Early Childbirth, Health Inputs and Child Mortality: Recent Evidence from Bangladesh

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Revised: November 2004

Abstract

This paper examines the relationship between early childbearing, parental use of health inputs and child mortality in Bangladesh. In order to account for the potential endogeneity of the age at birth and use of health inputs, (hospital delivery and child vaccination) in the child mortality regression, we jointly estimate mother's age at childbirth, hospital delivery, child vaccination and child mortality taking into account of unobserved mother level heterogeneity. There is evidence of significant self-selection in the use of health inputs especially among young mothers and that the failure to account for self-selection results in biased estimates. These estimates suggest 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 turns out to be less pronounced while the favourable effects of use of health inputs on child survival still remains significant in our sample

JEL Classification: D13, I12, O15

Key words: Family formation, Adolescent childbearing, Hospital Delivery, Child vaccination, Child mortality, Unobserved Heterogeneity, Correlated estimates.

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[*] Corresponding author. Funding provided by a Faculty Research Grant, Faculty of Business and Economics, Monash University. This research has benefited from discussions with Stan Panis. We would like to thank Lisa Farrell, participants at the AHES Annual Conference 2004 held in Melbourne, two anonymous referees and the editor (Junsen Zhang) for their comments and suggestions. The usual disclaimer applies.

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1. Introduction

This paper examines the relationship between early childbearing, parental use of health inputs and child mortality in Bangladesh. Though in the last two decades or so Bangladesh has experienced a substantial decline in child mortality rates (see Huq and Cleland (1992), Cleland and Streatfield (1992), 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. Of the South Asian countries Bangladesh is at one extreme in terms of high teenage fertility (with Sri Lanka being at the other extreme). Early marriage (average age at first marriage is only about 15 years in Bangladesh) combined with low levels of contraceptive has also resulted in children being born early: for example in 1996–97 36% of all teenage women (age 13 – 19 years) were either pregnant or already had a child.

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 childbirth and child mortality.[1] Evidence

[1] 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. If a woman is either too young or too old, her uterus and cervix may be unable to sustain a normal pregnancy. Also, since a teenage woman is "biologically immature" the needs of her developing body compete with the demands of the fetus. Thus,

from the National Family Health Survey 1998-99 data set from neighbouring India suggests 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. What this suggests is that there is substantial potential for reducing child mortality by designing policies aimed at these high-risk women. An analysis of 1999-2000 Demographic Health Survey data from Bangladesh however does not suggest 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.[2]

There is a large and growing literature on child mortality in low-income countries. A large number of these studies focus on the effects of various socio-economic factors on child mortality, e.g., mother's literacy (Glewwe (1999)), household income (Pal (1999)), inadequate use of available health inputs (Panis and Lillard (1994), Maitra (2004)). Among other things, this literature documents that parental use of health inputs has a direct and favourable impact on child health outcomes.

We argue that the high child mortality among adolescent mothers could be related to use of available health inputs (for example hospital delivery or a range of

she may be incapable of providing the fetus with adequate nutrients. For an older woman, changes in the circulatory system are important but less serious than the declining quality of her eggs as she ages.

[2] Of course it is worth noting that the proportion of cases of late childbirth, e.g., when women were aged above 30 years, was very small in our sample.

vaccinations). 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 fewer intrinsic advantages. They are likely to have less information about the advantages of using available health inputs and/or may 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 unsafe abortion, anaemia, haemorrhage, sepsis, preeclampsia, 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. Given this private health information, there could be some important selection issues that need to be resolved. For example, 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). In particular, it is possible that young women experiencing complications in pregnancy are more likely to go to the hospital for a delivery and/or vaccinate the child, which in turn, affect the child health outcomes. There could again be a combination of these possibilities, for example, given private health information although a young mother may end up having home delivery, (though she might prefer to vaccinate the child). The essential implication of these arguments is that effects of adolescent child birth on child mortality become closely correlated not only with the use of health inputs, but also

with the observable (income/wealth, literacy, women's say in various household decision making) and unobservable (e.g., those related to health endowment/information) characteristics of the parents/households.

We use 1999–2000 Demographic and Health Survey data set from Bangladesh for our empirical analysis. Information on health inputs used are available for children born in the five years immediately preceding the survey and we thus restrict our sample to children born during this period. Child mortality in this framework is assumed to depend on mother's age at birth, use of different health inputs, in addition to other observable individual, parental/household and community characteristics and unobserved heterogeneity. Although in a single cross-section framework, we could assume parental age difference, literacy levels or role in family decision making process to be exogenously given, we cannot ignore the potential problem of endogeneity in the mother's age at the time of the birth of the child and the use of health inputs in the child mortality regression.

While many existing papers control for the age of the mother at the time of the birth of the child, none of these explicitly accounts for the potential endogeneity of this variable in the child mortality regression. Age at childbirth is however potentially endogenous because it is related to parental choices regarding the timing and duration between successive births. Parental choices regarding timing and spacing between births in turn mean that they are choosing the age at which they want to have the next child. Thus mother's age at birth becomes endogenous to child health outcomes and is also directly related to the whole issue of early childbearing. Similarly use of health

inputs for female/child health is chosen by the couple in question and could be endogenous in the child mortality regression. Ignoring this self-selection would result in biased estimates. One may adopt an instrumental variable estimation to address the potential problem of endogeneity though it is rather difficult to find appropriate instruments variables to solve the identification problem. Our approach to address this problem of endogeneity is to estimate child mortality jointly with mother's age at the birth of the child as well as use of some health inputs, namely, hospital delivery and child vaccination. In doing so we also allow for cross-correlations between the unobserved heterogeneity terms between child mortality on the one hand and mother's age at birth, hospital delivery and child vaccination on the other. In the absence of any better alternatives (instruments), we think that this approach will yield more efficient estimates. However note that identification in this framework does not depend only on the non-linear nature of the likelihood function – we also include a set of identifying variables in each of the equations that we estimate. We discuss this in detail below.

The rest of the paper is organised as follows. Section 2 discusses the methodology while section 3 describes the data. Section 4 presents and analyses the results and finally section 5 concludes.

2. Data & 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

Adolescent (teenage) childbearing is widely prevalent in our sample: as many as 76% of the sample 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 was 10.4% (the corresponding number for the full sample was 7.4%). As indicated earlier, this could be related to the use of health inputs. For example, 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 of 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.

Next we identify the socio-economic characteristics of parents experiencing adolescent childbirths from all other parents. This is summarised in Table 3, which focuses on differences in religion, literacy and women's say in various family decisions. The

reference group here is the couples having later childbirths (after the twentieth birthday of the mother). On an average, compared to couples having later childbirths, about 7% more Muslim women and 5% less Hindu women experience early childbirth. Thus it is more likely for a Muslim couple to have adolescent childbirth. The latter may be related to the contraceptive use among Muslims in general. Secondly, parental literacy levels, especially mother's literacy levels seem to be lower for the couples experiencing early childbirth. In other words, less educated women are more likely to have early childbirth, which is to be expected. The latter is again reflected in the women's say in female/child health care decisions. In particular, about 7-8% less women experiencing early childbirth have any say in female/child health care decisions.

As noted in the introduction, much has been written about the u-shaped relationship between the age of the mother at the time of the birth of the child and child mortality (or the inverse u-shaped relationship between mother's age at birth and child health). However most of the evidence on this u-shaped relationship pertains to data from developed countries. The issue has not been examined in great detail using data from developing countries. What is interesting is that at least in the context of Bangladesh we do not find evidence of this kind of a u-shape. The descriptive statistics, presented in Table 1, show that child mortality rates are indeed higher for young/adolescent/early mothers (aged below 20) but beyond the age of 20, child mortality rates are almost constant. It appears that early childbirth is a more serious problem than later childbirth. Part of the reason may be that very few births actually occurred once the woman is

more than 30 years old.[3] The rest of the paper thus focuses on the effects of early child bearing on child outcomes.

3. Estimation Methodology & Explanatory Variables

The main variable of interest in our analysis is child mortality. The unit of analysis is a child (j) born to a particular woman (w) residing in a particular household (h). Remember that there may be multiple children born to the same mother during the period under considering.

We model child mortality as a probit equation[4] where the dependent variable $CHDEAD$ is defined as follows:

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

This paper considers both infant and child mortality. Infant mortality refers to the case where the child dies before reaching his/her first birthday. Child mortality on the other hand considers the cases where the child dies before reaching his/her fifth birthday. Including both infant and child mortality allows us to distinguish biological factors from other socio-economic factors affecting child mortality. The estimating equation for child mortality is specified as follows:

$$CHDEAD = b Z_c + h_c + e_c \qquad (1)$$

where Z_c is a vector of individual (including health inputs provided), parental/household and other characteristics that can potentially affect child health and

[3] This is also corroborated by modelling mother's age at each birth as an ordered probit along with hospital delivery, child vaccination and child mortality equations. This clearly showed that effects of childbirth to mothers aged 20 or more were not significant in the mortality equation.
[4] Later we also estimate child mortality using a hazard model. See Section 4.6 and Table 10.

child mortality.[5] h_c captures mother 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 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 $h_c \sim N\left(0, s_c^2\right)$ is assumed to be uncorrelated with the other covariates. Finally all other residual variation is captured by e_c where $e_c \sim IIDN(0,1)$.

The set of explanatory variables (Z_c) includes individual, parental/household characteristics, health inputs and other community characteristics. The individual characteristics include a dummy for the male child, whether the child was born in a hospital, whether the child received any vaccination, a dummy to indicate whether age of the mother at the time of the birth of the child was less than 20. The parental/household level variables include the highest education attained by the mother and the father, a dummy for rural residence, household religion, a dummy to indicate whether the mother ever received tetanus vaccination and an index of household assets.[6] 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 as highlighted in Table 2A. In view of the evidence presented in Table 2B, we argue that, these region dummies control for the

[5] Note that we suppress both child (j) and mother (w) subscripts for notational convenience.
[6] 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.

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 explanatory variables above (Z_c) includes a number of individual (child specific) characteristics, including three binary variables indicating if the child was born before the twentieth birthday of the mother, if the child was delivered in a hospital and if the child was vaccinated against major childhood diseases, and several endogeneity issues arise in this context. The first relates to the age of the mother at the time of the birth of the child. As explained earlier, 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. 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 growth is incomplete is likely to bring disproportionate health risks for the child, e.g., low birth weight, which in turn may be responsible for early child death. It may also cause reproductive health hazards for the mother, e.g., haemorrhage, sepsis, pregnancy induced hypertension including preeclampsia, eclampsia, obstructed labour caused by cephalopelvic disproportion,

complications of unsafe abortion and iron deficiency (anaemia). Young mothers face greater risks than older women of hypertension, cephalopelvic disproportion, iron-deficiency and unsafe abortion. It is also argued that mother's age at birth of the child is also an indicator of the socio-economic status of the mother. Young mothers may be more likely to be less affluent, less educated and employed in positions with higher demands and lower control over their environment as compared to older mothers. There are also further socio-economic and personal consequences of early child bearing and it is important to distinguish these consequences from the initial disadvantages that may lead to early marriage/early pregnancy (often beyond the influence of the mother in patriarchal societies as in most parts of Bangladesh). We thus focus on analysing the effects of early pregnancy on child health. Accordingly we define a binary variable $EARLY$ as follows:

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

The estimating equation for $EARLY$ is:

$$EARLY = xZ_m + h_m + e_m \qquad (2)$$

where Z_m refers to a vector of explanatory variables that affect the probability of having an early childbirth, h_m captures unobserved heterogeneity and e_m captures any other residual variation. Here $h_m \sim N\left(0, s_m^2\right)$ and is uncorrelated with all other covariates and $e_m \sim IIDN(0,1)$. Equation (2) is estimated as a probit. See Table A1 for a list of the explanatory variables used.

It could be argued 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). Age at first birth is essentially a mother level fixed-effect. In an earlier version of the paper we had jointly estimated the age at first birth (as a continuous variable) with the use of health inputs and child mortality. However we could not, in that case, separate the component of the error due to the mother level unobserved heterogeneity from all unobserved determinants of the age at first birth. In this version we focus our attention on the mother's age at birth (of each individual child). Note that we were unable to include both age at birth and age at first birth as explanatory variables in the same regression because of convergence problems.

As noted in the introduction, there does not appear to be a u-shaped relationship between mother's age at birth and child mortality rates (see Table 1). 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 thus corroborated the descriptive statistics presented in Table 1.

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 are hence susceptible to increased risk of infections and hence child mortality. Other ways of reducing the risk of child mortality would be, for example, to vaccinate the mother with tetanus during pregnancy or to vaccinate the child after birth. Tetanus has long been a major killer of the newborn in Bangladesh, especially in rural areas. However this is preventable in newborns as immunity can be transmitted from mother to the child through the placenta. Two doses of tetanus toxoid vaccine given one month apart during pregnancy prevent nearly all tetanus infections in both mothers and their newborn children. If the mother received two doses less than 3 years earlier during a previous pregnancy, a single booster is adequate.

Yet another way of reducing child mortality rates is to provide the full set of recommended vaccination to the child. 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)). In addition, immunization has been shown to have large social externalities – for example the "herd immunity" effect of measles vaccination. 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. In South Asia by 1995-96, 93% of children had

received BCG immunization, 83% had been immunized by the DPV and OPV and 77% were immunized against measles. Unfortunately the program had a late start in Bangladesh – for example 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. Essentially, 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 mothers. It is likely that these women are those who anticipate complications at birth or other 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 lead to an under estimate of 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.[7] Ignoring favourable self-selection actually causes the effects of health inputs on birth outcomes to be overstated. What all this implies is that health inputs are endogenous in the health outcome (child mortality) regression. To account for this potential endogeneity, following Panis and Lillard (1994), we use a model where child health outcomes are jointly estimated with a behavioural model where the

[7] This definition of favourable self-selection is due to Gortmaker (1979). See also Panis and Lillard (1994) and Maitra (2004). .

health inputs are themselves choices. 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. This choice of health inputs is essentially driven by data availability. 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. If we indeed find that increased use of health inputs (like hospital delivery or child vaccination) have significantly positive effect on child health we would have identified important policy tools. There is however one caveat that one needs to mention: omitted variable bias. If there are other health inputs (which might be unobserved) that can affect child health and if these inputs are omitted from the set of explanatory variables that are used to explain child health, the beneficial effects of hospital delivery and child vaccination on child health could be over-estimated. One therefore needs to be careful in designing policies based solely on the results obtained.

We define 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, parental/household and other characteristics and a term that captures unobserved heterogeneity $(h_i; i = h, v)$ that is assumed to apply to all children born to the same mother. Denote $Z_i; i = h, v$ as the relevant vector

of explanatory variables in the equations characterising the decision to have the baby in a hospital $i = h$ and characterising the decision to vaccinate the child $i = v$. Once again the heterogeneity components are assumed to be uncorrelated with the other covariates and $h_i \sim N\left(0, s_i^2\right); i = h, v$. So the estimated equations are as follows:

$$HOSPDEL = a Z_h + h_h + e_h \tag{3}$$

and

$$VACCN = y Z_v + h_v + e_v \tag{4}$$

All other residual variation is captured by $e_i; i = h, v$ where $e_i \sim IIDN(0,1)$. We estimate *HOSPDEL* and *VACCN* as separate probits. See Table A1 for a list of the explanatory variables used.

3.2 Joint Estimation:

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

$$\int_{h_m}\int_{h_h}\int_{h_v}\int_{h_c}\left[\prod L^m(h_m)\prod L^h(h_h)\prod L^v(h_v)\prod L^c(h_c)\right] f(h_m, h_h, h_v, h_c)\, dh_m\, dh_h\, dh_v\, dh_c \tag{5}$$

where $f(h_m, h_h, h_v, h_c)$ is the joint distribution of the unobserved heterogeneity components. Here $f(h_m, h_h, h_v, h_c)$ is a four dimensional normal distribution characterised as follows:

$$\begin{pmatrix} h_m \\ h_h \\ h_v \\ h_c \end{pmatrix} \sim N\left(\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \begin{pmatrix} s_m^2 & & & \\ r_{mh} s_m s_h & s_h^2 & & \\ r_{mv} s_m s_v & r_{hv} s_h s_v & s_v^2 & \\ r_{mc} s_m s_c & r_{hc} s_h s_c & r_{vc} s_v s_c & s_c^2 \end{pmatrix}\right) \tag{6}$$

The model is estimated using Full Information Maximum Likelihood (FIML) Method. All the other variables as defined as above.

Remember that *EARLY*, *HOSPDEL* and *VACCN* are endogenous in the child mortality regression because the unobserved characteristics of the mother that affect these choices might also affect child mortality in other ways. Here we make an important assumption: we assume that all such unobservables are captured by the mother specific heterogeneity terms: h_m, h_h, h_v, h_c. However conditional on the h residuals the choices are independent of one another and of child mortality. So conditional on all the heterogeneity terms, the equations are independent and the conditional joint likelihood can be obtained by simply multiplying the individual log likelihoods. The marginal joint likelihood is obtained by integrating out the heterogeneity terms.

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) and private information about their own health and might choose to deliver their children in a hospital and/or choose to vaccinate their children. All of this essentially means that the correlation between the heterogeneity terms in the age at birth equation, the place of delivery equation, the vaccination equation and the child mortality equation could be non-zero: i.e., $r_{i,j} \neq 0; i, j = m, h, v, c; i \neq j$. It is however worth noting that the assumption that the h residuals (unobserved heterogeneity) capture all selection is a very strong one.

Typically the joint estimates give us the lower bounds for the effect of these endogenous variables on child mortality. An example will make this clear. 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. [8]

3.3 Identifying restrictions

Given the potential endogeneity issues involved, we need to choose as instruments variables that are highly correlated with the endogenous variable under consideration and not correlated with the unobserved determinants of child mortality. As instruments for early childbirth $(EARLY)$ we use three variables that are likely to affect the decision to have an early child (but are unlikely to have a direct effect on child mortality): the age difference between the mother and the father; whether the father is an unskilled agricultural labourer; and an indicator variable for first use of contraception.[9] A smaller age difference between the wife and the husband is typically indicative of increased balance of power within the marriage and is likely to be associated with early childbearing (in the case of the first child) or more children born before the woman turns 20 (in the case of multiple children). 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

[8] We would like to thank an anonymous referee for pointing this to us.
[9] Note that parental current use of contraception too could be regarded as an endogenous variable. In an attempt to reduce this potential endogeneity bias, we instead generate a binary variable called FSTUSE from the observation relating to number of children at first use of contraception. The variable takes a value 0 if parents do not indicate the number of children at first use.

se. Finally it is expected that contraceptive use should be highly (and negatively) correlated with early childbearing and there is no reason to assume that it will be directly correlated with child mortality.

The choice of instruments is difficult for the hospital delivery and child vaccination equations. We use bargaining power within the household for the wife as instruments in these two equations. Here we assume that relative bargaining power of the husband and the wife cannot directly affect child health (and child mortality) but can directly 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. For the hospital delivery equation we use as instrument "whether the woman has any say on female health care" and for the child vaccination equation we use as instrument "whether the woman has any say on child health care". For the child vaccination regression we also include an indicator dummy for firstborn male. In many societies, including those in South Asia firstborn males receive preferential treatment (in terms of inputs, health and educational) and this kind of parental preferences might be reflected in the fact that vaccination rates are higher for first-born males.

Finally in this correlated framework, identification of the child mortality equation is more obvious. In particular, mortality equation is identified by including a number of binary variables, namely, if the child is a male, if the child was born to a young mother and a number of health inputs use variables. The latter included whether the child was delivered in a hospital, whether the child received vaccination and whether the mother received tetanus vaccination while pregnant.

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. The estimated equation in this case is $CHDEAD = b Z_c + e_c$, essentially a restricted version of equation (1). 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. So the estimating equation is now given by equation (1). Specification 2 corresponds to the case zero correlations between the unobserved mother-specific heterogeneity terms $r_{mc} = r_{hc} = r_{vc} = 0$. 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 $r_{mc} \neq 0$, but $HOSPDEL$ and $VACCN$ are assumed to be exogenous (i.e., $r_{hc} = r_{vc} = 0$). In *specification 4, EARLY* is assumed to be exogenous (i.e., $r_{mc} = 0$) but $HOSPDEL$ and $VACCN$ are assumed to be endogenous so that we allow for the possibility that $r_{hc} \neq 0; r_{vc} \neq 0$. Here we also allow for the possibility that $r_{hv} \neq 0$. Finally in *specification 5* (corresponding to the complete specification) $EARLY$, $HOSPDEL$ and $VACCN$ are all assumed to be endogenous so that $r_{mc} \neq 0, r_{hc} \neq 0, r_{vc} \neq 0$.

4.1 Unobserved Heterogeneity:

Table 4 presents the estimates for the unobserved heterogeneity components. Note that these estimates correspond to the complete model (*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 safely argue that ignoring unobserved heterogeneity and the correlation between the unobserved heterogeneity coefficients would result in inconsistent 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, to highlight the consequences of ignoring unobserved heterogeneity.

4.2 Child Mortality:

We start with a discussion of the estimates for the child mortality equations. The coefficient estimates and the associated standard errors are presented in Table 5. A positive (negative) and statistically significant coefficient estimate in the probit estimation of child mortality implies that the particular explanatory variable increases (decreases) the probability of child mortality.

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. Surprisingly, however, the estimate from the complete specification 5 implies that early childbirth has a negative but statistically insignificant effect on the probability of child mortality. This is quite a surprising result, especially in view of Table 1 that clearly suggests that the unconditional child mortality rates are higher for early childbirths. Thus after correcting for the selectivity bias, the adverse effects of early childbirth on child mortality turns out to be rather insignificant in our sample.

So how do we explain this rather surprising result? We believe that this is partly explained by the fact that 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. In order to investigate this, we jointly estimated (a) *EARLY* (equation (2)) and *HOSPDEL* (equation (3)) and also (b) *EARLY* (equation (2)) and *VACCN* (equation (4)). So, in addition to the set of explanatory variables included in Z_h and Z_v here we also included *EARLY* as an additional explanatory variable in each of HOSPDEL and VACCN equations.[10] The coefficient estimates for *EARLY* in the two regressions are presented in Table 6. Note that we estimate (and present) three different specifications: first where *EARLY* is exogenous in the *HOSPDEL* and *VACCN* regressions and we

[10] A discussion of the probit regression results for *HOSPDEL* and *VACCN* are presented below.

do not account for 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 $r_{mh} \neq 0$ and $r_{mv} \neq 0$). When we allow for endogeneity of early childbirth in the health input regressions, 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. These estimates essentially indicate that women who have children early behave quite differently compared to others, at least in terms of use of health inputs.

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). 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 under estimated when we do not take into account 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.

The probability of child mortality is significantly lower if the mother has ever received tetanus vaccination. In South Asia, tetanus has long been a major killer of newborn and very young children. It has been documented that two doses of the tetanus toxoid vaccine given to the mother when she is pregnant prevents nearly all tetanus infections in both the mother and the newborn child. Evidence from neighbouring India shows that the maternal tetanus immunisation program instituted by the Government of India in 1975-76 has had significant effects on both neo-natal and early childhood mortality (Luther (1998)). We do not have data on whether the mother received tetanus vaccination when pregnant with each child – rather we have data on whether the woman ever received tetanus vaccination. It is worth noting however that the mother ever receiving tetanus vaccination is 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. Our results are indicative of a strong relationship between maternal tetanus vaccination and child health. Alternatively the woman ever receiving tetanus vaccination could be viewed as a proxy for increased overall awareness of the woman on matters regarding health. There is a flow through effect of this increased awareness to better health outcomes for the child, which has nothing to do with the effect of maternal tetanus vaccination per se.

The estimation results indicate that parental educational attainment does not generally have a particularly strong effect on child mortality. 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. However one might note that the parental educational attainment dummies included as explanatory variables in the child mortality regressions capture only the direct effect of parental education on child mortality, which turns out to be not particularly strong. However there are indirect effects: parental education has significant effects on early childbirth, hospital delivery and child vaccination. We will discuss the results below (sections 4.3 – 4.5). However the fact that even the direct effect of the mother having more than primary school is statistically significant in the child mortality regressions underscores 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 however also indicative of a threshold level of education that must be attained before educational attainment starts having a statistically significant effect on child mortality.

Interestingly the sex of the child, the religion of the household (Muslim household), wealth of the household (captured by a composite asset index) or sector (rural/urban) of residence do not have statistically significant effects on child mortality (irrespective of the estimation method used). 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 the child is the only child. Our results are therefore indicative of significant life-cycle effects on child mortality.

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 Khulna (the reference category), child mortality rates are higher in other regions (this corroborates the descriptive statistics presented in Table 2A). We argue that these region dummies capture 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 (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.

4.3 Early Childbirth

The coefficient estimates for the probit estimates of early childbirth are presented in Table 7. We present the coefficient estimates of *EARLY* obtained from maximizing the joint likelihood function in equation (4). Educational attainment of the woman has a statistically significant effect on the age at birth. However the sign of the estimates are quite unexpected. The probability of early childbirth is positive and statistically significant for educated women compared to women who have no education. Though it is interesting to note that the coefficient estimate on more than primary schooling is smaller than the coefficient estimate on less than primary schooling, implying that while some education attained by the woman has a stronger effect on the probability of early childbirth, the effect becomes weaker for more educated women.

Some (less than primary) educational attainment of the husband actually does not have a statistically significant effect on the probability of early childbirth, but interestingly more than primary schooling attained by the husband actually reduces the probability of early childbirth. How can one interpret this result? The father's educational attainment might be viewed as a proxy for household permanent income (particularly in the absence of any data on household income/expenditure). The permanent income of the household is likely to be higher when the husband has more than primary schooling and hence one way of interpreting these results is that child bearing is delayed by women who reside in richer households. This result is confirmed by the negative and statistically significant coefficient associated with the asset index of the household. The probability of early childbirth is significantly higher for Muslim women and for women who reside in rural areas. Relative to the reference category, probability of early childbirth is significantly higher for women that are married to

men whose primary occupation is farming or unskilled labourer. Increased age difference between the husband and the wife is associated with an increase (weakly significant) in the probability of early childbirth. Not surprisingly, contraception use significantly reduces the probability of early childbirth. Finally several of the provinces of residence dummies are statistically significant indicating that there is fairly significant regional variation in the probability of having a child early.

4.4 Hospital Delivery:

We now turn to the results from the hospital delivery probit regression, which are presented in Table 8. These are the coefficient estimates of *HOSPDEL* obtained from maximizing the joint likelihood function in equation (4). The probability of hospital delivery is significantly higher if the mother has more than primary schooling, and it is worth noting that the coefficient estimate of mother's education being less than completed primary school is negative though not statistically significant. The results are therefore again indicative of a threshold level of education that must be attained before mother's education starts having a statistically significant effect on the probability of delivering the child in a hospital. The probability of hospital delivery is again positive and statistically significant if the husband has more than primary schooling and interestingly the husband having less than primary schooling actually has a negative, though not statistically significant, effect on the probability of hospital. The fact that more than primary schooling for both the mother's and the father's educational attainment have positive and statistically significant effect on the probability of hospital delivery could be indicative of some form of assortative matching in the marriage market – more educated men are more likely to choose to

marry more educated women and both parents are therefore likely to be aware of the potential benefits of choosing to deliver the child in a hospital. Additionally, as in the early childbirth regression, the father's educational attainment might be viewed as a proxy for household permanent income. Richer households might be more aware of the health services available and also maybe better able to afford to choose hospital delivery. This result is confirmed by the positive and statistically significant coefficient associated with the asset index of the household.

The probability of choosing hospital delivery is greater if the woman has any say (either independently or jointly with her husband) on matters relating to the health of the female members in the household. Having a greater say on health care matters can be viewed as a proxy for greater power within the household. One might therefore argue that increased power for women within the household is likely to be associated with increased probability of choosing hospital delivery. This is nothing new and there is actually a fairly long literature that shows that increased power for women in household decision making is associated with increased allocation of resources (health, educational and nutritional) for the next generation within the household. The probability of hospital delivery is significantly lower for women residing in rural areas and this is possibly a reflection of the lack of adequate health services in rural regions.

4.5 Child Vaccination:

We now turn to the child vaccination probit results, presented in Table 9. Surprisingly more than primary schooling of the mother is associated with a statistically significant reduction in the probability of child vaccination. However, more than primary

schooling for the father is associated with a statistically significant increase in the probability of child vaccination. Once again this could be thought of as an income effect, though in this case the composite asset index of the household is not statistically significant. The probability of child vaccination is greater if the woman has any say (either independently or jointly with her husband) on matters relating to the health of the children. Once again we could view this as a bargaining power effect: a confirmation of the hypothesis that increased power for women in household decision making is associated with increased allocation of resources (health, educational and nutritional) for the next generation within the household. The probability of child vaccination is higher if the woman resides in a rural region. While this is a rather surprising result, it could be the result of EPI programme of the WHO, which specifically aimed at increasing the vaccination rate among children in rural households.

The sign and significance of the birth order variables are interesting. The probability of child vaccination is significantly lower if the child is the youngest child or is the only child. We include a dummy variable to indicate whether the child is the first-born male – to account for the possibility that the first-born male might be given more preferential treatment. In many societies the firstborn male have a "special place" – he has the responsibility of taking care of his parents when they are old and also has to light the "funeral pyre". We do not find any evidence of any preferential treatment for the firstborn males (at least in terms of vaccination) in our sample.

Now the decision on the place of delivery is made prior to the birth of the child (and to the extent that advanced ultra-sound facilities are not readily available, parents are unlikely to know the sex of the yet unborn child) and the decision on vaccination is made after the child is born. So by looking at the choice of these two inputs one can determine whether there is a change in parental preferences once the child is born. This is an important issue in the context of South Asia where son preference is quite prevalent. This preference for sons often takes the form of significantly lower inputs (health, educational and nutritional) to girls. We re-estimated the child vaccination equation but this time we included a male dummy in the set of explanatory variables. This variable turned out to be not statistically significant, implying that there is no difference in vaccination rates between boys and girls in Bangladesh. This is quite an interesting result and shows that the behaviour of parents is quite different from other parts of South Asia – for example data from neighbouring India seem to indicate that controlling for other individual, household and community characteristics girls are less likely to be immunized. (See for example Pande (2003) and Maitra (2004)).

4.6 Modelling Child Mortality using a Hazard Model

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. We use a proportional hazard model with covariates and unobserved heterogeneity shifting the baseline hazard. The log hazard of mortality for a child at time t is given by:

$$\ln h(t) = g_0 + g_1 T(t) + g_2 Z_c + h_c + e_c \qquad (2)$$

where once again Z_c is a vector of individual (including health inputs provided), parental/household and other characteristics that can potentially affect child health and child mortality; h_c captures mother level unobserved heterogeneity that affects the health of all children born to the same woman and all other residual variation is captured by e_c where $e_c \sim IIDN(0,1)$. The set of variables included in Z_c are the same as those included in the probit estimation of child mortality. $T(t)$ is a spline in time beginning with the time the child enters the risk of dying (in this case the moment the child is born). Denote the time at which the child enters the risk of dying by t_0 and subdivide the duration $t - t_0$ into K discrete periods. Then the baseline log hazard function is defined as a spline or a piecewise linear function and the log hazard of the event will have different slopes over the duration. The baseline hazard function can be written as:

$$g_0 + g_1 T(t) = g_0 + \sum_{k=1}^{K} g_{1k} T_k(t)$$

The baseline hazard function is therefore the sum of the effects of the various sources of time dependence within the period of risk for an individual and the resulting log hazard equation is piecewise linear in time since the individual enters the risk of the event. The conditional likelihood of child mortality is given by:

$$\tilde{L}^C(h_c) = \begin{cases} S^c = \Gamma\left(t, Z\left(t^c\right), h_c\right) & \text{if censored} \\ S^u = \Gamma\left(t, Z\left(t^u\right), h_c\right) & \text{if uncensored} \end{cases}$$

Remember that 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.

As before when early childbirth $(EARLY)$, hospital delivery $(HOSPDEL)$ and child vaccination $(VACCN)$ are treated as endogenous in the child mortality hazard regression, the joint marginal likelihood function can be written as:

$$\int_{h_m}\int_{h_h}\int_{h_v}\int_{h_c}\left[\prod L^m(h_m)\prod L^h(h_h)\prod L^v(h_v)\prod L^c(h_c)\right] f(h_m,h_h,h_v,h_c)dh_m dh_h dh_v dh_c \quad (7)$$

where $f(h_m,h_h,h_v,h_c)$ is the joint distribution of the unobserved heterogeneity given by equation (6).

The FIML estimates of equation (7) for the child mortality hazard regressions are presented in Table 10. The results that we present therefore correspond to the specification where *EARLY*, *HOSPDEL* and *VACCN* are all regarded as endogenous in the child mortality regressions (the complete specification 5). The results for the early childbirth (probit), hospital delivery (probit) and child vaccination (probit) are available on request, as are the hazard estimates for child mortality when *EARLY*, *HOSPDEL* and *VACCN* are assumed to be exogenous (with and without unobserved heterogeneity).

A negative coefficient estimate implies that the relevant variable reduces the hazard of child mortality (and increases the duration the child was alive) while a positive coefficient estimate implies that the relevant variable increases the hazard of child mortality (and decreases the duration the child was alive). The baseline hazard model is estimated as a piece-wise constant log hazard model with one node at 6 months i.e., there are two intervals (0, 6) and (6+). The slope of the log hazard is assumed to be constant within each interval but may vary between the intervals. The configuration of

signs of DUR06 and DUR6+ indicate that the hazard of child mortality is increasing in the first 6 months following childbirth but is decreasing thereafter.

Effects of early childbirth and use of health inputs seem to be similar to those obtained from the child mortality probit equation (Specification 5, Table 5) – early childbirth reduces the hazard of child mortality (significant only at 10% level), as does hospital delivery (though it is not statistically significant) and child vaccination (significant at 1% level). Taken together, these selectivity corrected estimates tend to suggest that the adverse effects of early childbirth on child mortality could be mitigated significantly if couples are encouraged to make use of available health inputs.

5. Conclusion

Though adolescent child bearing is widely prevalent in many low-income regions with high child mortality, there is very little systematic investigation of the relationship between mother's age and child mortality in these regions. This paper examines the relationship between early childbearing and child mortality in Bangladesh, a country where adolescent childbearing is of growing concern. In doing so it specifically focuses on the role of available health inputs on child health (and child mortality), addresses the implicit problems of endogeneity and self-selection and identifies convenient policy instruments.

However, mother's age at birth as well as use of various health inputs, e.g., hospital delivery, child vaccination, are chosen by the couple in question i.e., are potentially endogenous. Failure to account of the potential endogeneity could result in biased

estimates. In an attempt to reduce this bias we jointly estimate child mortality, mother's age at birth, and demand for health inputs, allowing for the cross-correlation between the unobserved components of the residual terms in these equations. These estimates suggest that there are significant correlations between these equations so that these correlated estimates are an improvement over the uncorrected estimates of child mortality.

There is evidence of significant self-selection in our sample in that women having early childbirth tend to use health inputs differently from all other women. While uncorrected estimates of child mortality emphasize the adverse effects of early childbirth on child mortality, this effect tends to be much less pronounced, once we take account of the possible endogeneity of early childbirth and use of health inputs on child mortality. More interestingly, these estimates suggest that use of health inputs is one possible way of mitigating the adverse effects of early childbirth. In particular, there is strong evidence that children delivered in hospitals as well as vaccinated against major childhood diseases have better chances of survival.

From a policy point of view this is an extremely important issue. 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 human capital formation. The finding that the adverse effect of adolescent childbirth on child survival is correlated with the use of health

input like hospital delivery implies that one has in principle identified a convenient policy instrument of encouraging use of health inputs among young mothers rather than 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

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	Head count index of poverty	Access to sanitary latrine		Access to safe drinking water		Rate of Immunization DPT 12-23 months		Govt. expenditure on health Taka 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); Ranna-Eliya and Somanathan (2003)

Table 3: Selected Parental Characteristics

	Adolescent birth	Non-adolescent birth
Muslim	0.9152	0.8590
	(0.28)	(0.348)
Hindu	0.0805	0.1267
	(0.27)	(0.33)
Mother has primary or higher schooling	0.5162 (0.499)	0.5703 (0.50)
Father has primary or higher schooling	0.5297 (0.4)	0.6310 (0.48)
Mother's education in single years	2.54	3.78
	(3.1)	(4.237)
Say in female health care	0.4284	0.5007
	(0.49)	
Say in child health care	0.4975	0.5828
	(0.50)	(0.49)

Table 4: Structure of Unobserved Heterogeneity

Child Mortality estimated using a Probit model

	Early Childbirth	Hospital Delivery	Child Vaccination	Child Mortality
Early Childbirth (h_m)	1.4139 *** (0.0585)			
Hospital Delivery (h_h)	-0.2985 *** (0.0418)	2.0171 *** (0.1688)		
Child Vaccination (h_v)	0.0153 (0.0383)	-0.1016 * (0.0528)	0.8035 *** (0.0548)	
Child Mortality (h_c)	0.2699 ** (0.1193)	0.3592 *** (0.1367)	0.6741 *** (0.1120)	1.1241 *** (0.1678)

Notes:
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.

Table 5: Probit Estimates of Child Mortality

	All Exogenous (No Unobserved Heterogeneity)	All Exogenous (With Unobserved Heterogeneity)	Early Child birth Endogenous	Health Inputs Endogenous	All Endogenous
Constant	-0.6971 ***	-0.9840 ***	-0.8944 ***	-0.3619	-0.2876
	(0.1596)	(0.2572)	(0.2537)	(0.2642)	(0.2619)
Male Child	0.0525	0.0903	0.1015	0.0866	0.0977
	(0.0625)	(0.0859)	(0.0854)	(0.0898)	(0.0896)
Oldest Child	0.2080	0.3392 *	0.4341 **	0.3114	0.3979 **
	(0.1364)	(0.1903)	(0.1940)	(0.1950)	(0.1996)
Youngest Child	-0.6624 ***	-0.8292 ***	-0.7975 ***	-0.9291 ***	-0.8862 ***
	(0.1031)	(0.1399)	(0.1376)	(0.1431)	(0.1418)
Only Child	-0.4870 ***	-0.5872 ***	-0.5366 ***	-0.6728 ***	-0.6012 ***
	(0.1194)	(0.1632)	(0.1613)	(0.1703)	(0.1697)
Education of Mother Less	-0.0877	-0.1302	-0.0866	-0.1804	-0.1367
than Primary School	(0.0813)	(0.1196)	(0.1199)	(0.1262)	(0.1269)
Education of Mother More	-0.2072 **	-0.2903 **	-0.2544 **	-0.3390 **	-0.2992 **
than Primary School	(0.0822)	(0.1226)	(0.1222)	(0.1323)	(0.1315)
Education of Father Less	0.0108	0.0114	0.0148	-0.0078	-0.0024
than Primary School	(0.0739)	(0.1083)	(0.1078)	(0.1154)	(0.1145)
Education of Father More	-0.1093	-0.1682	-0.2221 *	-0.0672	-0.1124
than Primary School	(0.0844)	(0.1226)	(0.1234)	(0.1316)	(0.1321)
Muslim	-0.0671	-0.0570	-0.0100	-0.1137	-0.0737
	(0.0899)	(0.1328)	(0.1334)	(0.1413)	(0.1427)
Asset Index	-0.0521	-0.0755	-0.1027 *	-0.0205	-0.0426
	(0.0382)	(0.0552)	(0.0562)	(0.0602)	(0.0610)
Rural Resident	0.0025	-0.0102	0.0160	-0.0646	-0.0327
	(0.0662)	(0.0964)	(0.0958)	(0.1133)	(0.1093)
Mother Ever had Tetanus	-0.3012 ***	-0.4556 ***	-0.4635 ***	-0.5337 ***	-0.5452 ***
Vaccination	(0.0879)	(0.1263)	(0.1253)	(0.1338)	(0.1329)
Early Child Birth	0.1557 ***	0.1742 **	-0.3008	0.1973 **	-0.2491
(EARLY)	(0.0587)	(0.0846)	(0.1933)	(0.0889)	(0.2006)
Hospital Delivery	0.1710 *	0.2407 *	0.2448 *	-0.4808	-0.5037 *
(HOSPDEL)	(0.1007)	(0.1435)	(0.1420)	(0.3150)	(0.3029)
Child Vaccination	-2.6387 ***	-3.5664 ***	-3.5683 ***	-4.5209 ***	-4.4690 ***
(VACCN)	(0.2075)	(0.3922)	(0.3945)	(0.5234)	(0.5181)
Resident of Barisal	0.3434 ***	0.4830 **	0.4766 **	0.4557 **	0.4629 **
	(0.1255)	(0.1926)	(0.1914)	(0.2008)	(0.1987)
Resident of Chittagong	0.2230 **	0.3199 *	0.3101 *	0.3216 *	0.3205 *
	(0.1095)	(0.1637)	(0.1615)	(0.1743)	(0.1718)
Resident of Dhaka	0.4271 ***	0.5790 ***	0.5740 ***	0.5975 ***	0.6006 ***
	(0.1046)	(0.1625)	(0.1608)	(0.1697)	(0.1685)
Resident of Rajsahi	0.3600 ***	0.4905 ***	0.5233 ***	0.5332 ***	0.5589 ***
	(0.1099)	(0.1695)	(0.1690)	(0.1779)	(0.1775)
Resident of Sylhet	0.4529 ***	0.6306 ***	0.6199 ***	0.5887 ***	0.5731 ***
	(0.1106)	(0.1741)	(0.1725)	(0.1812)	(0.1795)
Log Likelihood	-12038.78	-11638.02	-5498.59	-7314.88	-11591.61

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

Table 6: 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 7: Probit Estimates for Early Childbirth

	Estimates obtained from Full Specification
Constant	-0.8120 ***
	(0.1583)
Education of Mother Less than Primary School	0.4000 ***
	(0.0892)
Education of Mother More than Primary School	0.3100 ***
	(0.0865)
Education of Father Less than Primary School	0.0162
	(0.0820)
Education of Father More than Primary School	-0.4177 ***
	(0.0895)
Age Difference between Mother and Father	0.0003 *
	(0.0002)
Contraceptive Use	-0.1922 **
	(0.0763)
Muslim	0.4832 ***
	(0.0939)
Asset Index	-0.2023 ***
	(0.0404)
If Father is Unskilled Labourer	0.1466 **
	(0.0724)
Rural Residence	0.2134 ***
	(0.0715)
Resident Of Barisal	-0.0864
	(0.1282)
Resident of Chittagong	-0.1730 *
	(0.1043)
Resident of Dhaka	-0.1938 *
	(0.1006)
Resident of Rajsahi	0.1407
	(0.1067)
Resident of Sylhet	-0.3009 **
	(0.1195)

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

Table 8: Probit Estimates for Hospital Delivery

	Estimates obtained from Full Specification
Constant	-2.2482 ***
	(0.3368)
Oldest Child	0.9819 ***
	(0.2528)
Youngest Child	0.6395 ***
	(0.2162)
Only Child	1.5198 ***
	(0.2586)
Education of Mother Less than Primary School	-0.0720
	(0.1986)
Education of Mother More than Primary School	0.5237 ***
	(0.1888)
Education of Father Less than Primary School	-0.1218
	(0.1890)
Education of Father More than Primary School	0.6432 ***
	(0.1849)
Muslim	-0.8268 ***
	(0.1843)
Asset Index	0.7361 ***
	(0.0849)
Rural Residence	-1.8279 ***
	(0.1746)
If Woman has say on Female Health Care	0.3550 ***
	(0.1224)
Resident Of Barisal	-1.3259 ***
	(0.2626)
Resident of Chittagong	-1.1171 ***
	(0.2076)
Resident of Dhaka	-0.7047 ***
	(0.1833)
Resident of Rajsahi	-0.4339 **
	(0.1873)
Resident of Sylhet	-0.4589 *
	(0.2432)

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

Table 9: Probit Estimates for Child Vaccination

	Estimates obtained from Full Specification
Constant	0.0435
	(0.1103)
Oldest Child	-0.0501
	(0.1044)
First Born Male	-0.1426
	(0.1276)
Youngest Child	-0.2260 ***
	(0.0554)
Only Child	-0.3752 ***
	(0.0692)
Education of Mother Less than Primary School	-0.0721
	(0.0614)
Education of Mother More than Primary School	-0.2572 ***
	(0.0623)
Education of Father Less than Primary School	0.0170
	(0.0560)
Education of Father More than Primary School	0.1957 ***
	(0.0634)
Muslim	0.0556
	(0.0661)
Asset Index	0.0025
	(0.0271)
Rural Residence	0.2254 ***
	(0.0500)
If Woman has say on Child Health Care	0.1269 ***
	(0.0424)
Resident Of Barisal	0.0201
	(0.0871)
Resident of Chittagong	0.1525 **
	(0.0700)
Resident of Dhaka	0.1872 ***
	(0.0688)
Resident of Rajsahi	0.0944
	(0.0716)
Resident of Sylhet	-0.1704 **
	(0.0800)

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

Table 10: Hazard Estimates of Child Mortality

	All Endogenous
DUR_0_6	0.5238 ***
	(0.0672)
DUR6+	-0.6133 ***
	(0.0638)
Constant	-5.2101 ***
	(0.5011)
Male Child	0.1029
	(0.1279)
Oldest Child	0.4592 *
	(0.2450)
Youngest Child	-1.2421 ***
	(0.2059)
Only Child	-0.7516 ***
	(0.2474)
Education of Mother Less than Primary School	-0.1413
	(0.1746)
Education of Mother More than Primary School	-0.3730 **
	(0.1781)
Education of Father Less than Primary School	0.0696
	(0.1555)
Education of Father More than Primary School	-0.1237
	(0.1837)
Muslim	-0.0477
	(0.1969)
Asset Index	-0.0719
	(0.0828)
Rural Resident	0.1010
	(0.1489)
Mother Ever had Tetanus Vaccination	-0.5465 ***
	(0.1872)
Early Child Birth (EARLY)	-0.5185 *
	(0.2787)
Hospital Delivery (HOSPDEL)	-0.4941
	(0.3763)
Child Vaccination (VACCN)	-7.5757 ***
	(0.8432)
Resident of Barisal	0.5236 *
	(0.2754)
Resident of Chittagong	0.5206 **
	(0.2342)
Resident of Dhaka	0.8939 ***
	(0.2249)
Resident of Rajsahi	0.7295 ***
	(0.2364)
Resident of Sylhet	0.7713 ***
	(0.2357)
Log Likelihood	-12285.41

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

Table A1: Explanatory Variables Used

	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	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
If Woman has say on Female Health Care		X		
If Woman has say on Child Health Care			X	
Age Difference between Mother and Father	X			
If Father is Unskilled Labourer	X			
Contraceptive Use	X			
Province Dummies	X	X	X	X