Cause-specific Neonatal Deaths: Levels, Trend and Determinants in Rural Bangladesh, 1987-2005¹

Unnati Rani Saha², Arthur van Soest³ and Govert E. Bijwaard⁴

January 2012

Running title: Cause-specific neonatal deaths

Key words: millennium development goals, neonatal deaths, competing risks models, unobserved heterogeneity, Bangladesh

¹ We thank Katie Carman, Sabu Padmadas and Frederic Vermeulen for their valuable comments that helped to improve the paper.

² Tilburg University, 5037 ÅB, 5000 LE Tilburg, The Netherlands; ICDDR,B, Dhaka, Bangladesh; Emails:rinku7us@yahoo.com; unnati.saha01@gmail.com

³ Tilburg University, 5037 AB, 5000 LE Tilburg, The Netherlands; Email:avas@uvt.nl ⁴ Netherlands Interdisciplinary Demographic Institute (NIDI); Email: <u>bijwaard@nidi.nl</u>

Abstract

Reducing neonatal mortality is a particularly important issue in Bangladesh. We employ a competing risks model incorporating both observed and unobserved heterogeneity and allowing the heterogeneity terms for various causes to be correlated. Data come from the Health and Demographic Surveillance System (HDSS), Matlab. The results confirm the general conclusion on levels, trends and patterns of causes of neonatal deaths in the existing literature, but also reveal some remarkable socioeconomic differences in the risks of cause-specific deaths. Deaths due to low birth weight and other causes (sudden infant death, unspecified or specified) are better explained from the socio- economic covariates than deaths due to neonatal infections or obstetric complications. The analysis highlights the role of maternal and child health interventions (particularly tetanus toxoid immunization for pregnant women, nutrition programs, and high coverage health services: distance to nearest health centre). Policies that increase quality and equity in child births may help to further reduce neonatal mortality.

1. Introduction

Of the 130 million children born alive each year worldwide, about four million die in the first four weeks after birth. 99% of these deaths occur in low and middle income countries; 4% occur in Bangladesh (Lawn et al. 2005). Achieving the fourth Millennium Development Goal (MDG-4) of reducing under-5 child mortality by two-thirds between 1990 and 2015 remains one of the United Nations' global priorities (United Nations 2001). The recent trends in mortality suggest that without substantial reductions in neonatal mortality, MDG-4 will not be achieved (Lawn et al. 2005). Global reviews suggest that almost 60% of childhood deaths can be prevented by increasing the coverage of existing newborn and child health interventions (Jones et al. 2003). The information on causes of neonatal and child death is important here, since - it can be used to prioritize and to increase the effectiveness of disease-specific interventions (Baqui et al. 2001; Lawn et al. 2006).

Reducing neonatal mortality is a particularly important issue in Bangladesh. Although child mortality rates in Bangladesh declined sharply during the last decades of the previous century, the reduction is slowed down particularly in the neonatal period. Among child deaths, those that occur during the first month represent an increasing proportion. Estimates based upon the Bangladesh Demographic and Health Survey (BDHS) suggest that 70% of all under-five deaths occur in the first year of life and 80% of these occur in the neonatal period (see Figure 1). It is therefore a significant challenge to reduce neonatal mortality in order to meet MDG-4 of reducing under-five mortality from 133 per 1,000 live births to two-thirds between 1990 and 2015. This study analyzes the levels and the trend of cause-specific neonatal deaths in Bangladesh and associated risk factors, both observed and unobserved. The findings may help to design policies that reduce neonatal mortality in Bangladesh in particular, but also are potentially relevant for many other countries in the developing world.

We estimate a flexible competing risks model of causes of death until 28 days after birth (neonatal deaths), considering children who survive the neonatal period as censored observations. Our modelling approach is more flexible than in many existing studies of determinants of causes of deaths. It combines a piecewise constant baseline hazard with proportionality assumptions concerning the influence of observed and unobserved risk factors for each cause of death. The model allows the unobserved heterogeneity components in the hazard rates for the various causes of death to be correlated.

Our estimations are based upon prospective panel data from the Matlab region in Bangladesh, following mothers and children over time from 1987 until 2005. Two sets of villages are covered: an intervention area with non-standard health services (International Centre for Diarrhoeal Disease Research, Bangladesh or ICDDR,B area), and an area with standard government-provided health care facilities (comparison area); the differences between the two areas give insight into how the additional health care services shape the child health epidemiology over the period.

2. Background

The disease structure of neonatal deaths is different from that of post-neonatal deaths or deaths of children older than one year (Bhatia 1989). Neonatal deaths are associated with biological characteristics of the mother and with problems during pregnancy and child birth, which can be improved by targeted interventions such as tetanus toxoid to pregnant women, nutrition education, and increasing use of antenatal care, or by ensuring safe delivery. On the other hand, socio-economic and programmatic factors that focused on reducing post-neonatal and child deaths become more important during the post-neonatal period, when deaths are more often caused by infectious diseases or accidents. For example, it is obvious that immunization of the children or oral rehydration therapy will not prevent deaths during the

neonatal period. It is, therefore, more useful to work with separate models for the determinants of competing risks of neonatal and post-neonatal death. In this study, we only consider the neonatal period.

In many countries, information on causes of death is not available. Verbal autopsy (VA) is a tool used in a retrospective interview with family members about the circumstances of a death to ascertain the underlying cause of death (Chen et al. 1980; Bhatia 1989; Baqui et al 2001; Karar et al. 2009). The interview is usually held 22 days after the date of death with the mother, or a close relative or neighbour in absence of the mothers (Karar et al. 2009). VA is not often used because it can be prohibitively expensive or difficult. The Health and Demographic Surveillance System (HDSS) of ICDDR,B in Matlab, Bangladesh, however, routinely records all births, deaths and causes of deaths through VA for a total population of about 220,000 (ICDDR,B 2006). It also incorporates information on several indicators of socioeconomic status of each household. The Matlab HDSS plays an important role in providing accurate information on vital events (e.g. births, deaths) and causes of death that are not often available in many resource-constrained setups.

In 1977, ICDDR,B started to provide extensive maternal-child health and family planning (MCH/FP) services, in addition to existing government health services, in half of the HDSS area called ICDDR,B area. The other half, called the comparison area, continued to get only the standard government health services. The MCH/FP project includes provision of domiciliary family planning services, simple nutrition education, tetanus toxoid immunization for pregnant women (which was modified in 1981 to include all women of reproductive age), community-based oral rehydration therapy, and measles immunization. These services were introduced incrementally phase by phase (Phillips et al 1984). In the ICDDR,B area there are several ICDDR,B sub-centres providing treatment for minor

illnesses and, basic emergency obstetric care (EOC), and a permanent hospital that provides treatment for diarrhoeal diseases. In order to understand the way in which better health services shape child health, we analyze the data from the area with the better health care services in addition to the government health services (ICDDR,B area) as well as the data from an area with standard government health services only (the comparison area - a typical rural area of Bangladesh).

Most of the studies of cause-specific neonatal deaths in developing countries, including those in Matlab, Bangladesh, reported neonatal mortality rates by age-group, giving insight in when and why the child deaths occur (Chen et al. 1980; Bhatia 1989; Baqui et al. 2001; Lawn et al. 2006; Karar et al. 2009; Chowdhury et al. 2010). The trends in mortality, however, reveal that factors other than the health and family planning interventions influence the levels of mortality in the ICDDR,B and comparison areas (Bhatia 1989). To strengthen the targeting of interventions, it may therefore be important to analyze what characterizes families and children at risk of neonatal death due to various causes. In other words, what are the underlying factors that are associated with cause-specific deaths? Our study contributes to answering this question.

The analysis of cause-specific death is related to the concept of competing risks (Cornfield 1957; Fine and Gray 1999; Coviello and Boggess 2004). In a competing risk situation, the analysis of one specific cause of death has to account for competing causes of death. For example, in one study it was found that the probability of a female developing cancer at some point during her life has increased by 25 per cent over a seven years period, but the largest part of this increase was accounted for by decreases in other causes of mortality (Goldberg et al. 1956). To our knowledge, our study is the first that applies this type of model to neonatal mortality Bangladesh.

Duration models are often used in demographic and epidemiological research where the events to be modelled are associated with time, such as time till marriage, time from marriage till birth of the first child, or time till death (Cox 1959; Heckman et al. 1985). Analyzing the duration to competing causes of death leads to knowledge about when (time) and why (disease types) deaths occur, which is useful for targeting policies of early prevention.

Studies of child deaths have shown that survival chances of several children in a family are correlated and that variation in death risks across families remains after controlling for observed covariates (such as age of mother at birth, gender of the child, or race), and can be attributed to unobserved family level heterogeneity. Examples are adverse genetic traits, inability to take care of the child (behavioural factors), or (unobserved) environmental factors (Mosley and Chen 1984; DasGupta 1990; Arulampalam and Bhalotra 2006). This is also relevant for specific causes of death. For example, if a mother has a high propensity to give birth to children with too low birth weight, it may well happen that all births to this same mother expose too low weight at birth (genetic traits). Furthermore, closely spaced births of the same mother may be affected by infectious diseases of siblings (disease contamination) or environmental factors such as unsafe water supply or limited access to health care. Finally, as emphasized by DasGupta (1990), some mothers may be less resourceful in caring for their child than others, reflecting a behavioural effect.

3. Data

We combined the HDSS data of all live births and deaths of children for the ICDDR,B and comparison area obtained between 1987 and 2005. The data set has records of 107,367 singleton live births (57,830 from the comparison area and 49,837 from the ICDDR,B area) and 4,047 neonatal deaths and their causes (2,446 from the comparison area and 1,601 from

the ICDDR,B area). Data on education of both mother and father, occupation of the father, and source of drinking water were obtained from the 1996 and 2005 census. For our purposes, we model the two areas separately.⁵

3.1. Causes of death

Since 1966 HDSS has recorded data on causes of death, with particular emphasis on child and maternal deaths. Before 1987, the cause of death was assigned by the health assistant, but from 1987 onwards the death form was revised and read by physicians who assigned a cause of death. A single three-digit code was selected from a list of 97 possible codes derived from the 'basic tabulation list' of the World Health Organization International Classification of Diseases, Injuries and Causes of Death (World Health Organization 1977). The death of a child often has more than one cause. The assignment of cause of death followed a hierarchical process whereby certain diagnoses were viewed as more certain than others, and thus given priority as primary and underlying cause of death. The assignment of causes of death is described in detail in the literature (Fauveau et al. 1994; Adjuik et al. 2006; Lawn et al. 2006; Karar et al. 2009; Ronsmans et al. 2010).

To reduce the sampling error in our statistical analyses resulting from small numbers, the causes of neonatal deaths are first recoded into two categories (1) communicable diseases (CDs): acute respiratory infections, diarrhoea, dysentery, sepsis, meningitis, hepatitis, chicken pox, and neonatal tetanus or Extended Program on Immunization (EPI) related diseases and other viral diseases, (2) non-communicable diseases (NCDs): preterm

⁵ Some efficiency could possibly be gained by analyzing the two areas jointly, but this would also require some interactions of covariates with an area dummy. We experimented with this but found no improvements.

delivery/low birth weight(LBW), deaths related to neonatal conditions (birth asphyxia, birth trauma/cord haemorrhage, congenital abnormality, neonatal infections, obstetric complications of new born, sudden infant death, unspecified neonatal death and miscellaneous neonatal deaths). Second, for an analysis at a more disaggregate level, exits due to non-communicable diseases (NCDs) of neonatal deaths were split into three categories: (1) LBW: preterm delivery/low birth weight, (2) NCs: deaths related to neonatal conditions (BA: birth asphyxia, CA: congenital abnormality or birth trauma/cord haemorrhage, NEO: neonatal related other conditions, OBSCOMP: obstetric complications), (3) Other (sudden infant death/unspecified or miscellaneous). See Table 1 in the annex for further details.

3.2 Socio-demographic variables

The covariates in the model refer to gender of the child, child birth cohorts (1987-1992, 1993-1999 or 2000-2005), religion of the family (Muslim or Hindu), dummies for birth order, education of the parents, employment status of the mother and occupation of the father (day labourer or not), source of drinking water (piped water or not), the mother's age at birth, and the distance to the nearest health facility. Maternal education is a proxy of child care skills and the ability to use modern health care services. Both paternal education and occupation are considered here as household socioeconomic indicators. The birth cohort of child can capture the time trends of cause-specific mortality risks. The distance variable captures the availability of health services, and sources of drinking water the environmental effects. Birth order variables are included to capture sibling effects on the risks of cause specific mortality. Summary statistics of these variables are presented in Table 1.

3.3. Distribution of causes of deaths

Table 2 depicts the percentage distribution of causes of neonatal death in the neonatal period (0-28 days). Of all neonatal deaths recorded in Matlab during 1987-2005, deaths due to

NCDs comprised 87% and 78% in the ICDDR,B and comparison areas, respectively. The specific types of NCDs demonstrate that prematurity or low birth weight is a leading cause of death, followed by deaths 'unable to specify'. Among deaths due to CDs, the majority were due to acute respiratory infections (10.79 and 15.35 percent).

Figures 2 and 3 demonstrate that over the period 1987-2005, the fraction of neonatal deaths due to prematurity or low birth weight decreased, particularly in the ICDDR,B area. The fraction of deaths due to miscellaneous causes (sudden infant death, unable to specify, other disorders originated in the perinatal period etc.) also fell over time. Figures 4 and 5 show that deaths due to acute respiratory infections form an inreasing share of death due to communicable diseases in the comparison area, with neonatal tetanus or EPI related deaths disapprearing in both areas.

3.4. Cumulative incidence of cause-specific death

Before proceeding with advanced competing risks modelling we calculate the nonparametric cumulative incidence functions for the different causes of death to show how the cause-specific mortality changes with the age of the child. The value of the *cumulative incidence function* of cause *j* at time *t* is the probability of dying due to cause *j* before age *t*. The cumulative incidence is a function of the hazards of all the competing events and not solely of the hazard of the event to which it refers. See, for example, Coviello and Boggess (2004). Figures 6 and 7 show the cumulative incidence functions for neonatal deaths due to different causes based upon the complete samples in the ICDDR,B and comparison areas. They show that about 15 deaths per 1,000 live births accounted are ascribed to low birth weight (LBW) in both areas. About ten of these deaths occur in the first three days after birth. The second most frequent cause is unspecified or miscellaneous (OTHER), with about six and nine neonatal deaths per 1,000 births in the ICDDR,B and comparison area, respectively.

These deaths are somewhat less concentrated in the first few days. This applies even more to deaths due to communicable diseases ARI and EPI, which together account for almost ten deaths per 1,000 births in the comparison area and five in the ICDDR,B area. Only about half of these are in the first week after birth. On the other hand, deaths due to obstetric complications (OBSCOMP) and birth asphyxia (BA) are almost exclusively concentrated in the first few days of life. Overall, although levels of neonatal deaths due to the various causes are substantially different in the two areas, the patterns of how these deaths are distributed over the 28 days of the neonatal period are similar in both areas.

4. Model

The modelling approach used builds on the concept of competing risks. We observe $\delta_{ij} = 1$ if child *i* dies from cause *j* (*j* =1,...,*k*) during the first 28 days after birth. We assume each neonatal death is associated with one single cause; there are in total *k* possible causes; we will estimate models with *k*=2 and with *k*=4. The hazard of dying from cause *j* (*j*=1,...,*k*) at time *t* is denoted by $\lambda_j(t)$, where *t* refers to the age of child in days (0-28). In the competing risks model, we only observe the time till the first of *k* possible exits or until the end of the neonatal period, so that the observed survival time is given by $T_i = \min(T_{il}, \ldots, T_{ik}, C_i)$, where in our case $C_i = 29$ days for each child *i*. So T_i is time of death in case of a neonatal death and 29 days in case of neonatal survival.

For the two exits model, $\lambda_1(t)$ is the hazard of dying due to CDs and $\lambda_2(t)$ the hazard of dying due to NCDs at time t. The hazard of dying at time t due to any cause is given by $\lambda_1(t) + \lambda_2(t)$. In general, the hazard of dying at time t is given by $\lambda_1(t) + \dots + \lambda_k(t)$. This sum corresponds to the single hazard $\lambda(t)$ of dying in the basic hazard model. The hazard

rates are specified as the following mixed proportional hazards (see for example Manton et al. 1981 or Lancaster 1979):

$$\lambda_i(t \mid x, v_i) = \lambda_{0i}(t) \exp(x\beta_i) v_i, j = 1, \dots, k$$
(1)

The hazard rates are functions of time *t*, explanatory variables *x* which in our case do not vary over the neonatal period (child, mother and community level observed characteristics), and time constant mother-specific heterogeneity v_j . The explanatory variables *x* are assumed to enter through linear indexes $x\beta_j$, j = 1,...,k. Time dependence is incorporated with a piecewise constant baseline hazard $\lambda_{0,i}(t)$: for each cause of death j (j=1,...,k), we have

$$\lambda_{0j}(t) = \exp(\beta_{0j}) \sum_{h=1}^{H} \exp(\gamma_{hj}) I_h(t) \text{ with } I_h(t) = I[t_{h-1} \le t < t_h], \text{ the indicator function for the}$$

interval $[t_{h-1}, t_h]$, and $t_0 = 0$, $t_H = 28$ days. Any duration dependence can be approximated arbitrarily closely by increasing the number of intervals. We experimented with several partitions of [0, 28] into several intervals and found the best model performance for the H=5intervals [0,1), [1,2), [2-3), [3,7) and [7,28]. For identification we need to restrict one of the γ_{hj} (h=1,...,H) to zero for each *j*. We choose $\gamma_{Hj} = \emptyset$. Thus, β_{0j} determines the hazard in the last interval. The other γ_{hj} determine the ratio of the hazard in each interval compared to this last interval. The baseline hazard at $t \in [t_{h-1}, t_h)$ is higher than the baseline hazard for a duration of $t > t_h$ if $\gamma_h > 0$ and lower if $\gamma_h < 0$.

Our emphasis is on the specification of unobserved heterogeneity v_j , j = 1,...k, capturing unobserved factors that affect survival of a child. Ignoring these factors may bias the parameter estimates. In principle, unobserved heterogeneity can be child specific, mother specific, or community specific. Following several existing studies emphasizing the role of mother specific heterogeneity or 'frailty,' (Sastry 1997; Arulampalam and Bhalotra 2006) we

model mother specific heterogeneity only. The unobserved heterogeneity terms $v_j > 0$ are time-independent and independent of observed characteristics *x*. Many different choices for the distribution of the unobserved heterogeneity exist. One issue is that the unobserved heterogeneity terms v_j of different causes of death *j* can be correlated. To address this we adopt a two factor loading model, with two independent fundamental factors W_1 and W_2 both having a discrete distribution on $\{-1,1\}$. This implies that

$$v_{j} = \exp(\alpha_{j1}W_{1} + \alpha_{j2}W_{2}), j = 1, ..., k$$
(2)

Let $W = (W_1, W_2)'$ and let A be the matrix of factor loadings with rows $A_j = (\alpha_{j1}, \alpha_{j2})$. Then the covariance matrix of the logarithms of the unobserved heterogeneity terms $v = (v_1, ..., v_k)$ is given by $V(\ln(v)) = AV(W)A'$. One additional restriction is needed for identification, we choose $\alpha_{12} = 0$. The probabilities for the discrete distributions for W_1 , W_2 are $Pr(W_1 = -1) = p_1$ and $Pr(W_2 = -1) = p_2$. We assume for both p_1 and p_2 a logit form, i.e. $p_1 = e^{\theta l}/(1 + e^{\theta l})$ and $p_2 = e^{\theta 2}/(1 + e^{\theta 2})$ and we estimate the θ 's. Thus, for example, for our model with two exits (CDs and NCDs) the unobserved heterogeneity terms have the following distribution:

$$P(\ln v_1 = -\alpha_{11}, \ln v_2 = -\alpha_{21} - \alpha_{22}) = p_1 p_2;$$

$$P(\ln v_1 = -\alpha_{11}, \ln v_2 = -\alpha_{21} + \alpha_{22}) = p_1(1 - p_2);$$

$$P(\ln v_1 = \alpha_{11}, \ln v_2 = \alpha_{21} - \alpha_{22}) = (1 - p_1)p_2;$$

$$P(\ln v_1 = \alpha_{11}, \ln v_2 = \alpha_{21} + \alpha_{22}) = (1 - p_1)(1 - p_2).$$

The parameters can be estimated jointly with maximum likelihood in Stata; details on the likelihood function are available upon request. The covariance matrix of the unobserved heterogeneity terms can be estimated ex post, since it is a function of model parameters. This also applies to the *total survival* and *cumulative incidence functions*. The *total survival function* conditional on observed and unobserved heterogeneity is

$$S(t \mid x, v_1, ..., v_k) = \Pr\left(T \ge t \mid x, v_1, ..., v_k\right)$$
$$= \exp\left(-\sum_{j=1}^k v_j \int_0^t \lambda_{0j}(s) \exp\left(x\beta_j\right) ds\right)$$
(3)

The *cumulative incidence function* of cause j is the probability of dying due to cause j before age t. In section 3, we have already presented the empirical cumulative incidence functions for various causes for the complete samples in the two areas. Based upon the model, we can also estimate the cumulative incidence functions for specific child, that is, conditional on observed and unobserved heterogeneity. They are given by:

$$F_{j}(t \mid x, v_{1}, ..., v_{k}) = \int_{0}^{t} v_{j} \lambda_{0j}(s) \exp(x\beta_{j}) S(s \mid x, v_{1}, ..., v_{k}) ds$$
(4)

Integrating out the observed and unobserved heterogeneity, we can also obtain the total survival and cumulative incidence functions. Note that the sum of all cumulative incidence functions at a given age is equal to one minus the total survival function at that age, i.e.

$$\sum_{j=1}^{k} F_{j}(t \mid x, v_{1}, ..., v_{k}) = 1 - S(t \mid x, v_{1}, ..., v_{k}).$$

5. Estimation results

5.1. Communicable versus Non-communicable diseases

Tables 3 and 4 present the estimation results for the models distinguishing two causes of death (non-communicable (NCDs) and communicable diseases (CDs)) in the ICDDR,B area and the comparison area, respectively. We focus on the competing risk model introduced in the previous section, controlling for observed covariates in all the hazards and for mother specific unobserved heterogeneity terms that are allowed to be correlated across causes. For comparison, Tables 3 and 4 also present the parameter estimates of standard hazard models without unobserved heterogeneity for each cause of death separately ("traditional model"; first column in each table). In general, the estimated effects of the covariates are similar in the

traditional model and in the full model, in terms of sign, size, and significance level. Allowing for a general form of unobserved heterogeneity therefore has very little effect on the estimated duration dependence (the coefficients in the baseline intensity) or the estimated effects of the exogenous variables.

Tables 3 and 4 show that, in both areas, male children are more likely to die of CDs or NCDs than female children in similar families and circumstances. The gender differences are larger and more significant in the ICDDR,B area, and, in relative terms, larger for CDs than for NCDs. The magnitude of the differences is substantial. For example, in the ICDDR,B area, the chances of dying of a communicable disease on a given day in the neonatal period are about 70% higher (exp(0.54)-1)*100%) for a boy than for a girl (ceteris paribus). For a reference individual this is a difference of about 11 deaths per 1,000 over the complete neonatal period.

Religion, father's occupation and distance to the nearest health facility play no significant role in the ICDDR,B area. In the comparison area, however, a child is significantly more likely to die due to a CDs if the mother is Hindu (rather than Muslim), if the father is a day labourer, or if the distance to the nearest health facility is larger. The latter also applies to NCDs. It seems plausible that distance to the nearest health facility is more important in the comparison area than in the ICDDR,B area, since distances are much larger in the comparison area (cf. Table 1), making limited access to a health facility a more common concern there (Bhatia 1981).

A higher education level of the mother significantly reduces the NCDs hazard in both areas, whereas it has a negative but insignificant effect on death due to CDs. On average, if the mother is educated up at least secondary level this reduces the number of deaths per 1,000

live births due to NCDs on the first day after birth by 9 in the ICDDR,B area and by 14 in the comparison area.

In line with the demographic literature, first born children and children born to mothers aged less than 20 years old are at higher risk of neonatal mortality compared to the reference categories (20-24 years old mother and higher order births). The differences are much larger and more significant for NCDs than for CDs. In the ICDDR,B area, there is some evidence that children of older mothers (age 25 and older) have a lower risk of dying from CDs than the benchmark category (ages 20-24). There are no significant differences amongst birth orders 2 or higher.

A decreasing trend of neonatal death is observed in both CDs and NCDs in both areas, but it is not always significant. The degree of decline is strongest for CDs in the comparison area, where the risk has fallen substantially in the period 2000-2005. In the ICDDR,B area, there has been a significant reduction in the risk due to NCDs from the first to the second time period considered (a reduction of more than 25% from 1987-1992 to 1993-1999). In the comparison area, a similar reduction occurred a few years later.

A monotonically decreasing pattern trend is observed in the baseline intensity of dying due to NCDs in both areas: the hazard of dying is largest on the day of birth and already much lower one day after birth, and decreases further during the neonatal period. On the other hand, the pattern is quite different for communicable diseases, for which the hazard declines much less during the first week (and even increases from day 0 to day 1). This difference is in line with Figures 6 and 7, where we already saw that deaths due to CDs less often occurred on the first few days after birth.

The bottom panels of Tables 3 and 4 show that there is evidence of unobserved heterogeneity in both areas. In the ICDDR.B area, the covariance between the (mother

specific) unobserved heterogeneity terms in the two competing hazards is significantly positive, implying a correlation coefficient of 0.28. In the comparison area the implied correlation is 0.66, but the estimated covariance is not significant. In both areas, only one of the variances is significantly different from zero, suggesting that it is hard to identify the covariance structure of the unobserved heterogeneity terms, possibly due to the fact that, fortunately, neonatal death due to each of the specific causes is not such a common event and more than one neonatal death in the same family is rare.

5.2. Model with four causes of death

In order to get a better understanding of the deaths due to NCDs, we also split NCDs into three: (1) low birth weight (LBW); (2) neonatal conditions (NCs) which includes CA, NEO, BA, OBSCOMP; and (3) Other: sudden infant deaths/ unspecified or miscellaneous (miscellaneous: with a range of 27-30 cases in each area). Together with CDs, this gives four different causes of neonatal death. The results of these models for the complete model allowing for correlated unobserved heterogeneity in all four hazard rates are reported in Tables 5 and 6 for the ICDDR,B area and the comparison area, respectively. (Results of the corresponding traditional models generally give similar effects for the covariates; details are available upon request from the authors.) In general, the effects of many covariates are quite different for the three causes of death due to non-communicable diseases, showing that treating these causes separately is worthwhile.

The disadvantage for boys in NCDs that we already found in Tables 3 and 4 can be attributed to their larger vulnerability to NCs and, in the ICDDR,B area, to death due to low birth weight. We find no significant difference between boys and girls for the category "Other." As in Table 3, no significant religion difference is found in the ICDDR,B area. In the comparison area, however, a child born to a Hindu mother is not only more likely than an

otherwise similar child from a Muslim mother to die from a communicable disease, but is also more vulnerable to death related to NCs.

In both areas, higher education of the mother substantially reduces the risk of death due to LBW. The effects of mother's education on the other causes of death are much weaker, though some are still significant at the 5% level. Children born to mothers younger than 20 years of age are more likely to die of LBW and "other" causes in both areas. The effects of birth order show that first born children are at higher risk of death due to any cause than higher order births. The differences are large, sometimes more than a factor two; the only exception is death due to CDs in the ICDDR,B area where birth order appears to play no role.

Some of the results for the father's education level and type of occupation seem puzzling at first sight. In particular, we find that children of fathers with primary education rather than no education have higher risk to die because of NCs in both areas, and the effect is significant at the 5% level (but not at 1%). Moreover, if the father is a day labourer, this reduces the risk to die from NCDs in the ICDDR,B area, whereas being a day labourer is a negative index of socio-economic status. The finding that death due to NCs is positively associated with socio-economics status is in line, however, with data from the nationally representative BDHS 2004 (NIPORT et al. 2005), which show that deaths due to birth asphyxia are more common amongst mothers with higher education. Chowdhury et al. (2010) suggest this may be related to delivery at a health centre instead of at home, and this finding is in line with large unobserved heterogeneity for deaths due to NCs and increased institutional delivery (which includes mainly birth asphyxia/neonatal infections and delivery

complications) in the ICDDR,B area.⁶ On the other hand, secondary education of the father reduces the risk to die from "other" causes in the comparison area, and in the same area, being a day labourer increases the risk of death due to CDs (as in Table 4), as expected.

Lack of access to running water has the expected effect of increasing mortality due to CDs in the ICDDR,B area. It also increases the risk of dying from LBW. On the other hand, it has no significant effects in the comparison area. In the comparison area, a larger distance to the nearest health centre increases the risk of CDs related death (as in Table 4) and of LBW related death. As in Table 3, it has no significant effect in the ICDDR,B area where distances are smaller. In both areas, we find substantial differences among cohort effects for different causes of death. In particular, the risk of NCs related death has increased in 2000-2005 compared to 1987-1999, while the risk of dying from the other causes has fallen. Particularly for the "other" category (which includes sudden infant deaths, among others) the reduction in the period 2000-2005 is remarkably large in both areas.

The baseline intensities of dying due to LBW, NCs, and other non-communicable diseases follow similar patterns, which are also similar to the patterns in Tables 3 and 4 for all NCDs combined: the risk is very high on the first day of life, and reduces quickly after a few days. The pattern is quite different from that for non-communicable diseases, for which the

⁶ Anwar et al. (2004) found that in Matlab 19% of births took place in ICDDR,B facilities, 4% occurred in other (public and private) facilities, 2.6 births were attended by ICDDR,B midwives at home, and the remaining deliveries took place at home. However, according to BDHS 2004, nationally 90% of all births took place at home, which is comparable with our comparison area, a typical rural area of Bangladesh with the usual standard health facilities.

hazard shows a much less clear duration dependence pattern over the neonatal period, as we already saw in Tables 3 and 4.

We find some evidence of unobserved heterogeneity: two of the four variances are significant in the ICDDR,B area and one in the comparison area. Moreover, in the ICDDR,B area, the covariance between the unobserved heterogeneity terms in the hazards for communicable diseases and low birth weight related deaths is significant at the 1% level. The other covariances are insignificant at the 5% level. Still, at least in the ICDDR.B area, the covariance matrix of the unobserved heterogeneity terms seems easier to estimate in this model than in the model with only two causes of death – we no longer find the very large standard error found for NCDs in Table 3. This suggests that this large standard error might be due to aggregation of rather different causes of death. (That this problem does not arise in the comparison area may be because of the larger death rates there.)

5.3. Cumulative incidences functions

The cumulative incidence functions corresponding to the models with four causes of death for the benchmark cases are shown in Figures 8 (ICDDR,B area) and 9 (comparison area). These rates are substantially different from the rates for the complete sample (see Figures 6 and 7), since the socio-economic characteristics of the benchmark case are not representative for the sample average. The patterns over time confirm what we concluded from the baseline hazards in Tables 5 and 6: they are much steeper in the first few days for the various types of non-communicable diseases than for communicable diseases. At each point of time, the cumulative number of deaths due to LBW is larger than the numbers for all three other causes. In the ICDDR,B area, about 16 deaths per 1,000 live births are due to LBW within one day after birth, rising to about 28 per 1,000 after 28 days. The patterns over time are similar in the

two areas, but the levels are not: the hazards for CDs and for "other" NCDs are much larger in the comparison area than in the ICDDR,B area.

Figures 10 and 11 show the same cumulative incidence functions as Figures 8 and 9, but now for a benchmark birth in the period 2000-2005 instead of 1987-1992. The epidemiological shifts are similar in the two areas. In both areas, the largest difference between the two time periods is the significant reduction of the number of deaths due to LBW, about 12 per 1,000 in the ICDDR,B area and 8 per 1,000 in the comparison area during the whole neonatal period, and concentrated in the first few days after birth. On the other hand, the number of deaths due to NCs surprisingly increases substantially in the period 2000-2005, by about 5-6 deaths per 1,000 live births compared to the reference period 1987-1992. In the competing risks situation, the decrease of deaths due to LBW is perhaps partly substituted by an increase of deaths due to neonatal infections or obstetric complications at birth.

6. Discussion and conclusion

This study analyzes causes of neonatal death, derived from open-ended death history data reported by the mother or a close relative or neighbour (in absence of the mother) and recorded by non-medically trained field workers. Three physicians independently reviewed all death records and reached consensus. The uniform death registration form and assessment of causes of death by physicians during 1987-2005 is an important strength for the comparison the patterns of causes of death over the years. This Verbal Autopsy method was recommended by WHO to attain the reliable epidemiological data on mortality.

During 1987-2005, recorded neonatal mortality per 1,000 live births was 32.3 in the ICDDR,B area (which, in addition to government services, gets high quality health care services) and 42.3 in the comparison area (with standard government services). In

Bangladesh, the national neonatal mortality rate is about 41 per 1,000 live births (BDHS, 2004) and this rate is close to the rate of comparison area, a typical setup in rural Bangladesh.

A remarkable success is the reduction in the number of neonatal deaths due to neonatal tetanus or EPI, which mainly explains the reduction of total neonatal mortality in ICDDR,B area, and which is well noticed in other studies (Bhatia 1989; Baqui et al. 2001). This is supported by the cause-of-death data, indicating that mortality rates due to neonatal tetanus were 1.0% and 5.9% per 1,000 live births in ICDDR,B and comparison areas, respectively. During the study period, low birth weight was the foremost leading cause of neonatal deaths in both ICDDR,B and comparison area. Our findings are in agreement with global findings pointing at preterm birth or low birth weight as a major cause of neonatal death in the world and particularly in Bangladesh (Lawn et al. 2006).

On the other hand, death due to NCs - mainly neonatal infections or obstetric complications - became a primary cause of neonatal death in both areas in 2000-2005 (see also, Chowdhury et al., 2010). Compared to the 1987-1992 period, for otherwise similar children, the hazard to die due to NCs increased by about 77% in the ICDDR,B area and 46% in the comparison area. The increase in this rate in the ICDDR,B area is remarkable. As indicated in an earlier study, the absence of appropriate antenatal, intra-partum, and postnatal care in both areas takes an unnecessary toll on infant lives, which could easily be prevented with appropriate interventions (Bhatia 1989; Bari et al. 2002; Bang et al. 2005; Velaphi and Pattinson 2007).

Although a downward trend since 1993 is observed in neonatal death due to NCDs in both areas, this decline is faster in the ICDDR,B area, specifically for deaths due to LBW. This finding can be related to the large scale nutrition programs in the ICDDR,B area, which attempt to improve nutrition of pregnant mothers with the goal of increasing birth weight (see

<u>www.icddrborg/what-we-do/health-programmes/nutrition</u>). Since villages in the comparison area are contiguous to those in the ICDDR,B area, spill-over effects of these programs, changing information and behaviour in the comparison area also, may explain why mortality due to LBW has also significantly declined in the comparison area (Phillips et al. 1988).

The decline in childhood deaths due to CDs is widely discussed in the epidemiology literature. It is mainly due to neonatal tetanus or EPI, which is no longer an existing cause of death after 2000 in either area. Studies for India (Reddiah and Kapoor 1988) and Bangladesh (Bhatia 1989) show that the numbers of deaths due to CDs (including acute respiratory infections) remained almost unchanged during earlier periods, in line with what we find for the period 1993-2005. In contrast, a recent study in Bangladesh found a reduction of 79% in child or infant deaths due to respiratory infections during 1986-2006 (Karar et al. 2009). It may be noteworthy to mention that lack of consistent case definitions and rules in the hierarchical assignment of causes may hinder comparisons across time and studies.

The time of exposure to a disease (the number of days after birth) is an important phenomenon in epidemiological studies. This study finds that the number of children dying during days 1-6 due to all types of NCDs falls over the period, but on the other hand the number of deaths due to CDs increases. Studies in Matlab (Bhatia 1989; Chowdhury et al. 2010) reported that the ICDDR,B program significantly reduced neonatal mortality within one day after birth, which is also apparent in our estimations (Figures 10 and 11).

Our result confirms the general conclusion of the levels, trend and pattern of causes of neonatal deaths, but we find some remarkable socioeconomic differences in the causespecific deaths. Cause-specific deaths due to low birth weight and other causes (sudden deaths, specified, unspecified) are better explained from the socio-economic covariates than the others. Secondary education of the mother reduces deaths due to LBW significantly and

thus it seems that education helps women to improve general socioeconomic status or overcome the barriers set by low autonomy in traditional society. Education improves women's innate ability in pregnancy management and in caring for their child and management of household work (DasGupta 1990).

First-borns and children born to a young mother (age below 20) are more likely to die due to LBW in both areas, but particularly in the comparison area. This reflects an advantage of high quality primary care services and interventions for the risk of low birth weight (LBW) in the ICDDR,B area. Father's education leads to lower risk of neonatal mortality due to LBW in the ICDDR,B area, possibly as an indicator of the family's general socioeconomic status, which helps to take advantage of high quality services in ICDDR,B area. In the comparison area on the other hand, the father's being a day labourer, another index of poor socio-economic status, makes neonatal death due to CDs more likely.

Neonatal death is more likely among Hindu families (due to CDs and NCs) in the comparison area and a similar trend is observed in a study for India (Bhalotra et al. 2010a,b). In the ICDDR,B area, extensive health services apparently annihilate this religion difference. Male children are more likely to die than female children due to CDs and NCDs in both areas. The (relative) difference is largest for CDs in the ICDDR,B area (almost 72%). Furthermore, gender discrepancies in deaths due to NCDs are mainly related to NCs. This is in line with a study that finds that infant mortality is inherently larger for boys than for girls, but that this can be reversed by environmental disadvantages for female children. (Waldron 1983; Chowdhury et al. 2010). The influence of such environmental factors can be reduced by the extensive health services in the ICDDR,B area. This finding gives an insight of what causes gender discrepancies in child deaths compared to an earlier study which only revealed overall improvement of female child survival in the ICDDR,B area (Datta and Bairagi 2000). No

significant gender difference is observed for neonatal deaths due to LBW in the ICDDR,B area, perhaps since nutrition programs in ICDDR,B area diminished the excess deaths due to malnutrition among female children, where earlier studies reported excess female deaths (Bhuiya et al. 1986; Fauveau et al. 1991).

Keeping constant socioeconomic indicators (parental education and occupation) in the model, the risks for first-borns and children of young mothers remain significantly higher than for others, probably pointing at a role of physiological factors rather than socio-economic factors (Bhatia 1989).

An additional contribution of our study is to allow for a flexible form of unobserved heterogeneity. We could not include some potentially relevant covariates, such as use of antenatal care and birth practice, which might lead to unobserved variation in the outcomes of our interest. Unobserved heterogeneity in death due to LBW may reflect the importance of extra attention to warmth, feeding and prevention or early treatment of infections (Conde-Agudelo et al. 2003). Point estimates of large unobserved heterogeneity in the hazards of NCs (mainly neonatal related infections or obstetric complications) suggest disadvantages or mistreatment of modern health technology or lack in quality of care. For example, unnecessary administration of oxytocics to augment labour in child birth or inadequate foetal monitoring by health workers increased neonatal deaths significantly (Bari et al. 2002; Bang et al. 2005; Velaphi and Pattinson 2007).

Our findings highlight the role of maternal and child health interventions for child survival, particularly tetanus toxoid immunization for pregnant women, nutrition programs, and high coverage health services (distance to health centre and information dissemination). Death due to EPI has been eliminated, but in order to achieve MDG-4 of reducing child mortality, strategies targeting acute respiratory diseases remain necessary.

For further reduction of neonatal mortality due to low birth weight it is important to add strategies to ensure equitable utilization of services by various socio-economic groups to the existing programs, such as for low educated mothers, and particularly for their first pregnancy. On the other hand, the finding that unobserved heterogeneity in the ICDDR,B area is much larger for deaths due to NCs than in the comparison area suggests more death tolls because of poor quality of institutional delivery and in foetus monitoring. It also may mean that not everyone benefits equally from the health interventions, so that policies that increase quality and equity in interventions may help to further reduce neonatal mortality.

References

- Adjuik, Martin, Tom Smith, Sam Clark, Jim Todd, Anu Garrib, Yohannes Kinfu, Kathy Kahn,
 Mitiki Mola, Ali Ashraf, Honorati Masanja, Ubaje Adazu, Jahit Sacarlal, Nurul Alam,
 Adama Maria, Adjima Gbangou, Eleuther Mwageni, and Fred Binka. 2006. Causespecific mortality rates in sub-Saharan Africa and Bangladesh, *Bulletin of the World Health Organization* 84(3): 181-188.
- Anwar, Iqbal., Japhet Killewo, Mahbub–E-Elahi K Chowdhury, and Sushil Kanta Dasgupta.
 2004. Bangladesh: Inequalities in utilization of maternal health care services evidence from Matlab, *Health, Nutrition and Population* (HNP) discussion paper Number 2.
- Arulampalam, Wiji and Sonia Bhalotra. 2006. Sibling death clustering in India: state dependence versus unobserved heterogeneity, *Journal of Royal Statistical Society A* 169(4): 829-848.
- Bang, Abhay T., Rani A. Bang, Sanjay B. Baitule, Hanimi H. Reddy, and Mahesh D.
 Deshmukh. 2005. Management of birth asphyxia in home deliveries in rural Gadchiroli: the effect of two types of birth attendants and of resuscitating with mouth-to-mouth, tube-mask or bag-mask. *Journal of Perinatology*, 25(Suppl): S82-S91.
- Baqui, Abdullah, Ahmed Al Sabir, N Begum, Shams El Arifeen, S N Mitra, and Robert E
 Black. 2001. Causes of childhood deaths in Bangladesh: an update. *Acta Paediatrica*, 90: 682-690.
- Bari, Wasimul, Rafiqul I. Chowdhury, Ataharul M. Islam, Nitai Chakraborty, and Halida Hanum Akhter. 2002. The differentials and determinants of perinatal mortality in rural Bangladesh. *European Journal of Contraception and Reproductive Health Care* 7: 216-222.

- Bhatia, Shushum. 1981. Traditional childbirth practices: implications for a rural MCH program, *Studies in Family Planning* 12 (2): 66-75.
- Bhatia, Shushum. 1989. Patterns and causes of neonatal and postneonatal mortality in rural Bangladesh, *Studies in Family Planning* 20(3): 136-146.
- Bhuiya, Abbas, Susan Zimicki, and Stan D'Souza. 1986. Socioeconomic differentials in child nutrition and morbidity in a rural area of Bangladesh. *Journal of Tropical Pediatrics* 32(1): 17-23.
- Chen, Lincoln C, Mizanur Rahman, and A M Sarder. 1980. Epidemiology and causes of death among children in a rural area of Bangladesh, *International Journal of Epidemiology* 9(1): 25-33.
- Chowdhury, Hafizur Rahman, Sandra Thompson, Mohammad Ali, Nurul Alam, Md Yunus, and Peter Kim Streatfield. 2010. Causes of neonatal deaths in a rural subdistrict of Bangladesh: Implications for intervention, *Journal of Health Population Nutrition* 28(4): 375-382.
- Conde-Agudelo, Agustin, Jose M Belizan, and Jose Diaz-Rossello. 2003. Kangaroo mother care to reduce morbidity and mortality in low Barth weight infants (Cochrane Review). In: *The Cochrane Library, Issue 4*, Oxford: Update Software.
- Cornfield, Jerome. 1957. The estimation of the probability of developing a disease in the presence of competing risks, *Disease Probability* 47(5): 601-607.
- Coviello, Vincenzo and May Boggess. 2004. Cumulative incidence estimation in the presence of competing risks, *The Stata Journal* 4(2): 103-112.
- Cox, David Roxbee. 1959. The analysis of exponentially distributed life-times with two types of failures. *Journal of the Royal Statistical Society* B, 21: 411-421.

- DasGupta, Monica. 1990. Death clustering, mothers' education and the determinants of child mortality in rural Punjab, *India Population Studies* 44(3): 489-505.
- Datta, Ashish Kumar and Radheshyam Bairagi. 2000. Improvement in female survival: A quite revolution in Bangladesh, *Asia Pacific Population Journal* 15(1): 19-40.
- Fauveau, Vincent, Michael A Koenig, and Bogdan Wojtyniak. 1991. Excess female deaths among rural Bangladeshi children: An examination of cause-specific mortality and morbidity, *International Journal of Epidemiology* 20(3): 729-735.
- Fauveau, Vincent, Bogdan Wojtyniak, Hafizur Rahman Chowdhury, and A M Sarder. 1994.
 Assessment of cause of death in the Matlab Demographic Surveillance System. In:
 Fauveau, V. (ed.), *Matlab: Women, Children and Health*. Dhaka: International Centre for Diarrhoeal Diseases Research: 65-86.
- Fine, Jason P and Robert J Gray. 1999. A proportional hazards model for the subdistribution of a competing risk, *Journal of the American Statistical Association* 94(446): 496-509.
- Goldberg, I.D., Morton L Levin, Paul R Gerhardt, Vincent H Handy, & R. E Cashman. 1956.
 The probability of developing cancer, *Journal of the National Cancer Institute* 17: 155-173.
- Heckman, James J, Joseph V Hotz, and James R Walker. 1985. New evidence on the timing and spacing of births, *American Economic Review* 75(2): 179-184.
- ICDDR,B Centre for Health and Population Research, Dhaka, Bangladesh. 2006. Health and Demographic Surveillance System-Matlab: registration of health and demographic events 2004, *Scientific Report* 93
- Jones, Gareth, Richard W Steketee, Robert E Black, Zulfiqar A Bhutta, Saul S Morris, and the Bellagio Child Survival Study Group. 2003. How many child deaths can we prevent this year? *Lancet* 362:65-71.

- Karar, Zuniad A, Nurul Alam, and Peter Kim Streatfield. 2009. Epidemiological transition in rural Bangladesh, 1986-2006, *Global Health Action* DOI:10.3402/gha.v2i0.1904.
- Lancaster, Tony. 1979. Econometric methods for the duration of unemployment, *Econometrica* 47(4): 939-956.
- Lawn, Joy E, Simon Cousens and Jelka Zupan. 2005. 4 million neonatal deaths: When? Where? Why? *Lancet* 365(9462): 891-900.

Lawn, Joy E, Katarzyna Wilczynska-Ketende and Simon Cousens. 2006. Estimating the causes of 4 million neonatal deaths in the year 2000, *International Journal of Epidemiology* 35: 706-718.

- Manton, Kenneth G, Eric Stallard, and James W Vaupel. 1981. Methods for the mortality experience of heterogeneous populations, *Demography* 18(3): 389-410.
- Mosley, W Henry and Lincoln C Chen. 1984. An analytical framework for the study of child survival in developing countries, *Population Development Review* 10(Suppl.): 25-45.
- National Institute of Population Research and Training (NIPORT), Mitra and Associates, and ORC Macro. (2005). Bangladesh Demographic Health Survey 2004. Dhaka, Bangladesh and Calverton, Maryland.
- Phillips, James F, Ruth Simmons, J Chakraborty, and A I Chowdhury. 1984. Integrating health services into an MCH-FP program: Lessons from Matlab, Bangladesh, *Studies in Family Planning* 15(4): 153-161.
- Phillips, James F, Ruth Simons, Michael A Koenig, and J Chakraborty. 1988. Determinants of reproductive change in a traditional society: Evidence from Matlab, Bangladesh. *Studies in Family Planning* 19(6): 313-334.
- Reddiah, Vankadara P and Suresh Kumar Kapoor. 1988. Acute respiratory infections in rural underfives, *Indian Journal of Pediatrics* 55(3): 424-426.

- Ronsmans, Carine, Mahbub E Chowdhury, Shushil Kanta DasGupta, Anisuddin Ahmed, and Marge Koblinsky. 2010. Effect of parent's death on child survival in rural Bangladesh: a cohort study, *Lancet* 375 (9730): 2024-2030.
- Sastry, Narayan. 1997. Family-level clustering of childhood mortality risk in Northeast Brazil, *Population Studies* 51(3): 245-261.
- United Nations, General Assembly, 56th session, Road map towards the implementation of the United Millennium Declaration: Report of the Secretary-General, New York: United Nations, 2001.
- Velaphi, Sithembiso & Robert Pattinson. 2007. Avoidable factors and causes of neonatal deaths from perinatal asphyxia-hypoxia in South Africa: National perinatal survey, *Annals of Tropical Paediatrics: International Child Health* 27(2): 99-106.
- Waldron, Ingrid. 1983. Sex differences in human mortality, *Social Science & Medicine* 17(6): 321-333.
- World Health Organization. Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death. 9th revision. Geneva: WHO, 1977.

Table 1: Summary statistics of Variable	ICDDR,B area	Comparison area		
Gender of index child				
Male	50.78	50.76		
Child's birth cohort				
Before 1993	32.86	36.53		
1993-1999	34.63	34.26		
2000-2005	32.51	29.22		
Birth order				
1	30.97	26.65		
2-3	43.07	38.23		
4 +	25.96	35.12		
Religion: Hindu	14.67	8.91		
Mother's education				
No education	49.00	53.10		
Primary education	25.15	25.05		
At least secondary educat	tion 25.86	21.85		
Mother's age at birth				
<20	12.19	11.54		
20-24	33.31	33.13		
25-29	28.55	28.39		
30 +	25.94	17.8		
Father's education				
No education	57.26	58.72		
Primary education	ation 21.70	23.00		
At least secondary educ		18.28		
Father's occupation				
Day labourer	15.75	18.37		
Source of drinking water				
No tube-well/pipewate	er 25.31	24.10		
Distance to health centre	1.86 (0.96)	7.29 (3.96)		

Table 1: Summary statistics of explanatory variables.

Note: Percentages of outcome 1 for all dummy variables; mean and standard deviation (in parentheses) for continuous variables.

	ICDDR,B area	Comparison area
	Neonatal deaths	Neonatal deaths
Cause of death	(0-28 days)	(0-28 days)
Communicable diseases (CDs)	13.09	21.93
Hepatitis	0.06	0.00
Septecaemia	0.31	0.00
Acute respiratory infections (ARI)	10.79	15.35
Diarrheal diseases	0.93	0.73
Neonatal tetanus or EPI related (EPI)	1.00	5.85
Non-communicable diseases (NCDs)	86.57	77.90
Congenital abnormality	2.43	1.71
Prematurity/low birth weight (LBW)	45.95	36.50
Birth asphyxia (BA)	5.25	3.68
Obstetric complications of new born (OBSCOMP)	8.74	9.20
Birth trauma/cord haemorrhage	1.69	1.02
Other neonatal related conditions* (NEO)	4.10	4.15
Miscellaneous**	2.05	1.17
Diagnosis not possible***	16.36	20.47

Table 2: Percentage distribution of cause-specific neonatal deaths.

* includes neonatal infections, respiratory and cardiovascular specific disorder to the perinatal period

**includes skin infections, fever, jaundice, intestinal obstruction, Oedemas, external cause (injury), homicide

*** includes sudden infant death, unspecified cause, other disorder in the perinatal period

communicable			e Diseases				cable Dise	0505
Variable	Comm	umcabie	Diseases	(CDS)		(NC		ases
	Traditi	onal	With Ur	observed	Tradi	(With U	Inobs
	Taun	onai		geneity	ITaur	uonai	Heterog	
Male	0.53**	(0.14)	0.54**	(0.14)	0.21**	(0.05)	0.23**	(0.06)
Hindu	-0.02	(0.14) (0.20)	-0.03	(0.14) (0.20)	0.01	(0.03) (0.08)	0.25	(0.00) (0.08)
Mother's education level	0.02	(0.20)	0.05	(0.20)	0.01	(0.00)	0.001	(0.00)
At least primary	-0.17	(0.18)	-0.19	(0.18)	-0.13*	(0.07)	-0.15*	(0.07)
At least secondary	-0.19	(0.13) (0.23)	-0.23	(0.10) (0.22)	-0.35**	0.09)	-0.39**	(0.09)
Mother's age at birth	0.19	(0.25)	0.20	(0.22)	0.50	0.07)	0.09	(0.0))
<20 years	0.30	(0.21)	0.29	(0.22)	0.20*	(0.08)	0.20*	(0.08)
25-29 years	-0.50*	(0.23)	-0.47*	(0.21)	-0.08	(0.09)	-0.05	(0.09)
≥30	-0.58*	(0.26)	-0.52*	(0.26)	0.13	(0.10)	0.20*	(0.10)
Birth order								
2-3	-0.02	(0.20)	-0.08	(0.20)	-0.67**	(0.08)	-0.73**	(0.08)
≥4	0.26	(0.28)	0.13	(0.29)	-0.51**	(0.11)	-0.65**	(0.11)
Father's education level								
At least primary	-0.17	(0.18)	-0.17	(0.18)	0.17*	(0.07)	0.17*	(0.07)
At least secondary	-0.30	(0.21)	-0.30	(0.22)	-0.08	(0.08)	-0.07	(0.09)
Father day labourer	0.20	(0.18)	0.18	(0.18)	-0.02	(0.08)	-0.03	(0.08)
No tube-well/pipe	0.31*	(0.15)	0.31*	(0.16)	0.12*	(0.06)	0.11	(0.07)
water								
Distance to health	0.01	(0.07)	0.01	(0.07)	-0.01	(0.03)	-0.005	(0.03)
centre ^d								
Birth cohort child								
1993-1999	0.08	(0.16)	0.05	(0.16)	-0.29**	(0.07)	-0.31**	(0.07)
2000-2005	-0.30	(0.21)	-0.32	(0.20)	-0.31**	(0.07)	-0.33**	(0.08)
Baseline intensity								
Day 0	0.80*	(0.29)	0.76*	(0.29)	4.11**	(0.07)	4.06**	(0.07)
Day 1	1.28**	(0.24)	1.26**	(0.24)	2.55**	(0.10)	2.52**	(0.10)
Days 2	1.02**	(0.27)	1.00**	(0.27)	2.01**	(0.12)	1.99**	(0.12)
Days 3-6	0.55**	(0.18)	0.54**	(0.18)	1.15**	(0.10)	1.14**	(0.10)
Constant	-9.07**	(0.21)	-7.97**	(0.37)	-7.83	(0.11)	-7.59**	(2.19)
Unobserved heterogeneit	у							
Variance	-	-	0.14*	(0.07)	-	-	1.58	(3.01)
Covariance	-	-	0.13*	(0.06)	-	-	-	-

 Table 3. Parameter estimates of competing risks model for neonatal deaths due to communicable and non-communicable diseases, ICDDR,B area.

Notes: ^d centered around its mean in each area; * p-value<0.05, ** p-value<0.01, standard errors are in parenthesis Reference category: gender is female, religion is Muslim, mother and father have no education, mother's age at birth 20-24 years, father is not day-labourer, source of drinking water is tube-well/pipewater, living at average

distance to health centre, child birth cohort 1987-1992, baseline intensity 7-28 days.

	Communicable Diseases (CDs)			Non-communicable Diseases (NCDs)				
Variables								
	Tradit	ional	With U	Inobs.	Tradi	tional	With Un	observed
			Heterog	geneity			Heteroge	eneity
Male	0.19*	(0.09)	0.20*	(0.09)	0.11*	(0.05)	0.11*	(0.05)
Hindu	0.28*	(0.14)	0.28*	(0.14)	0.13	(0.08)	0.14	(0.08)
Mother's education level								
At least primary	-0.21	(0.12)	-0.22	(0.12)	-0.10	(0.06)	-0.11	(0.06)
At least secondary	-0.23	(0.15)	-0.25	(0.15)	-0.35**	(0.08)	-0.37**	(0.08)
Mother's age at birth								
<20 years	0.12	(0.14)	0.12	(0.14)	0.27**	(0.07)	0.26**	(0.07)
25-29 years	-0.27	(0.13)	-0.25	(0.13)	-0.03	(0.07)	0.001	(0.07)
30 years plus	-0.14	(0.17)	-0.10	(0.16)	0.05	(0.09)	0.11	(0.09)
Birth order								
2-3	-0.33**	(0.12)	-0.38**	(0.13)	-0.63**	(0.67)	-0.68**	(0.07)
4 plus	-0.20	(0.17)	-0.29	(0.17)	-0.61**	(0.09)	-0.71**	(0.09)
Father's education level								
At least primary	0.01	(0.11)	0.014	(0.11)	0.04	(0.06)	0.06	(0.06)
At least secondary	-0.20	(0.15)	-0.20	(0.14)	-0.03	(0.07)	0.03	(0.07)
Father day labourer	0.46**	(0.10)	0.48**	(0.10	0.12*	(0.06)	0.13*	(0.06)
No tube-well/pipe	0.03	(0.11)	0.03	(0.11)	0.04	(0.06)	0.04	(0.06)
Water								
Distance to health	0.03*	(0.01)	0.03*	(0.01)	0.02**	(0.01)	0.02**	(0.01)
centre ^d								
Birth cohort child								
1993-1999	-0.20	(0.11)	-0.21	(0.11)	-0.10	(0.06)	-0.11	(0.06)
2000-2005	-0.49**	(0.13)	-0.50**	(0.13)	-0.28**	(0.07)	-0.29**	(0.07)
Baseline intensity								
Day 0	1.02**	(0.16)	0.99**	(0.16)	3.96**	(0.06)	3.92**	(0.06)
Day 1	0.61**	(0.20)	0.58**	(0.20)	2.54**	(0.08)	2.51**	(0.08)
Days 2	0.37	(0.22)	0.35	(0.22)	1.90**	(0.11)	1.88**	(0.11)
Days 3-6	0.77**	(0.10)	0.76**	(0.10)	1.18**	(0.08)	1.17**	(0.08)
Constant	-8.05**	(0.16)	-7.57**	(0.58)	-7.69**	(0.10)	-6.80**	(0.33)
Unobserved heterogeneity								
Variance	-	-	0.41	(0.44)	-	-	0.38*	(0.13)
Covariance	-	-	0.26	(0.23)	-	-	-	-

Table 4. Parameter estimates of competing risks model for neonatal deaths due to communicable and non-communicable diseases, comparison area.

Notes: ^d centered around its mean in each area; * p-value<0.05, ** p-value<0.01, standard errors are in parenthesis Reference category: gender is female, religion is Muslim, mother and father have no education, mother's age at birth

20-24 years, father is not day-labourer, source of drinking water is tube-well/pipewater, living at average distance to health centre, child birth cohort 1987-1992, baseline intensity 7-28 days.

		inicable			municable		s (NCDs)	
Variables	disease	s (CDs)						
	Cl		LBW	/ ^a	NCs ^b)	Othe	er ^c
Male	0.54**	(0.14)	0.26**	(0.08)	0.30**	(0.11)	0.07	(0.12)
Hindu	-0.03	(0.20)	-0.15	(0.12)	0.16	(0.15)	0.15	(0.16)
Mother's education level						. ,		
At least primary	-0.19	(0.18)	-0.07	(0.10)	-0.12	(0.14)	-0.38*	(0.16)
At least secondary	-0.23	(0.22)	-0.44**	(0.13)	-0.34*	(0.16)	-0.36	(0.19)
Mother's age at birth		. ,		. ,				
<20 years	0.29	(0.22)	0.24*	(0.11)	-0.04	(0.16)	0.38*	(0.18)
25-29 years	-0.47*	(0.21)	-0.21	(0.12)	0.09	(0.16)	0.16	(0.18)
30 years plus	-0.52*	(0.26)	0.16	(0.15)	0.30	(0.20)	0.23	(0.22)
Birth order						. ,		
2-3	-0.08	(0.20)	-0.67**	(0.11)	-0.96**	(0.15)	-0.57**	(0.17)
4 plus	0.13	(0.29)	-0.67**	(0.16)	-0.77**	(0.22)	-0.41	(0.24)
Father's education level		. ,		. ,				
At least primary	-0.17	(0.18)	0.12	(0.10)	0.33*	(0.13)	0.14	(0.15)
At least secondary	-0.30	(0.22)	-0.25*	(0.12)	0.11	(0.15)	0.08	(0.18)
Father day labourer	0.18	(0.18)	0.01	(0.10)	-0.51*	(0.18)	0.24	(0.15)
No tube-well/pipe water	0.31*	(0.16)	0.18*	(0.09)	-0.21	(0.14)	0.24	(0.13)
Distance to health centre ^d	0.01	(0.07)	-0.02	(0.04)	-0.04	(0.06)	0.08	(0.06)
Birth cohort child								
1993-1999	0.05	(0.16)	-0.36**	(0.09)	-0.24	(0.15)	-0.24	(0.14)
2000-2005	-0.32	(0.20)	-0.76**	(0.11)	0.57**	(0.14)	-0.66**	(0.17)
Baseline intensity								
Day 0	0.76*	(0.29)	4.09**	(0.10)	4.53**	(0.16)	3.46**	(0.15)
Day 1	1.26**	(0.24)	2.45**	(0.15)	2.93**	(0.21)	2.28**	(0.20)
Days 2	1.00**	(0.27)	2.08**	(0.17)	1.77**	(0.30)	1.97**	(0.23)
Days 3-6	0.54**	(0.18)	1.28**	(0.14)	0.80**	(0.26)	1.05**	(0.19)
Constant	-7.97**	(0.37)	-7.19**	(0.45)	-10.62**	(3.13)	-8.31**	(0.39)
Variance	0.14*	(0.07)	0.58*	(0.24)	3.42	(10.3)	0.23	(0.20)
Covariance (row1)	-	-	0.17**	(0.06)	0.13	(0.07)	0.16*	(0.07)
Covariance (row2)	-	-	-	-	-0.97	(1.63)	0.05	(0.28)
Covariance (row3)	-	-	-	-	-	_	0.55	(1.15)

 Table 5. Parameter estimates of intensity to neonatal deaths due to communicable diseases and different types of non-communicable diseases, ICDDR,B area.

Notes: ^d centered around its mean in each area; * p-value<0.05, ** p-value<0.01, standard errors are in parenthesis Reference category: gender is female, religion is Muslim, mother and father have no education, mother's age at birth 20-24 years, father is not day-labourer, source of drinking water is tube-well/pipewater, living at average distance to health centre, child birth cohort 1987-1992, baseline intensity 7-28 days.

^a Low birth weight/prematurity

^b Neonatal infections, birth asphyxia, obstetric complications, respiratory disorders, birth trauma, cord haemorrhage congenital abnormalities

^c skin infections, fever, jaundice, intestinal obstruction, Oedemas, external cause (injury), homicide, and sudden infant death, unspecified cause, other disorder in the perinatal period

	Comm	unicable		Non-cor	nmunicable	e Disease	s (NCDs)	
Variables		es (CDs)						
	C	Ds	LBV	V ^a	NCs ^b		Other ^c	
Male	0.19*	(0.09)	0.08	(0.07)	0.31**	(0.09)	-0.01	(0.09)
Hindu	0.28*	(0.14)	0.03	(0.12)	0.37*	(0.15)	0.08	(0.15)
Mother's education level								
At least primary	-0.21	(0.12)	-0.15	(0.09)	-0.03	(0.12)	-0.07	(0.11)
At least secondary	-0.23	(0.15)	-0.61**	(0.12)	-0.03	(0.14)	-0.36*	(0.15)
Mother's age at birth								
<20 years	0.12	(0.14)	0.36**	(0.10)	-0.09	(0.14)	0.44**	(0.14)
25-29 years	-0.27	(0.13)	-0.10	(0.11)	0.11	(0.14)	0.09	(0.13)
30 years plus	-0.14	(0.17)	0.09	(0.13)	0.14	(0.17)	0.14	(0.16)
Birth order								
2-3	-0.33**	(0.12)	-0.60**	(0.10)	-1.00**	(0.13)	-0.49**	(0.13)
4 plus	-0.20	(0.17)	-0.79**	(0.14)	-0.88**	(0.18)	-0.39*	(0.17)
Father's education level								
At least primary	0.004	(0.11)	0.10	(0.09)	0.23*	(0.11)	-0.16	(0.11)
At least secondary	-0.20	(0.15)	0.03	(0.11)	0.15	(0.13)	-0.32*	(0.15)
Father day labourer	0.46**	(0.10)	0.11	(0.09)	0.14	(0.12)	0.14	(0.11)
No tube-well/pipe water	0.03	(0.11)	0.17	(0.09)	0.024	(0.13)	-0.15	(0.11)
Distance to health centre ^d	0.03*	(0.01)	0.03**	(0.01)	0.02	(0.01)	-0.002	(0.01)
Birth cohort child								
1993-1999	-0.20	(0.11)	-0.04	(0.09)	-0.19	(0.13)	-0.15	(0.11)
2000-2005	-0.49**	(0.13)	-0.50**	(0.11)	0.38**	(0.13)	-0.78**	(0.14)
Baseline intensity								
Day 0	0.99**	(0.16)	3.93**	(0.09)	4.58**	(0.14)	3.37**	(0.11)
Day 1	0.58**	(0.20)	2.53**	(0.12)	2.81**	(0.19)	2.32**	(0.15)
Days 2	0.35	(0.22)	1.89**	(0.15)	1.62**	(0.29)	1.97**	(0.17)
Days 3-6	0.76**	(0.10)	1.15**	(0.12)	1.52**	(0.19)	1.01**	(0.14)
Constant	-8.05**	(0.16)	-7.34**	(0.40)	-9.20**	(0.43)	-7.79**	(0.44)
Variance	0.35	(0.29)	0.52*	(0.23)	0.16	(0.12)	0.26	(0.19)
Covariance (row1)	-	-	0.36	(0.24)	0.02	(0.13)	0.01	(0.11)
Covariance (row2)	-	-	-	-	0.18	(0.14)	0.21	(0.17)
Covariance (row3)	-	-	-	-	-	-	0.20	(0.14)

 Table 6. Parameter estimates of intensity to neonatal deaths due to communicable diseases and different types of non-communicable diseases, comparison area.

Notes: ^d centered around its mean in each area; * p-value<0.05, ** p-value<0.01, standard errors are in parenthesis Reference category: gender is female, religion is Muslim, mother and father have no education, mother's age at birth 20-24 years, father is not day-labourer, source of drinking water is tube-well/pipewater, living at average

distance to health centre, child birth cohort 1987-1992, baseline intensity 7-28 days.

^a Low birth weight/prematurity

^b Neonatal infections, birth asphyxia, obstetric complications, respiratory disorders, birth trauma, cord haemorrhage congenital abnormalities

^c skin infections, fever, jaundice, intestinal obstruction, Oedemas, external cause (injury), homicide, and sudden infant death, unspecified cause, other disorder in the perinatal period





Figure 2: Neonatal deaths due to major non-communicable diseases, ICDDR,B area









Figure 3: Neonatal deaths due to major non-communicable diseases, comparison area



Figure 5: Neonatal deaths due to major communicable diseases, comparison area



Notes: NCDs: non-communicable diseases, LBW: low birth weight, NEO: neonatal related other conditions (infections, respiratory and cardiovascular disorder specific to the perinatal period), BA: birth asphyxia, OBSCOMP: obstetric complications, OTHER: sudden infant death, unspecified, other disorders originated in

the perinatal period etc. CDs: communicable diseases, ARI: acute respiratory infections/pneumonia, EPI: extended program for immunization related diseases.







Figure 8:Parametric estimates of cause-specific neonatal mortality (ref. individuals), ICDDR,B area, 1987-1992

Figure 9:Parametric estimates of cause-specific neonatal mortality (ref. individuals), comparison area, 1987-1992





Figure 10:Parametric estimates of cause-specific neonatal mortality (ref. individuals), ICDDR,B area, 2000-2005

Figure 11:Parametric estimates of cause-specific neonatal mortality (ref. individuals), comparison area, 2000-2005



Annex

Table A1: Assignment of causes of neonatal death, 1987-2005, HDSS, Matlab, Bangladesh.

Codes (ICD9, ICD10)	Labels of code	Categories used (Table 2)	Categories used (Table 5 & 6) LBW	
190, 192, 193,452,458,P05,P07	Preterm delivery/low birth weight	Low birth weight (LBW)		
454,P21	Birth asphyxia	Birth asphyxia (BA)		
457, 456, P22-P29, P35,P36,P51,P76,P80	infections, respiratory and cardiovascular disorder specific to the perinatal period	Neonatal related conditions (NEO)	NCs	
449, Q01, Q02, Q03,Q24, Q35,Q37, Q42,Q45,Q89	Congenital abnormalities	Congenital abnormalities (CA)		
453, P15	Birth trauma	Birth trauma		
451,P00,P01,P02,P03	Obstetric complications	OBSCOMP	-	
P59	Haemorragic	Haemorragic	-	
P90-P96	Other disorders originated in the perinatal period	unspecified	OTHER	
990,998,999,R34,R95,R96,R99	Unspecified causes	-		
R95,450,459	Sudden death	-		
K75, W75,X91, 293,344,420,460,461,552,555,559,691,738	Other specific	Other specific		
, 010,013,014	Acute watery diarrhoea, dysentery, acute non-watery diarrhoea	Diarrhoea, dysentery		
038,046	Septicamia, viral hepatitis	Septicemia, hepatitis		
321, 325, 328, 191	Pneumonia, ALRI, Pneumonia with diarrhoea, Pneumonia severe	ARI	CDs	
A41,B01,G03,J11,J18, A03	Other bacterial diseases, viral infections characterized by skin and mucous membrane lesions	ARI		
456	EPI	EPI	1	