	0	0.2034	-
2: Early birth, hospital delivery & no vaccination	1	1	0	0.0962	0.4723
3: Early birth, vaccination and home delivery	1	0	1	0.0134	0.0661
4: Early birth, hospital delivery and vaccination	1	1	1	0.0073	0.0360

Table 7: Robustness to Alternative Specifications

	FE Logit[#] (Women with Multiple Births) (1)	RE Probit (2)	IV Probit - Two Step (3)	Correlated Probit (4)	Correlated Probit (5)
Constant		-0.8164***	0.9433	-0.2063	-0.2177
		(0.1855)	(0.5814)	(0.1390)	(0.1392)
Male Child	0.3990	0.0922	0.0784	0.1027	0.1025
	(0.3514)	(0.0651)	(0.0688)	(0.0868)	(0.0870)
Oldest Child	1.2439*	0.3577***	0.2377	0.3686 **	0.3627 **
	(0.5987)	(0.1174)	(0.3880)	(0.1501)	(0.1505)
Youngest Child	-1.6793**	-0.7270***	-0.6924***	-1.2183 ***	-1.2225 ***
	(0.3222)	(0.1040)	(0.1161)	(0.1279)	(0.1282)
Only Child	0.4832	-0.5199***	-0.6094		
	(0.5071)	(0.1276)	(0.3807)		
Education of Mother Less than Primary School		-0.1073	-0.1944*		
		(0.0940)	(0.0992)		
Education of Mother More than Primary School		-0.2425**	-0.1879		
		(0.0985)	(0.1334)		
Education of Father Less than Primary School		0.0098	-0.0206		
		(0.0858)	(0.0896)		
Education of Father More than Primary School		-0.1382	0.2288*		
		(0.1010)	(0.1364)		
Muslim		-0.0605	-0.2646*		
		(0.1051)	(0.1602)		
Asset Index		-0.0593	0.1532		
		(0.0441)	(0.0971)		
Rural Resident		-0.0022	-0.4557		
		(0.0802)	(0.2862)		
Mother Ever has Tetanus Vaccination		-0.3614***	-0.2325**		
		(0.0929)	(0.1172)		
Early Child Birth (EARLY)	**2.61E-12**	**0.1589****	**0.5427**	**-0.0568**	**-0.0078**
	(7.56E+14)	**(0.0710)**	**(0.9487)**	**(0.1867)**	**(0.1872)**

Hospital Delivery (HOSPDEL)	**-0.5636**	**0.2019***	**-3.3337****	**-0.2786****	**-0.2845****
	(1.6140)	**(0.1199)**	**(1.6877)**	**(0.0977)**	**(0.0947)**
Child Vaccination (VACCN)	**-0.0567**	**-2.9776*****	**-4.3555*****	**-4.2249 *****	**-4.2347 *****
	(0.1021)	**(0.2483)**	**(0.7157)**	**(0.4632)**	**(0.4648)**
Resident of Barisal		0.3966***	0.0513		
		(0.1490)	(0.2004)		
Resident of Chittagong		0.2626**	0.0433		
		(0.1261)	(0.1784)		
Resident of Dhaka		0.4844***	0.3582**		
		(0.1208)	(0.1652)		
Resident of Rajsahi		0.4087***	0.2896**		
		(0.1260)	(0.1382)		
Resident of Sylhet		0.5226***	0.2912**		
		(0.1299)	(0.1393)		
σ_m				1.6160 ***	1.6456 ***
				(0.1321)	(0.1420)
σ_h				2.0850 ***	2.1137 ***
				(0.1670)	(0.1753)
σ_v				0.8205 ***	0.8205 ***
				(0.0549)	(0.0549)
σ_c				1.0296 ***	1.0302 ***
				(0.1565)	(0.1570)
ρ_{mh}				-0.4182 ***	-0.4352 ***
				(0.0445)	(0.0434)
ρ_{mv}				-0.0062*	-0.0013
				(0.0038)	(0.0398)
ρ_{mc}				0.2361 **	0.2006 *
				(0.1182)	(0.1180)
ρ_{hv}				-0.1418 ***	-0.1414 ***
				(0.0515)	(0.0513)
ρ_{hc}				0.0485	0.0581
				(0.1881)	(0.1860)
ρ_{vc}				0.6844 ***	0.6861 ***
				(0.1137)	(0.1139)

Note:

Standard errors are shown below the estimates. Significance: '*'=10%; '**'=5%; '***'=1%.
[#]: By the very nature of this model, all the mother/family-specific variables are dropped from this model.
In column 4 EARLY is estimated by including and intercept and the MUSLIM variable while in column 5 it includes the constant term and the AGEDIFF variable.

Table A1: Explanatory variables and identification of the system of four equations

	Early Childbirth	Hospital Delivery	Child Vaccination	Child Mortality
Male Child				X
Oldest Child		X	X	X
Youngest Child		X	X	X
Only Child		X	X	X
First Born Male			X	
Education of Mother Less than Primary School	X	X	X	X
Education of Mother More than Primary School	X	X	X	X
Education of Father Less than Primary School	X	X	X	X
Education of Father More than Primary School	X	X	X	X
Muslim	X	X	X	X
Asset Index	X			X
Rural Resident	X	X	X	X
Mother Ever had Tetanus Vaccination				X
Early Child Birth (EARLY)				X
Hospital Delivery (HOSPDEL)				X
Child Vaccination (VACCN)				X
Woman has say on Female Health Care		X		
Woman has say on Child Health Care			X	
Mother can't go to hospital without the husband		X		
Mother can't go anywhere without the husband			X	
There are no health facilities nearby			X	
Age Difference between Mother and Father	X			
Father is Unskilled Labourer	X			
Contraceptive Use	X			
Province Dummies	X	X	X	X