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PARTITIONING THE EFFECT OF INFANT AND CHILD DEATH ON SUBSEQUENT FERTILITY: AN EXPLORATION IN BANGLADESH

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ABSTRACT

The authors propose a method to partition the fertility impact of infant and child death into two components—a physiological and a behavioral effect—by application of the Cox hazards model with three dummy variables that indicate the time of child death and the status of lactation with reference to the return of menstruation. Analysis of results from the 1991 Bangladesh Contraceptive Prevalence Survey (BCPS) indicates that child death far outweighs any other factor in increasing the likelihood of an additional birth. The physiological effect of a child's death is 90 percent stronger than the behavioral effect, and both effects wane quickly over time. It appears that an early cessation of lactation is not the sole cause of the physiological effect, at least in Bangladesh. The relative strength of the two effects supports the observation that Bangladesh is in the early stage of fertility transition.

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INTRODUCTION

Classical demographic transition theory implies that fertility decline is preceded by mortality decline. For this reason, the interrelationship between mortality and fertility has been a keen concern of demographers as well as policymakers. Indeed, during the past few decades, with wide availability of data from large-scale fertility surveys, abundant empirical evidence has demonstrated an increased probability of pregnancy among women who experience infant or child loss.¹

A widely accepted hypothesis offers two possible linkages between infant/child mortality and subsequent fertility. One is the physiological or biological effect. By the death of a child the postpartum infecund period is shortened owing to an interruption of lactation. Thus this effect is applicable to non-contracepting, breastfeeding societies. The other possible linkage is the behavioral or replacement effect. Child death may cause the parents to make deliberate efforts to bear another child in the hope of replacing the lost child. This effect is closely related to the total number of children that parents desire and the practice of contraception when the desired family size is reached.

The relative magnitudes of the two effects may be indicative of the stage of fertility transition: when parents allow fertility to be determined "naturally," then child death may cause only a physiological effect. By contrast, when fertility is under perfect rational control, then child death will

¹ For instance, J. A. Harrington, "The effect of high infant and childhood mortality on fertility: The West African case," *Concerned Demography* 3(1971), pp.22-35; A. Adlakha "Fertility and infant mortality an analysis of Turkish data," *Demography India* 1(1973), pp. 56-76; S. O. Rutstein, "The influence of child mortality on fertility in Taiwan," *Studies in Family Planning* 5(1974), pp. 182-88; H. P. Vogel and E. G. Knox, "Reproductive patterns after stillbirth and early infant death," *Journal of Biosocial Science* 7(1975), pp.103-11; A.K.M.A. Chowdhury, A. R.Khan, and L. C. Chen "The effect of child mortality experience on subsequent fertility in Pakistan and Bangladesh," *Population Studies* 30(1976), pp.249-61; H. Ware, "The relationship between infant mortality and fertility: Replacement and insurance effects," *International Population Conference* 1(1977), pp. 205-22; C. B. Park, S. H. Han, and M. K. Choe, "The effect of infant death on subsequent fertility in Korea and the role of family planning," *American Journal of Public Health* 69(1979), pp. 557-65; R. J. Olsen, "Estimating the effect of child mortality on the number of births," Economics-Growth Center Discussion Paper (1980), Yale University; C. M. Suchindran and A. L. Adlakha, "Infant mortality and its effect on fertility: The experience of Sri Lanka," Paper presented at the Annual Meeting of the Population Association of America, Washington, D.C., March 1981; A. R. Chowdhury, "The infant mortality-fertility debate: Some international evidence," *Southern Economic Journal* 54(1988), pp.666-74; B. S. Mensch, "The effect of child mortality on contraceptive use and fertility in Colombia, Costa Rica, and Korea," *Population Studies* 39(1985), pp.309-27; G. Santow and M. D. Bracher "Child death and time to next birth in Central Java," *Population Studies* 38(1984), pp.241-53; O. E. M. Nur "An analysis of the child survival hypothesis in Jordan," *Studies in Family Planning* 16(1985), pp. 211-18; S. H. Cochrane and K. C. Zachariah "Infant and child mortality as a determinant of fertility," World Bank Staff Working Papers 556(1983); S. B. S. Parmar, "The impact of infant mortality on fertility behavior of women," *Journal of Family Welfare* 36(1990), pp. 43-47; United Nations Economic and Social Commission for Asia and the Pacific "The influence of infant and child mortality on fertility in selected countries of the Asian and Pacific Region," *Population Research Leads* 1985; C. E. E. Okojie, "Fertility response to child survival in Nigeria," *Research in Population Economics* 7(1991), pp. 93-112.

invoke the replacement effect only. In a society undergoing demographic transition, both effects should be operating.²

Although this two-component hypothesis appears to provide a reasonable explanation of the fertility impact of child death, in fact partitioning the total effect into two components has not been very satisfactory. Probably the most frequently used procedure in such efforts has been to compare subsequent fertility measures, such as parity progression ratio or birth interval, following different survival outcomes of preceding children born in succession.³ For two preceding births, there are four possible outcomes: “survived-survived,” “survived-died,” “died-survived,” and “died-died.” Fertility differences following different survival outcomes are considered to suggest different effects. For instance, the fertility contrast between the two sequences of “survived-survived” and “died-survived” is considered to represent the psychological motivation to replace a lost child, as the death of the first child should not cause any interruption of lactation of the surviving second child.⁴

The fertility difference following the sequences “died-survived” and “survived-died” is considered to represent the physiological effect, as the psychological motivation should be similar in both cases. To evaluate the replacement effect, net of the physiological effect, Mensch⁵ rigorously examined the probability of birth following different survival patterns of three preceding births, excluding from analysis all cases in which the last birth died. In this manner a replacement effect would not be confounded by a shortened postpartum amenorrheic period.

Other researchers believe that the physiological effects can be estimated by comparing birth intervals following a previous child that died and a previous child that survived, the rationale being that the physiological effect of the death of the previous child would be to shorten the birth interval. They also consider that the total effect of child death can be estimated by the parity progression ratio and the number of subsequent births a couple has, depending on the survival or death of their first few births.⁶ Sometimes median birth intervals following neonatal death and postneonatal death are compared

² United Nations Secretariat, “Interrelationship between child survival and fertility,” *Population Bulletin of the UN* 25 (1988), pp. 27–50.

³ Adlakha (1973); Chowdhury et al. (1976); Knodel (1977); Park, Han, and Choe (1979); Suchindran and Adlakha (1981).

⁴ Suchindran and Adlakha (1981).

⁵ Mensch, B.S. (1985).

⁶ Cochrane and Zachariah (1983).

separately with median birth intervals following children that survived, tacitly assuming that neonatal death would be associated predominantly with a physiological effect.⁷

In these approaches, however, several problems are obvious. There are logical difficulties in interpreting fertility differences following contrasting survival outcomes of preceding birth(s). Consider the comparison of the outcomes of two preceding births. The difference in the two sequences “survived-survived” and “died-survived” is supposed to show the replacement effect, but in the second sequence the first child who died may be considered to have already been replaced by the surviving second child. A similar argument may be applied to an analysis that considers three preceding births. As Mensch⁸ admits that “any response to the death of a child should be greatest during the interval in which the death occurs,” her approach actually underestimates the replacement effect. The contrast between the two sequences “died-survived” and “survived-died” is used to measure the physiological effect, but fertility following the latter sequence would reflect both physiological and behavioral effects, as the parents may be trying to replace the lost child.

It is equally incorrect to say that the difference in the subsequent birth interval represents the physiological effect, while the difference in the number of subsequent births represents the total effect. These two fertility measurements are functionally related. Theoretically, the reciprocal of the average birth interval is the hazard or risk of childbirth. Thus, a short birth interval results in a high parity progression ratio and consequently a large completed fertility.

Most previous studies have used aggregate analysis, rather than individual analysis. When the sample size is moderate or small, controlling for variables associated with birth interval and/or parity progression becomes problematic. When birth intervals are compared, open intervals have to be excluded unless the hazard model is applied. If the survival status of a minimum of two previous births is necessary to separate the two components of the effect of child death, then it may not be easy to obtain enough study material from low-fertility societies. In any case, conventional approaches do not offer an explicit partitioning of the effect.

In this paper we propose a new approach to distinguish the physiological from the behavioral effect of infant/child death on subsequent fertility, using data from Bangladesh. Essentially, the approach

⁷ Cochrane, S.H. and Zachariah, K.C. (1983).

⁸ Mensch, B.S. (1985).

is an application of the Cox hazard model⁹ with dummy variables that indicate the time of infant/child death and the lactation status of children that survive the postpartum amenorrheic period.

Conceptual Framework

The timing of the death of an index infant/child with reference to the mother's resumption of menstruation (RM) is crucial for partitioning the effect of that death on subsequent fertility. The resumption of ovulation postpartum signals the end of the infecund period following childbirth. It is difficult, however, to measure the return of ovulation directly without using such methods as taking daily temperatures, studying cervical mucus, or conducting endometrial biopsies. In the absence of a better measurement for the postpartum anovulatory period, the postpartum amenorrheic period is customarily equated with the period of infecundability. Empirical and endocrinological studies have shown that if lactation is interrupted, the postpartum amenorrheic period is shortened.¹⁰ Thus the physiological effect on the mother of a child's death is essentially a lactation effect, and it should arise only when a lactating child dies before the RM.

The behavioral effect of a child's death stems from the deliberate efforts of parents to produce another child to replace the one that was lost. In this case, if a child dies before the conception of the next child, regardless of the time of its death, then the desire for another child could arise, given that the number of living children is within the family size norm. If a child dies before the RM, both physiological and replacement effects could occur. By contrast, if a child dies after the RM, only the replacement effect should be operating as the mother's ovulation has already begun.

We now introduce the following dummy variables, depending on the survival status and the time of death of the index child:

$$\begin{aligned} z_1 &= 1, && \text{if the index child dies before the RM,} \\ &= 0, && \text{otherwise, and} \\ z_2 &= 1, && \text{if the index child dies between the RM and the conception of the next child (or} \\ &&& \text{the survey date, whichever comes first in a retrospective survey),} \\ &= 0, && \text{otherwise.} \end{aligned}$$

⁹ Cox, D.R. (1972).

¹⁰ Shyam Thapa, Roger V. Short, and Malcolm Potts, *Nature* 335(1988), pp. 679–82.

Assuming that the risk of childbirth is described by the hazard model, we are tempted to formulate the following model:

$$h_i(t) = h_o(t)e^{\gamma_1 z_1 + \gamma_2 z_2 + \sum \beta x}$$

where $h_i(t)$ and $h_o(t)$ are the hazard functions of childbirth following the index child for the i -th person and baseline person, respectively, and x is the vector of covariates that affect the hazard of childbirth. Then γ and β are the unknown coefficients associated with z and x . Thus, under this model γ_1 represents the combination of the physiological and behavioral effects, while γ_2 represents the behavioral effect only. Obviously, the difference between γ_1 and γ_2 should measure the physiological effect.

Unfortunately, $h_o(t)$ in the above model is not the baseline hazard function as long as the physiological effect of a lost child is treated as a covariate and is considered as a lactation effect. Note that the physiological effect is caused by an early cessation of breastfeeding during the amenorrheic period. A child that survives through the amenorrheic period but is never breastfed or is breastfed during only part of the amenorrheic period also causes the same fertility impact. Thus the reference group must be composed of children who survive and are breastfed throughout the amenorrheic period. Introduce another dummy variable z_3 that indicates the breastfeeding status of the surviving children. Let

$$\begin{aligned} z_3 &= 1 && \text{if the index child survives through the postpartum amenorrheic period but is} \\ &&& \text{never breastfed or is breastfed during only part of the amenorrheic period,} \\ &= 0 && \text{otherwise.} \end{aligned}$$

Our hazard model now becomes:

$$h_i(t) = h_o(t)e^{\gamma_1 z_1 + \gamma_2 z_2 + \gamma_3 z_3 + \sum \beta x}$$

If indeed the physiological effect consists solely of the lactation effect, the difference between γ_1 and γ_2 should be roughly equal to γ_3 , the coefficient associated with z_3 . Further refinements, such as time-dependency and interaction effects, may be incorporated into the model as necessary.

Data

The data for this study were derived from the 1991 Bangladesh Contraceptive Prevalence Survey (BCPS), which covered a representative sample of ever-married women under age 50. The survey used a two-stage "probability-proportional-to-size" (PPS) sample: rural villages or urban "mahallas" were

selected in the first stage, and households were selected in the second stage. Respondents were ever-married women under 50 years old in the selected households. A total of 12,347 eligible women were identified, and 12,050 of them were interviewed—8,873 from the rural stratum and 3,077 from the urban. The nonresponse rate was thus approximately 2 percent. Mitra, Lerman, and Islam (1993) describe technical aspects of the survey, sample characteristics, and overall findings in detail.¹¹

The 1991 BCPS was the sixth survey conducted in Bangladesh since 1979 that collected information on fertility and contraceptive use. The data are considered to be of good quality, with results similar to those of other surveys conducted during the same period such as the 1989 Bangladesh Fertility Survey.¹²

For this analysis, the index children are derived from all births that the BCPS respondents reported for the period 1986–91, five years preceding the survey date. However, births to mothers younger than 15 or older than 49 are excluded from analysis, as are multiple births. As the survey did not ask about the gestation period for live births, we arbitrarily assumed that the conception date for each child was nine months prior to the birth date. For women who were pregnant at the time of the survey, however, the conception date was determined by the length of the pregnancy reported. When the RM between two births was not reported, the date of the RM was set as the conception date of the second child.

To study the effect of infant/child death on subsequent fertility, we used the Cox proportional hazards model. The input variable is the birth interval following the birth of each index child. The endpoint is the birth of the next child. Open birth intervals at the time of the survey are, of course, a censored case. The analysis is based on 10,925 intervals. There were 575 deaths before the RM and 217 deaths between the RM and the conception of the next child or the survey date. Thus the infant and child death rate during the referred period was 72.5 per 1,000 births—this is slightly lower than the infant mortality rate found in the BCPS,¹³ most likely because high-risk children (births to very young or very old women and multiple births) were excluded from the pool of index children.

For the covariates, we have included variables that are known to be associated with fertility. They are: age of mother at the index birth, educational attainment of mother, birth order of the index

¹¹ S. N. Mitra, Charles Lerman, and Shahidul Islam, *Bangladesh Contraceptive Prevalence Survey, 1991: Final report* (1993), Dhaka: Mitra and Associates.

¹² John Cleland, Nawab Ali, Edward Ebanks, M.A. Rashid, and Lokky Wai (eds.), *Secondary analysis of BFS, 1989 data* (1993), Dhaka: National Institute of Population Research and Training.

¹³ Mitra, et al. (1993) report the mortality rate as 81 in 12–59 months before the survey.

child, place of residence, employment status of mother, and sex of the index child. Because the age of mother and birth order are suspected to have nonlinear relationships with fertility, we introduced the squares of these variables into the model. In later stages of the analysis, we examined the interactions and the time dependency of the child-death effect. For a description of the variables, see Table 1.

Results

Life-table analysis. Before partitioning the fertility impact of child death, we first compare the probability of an additional birth by the survival and lactation status of the index child. Table 2 presents the cumulative probability, $1-S(t)$, that a woman will bear an additional child at each of three successive months following the birth of an index child. For the entire sample, the probability of birth increases almost linearly with time. Five years after a birth, two-thirds of women have born an additional child.¹⁴ Half of the women bear an additional child slightly more than 3.5 years (median time) after the previous birth, while the first quartile point is a little less than two years.

The probability of an additional birth is consistently higher if a child dies before the RM. At two years after a birth, the probability of an additional birth is more than three times higher for a woman whose previous child died before the RM than for a woman whose child survived. The difference between the two groups gradually diminishes, however: at four years after the first birth, the probability of an additional child is only about 35 percent higher for the “died” group than for the “survived” group.

Actually, there are no additional births in the “died” group after four years, while in the “survived” group the probability of an additional birth continues to increase. In other words, if a child dies during the amenorrheic period, the likelihood of a subsequent birth begins early and ends early, but if a child survives the amenorrheic period, the opposite is true. The overall difference in the two probability curves is highly significant by any commonly used test, such as the generalized Wilcoxon test ($p < 0.0001$).

There are similar, but smaller, differences in the probability of an additional birth between mothers whose child “survived” through the fecund period and those whose child “died” during the same period. Two years after the birth of the index child, there is a twofold difference between the two

¹⁴ Because our index children are all births that occurred during the five years 1986–91, the maximum length of birth interval is necessarily five years.

Table 1. Covariates considered in the study

Covariate	Description	Coding
Z1	Child death before RM	0 if survived, 1 otherwise
Z2	Child death between RM and conception of the next child or survey date, whichever is earlier	0 if survived, 1 otherwise
Z3	Breastfeeding before RM among children survived beyond RM	0 if fully breastfed, 1 otherwise
EMP	Employment status of mother	1 if employed for payment, 0 otherwise
RES	Place of residence	1 if urban, 0 rural
SEX	Sex of the index child	1 if male, 2 female
EDU	Education of mother	0 = no education, 1 = primary school, 2 = high school, 3 = college/professional
OCU	Occupation of father	1 if white collar, 0 otherwise
AGE	Age of mother at birth of index child	In years
AGE ²	Square of AGE	
BOR	Birth order of index child	Order (1 to 18)
BOR ²	Square of BOR	

RM: resumption of menstruation

Table 2. Probability of bearing an additional child after the index birth

Months after birth of index child	Entire sample	Before RM, child		After RM, child		Before RM, child was	
		Survived	Died	Survived	Died	Breastfed	Not Breastfed
9.0	0.000	0.000	0.000	0.000	0.000	0.000	0.000
12.0	0.006	0.004	0.030	0.004	0.010	0.004	0.004
15.0	0.028	0.020	0.170	0.019	0.077	0.020	0.022
18.0	0.046	0.035	0.242	0.034	0.114	0.034	0.040
21.0	0.080	0.066	0.312	0.065	0.136	0.065	0.072
24.0	0.129	0.114	0.385	0.112	0.220	0.111	0.132
27.0	0.210	0.192	0.510	0.189	0.347	0.187	0.217
30.0	0.268	0.251	0.570	0.246	0.435	0.247	0.271
33.0	0.327	0.310	0.627	0.306	0.466	0.321	0.337
36.0	0.386	0.370	0.649	0.369	0.511	0.362	0.402
39.0	0.446	0.432	0.691	0.428	0.574	0.421	0.470
42.0	0.497	0.484	0.714	0.482	0.597	0.474	0.522
45.0	0.530	0.519	0.737	0.515	0.654	0.510	0.551
48.0	0.569	0.559	0.751	0.556	0.654	0.545	0.600
51.0	0.608	0.598	0.767	0.597	0.666	0.584	0.639
54.0	0.632	0.624	0.767	0.623	0.666	0.608	0.666
57.0	0.651	0.644	0.767	0.644	0.666	0.626	0.686
60.0	0.666	0.660	0.767	0.660	0.666	0.638	0.703
Median time	42.3	43.4	26.8	43.6	35.3	44.2	40.7
N	10,925	10,350	575	10,133	217	8,991	1,359
Mantel		223.8***		14.8***		17.9***	
Breslow		405.4***		34.8***		12.3**	
Peto-Prentice		320.9***		25.2***		15.4**	
		***P < 0.0001		**P < 0.001			

groups, but five years after the birth of the index child there is only a 10 percent difference. The difference in the probability distribution was highly significant. Among the mothers with children who survive the postpartum amenorrheic period, the probability of an additional birth for the breastfed group is significantly lower than that for the never- or partially-breastfed group. In contrast to the previous two cases, the difference in the probability of an additional birth between the breastfed and the never- or partially-breastfed groups tends to increase over time.

Contrasts in the cumulative probability of an additional birth by survival and breastfeeding status of the index child may be examined by the median time to child birth. If the index child dies before the RM, the median time before the next birth is 40 percent shorter than if it survives; if the child dies after

the RM, the median time before the next birth is 20 percent shorter than if it survives. Among cases where the index child survives beyond the RM, the median time before an additional birth is less than 10 percent longer if the index child was breastfed throughout the amenorrheic period than if it was never breastfed or was breastfed during only part of the period.

The estimated hazard (instantaneous) rate of childbirth at the midpoint of each interval was compared by survival outcome and lactation status. In the entire group the hazard rate first increases with time, reaching a plateau at 25–50 months after the index birth, and then begins to decline (Table 3). The hazard rate for index children that died before the RM is much higher in the early period than the rate for children that survived: at 12 months the rate for the former group is nearly 10 times the rate for the latter. The relative difference becomes steadily smaller with time, however, and a crossover occurs at 33 months. From 51 months, the hazard of childbirth vanishes for the “dead” group, while that for the “survived” group still maintains considerable strength.

Among index children that survived the RM but died subsequently, large excesses in the hazard rate are found only in early periods. Between 30 and 40 months, the hazard rates for those that died and those that survived are practically on a par, and after 50 months the hazard for the “died” group vanishes (Table 4). As in the cumulative probability of an additional birth, the degree of difference in the hazard rate is much smaller between index children that survived or died after the RM than between those that survived or died before the RM: at 13.5 months, the difference in hazard rates is nearly 10 times for those that survived or died before the RM, compared with nearly five times for those that survived or died after the RM.

The effect of breastfeeding status before the RM is totally different. The relative difference in hazard rates between breastfed and the never- or partially-breastfed children remains consistent throughout the entire period of observation, with the former only slightly less than the latter.¹⁵ As the gap between the two hazard rates is about the same through the entire period, the difference in the cumulative probability widens with time.

Multivariate survival analysis. To evaluate the individual components of the child-death effect controlling for other covariates, the Cox proportional hazards model was applied to the data. Several possible models are shown in Table 5. In the full model that includes all the variables listed in Table 1 (Model 1), the effects of occupation of father, birth order, and the square of birth order are not significant by the Wald test. Although age of mother is not significant *per se*, its square is. In Model 2,

¹⁵ Toward the end of the reference period the difference becomes larger, but it is not statistically significant.

Table 3. Hazard rate of subsequent childbirth by survival status of the index child before the resumption of menstruation

Time (months)	Entire sample	Survived	Died	Ratio
10.5	0.0019	0.0014	0.0101	7.2
13.5	0.0077	0.0055	0.0518	9.4
16.5	0.0062	0.0051	0.0301	5.9
19.5	0.0118	0.0109	0.0327	3.0
22.5	0.0184	0.0176	0.0371	2.1
25.5	0.0323	0.0307	0.0753	2.4
28.5	0.0258	0.0252	0.0435	1.7
31.5	0.0278	0.0272	0.0472	1.7
34.5	0.0303	0.0306	0.0201	0.6
37.5	0.0343	0.0341	0.0422	1.2
40.5	0.0323	0.0325	0.0265	0.8
43.5	0.0229	0.0228	0.0275	1.2
46.5	0.0289	0.0291	0.0183	0.6
49.5	0.0311	0.0313	0.0222	0.7
52.5	0.0213	0.0219	0.0000	0.0
55.5	0.0177	0.0183	0.0000	0.0
58.5	0.0146	0.0149	0.0000	0.0

therefore, we eliminate the three nonsignificant covariates but retain the other covariates plus maternal age and its square. This model provides a good fit: the difference in the log likelihood (-2LL) between Model 1 and Model 2 is negligible ($p > 0.10$). By contrast, the exclusion of age-related variables (Model 3) entails a drastic increase in -LL, indicating the importance of mother's age in the risk of an additional birth. Models 4-6 demonstrate the importance of survival and lactation status (i.e., z-related) variables. An examination of -LL clearly suggests that none of the z-related variables should be omitted from the analysis. The nonsignificance of birth order may indicate that the family size norm is still large in Bangladesh so that the number of children already born does not affect the likelihood of an additional birth.

Model 2 provides the best description of the relationship between the covariates considered and the hazard of an additional birth. This is a proportional model, as no time-dependent effects are considered. The estimates of coefficients, standard errors, and relative risks (hazard ratios) are shown in

Table 4. Hazard rate of subsequent childbirth by survival status of index child after the resumption of menstruation (Z2) and breastfeeding status during the postpartum amenorrhic period (PPA) in index child survived through PPA (Z3)

Time (months)	z2			z3		
	Survived	Died	Ratio	Breastfed	Not breastfed	Ratio
10.5	0.0014	0.0033	2.4	0.0014	0.0015	1.1
13.5	0.0051	0.0233	4.6	0.0054	0.0061	1.1
16.5	0.0049	0.0137	2.7	0.0049	0.0059	1.2
19.5	0.0109	0.0083	0.8	0.0108	0.0114	1.1
22.5	0.0173	0.0341	2.0	0.0166	0.0223	1.3
25.5	0.0301	0.0589	2.0	0.0299	0.0342	1.1
28.5	0.0246	0.0485	2.0	0.0255	0.0240	0.9
31.5	0.0274	0.0185	0.8	0.0260	0.0314	1.2
34.5	0.0307	0.0294	1.0	0.0294	0.0347	1.2
37.5	0.0338	0.0462	1.4	0.0323	0.0397	1.2
40.5	0.0328	0.0182	0.6	0.0316	0.0350	1.1
43.5	0.0221	0.0507	2.3	0.0237	0.0206	0.9
46.5	0.0298	0.0000	0.0	0.0250	0.0386	1.5
49.5	0.0318	0.0121	0.4	0.0301	0.0338	1.1
52.5	0.0225	0.0000	0.0	0.0197	0.0256	1.3
55.5	0.0187	0.0000	0.0	0.0159	0.0214	1.3
58.5	0.0152	0.0000	0.0	0.0107	0.0185	1.7

Table 6. If a child dies during the postpartum amenorrhic period (Z1), its effect on subsequent fertility is extremely large: the relative risk of an additional birth is increased by 180 percent. Since the effect of z_1 is considered to be a combination of physiological and behavioral effects, we estimate the physiological effect by subtracting the coefficient of z_2 from that of z_1 . This is 0.631, which indicates that the physiological effect of child death increases the hazard of an additional birth by nearly 90 percent ($e^{0.631}=1.88$).

The behavioral effect is much weaker than the physiological effect, but the relative risk is still 50 percent higher than unity. The effect of lactation during the amenorrhic period (Z3) is much weaker than the physiological effect, with a relative risk of 1.25.

The directions of the effects of other covariates are reasonable, but their magnitudes are generally small relative to the effects of child death or lactation. Urban women and employed women have similar lower risks of an additional birth. As women's educational attainment goes up, the risk of an

Table 5. Comparison of some possible Cox models

Covariate	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Z1	1.025	1.030	0.925		0.979	
Z2	0.395	0.399	0.378		0.368	
Z3	0.227	0.226	0.194			0.153
EMP	-0.127	-0.128	-0.134	-0.107	-0.128	-0.107
RES	-0.140	-0.137	-0.115	-0.138	-0.132	-0.143
SEX	0.113	0.113	0.111	0.106	0.109	0.108
EDU	-0.166	-0.167	-0.128	-0.185	-0.168	-0.185
OCU	0.009 ^a					
AGE	0.047 ^a	0.042		0.032 ^a	0.045	0.029 ^a
AGE2	-0.002	-0.002		-0.002	-0.002	-0.002
BOR	-0.002 ^a					
BOR2	0.002 ^a					
Log likelihood (-LL)	3296.3	30297.3	30484.5	30413.2	30312.6	30406.0
Degrees of freedom (df)	12	9	7	6	8	7

^a p > 0.05

additional birth declines sharply. Note that the education variable is not dichotomous, but rather is grouped into four classes. Between women of no education and the highest education level, the relative risk declines by about twofold ($e^{4 \times -0.167} = 0.51$).

The effect of maternal age is nonlinear. While the direction of the effect of AGE itself is positive, that of its square is negative, indicating that the increase in the risk of an additional birth gradually diminishes with mother's age, and the risk eventually declines at old age. The maximum risk is reached at 23.4 years by taking the derivative of the risk function of age and its square $0.0421t - 0.0018t^2$, where t is mother's age in years.

The sex of the index child has a significant effect on the risk of an additional birth. The risk following a female birth is 12 percent higher than that following a male birth; apparently parents are more likely to continue childbearing after having a daughter with a hope that the next birth will be a son. Several authors have indicated a strong son preference in Bangladesh.¹⁶

¹⁶ R. Bairagi and R. L. Langsten, "Sex preference for children and its implication for fertility in rural Bangladesh," *Studies in Family Planning* 17(1986) pp.302-7; R. Amin and A. G. Mariam, "Son preference in Banglaesh: An emerging barrier to fertility regulation," *Journal of Biosocial Science* 19(1987), pp.221-28; A. J. M. Sufian and N. E. Johnson, "Son preference and child replacement in Bangladesh: a new look at the child survival hypothesis," *Journal of Biosocial Science* 21(1989), pp.207-16; M. K. Chowdhury and R. Bairagi, "Son preference and fertility

Table 6. Estimated effects of covariates on the hazard of additional childbirth (see Table 1 for covariates)

Covariate	Coefficient	Standard Error	Relative Risk
Z1	1.030	0.063	2.80
Z2	0.399	0.097	1.49
Z3	0.226	0.040	1.25
EMP	-0.128	0.037	0.88
RES	-0.137	0.041	0.87
SEX	0.113	0.033	1.12
EDU	-0.167	0.028	0.84
AGE	0.042	0.020	1.04
AGE2	-0.002	0.000	1.00

If the sex of an index child affects the risk of an additional birth, then the effect of an index child's death should also vary according to the dead child's sex. Specifically, the replacement effect of a lost boy should be stronger than that of a lost girl. To consider this proposition, we introduce four new indicator variables: $z_4 = 1$ if a boy dies before RM and $z_4 = 0$ if otherwise, $z_5 = 1$ if a girl dies before RM and $z_5 = 0$ if otherwise, $Z_6 = 1$ if a boy dies between RM and the conception of the next child or the survey date, whichever is earlier, and $z_6 = 0$ if otherwise, and $Z_7 = 1$ if a girl dies between RM and the conception of the next child or the survey date, whichever is earlier, and $z_7 = 0$ if otherwise.

By substituting these new variables for Z1 and Z2 in Model 2, we evaluate the effect of the sex of a child who dies. The excess replacement effect of a male death, compared with a female death, is given by $\gamma_6 - \gamma_7$; the excess physiological effect is given by $(\gamma_4 - \gamma_6) - (\gamma_5 - \gamma_7)$, where γ_i is the coefficient associated with z_i , $i = 4, \dots, 7$. As shown in Table 7, at least in terms of a point estimate, there is evidence of a stronger replacement effect when a boy dies than when a girl dies: the relative risk of an additional birth is 20 percent higher ($e^{0.186}$) following the death of a boy. In terms of the confidence interval, however, the results are not conclusive. More convincing evidence may require a larger sample. There is little difference in the physiological effect according to the sex of a dead child. Here, the relative risk of an additional birth is less than 4 percent higher if the dead child is a boy, even by the point estimate.

in Bangladesh," *Population and Development Review* 16(1990), pp.749–57; A. I. Chowdhry, R. Bairagi, and M. A. Koenig, "Effects of family sex composition of fertility preference and behaviour in rural Bangladesh," *Journal of Biosocial Science* 25(1993), pp. 455–64; M. Rahman and J. DaVanzo, "Gender preference and birth spacing in Matlab, Bangladesh," *Demography* 30(1993), pp. 315–32.

Table 7. Estimated effects of sex of dead child and other covariates (see Table 1 for covariates)

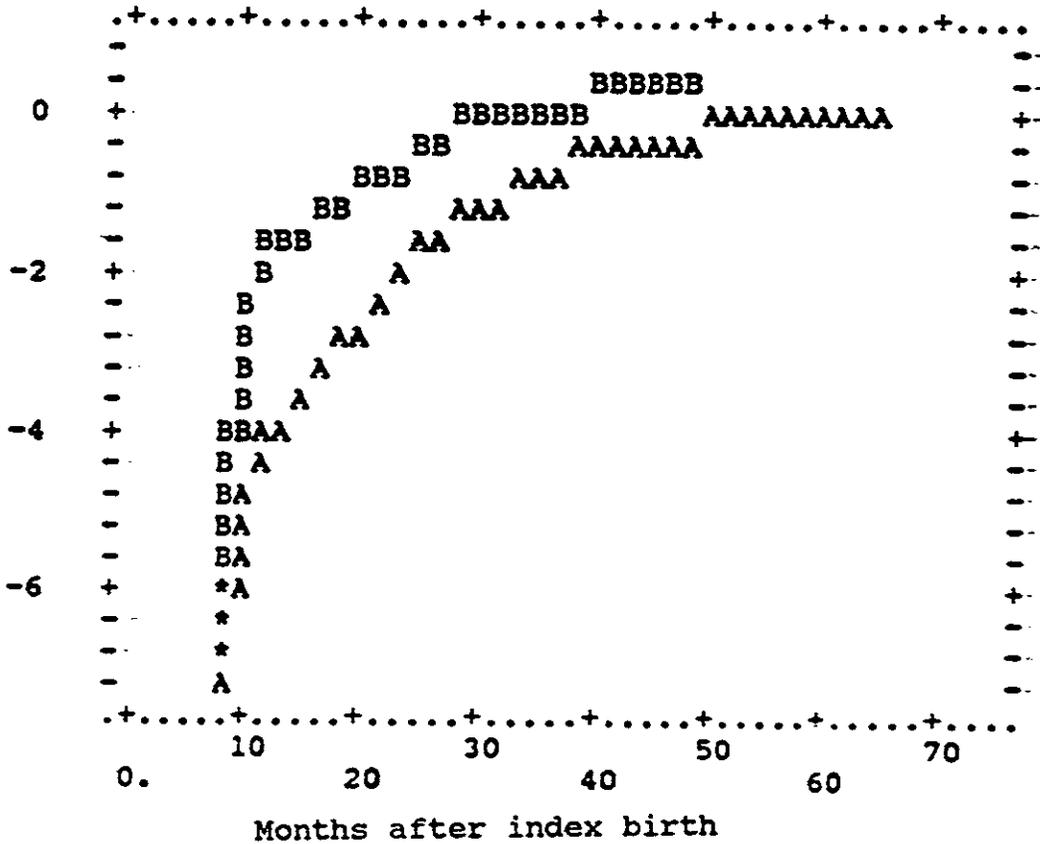
Covariate	Entire sample		
	Coefficient	Standard Error	Relative Risk
Z3	0.227	0.041	1.25
Z4	1.135	0.084	3.11
Z5	0.913	0.093	2.49
Z6	0.504	0.144	1.65
Z7	0.318	0.132	1.37
EMP	-0.131	0.037	0.88
RES	-0.139	0.041	0.87
SEX	0.136	0.035	1.14
EDU	-0.168	0.028	0.84
AGE	0.044	0.020	1.04
AGE2	-0.002	0.000	1.00

The actuarial analysis suggests that child death has time-dependent effects on the risk of an additional birth. To investigate a possible nonproportionality in the z-related effects, we plot the “log minus log survival functions” $\log(-\log S(t))$ for the two strata of each z-variable, where $S(t)$ is the “survival” function (probability of not bearing another child) at t time since the index birth. A strong deviation from the parallel in the two curves for z_1 strata (Figure 1) suggests that the effect of early child death is not constant over time. On the other hand, deviations from the parallel are not readily noticeable for z_2 , and especially for z_3 (Figures 2 and 3), suggesting weak or no time-dependent effects.

Assuming a simple form of time dependency for z's, we introduce the arbitrary functions $Tz_i(t) = Z_i * (\lg(t-8))$, $i = 1, 2, 3$. The addition of these three time-dependent effects results in a significantly large reduction in $-2LL$ from that of Model 2, but $TZ_3(t)$ is not significant according to the Wald test. We thus consider a model that includes time-dependent effects of z_1 and z_2 only. This time-dependent model reduces the log likelihood from Model 2 by 131.0 for 2 degrees of freedom ($p < 0.001$). According to the new model, the main effects of z_1 and z_2 are extremely high but decline quickly over time: each month, the relative risk is reduced by 60 percent ($e^{-0.986}$) for z_1 and by 50 percent ($e^{-0.715}$) for z_2 (Column A of Table 8).

Child-survival and lactation variables may interact with other covariates. We provisionally determined the possible significant interactions by examining differences in the coefficients of the z-

Figure 1. Log minus log survivor functions for Z1 = 0 (A) and Z1 = 1 (B)

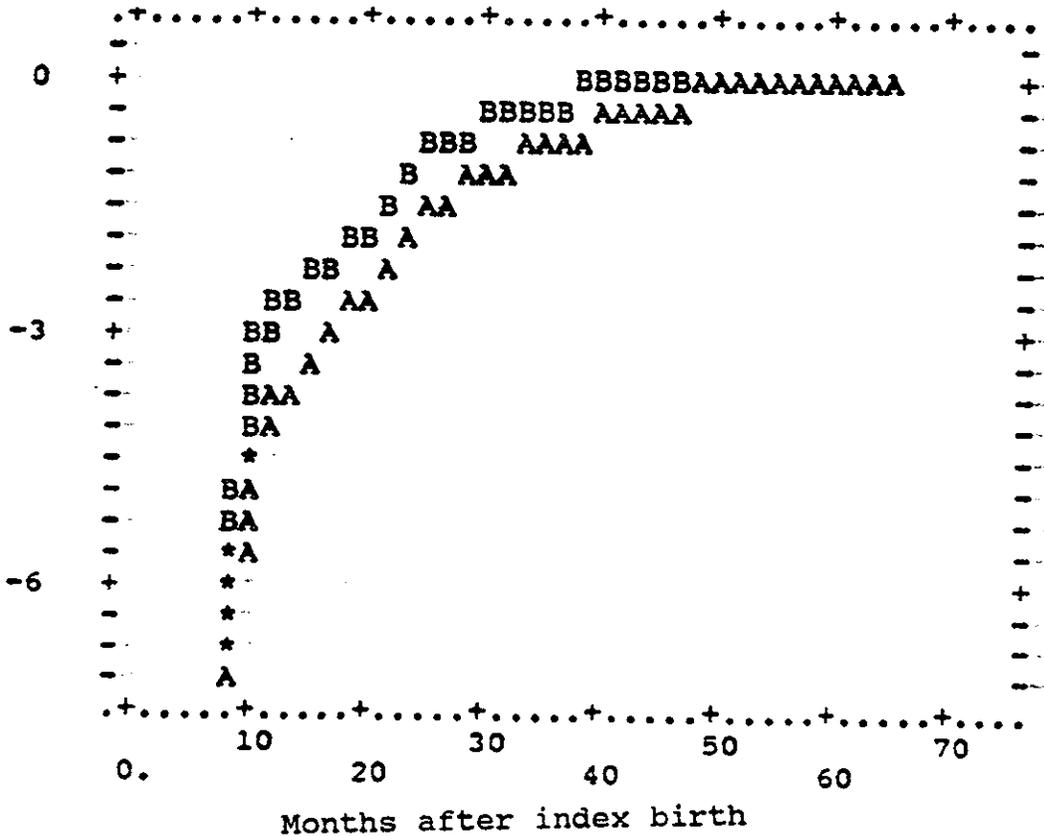


variables when the hazards model is applied separately to two levels of a given covariate.¹⁷ By including these possible interaction terms in the time-dependent model, we identified five significant interactions: age and education with z1 (z1*AGE and z1*EDU), education with z2 (z2*EDU), and employment and sex of the index child with z3 (z3*EMP and z3*SEX).

The fit of the model that incorporates both time dependency and the significant interactions is better than that any of the earlier models (Column B of Table 8). The difference in -2LL from Model 2 is 153.2 for 7 degrees of freedom, and the difference in -2LL from the time-dependency model is 22.2 for 5

¹⁷ For simplicity, we dichotomized all the variables. For maternal age, we arbitrarily set 25 years of life as the cutoff point (n = 6,359 for age < 25 and n = 4,566 for age ≥ 25). For education, women with no education formed one group with n = 9,426, and women with any degree of formal education (n = 1,499) the other. We also considered birth orders 2 and 3 as the “moderate” family size (n = 3,955) and all others as the “extreme” size (n = 6,970). If the existing family size is moderate, the parent’s desire to replace a dead child is likely to be strong, but if the family size is large, the desire to replace a dead child might not be urgent. If the parents have only one child, they will go on to have another whether the first child survives or dies.

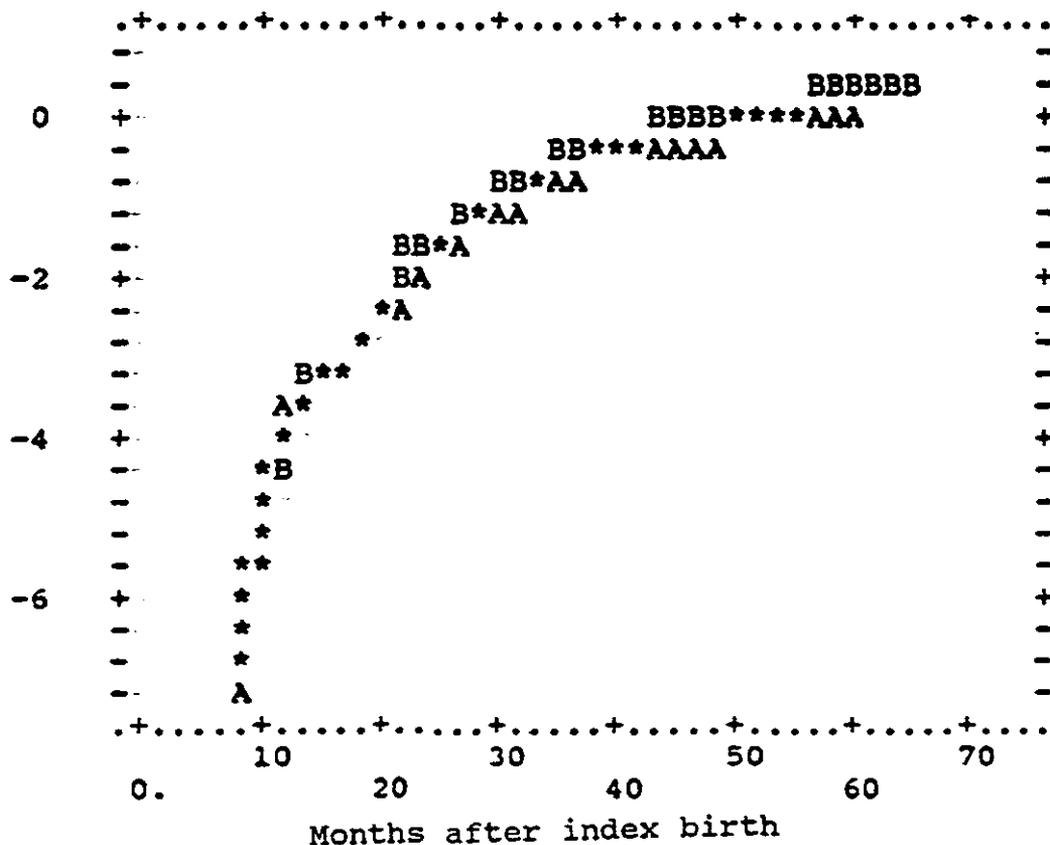
Figure 2. Log minus log survivor functions for $Z_2 = 0$ (A) and $Z_2=1$ (B)



degrees of freedom (in both cases $p < 0.001$). In general appearance, this new model resembles the time-dependency model (Table 8 Column A). In the new model, however, the main effect of z_1 becomes extremely strong and rapidly diminishes over time. Although the main effects of z_3 and SEX are nonsignificant, the interaction between them is large.

The magnitudes and directions of the interaction effects seem to be reasonable. The strong positive interaction between education and z_2 (with a relative risk of nearly 2.7) suggests a more rational reproductive behavior among the more educated. The fairly large interaction effect between z_1 and EDU may indicate a strong replacement effect among the highly educated, as z_1 represents both physiological and replacement effects. The negative effect of $z_1 * AGE$ suggests that the risk of an additional birth declines if the mother is old, even if a child died before RM. A never- or partially-breastfeeding mother has a lower risk of an additional birth if she is working, but the risk becomes higher if the index child is female.

Figure 3. Log minus log survivor functions for Z3 = 0 (A) and Z3=1 (B)



Discussion

Many researchers have demonstrated that a woman whose infant or child dies has an elevated probability of pregnancy. A theory has been advanced asserting that the fertility impact of child death consists of two components—physiological and behavioral. The first effect is linked to a shortened postpartum anovulatory period owing to an early cessation of breastfeeding when a child dies. The second effect reflects the parents' desire to replace the lost child. In a society that is noncontracepting, the fertility effect of a child's death should be largely physiological, whereas in a society at an advanced stage of demographic transition, fertility after a child's death should largely reflect the desire to replace the child.

Assuming that the return of menses (RM) marks the resumption of fecundability, subsequent fertility following a child's death after the RM should be attributable to the replacement effect, whereas fertility following a child's death before the RM should result from both physiological and replacement effects. We have proposed a method to partition the total effect of child death on subsequent fertility into

Table 8. Estimated coefficients and relative risks of covariates in nonproportional hazard model

Effects	Time-dependent effects only (A)			Time-dependent effects with interaction effects (B)		
	Coefficient	Standard Error	Relative Risk	Coefficient	Standard Error	Relative Risk
Main effect						
Z1	3.629	0.238	37.68	4.094	0.308	60.00
Z2	2.446	0.408	11.55	2.282	0.416	9.80
Z3	0.212	0.040	1.24	-0.042	0.128	0.96
EMP	-0.119	0.037	0.89	-0.117	0.037	0.89
RES	-0.134	0.041	0.88	-0.089	0.046	0.91
EDU	-0.170	0.028	0.84	0.336	0.056	0.71
SEX	0.112	0.033	1.12	0.068	0.038	1.07
AGE	0.044	0.020	1.04	0.044	0.020	1.04
AGE2	-0.002	0.000	1.00	-0.002	0.000	1.00
Time-dependent effect						
TZ1	-0.986	0.092	0.37	-0.947	0.093	0.39
TZ2	-0.715	0.145	0.49	-0.682	0.146	0.50
Interaction effects						
Z1*AGE				-0.026	0.010	0.97
Z1*EDU				0.587	0.230	1.80
Z2*EDU				0.985	0.302	2.68
Z3*EMP				-0.198	0.094	0.82
Z3*SEX				0.194	0.078	1.21
TZ1: Z1*(log(t-8))						
TZ2: Z2*(log(t-8))						

these two components using data from Bangladesh. This method is based on a simple application of the Cox hazards model using three dummy variables that indicate the time of child death with respect to RM and the early breastfeeding status of a surviving child.

Our analysis suggests that the death of a child far outweighs any other factor, biological or social, such as the age or employment status of the mother, in influencing the probability of an additional birth. Considering the fact that infant mortality in Bangladesh is nearly 100 per 1,000 births, prevention of infant and child death should contribute significantly to reducing fertility. The effect of a child death before RM is much stronger than that of a later death. Both physiological and replacement effects appear to wane quickly over time, particularly the physiological effect.

The rapid decline in the physiological effect is reasonable, as an earlier infant death should cause an earlier return of fecundability. It is not clear why the desire to replace a lost child should decrease

with time, however: here, the underlying mechanism requires further research. By contrast, the lactation effect does not appear to be time dependent, possibly explained by an association between limitations on breastfeeding and the practice of family planning.

If the physiological effect is caused solely by an early cessation of breastfeeding, it is nothing but a lactation effect. Our proportional hazard model suggests, however, that the imputed relative risk of the physiological effect ($e^{t^1-r^2}$) is 50 percent stronger than that of the lactation effect (e^{t^3}). The time-dependency model offers evidence of an even larger difference in early periods following the index birth. The discrepancy between the physiological and the lactation effects suggests two possible explanations: (a) mothers who never breastfed or weaned their children early practiced family planning earlier and more effectively than mothers with who lost a child before RM or (b) the so-called physiological effect consists of something more than the lactation effect associated with child loss.

Although individual correlations are not presented, The BCPS report indicates that for every characteristic—such as place of residence, educational level, or employment status—women with a shorter mean duration of breastfeeding have a higher rate of current use of modern contraception than the group as a whole.¹⁸ Women's knowledge of the contraceptive effectiveness of breastfeeding may also contribute to the difference between physiological and lactation effects. While women who never breastfeed or who wean their children early may be more likely to use contraceptives to avoid pregnancy, women who continue to breastfeed until a child dies may not feel the need for family planning: before the death they are protected by breastfeeding and afterwards they want to replace the lost child. In addition, women who experience the death of a child may have a heightened risk of pregnancy related to overall family size.

The difference between physiological and replacement effects may be caused, at least in part, by postpartum abstinence or spousal separation. In Bangladesh, sexual abstinence is enjoined on religious grounds for 40 days after a birth.¹⁹ Among Hindus, abstinence may last even longer, up to 70 or 100 days.²⁰ This rule of abstinence may be lifted, however, if a child dies. In Java, Indonesia, postpartum abstinence is sanctioned to protect the lactation and the child at breast. In this case, the death of a child

¹⁸ Mitra et al. (1993).

¹⁹ Therese Blanchet, *Women, pollution, and marginality meanings and rituals of birth in rural Bangladesh* (1984), Dhaka: University Press; K. M. A. Aziz and Clarence Maloney, *Life stages, gender and fertility in Bangladesh* (1985), Dhaka: International Centre for Diarrheal Disease Research, Bangladesh.

²⁰ Aziz and Maloney (1985).

obviates the need for abstinence.²¹ In Bangladesh, long and frequent separations of women from their husbands appear to bring down fertility from the biological maximum.²² One study over a period of 2.5 years found that nearly a quarter of married women were separated from their husbands for 1.5 years or more for domestic and business reasons.²³ The main domestic reason for separation was a woman's visits to her natal home, in some cases probably so that infants could be cared for by the woman's parents. If a child dies during such a visit, the mother is likely to return to her husband.

The behavioral effect comes into play when parents wish to replace a child that has died. If they have not yet reached their desired family size, parents will go on to bear more children whether a child has died or not. Conversely, if the number of living children has already exceeded the desired family size, the death of a child will not motivate parents to bear another. Operationally, the replacement motivation comes into effect only if contracepting parents willfully discontinue contraception upon a child's death, or noncontracepting parents willfully fail to adopt contraception, in contrast to the behavior of parents who have not experienced the death of a child.²⁴ Thus in a strict sense, the "population at risk" for the replacement effect is composed of parents who have reached exactly their desired family size, who do not use contraception or at least not a permanent method, and who experience the death of a child. By contrast, studies on the replacement effect of child loss usually include all women with dead children. Because of this dilution of the population at risk, the strength of the replacement effect is expected to be generally low for the population as a whole, even if the desire to replace a lost child is strong for the "population at risk."

In this respect the Bangladesh situation does not seem to be particularly favorable for demonstrating the presence of a replacement effect. Although the total fertility rate has fallen considerably in recent times,²⁵ the family size norm is still high. As mentioned earlier, existing family

²¹ Santow, Gigi, and Bracker (1984).

²² Aziz and Maloney (1985).

²³ A. K. Chowdhury and S. Becker, *Determinant of national fertility study: Methods and descriptive tables for the prospective fertility study 1975-78*, Vol. 1 (1981), Dhaka: International Centre for Diarrheal Disease Research, Bangladesh.

²⁴ We discard other possibilities, such as increased sexual activity or better nutritional intake with the hope of increased probability of pregnancy.

²⁵ Andrew Kantner, Charles Lerman, and Mohammed Yusuf, *What can we say about fertility trends in Bangladesh? An evaluation of the 1991 population census*, Asia-Pacific Research Reports 5 (1995), Honolulu: East-West Center, Program on Population.

size is not a significant factor in the decision to bear another child. As of 1991, only about 30 percent of married women of reproductive age were practicing modern contraception. Nevertheless, the proportional hazard model indicates that the replacement effect increases the relative risk of an additional birth by as much as 50 percent, reflecting a strong motivation to replace a dead child. The physiological effect of a child's death is even stronger, however. These findings are consistent with the observation that Bangladesh is in an early stage of fertility transition²⁶ despite the recent achievement in fertility decline.²⁷ Our analysis also seems to indicate the importance of education in reproductive behavior. In general, an educated woman is 30 percent less likely to have a birth than an uneducated women, but an educated women who loses a child is more than 150 percent more likely to have an additional child than an uneducated woman.

We use RM as a surrogate for the resumption of ovulation, which is not directly observable. While this may be a satisfactory approximation in general, it may also involve considerable error.²⁸ Women may erroneously recognize a vaginal bleeding episode at around 40 days postpartum as the RM.²⁹ The resumption of ovulation is often accompanied by a low level of fecundability,³⁰ and there is evidence that fecundability is substantially reduced by continued breastfeeding beyond the RM.³¹ Incorporation of these factors, admittedly complicated, into the model is likely to introduce considerable changes in the estimates of the z-related parameters.

Using the RM as the dividing line, we create two indicator variables for the timing of the death of an index child. In actuality, the time of a child's death is a continuous variable. In particular, the timing of an infant death during the amenorrhic period affects the timing of the RM. As noted, the

²⁶ United Nation Secretariat (1988).

²⁷ Kantner et al. (1995).

²⁸ S. S. Esmelani, R. H. Gray, R. Apelo, and R. Ramos, "The reliability of menses to indicate the return of ovulation in breastfeeding women in Manila, the Philippines," *Studies in Family Planning* (1990)21, pp.243–50.

²⁹ K. I. Kennedy, R. Rivera, and A. S. McNilly, "Consensus statement on the use of breastfeeding as a family planning method," *Contraception* (1989)39, pp.477–91.

³⁰ R. H. Gray and P. E. Doyle. "The epidemiology of conception and fertility," In S. L. Barron and A. M. Thompson (eds), *Obstetrical epidemiology* (1983).

³¹ D. Guz and J. Hobcraft. "Breastfeeding and fertility: A comparative analysis," *Population Studies* (1991)45, pp. 91–108; Dilip C. Nath, C. Kenneth, and Kaushalendra K. Singh, "The role of breastfeeding beyond postpartum amenorrhoea on the return of fertility in India: A life table and hazards model analysis," *Journal of Biosocial Science* (1994)26, pp. 191–206.

fertility effect of a child's death is strongly time dependent. A function that describes the child's age at death may elucidate the fertility impact more clearly, but it would make it difficult to partition the impact into two components as was done here.

Contraceptive use is a major factor confounding the effects of child death and lactation. In fact, a child's death can only have a physiological effect if the parents are noncontracepting, whereas the death can have a replacement effect in a society that is at least partially contracepting. It is thus likely that including contraceptive use in the model will significantly reduce both components of the child-death effect on fertility. It was not possible to include contraceptive use in this instance, however, because the 1991 BCPS does not provide adequate information on the timing of adoption and discontinuation of family planning between births. Further refinements and alternative approaches should be contemplated in modeling the effects of child death. Comparative studies across different cultural settings and stages of fertility transition may also provide insights into the effects of child death on subsequent fertility and the interactions between child death, contraceptive use, and fertility behavior.