Partitioning the Effect of Infant and Child Death on Subsequent Fertility: An Exploration in Bangladesh

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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.

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...

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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

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³ Adlakha (1973); Chowdhury et al. (1976); Knodel (1977); Park, Han, and Choe (1979); Suchindran and Adlakha (1981).
⁴ Suchindran and Adlakha (1981).
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

is an application of the Cox hazard model\(^9\) 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{align*}
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} \\
     &= 0, & \text{the survey date, whichever comes first in a retrospective survey),} \\
\end{align*}
\]

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\(^9\) Cox, D.R. (1972).

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

\[ h_i(t) = h_0(t) e^{\gamma_1 z_1 + \gamma_2 z_2 + \sum \beta x} \]

where \( h_i(t) \) and \( h_0(t) \) 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 \( \gamma \) and \( \beta \) are the unknown coefficients associated with \( z \) and \( x \). Thus, under this model \( \gamma_i \) represents the combination of the physiological and behavioral effects, while \( \gamma_i^* \) represents the behavioral effect only. Obviously, the difference between \( \gamma_i \) and \( \gamma_i^* \) should measure the physiological effect.

Unfortunately, \( h_0(t) \) 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

\[ z_3 = 1 \quad \text{if the index child survives through the postpartum amenorrheic period but is never breastfed or is breastfed during only part of the amenorrheic period,} \]

\[ = 0 \quad \text{otherwise.} \]

Our hazard model now becomes:

\[ h_i(t) = h_0(t) e^{\gamma_1 z_1 + \gamma_2 z_2 + \gamma_3 z_3 + \sum \beta x} \]

If indeed the physiological effect consists solely of the lactation effect, the difference between \( \gamma_1 \) and \( \gamma_2 \) should be roughly equal to \( \gamma_3 \), 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-
marrid 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
children.

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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. 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

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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

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Description</th>
<th>Coding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Z1</td>
<td>Child death before RM</td>
<td>0 if survived, 1 otherwise</td>
</tr>
<tr>
<td>Z2</td>
<td>Child death between RM and conception of the next child or survey date, whichever is earlier</td>
<td>0 if survived, 1 otherwise</td>
</tr>
<tr>
<td>Z3</td>
<td>Breastfeeding before RM among children survived beyond RM</td>
<td>0 if fully breastfed, 1 otherwise</td>
</tr>
<tr>
<td>EMP</td>
<td>Employment status of mother</td>
<td>1 if employed for payment, 0 otherwise</td>
</tr>
<tr>
<td>RES</td>
<td>Place of residence</td>
<td>1 if urban, 0 rural</td>
</tr>
<tr>
<td>SEX</td>
<td>Sex of the index child</td>
<td>1 if male, 2 female</td>
</tr>
<tr>
<td>EDU</td>
<td>Education of mother</td>
<td>0 = no education, 1 = primary school, 2 = high school, 3 = college/professional</td>
</tr>
<tr>
<td>OCU</td>
<td>Occupation of father</td>
<td>1 if white collar, 0 otherwise</td>
</tr>
<tr>
<td>AGE</td>
<td>Age of mother at birth of index child</td>
<td>In years</td>
</tr>
<tr>
<td>AGE²</td>
<td>Square of AGE</td>
<td></td>
</tr>
<tr>
<td>BOR</td>
<td>Birth order of index child</td>
<td>Order (1 to 18)</td>
</tr>
<tr>
<td>BOR²</td>
<td>Square of BOR</td>
<td></td>
</tr>
</tbody>
</table>

RM: resumption of menstruation
<table>
<thead>
<tr>
<th>Months after birth of index child</th>
<th>Entire sample</th>
<th>Before RM, child survived</th>
<th>Died</th>
<th>After RM, child survived</th>
<th>Died</th>
<th>Before RM, child was Breastfed</th>
<th>Not Breastfed</th>
</tr>
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<tbody>
<tr>
<td>9.0</td>
<td>0.000</td>
<td>0.000</td>
<td>0.000</td>
<td>0.000</td>
<td>0.000</td>
<td>0.000</td>
<td>0.000</td>
</tr>
<tr>
<td>12.0</td>
<td>0.006</td>
<td>0.004</td>
<td>0.030</td>
<td>0.004</td>
<td>0.010</td>
<td>0.004</td>
<td>0.004</td>
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<tr>
<td>15.0</td>
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<td>0.020</td>
<td>0.170</td>
<td>0.019</td>
<td>0.077</td>
<td>0.020</td>
<td>0.022</td>
</tr>
<tr>
<td>18.0</td>
<td>0.046</td>
<td>0.035</td>
<td>0.242</td>
<td>0.034</td>
<td>0.114</td>
<td>0.034</td>
<td>0.040</td>
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<tr>
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<td>0.080</td>
<td>0.066</td>
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<td>0.136</td>
<td>0.065</td>
<td>0.072</td>
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<tr>
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<td>0.114</td>
<td>0.385</td>
<td>0.112</td>
<td>0.220</td>
<td>0.111</td>
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<td>27.0</td>
<td>0.210</td>
<td>0.192</td>
<td>0.510</td>
<td>0.189</td>
<td>0.347</td>
<td>0.187</td>
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<td>0.251</td>
<td>0.570</td>
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<td>0.435</td>
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<td>0.310</td>
<td>0.627</td>
<td>0.306</td>
<td>0.466</td>
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<td>0.337</td>
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<td>0.386</td>
<td>0.370</td>
<td>0.649</td>
<td>0.369</td>
<td>0.511</td>
<td>0.362</td>
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</tr>
<tr>
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<td>0.432</td>
<td>0.691</td>
<td>0.428</td>
<td>0.574</td>
<td>0.421</td>
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<tr>
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<td>0.497</td>
<td>0.484</td>
<td>0.714</td>
<td>0.482</td>
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<td>0.474</td>
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<tr>
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<td>0.519</td>
<td>0.737</td>
<td>0.515</td>
<td>0.654</td>
<td>0.510</td>
<td>0.551</td>
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<tr>
<td>48.0</td>
<td>0.569</td>
<td>0.559</td>
<td>0.751</td>
<td>0.556</td>
<td>0.654</td>
<td>0.545</td>
<td>0.600</td>
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<tr>
<td>51.0</td>
<td>0.608</td>
<td>0.598</td>
<td>0.767</td>
<td>0.597</td>
<td>0.666</td>
<td>0.584</td>
<td>0.639</td>
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<tr>
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<td>0.626</td>
<td>0.686</td>
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<tr>
<td>60.0</td>
<td>0.666</td>
<td>0.660</td>
<td>0.767</td>
<td>0.660</td>
<td>0.666</td>
<td>0.638</td>
<td>0.703</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Median time</th>
<th>42.3</th>
<th>43.4</th>
<th>26.8</th>
<th>43.6</th>
<th>35.3</th>
<th>44.2</th>
<th>40.7</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>10,925</td>
<td>10,350</td>
<td>575</td>
<td>10,133</td>
<td>217</td>
<td>8,991</td>
<td>1,359</td>
</tr>
</tbody>
</table>

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. 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,
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 amenorrheic period (PPA) in index child survived through PPA (Z3)

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

Table 6. If a child dies during the postpartum amenorrheic 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 Z1 is considered to be a combination of physiological and behavioral effects, we estimate the physiological effect by subtracting the coefficient of Z2 from that of Z1. 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 amenorrheic 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

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
<th>Model 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Z1</td>
<td>1.025</td>
<td>1.030</td>
<td>0.925</td>
<td>0.979</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Z2</td>
<td>0.395</td>
<td>0.399</td>
<td>0.378</td>
<td>0.368</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Z3</td>
<td>0.227</td>
<td>0.226</td>
<td>0.194</td>
<td></td>
<td>0.153</td>
<td></td>
</tr>
<tr>
<td>EMP</td>
<td>-0.127</td>
<td>-0.128</td>
<td>-0.134</td>
<td>-0.107</td>
<td>-0.128</td>
<td>-0.107</td>
</tr>
<tr>
<td>RES</td>
<td>-0.140</td>
<td>-0.137</td>
<td>-0.115</td>
<td>-0.138</td>
<td>-0.132</td>
<td>-0.143</td>
</tr>
<tr>
<td>SEX</td>
<td>0.113</td>
<td>0.113</td>
<td>0.111</td>
<td>0.106</td>
<td>0.109</td>
<td>0.108</td>
</tr>
<tr>
<td>EDU</td>
<td>-0.166</td>
<td>-0.167</td>
<td>-0.128</td>
<td>-0.185</td>
<td>-0.168</td>
<td>-0.185</td>
</tr>
<tr>
<td>OCU</td>
<td>0.009*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGE</td>
<td>0.047*</td>
<td>0.042</td>
<td>0.032*</td>
<td>0.045</td>
<td>0.029*</td>
<td></td>
</tr>
<tr>
<td>AGE2</td>
<td>-0.002</td>
<td>-0.002</td>
<td>-0.002</td>
<td>-0.002</td>
<td>-0.002</td>
<td>-0.002</td>
</tr>
<tr>
<td>BOR</td>
<td>-0.002*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BOR2</td>
<td>0.002*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

* \(p > 0.05\)

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 \(e^{5x-0.167}=0.51\).

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.\(^{16}\)

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

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

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 \( Z_1 \) and \( Z_2 \) 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 \( \gamma_i \) is the coefficient associated with \( z_i, i = 4, \ldots, 7 \). 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.

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

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

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 \( T_{z_i}(t) = Z_i \ast (\log(t-8)) \), \( i = 1, 2, 3 \). The addition of these three time-dependent effects results in a significantly large reduction in \(-2\text{LL}\) from that of Model 2, but \( T_{z_3}(t) \) 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 \( z \)-
variables when the hazards model is applied separately to two levels of a given covariate. By including these possible interaction terms in the time-dependent model, we identified five significant interactions: age and education with $z_1$ ($z_1*AGE$ and $z_1*EDU$), education with $z_2$ ($z_2*EDU$), and employment and sex of the index child with $z_3$ ($z_3*EMP$ and $z_3*SEX$).

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

---

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 $\geq 25$). 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.
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 $\text{SEX}$ 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 $\text{EDU}$ may indicate a strong replacement effect among the highly educated, as $z_1$ represents both physiological and replacement effects. The negative effect of $z_1*\text{AGE}$ suggests that the risk of an additional birth declines if the mother is old, even if a child died before $\text{RM}$. 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.
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

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

TZ1: Z1*(log(t-8))
TZ2: Z2*(log(t-8))

des 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 \( e^{\tau} \) is 50 percent stronger than that of the lactation effect \( e^{\alpha} \). 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. 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. Among Hindus, abstinence may last even longer, up to 70 or 100 days. 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

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18 Mitra et al. (1993).


20 Aziz and Maloney (1985).
obviates the need for abstinence. In Bangladesh, long and frequent separations of women from their husbands appear to bring down fertility from the biological maximum. 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. 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. 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, the family size norm is still high. As mentioned earlier, existing family

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22 Aziz and Maloney (1985).
24 We discard other possibilities, such as increased sexual activity or better nutritional intake with the hope of increased probability of pregnancy.
size is not a significant factor in the decision to bear another child. As of 1991, only about 30 percent of 
mixed 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 transition despite the recent achievement in 
fertility decline. 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. 
Women may erroneously recognize a vaginal bleeding episode at around 40 days postpartum as the 
RM. The resumption of ovulation is often accompanied by a low level of fecundability, and there is 
evidence that fecundability is substantially reduced by continued breastfeeding beyond the RM. 
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 amenorrheic period affects the timing of the RM. As noted, the

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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 
29 K. I. Kennedy, R. Rivera, and A. S. McNilly, "Consensus statement on the use of breastfeeding as a family 
(eds), Obstetrical epidemiology (1983).
amenorrhoea on the return of fertility in India: A life table and hazards model analysis," Journal of Biosocial 
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.