

The Impact of Infant and Child Death on Subsequent Fertility in Ethiopia

David P. Lindstrom<sup>1</sup>  
Gebre-Egziabher Kiros<sup>2</sup>

<sup>1</sup>Population Studies and Training Center  
Box 1916, Brown University  
Providence, RI 02912  
401-863-3765  
David\_Lindstrom@brown.edu

<sup>2</sup>Institute of Public Health  
Florida A&M University  
Tallahassee, FL 32307  
850-412-7422  
ge.kiros@famu.edu

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**Abstract**

This paper uses hazard regression models to assess the impact of experienced infant and child mortality on the risk of subsequent conceptions in Ethiopia. The purpose of this paper is to test for the presence of a fertility response to an infant or child death, net of the effects of truncated breastfeeding on fecundity. Using retrospective birth history data from a national survey in Ethiopia, we find a significantly higher risk of a conception in the months following the death of an index child, even after controlling for postpartum amenorrhoea and breastfeeding status. The fertility response is strongest after the death of the fourth or fifth child, which is when most women in Ethiopia are at or near their desired family size. We, however, find no evidence of a fertility response to the death of a nonindex child. We attribute the higher risk of a conception following an index child's death to the intentional efforts of couples to reduce the waiting time to a next birth and thereby replace the deceased child. However, absent evidence of replacement fertility in response to the death of older nonindex children, we interpret the response to the death of an index child as an emotional response to child loss rather than a conscious strategy to meet a fertility target.

The impact of infant and child mortality on fertility has been closely examined by demographers because of the net effect of mortality and fertility on the rate of population growth (Bongaarts and Bruce 2001; Lehrer 1984; Preston 1978). Because surveys rarely measure parents' expectations of mortality, most studies examine the influence of experienced mortality on subsequent fertility. Individual-level analyses tend to focus on the effect of the death of an index child, that is the child whose birth opens an interval, on the timing of a subsequent conception or birth. Analyses of high fertility populations consistently find that the death of an index child is associated with a shorter than expected birth interval, and similarly a higher probability of conception or birth (Chowdhury et al. 1976; Kuate Defo 1998; Park et al. 1998; Santow and Bracher 1984).

Studies consider two possible mechanisms for linking an index child's death to a subsequent conception or birth; one is physiological, and the second is volitional in nature. The physiological effect relates to the impact of an index child's death on breastfeeding and the return of menstruation. The inhibiting effects of breastfeeding on fecundity, particularly during the first twelve months after a birth, are well established. In the absence of breastfeeding the postpartum amenorrheic period is about two months (Lloyd and Ivanov 1988). Prolonged breastfeeding can extend the amenorrheic period for up to 19 months or more (Bongaarts and Potter 1983; Berhanu and Hogan 1998). Even after the return of menstruation breastfeeding can continue to depress a woman's fecundity. The death of a breastfeeding infant prematurely ends lactation, and thereby shortens the length of the postpartum amenorrheic period. In noncontracepting populations the early return of menstruation contributes to a higher risk of conception, and thus a shorter than normal birth interval.

Parents may also intentionally try to replace a deceased child as soon as possible with a subsequent birth. Known as the replacement effect, this conscious effort to compensate for infant or child mortality by shortening the time to a next birth is generally believed to occur in populations where mortality has begun to decline, and contraceptive use is common (Lloyd and Ivanov 1988; Nur 1985; Park et al. 1998).

Most studies that attempt to assess the relative importance of these two mechanisms in pre- and early transition populations report strong physiological effects and comparatively weaker replacement effects (Chowdhury, Khan and Chen 1976; Park et al. 1979; Park et al. 1998; Taylor, Newman and Kelly 1976). These results are consistent with the belief that in noncontracepting populations couples exercise little conscious control over the timing of births, and that fertility is primarily determined by factors that affect the supply of children (Chandran 1989; Mensch 1985). If the number of surviving children is lower than the desired number of children, then fertility follows a natural pace and the death of a child will have little effect on subsequent fertility (Jensen 1997:4). On the other hand, if fertility is governed by the demand for children, then the death of a child may produce a strong fertility response (Mensch 1985).

Separating replacement and physiological effects using data on the death of an index child is difficult to accomplish in populations where breastfeeding is common: many infant deaths are closely followed by the return of menstruation and thus an increased risk of conception will occur simply because of the termination of breastfeeding (Kuate Defo 1998). As an alternative to focusing on the death of an index child, the death of a nonindex child during a conception interval provides a measure of the replacement effect that is not confounded with the physiological effect of breastfeeding. Relatively few studies consider the impact of the death of a nonindex child on the timing of a subsequent conception, and the few studies that do, report mixed results. In a study of Pakistan and Bangladesh, Chowdhury et al. (1976) found no difference in the median lengths of birth intervals by the survival status of nonindex children. Lehrer (1984) in a study of Malaysia, also found no significant effect of the death of a nonindex child on subsequent birth spacing. However, in a sample of women from Cameroon, Kuate Defo (1998) found clear evidence of replacement effects on par with the physiological effects associated with the death of an index child. Jensen (1997) in an analysis of birth intervals from Kenya, Zimbabwe and Botswana also found evidence that the death of a nonindex child increased the risk of a birth in subsequent intervals.

In this paper we use hazard regression models with time-varying dummy variables to assess the impact of experienced infant and child mortality on the risk of subsequent conceptions in Ethiopia. The objectives of this article are (1) to determine whether the death of a nonindex child produces any short- and long-term behavioral response, (2) to determine whether this response varies by parity, and (3) to estimate the behavioral response to an index child's death net of the physiological effects associated with early termination of breastfeeding. Importantly, this paper tests for the presence of intentional child replacement in a high fertility population.

### ***Modeling the Fertility Response to Infant and Child Death***

Our approach to measuring the fertility response uses information on the timing of births and deaths, and the duration of postpartum amenorrhoea and breastfeeding available from retrospective fertility histories collected in many demographic surveys. We define three variables to describe the different mortality scenarios under which replacement fertility can occur. These three scenarios include the death of the index child, the death of a nonindex child during the current interval, and the death of a nonindex child before the start of the current interval. We define  $D_1$  as a time-varying dummy variable that equals one in the period following the month in which an index child dies. We also define a time-varying dummy variable that equals one in the months corresponding to the postpartum amenorrheic period; and a time-varying dummy variable that equals one if the index child was breastfed during the immediately preceding month and the current month is in the period after the return of menses. Studies indicate that prolonged breastfeeding, even after the return of menses, extends the time to next conception (Guz and Hobcraft 1991; Singh and Suchindran 1992). After controlling for postpartum amenorrhoea and prolonged breastfeeding,  $D_1$  provides an estimate of volitional replacement. Because the three variables are time-varying they can be used to describe the risks of conception at different periods of the conception interval, and for alternative timings of an index child's death.

We define  $D_2$  as a time-varying dummy variable that equals one during the months that follow the death of a nonindex child in the current interval.  $D_2$  provides a measure of short-term

replacement fertility that is not confounded with physiological effects. If couples intentionally try to replace a lost child with a new birth, we should expect the risk of a conception to rise during the period following the death of a nonindex child.

Finally, we define  $D_3$  as a time invariant dummy variable that equals one if a nonindex child had died before the start of the current interval. Efforts to replace a deceased child need not be restricted to the interval in which the child died. The effects of a child death on fertility can be long-term. Parents can attempt to replace a lost child and meet their fertility target by reducing the spacing of subsequent birth intervals, or by delaying stopping behavior and going on to have higher order births than they would have had if no children had died. These long-term effects assume that parents are able to effectively control the timing and occurrence of births either through the use of modern contraceptives or the use of more traditional methods such as abstinence or changes in coital frequency.

The variables  $D_1$ ,  $D_2$ ,  $D_3$ , and the time-varying measures of postpartum amenorrhoea and breastfeeding can be incorporated into a multistate hazard regression model introduced by Heckman and others (Flinn and Heckman 1982; Heckman and Singer 1984; Heckman and Walker 1991). The conditional hazard of a conception in the interval opened by the  $i$ th birth at  $t$  months after the birth is defined as:

$$h_i(t_i|X, \mathcal{Z}) = \exp[\beta_{i0} + X(t_i)\beta_i + (t_i^\delta - 1)/\delta + c_i\mathcal{Z}]$$

where  $\beta_{i0}$  is a constant term,  $X(t_i)$  is a vector containing the values of the independent variables at time  $t_i$ ,  $\beta_i$  is a vector of coefficients,  $(t_i^\delta - 1)/\delta$  is a term for modeling duration dependence, and  $c_i\mathcal{Z}$  is a correction term for woman-specific unobserved heterogeneity.<sup>1</sup> In its multistate (or parity-specific) form, the model simultaneously estimates separate equations for each conception interval and therefore allows the effects of the covariates to vary by parity (for examples of studies that recognize that replacement may vary by parity see Kuate Defo 1998; Lehrer 1984; Rutstein and Medica 1978).

A common assumption in continuous-time hazard models is that all the variables relevant to determining the hazard rate are included in the model. This assumption is frequently unrealistic because many characteristics of an individual or a process are unobserved. The presence of unobserved heterogeneity due to the omission of influential variables can lead to biases in the estimated form of the baseline hazard and in the effects of the covariates (see Trussell and Richards 1985; Yamaguchi 1991). In the multistate model the correction term for unobserved heterogeneity captures the fertility effects of woman-specific, unobserved characteristics, such as fecundity and desired family size, that are correlated across birth intervals.

Kuate Defo (1998) adapted Heckman's generalized hazard model to examine the impact of infant and child deaths on subsequent fertility in Cameroon. He considered only the death of an index child in the first year of life, and he distinguished nonindex children who died in the current interval by their birth order. The variable  $D_1$  in our model considers an index child's death beyond the first year of life, but  $D_2$  in our model does not distinguish nonindex children who die in the interval by birth order.<sup>2</sup> Our time-varying measure of lagged breastfeeding is similar to what Grummer-Strawn et al. (1998) used in their analysis of DHS data, with the important difference that we take advantage of information on the duration of postpartum amenorrhoea to distinguish between the postpartum amenorrheic period and breastfeeding after the return of menses. We expect the former to have a more powerful depressing effect on fertility than the latter.

### ***Data and Background***

The data for this paper come from the 1990 National Family and Fertility Survey (NFFS) conducted by the Central Statistical Authority of the Ethiopian government. The NFFS was the first large-scale demographic survey completed in Ethiopia since the 1981 Ethiopian

Demographic Survey. The NFFS was designed to be nationally representative, although it excluded northern regions of the country that were areas of military conflict at the time of the survey. The survey used a multistage, stratified sampling plan, with major strata based on regional and rural-urban divisions. The primary target of the survey was women aged 15-49. The survey collected data on women's background characteristics, marital status, birth history, health and breastfeeding practices for the last two births, knowledge and use of contraception, fertility preferences, work and migration experience, and husband's background characteristics.<sup>3</sup> Interviews were completed with 8,757 women. In this paper we use the birth histories of 3,405 currently married women, and information on the two most recent births for 3,924 currently married women.<sup>4</sup>

Data quality is a concern in any study that uses retrospective information on the occurrence and timing of events. The omission of deceased children and the misreporting of surviving children's ages is particularly common among illiterate women. In a previous analysis of the retrospective birth histories available in the NFFS, Lindstrom and Berhanu (1999) found no evidence of age heaping in numbers ending in 0 or 5. Based on a careful analysis of infant mortality rates they also found no evidence of gross underreporting of deaths in the NFFS survey. In the current paper we restrict our analysis to births that occurred between 1976 and 1989 to reduce the amount of recall error in our sample data. The month of birth was recorded for approximately two-thirds of the births in our sample. In 30 percent of the births the season was recorded but not the month, and in slightly less than 3 percent of the births only the year was recorded. In the cases where the month was missing, we randomly assigned a birth month within the given season or year. We assigned individual months with equal probability based on the distribution of recorded birth months, which was approximately uniform.

The reported durations of breastfeeding and postpartum amenorrhoea in the NFFS both show clear patterns of heaping at 12, 18, 24 and 36 months (Berhanu and Hogan 1998). Some heaping of breastfeeding durations is genuine and reflects socially prescribed weaning times, however, because women exert considerably less control over the resumption of menses, heaping

for these durations likely reflects recall or response errors (Berhanu and Hogan 1998). In a hazards analysis of postpartum amenorrhoea durations, Berhanu and Hogan (1998) found that heaping at multiples of six was associated with longer than expected durations, suggesting that women tend to round-up their reported durations of postpartum amenorrhoea.<sup>5</sup> A tendency to round-up these durations will result in underestimates of both the negative effect of postpartum amenorrhoea and the positive effect of the death of an index child on the risk of a conception in our hazard model.<sup>6</sup>

Ethiopia provides an opportune context for examining the relationship between infant and child mortality and subsequent fertility. Total fertility in 1990 was estimated at 6.4 children per woman and the infant mortality rate during roughly the same period was estimated at 106 deaths per 1,000 births (CSA 1993:126,286). These figures place Ethiopia near the top of sub-Saharan countries in terms of both high fertility and high infant mortality. According to conventional explanations of the physiological and behavioral linkages between infant mortality and subsequent fertility, replacement effects should be weak or absent in Ethiopia.

### ***Data Analysis***

Our analysis is divided into two parts. First we estimate a multistate hazard model for conception intervals opened by the first through the sixth birth using the complete birth histories collected by the survey. We restrict our sample to women who were currently married and in their first union as of the time of the survey. Marriage is almost universal in Ethiopia with 80-85 percent of women age 20-44 currently married. Because women marry at relatively young ages most births occur within marriage. In urban areas of Ethiopia, where a larger proportion of women are not in unions, nonmarital fertility is exceptionally low and has actually declined (Lindstrom and Woubalem 2003; Sibanda et al. 2003). We excluded from our sample women who had been in more than one union or who were no longer married at the time of the survey because information on the end dates of unions was not collected by the survey.

We used information on the monthly timing of births and infant and child deaths to construct an event history file for each conception interval after the first birth. Each record in the file represents one month of exposure to the risk of conception. Intervals that were still open as of the time of the survey were right censored at 72 months. In the event history file intervals pertaining to the same woman are adjacent to one another and in ascending parity: this file structure permits the estimation of woman-specific unobserved heterogeneity that is correlated across intervals. We identified the month of conception by moving nine months back from the month of birth.

Information on the duration of postpartum amenorrhoea and breastfeeding was collected by the survey only for the last two births. Our multistate hazard model of conception therefore included the three dummy variables for child death,  $D_1$ ,  $D_2$  and  $D_3$ , without any control variables for postpartum infecundity. The variable  $D_1$ , death of the index child, thus measures both physiological and potential replacement effects. In order to estimate the replacement effect of an index child's death net of physiological effects, we estimate a second hazard regression model for the last two conception intervals that is not parity-specific.

Table 1 presents Kaplan-Meier estimates of the median length of conception intervals by the survival status of the index child. The death of an index child in the first year of life is associated with a reduction in the median of between 6 and 13 months, which in relative terms represents a reduction of between 29 and 48 percent of the interval length. The shorter time to next conception that is associated with an index child's death in the first year of life is consistent with the early return of fecundity that occurs when breastfeeding is truncated, in addition to couple efforts to speed-up the time to a next birth. The death of an index child after the first year of life, is also associated with a shorter median time to next conception (from 2 to 10 months shorter), but the reduction is not as great as when the death occurs in the first year. This result is consistent with the expectation that physiological factors play less a role in the timing of a next conception the older the index child is at the time of death. The other feature of Table 1 worth pointing out, is that as parity increases, the median length of intervals in which the index child

dies decreases, while the median length of intervals in which the index child survives increases. Because we would expect the physiological impact of an index child's death to be the same at all parities, this pattern of shorter times to conception as parity increases suggests the presence of replacement behavior (i.e., as parity increases and a woman is nearer her fertility target and/or the end of her reproductive span, the desire or urgency to replace a deceased child increases). Women with infant deaths after the first birth (and hence higher infant mortality), may also be selected for higher fertility, which could produce the association between shorter birth intervals and infant deaths shown in Table 1. The hazards regression models that we estimate in the following section control for selectivity on observed characteristics, as well as unobserved characteristics that may be jointly associated with differential risks of conception and infant mortality.

[Table 1 about here]

Table 2 describes the variables we include in our hazard models of conception. In addition to the child death variables ( $D_1$ ,  $D_2$  and  $D_3$ ), and the control variables for postpartum amenorrhoea and breastfeeding, we include a number of socio-economic and demographic control variables. For the mother's characteristics, we include education, place of current residence, whether the mother currently has more boys than girls, and mother's age at the start of the interval and age squared. Because of the strong overlap in Ethiopia of region and ethnicity, we did not include ethnicity in our models. We also found no significant effects of religion and dropped this variable as well. Because husband's education is highly correlated with wife's education, we excluded it from our models with no loss of information.

[Table 2 about here]

We estimated the hazard models presented in this article using alternative parametric specifications of duration dependence, and both with and without unobserved heterogeneity.<sup>7</sup> The principal advantage of this approach is that we can check the robustness of the results against different assumptions concerning the parametric form of the baseline hazard and the presence of unobserved heterogeneity. We selected the best fitting model based on the Bayesian

information criterion (BIC) specified by Yamaguchi (1992:286). Heckman and Walker (1987) demonstrate the value of this test statistic for comparing non-nested models. In both the parity-specific model and the model of the last two intervals, the Weibull specification of duration dependence and the inclusion of unobserved heterogeneity provided the best fit.

### *Analysis of Birth Histories*

Table 3 presents the results for the parity-specific hazard model of conception. We present the estimated effects of the child death variables and the control variables as hazard ratios ( $e^{\beta}$ ). In all of the conception intervals, with the exception of the interval following a sixth birth, the death of the index child significantly raises the risk of a conception in a given month by a factor of between 1.7 and 2.3. The effect appears to be bigger at higher parity intervals suggesting that parents may make a more concerted effort to replace a lost index child as they move closer to their desired family size (see Lehrer 1984; Mensch 1985).<sup>8</sup> The magnitude of the  $D_I$  effects in the intervals following the first, second and third birth are very close to those estimated by Kuate Defo (1998) for Cameroon using a similar modeling approach. At higher parities the  $D_I$  effects are slightly larger in Ethiopia compared to Cameroon, which is consistent with the higher fertility in Ethiopia. The larger effects of an index child's death at high parities is also consistent with the parity-specific differences in the median interval lengths shown in Table 1.

We must be cautious in the interpretation of  $D_I$ . Because the model does not control for postpartum amenorrhoea and breastfeeding,  $D_I$  confounds the effects of early termination of breastfeeding with intentional replacement. Nevertheless, the greater impact of the death of an index child on the risk of a conception at higher parities suggests the presence of replacement behavior, at the very least, in these intervals. In contrast to the death of an index child, neither the death of a nonindex child during the current interval, nor the death of a nonindex child before the start of the interval has a significant effect on the risk of a conception. There is no evidence to suggest that parents attempt to replace a lower order child during the interval in which the child

dies, nor in subsequent intervals. Also, there is no evidence to suggest that parents who have experienced a child death are more likely to move onto higher parities..

[Table 3 about here]

The duration dependence term ( $\gamma$ ) is less than one indicating a declining baseline hazard over time. The heterogeneity term was estimated with two points of support indicating the presence of two latent groups of women with very different underlying risks of conception. For approximately 15 percent ( $1 - p$ ) of the sample the monthly risk of a conception in the intervals following the first, second and third births is between one-fifth (0.197) and one-tenth (0.104) that of the rest of the women in the sample. Beyond the fourth birth the likelihood of a conception for this latent group of women is essentially zero. The heterogeneity terms in the model are picking up a component of subfecundity and sterility, or intentional spacing and stopping in the population, that is not associated with any of the covariates included in the model.

To summarize the findings from the parity-specific analysis of conception intervals, there is clear evidence in Ethiopia that the death of an index child results in a higher hazard of a next conception and a shorter conception interval. The impact of an index child's death on the time to a next conception is greatest at high parities when most women in Ethiopia are approaching their desired number of children or the end of the reproductive age span. There is no evidence, however, that the timing of a next conception is affected by the death of a nonindex child in the current interval or in a prior interval. To the extent that there is a fertility response to the death of a child in Ethiopia, it appears to only occur in the case of the most recent born, and perhaps is limited to higher order children.

### ***Analysis of Last Two Conception Intervals***

In the next model we introduce time-varying control variables for postpartum amenorrhoea and prolonged breastfeeding to estimate the fertility response to an index child's death net of physiological factors that affect fecundity. We include in the model control variables for parity. This analysis uses information from the last two births. The sample includes the same

women from the parity-specific analysis, plus women who were excluded from that analysis because they were missing information on earlier births. We first estimate a model without controls for postpartum amenorrhoea and breastfeeding to derive an estimate of the total impact of an index child's death ( $D_1$ ) on the monthly risk of conception. Next, we estimate a model that includes the two physiological control variables. If the increased risk of conception following an index child's death is due to the shorter length of postpartum amenorrhoea and breastfeeding, then once we control for these two factors the death of an index child should have no significant effect on the hazard of a conception. The estimate of  $D_1$  in this model represents the mean effect of replacement across all parities. Because the effect of  $D_1$  in the multistate model varied across parities, the estimate of  $D_1$  in the non-parity-specific model provides an overestimate of replacement at low parities and an underestimate of replacement at higher parities. We detected no significant effects of the death of a nonindex child in the parity-specific model, and therefore exclude  $D_2$  and  $D_3$  from the analysis of the last two conceptions.

Table 4 presents the results for the analysis of the two most recent conception intervals. In the model that excludes the control variables for postpartum amenorrhoea and breastfeeding the death of the index child increases the monthly risk of a conception by a factor of 1.8. This result is consistent with the findings from the parity-specific model of conception. After we introduce the control variables for postpartum amenorrhoea and breastfeeding, the hazard ratio for the death of an index child declines to 1.46, but remains highly significant. As expected an important component of the effect of  $D_1$  is due to the premature termination of breastfeeding and the return of menses that is associated with an index child's death in the first 1-2 years of life. During the postpartum amenorrhoeic period, women are at one-fifth (0.196) the risk of a conception. Even after the return of menses, women who are breastfeeding continue to be at a lower risk of conception (around 30 percent lower) compared to women who are not breastfeeding. However, once we take into account a woman's fecundity, we find that women who lost an index child are almost 50 percent more likely to conceive in the months following the child's death than women with a surviving index child. This finding represents a significant

fertility response to the death of an index child, and it is particularly note worthy given that it is found in a high fertility and high mortality population.

The magnitude of the replacement effect that we find in Ethiopia is very close to estimates of the replacement effect reported by other studies that have used hazard regression models to estimate the impact of an index child's death on the hazard of a subsequent conception or birth net of physiological factors. Table 5 presents the countries analyzed, the models used, and the estimated hazard ratios from four studies, and the current study. Using data from Malaysia and a Cox regression, Lehrer (1984) estimated a hazard ratio of 1.46 for the death of an index child in the fifth birth interval. Park et al. (1998) also using a Cox regression with data from Bangladesh estimated a hazard ratio of 1.53 for the death of an index child during the fecundable period, irrespective of parity. Both Lehrer and Park et al. included controls for breastfeeding in their models. Grummer-Strawn et al. (1998) estimated piecewise exponential hazard regressions for 44 countries using DHS data, and with a control for breastfeeding status. The hazard ratio associated with the death of an index child varied substantially across countries, from a low of 0.88 to a high of 2.81. The mean values of the hazard ratios for the three regions, Africa, Asia, and Latin America are 1.35, 1.46, and 1.44 respectively. Palloni and Fafalimanana (1999) also used a piecewise exponential hazard regression with DHS data from seven Latin American countries. They estimated separate effects for the death of an index child depending on whether or not the death truncated breastfeeding. When the death did not truncated breastfeeding, the associated hazard ratio of a conception ranged from 1.29 to 2.29 among the seven countries, with a mean of 1.75. The estimated hazard ratio of 1.46 that we find for Ethiopia is within the range of values reported for other countries, and in particular is in the middle of the range of values that Grummer-Strawn et al. (1998) report for a wide sample of African countries.

### ***Discussion***

The hazard regression model used in this paper provides a method for identifying the short and long-term responses to experienced infant and child mortality by incorporating the

current and prior survival status of lower birth order children in addition to the survival status of the index child. In using time-varying covariates the model accurately captures changes in a woman's fecundity and the timing of a child death, which in turn produce more precise estimates of the fertility response than those provided by static models. Using retrospective birth history data from a national survey in Ethiopia, we find a significantly higher risk of a conception in the months following the death of an index child, even after controlling for postpartum amenorrhoea and breastfeeding status. We, however, find no evidence of a fertility response to the death of a nonindex child. We attribute the higher risk of a conception following an index child's death in part to the intentional efforts of couples to reduce the waiting time to a next birth and thereby replace the lost index child. Our finding of replacement behavior is consistent with findings from several other studies that used hazard regression models with data from middle to high fertility countries.

How then do couples accelerate the timing of a conception? We identify three possible mechanisms: discontinuance of contraceptive use, early termination of postpartum abstinence, and increased coital frequency. We eliminate discontinuance of contraceptive use from consideration given the very low levels of current contraceptive use among Ethiopian women: 4.8 percent of currently married women aged 15-49 were currently using a contraceptive method in 1990 (CSA 1993:191). We also eliminate early termination of postpartum abstinence as a potential factor. In Ethiopia the traditional period of postpartum abstinence is relatively short, around 40 days. Cutting short the period of postpartum abstinence will have little or no effect on the timing of a next conception. We find increased coital frequency to be the most plausible method by which couples shorten the time to a next birth. Lindstrom and Berhanu (1999) in an analysis of the NFFS data also found strong evidence that couples in Ethiopia exercise significant control over the timing of conceptions even in the absence of widespread contraceptive use.

Why are there no signs of intentional replacement behavior in response to the death of a nonindex child? We offer two possible explanations. First, because births are already closely

spaced in Ethiopia and breastfeeding is near universal and prolonged, shortening the time to a next birth in the event of a nonindex child's death would require the early termination of breastfeeding. The median duration of breastfeeding in Ethiopia is around 19 months, and over a quarter of infants are breastfed for more than 24 months (Hasen et al. 1994; CSA 1993). The median length of conception intervals in Ethiopia range between 21 and 24 months. In the Ethiopian context of widespread food insecurity, breast milk is an essential component of an infant's nutritional in-take: premature weaning would raise the risk of an infant's death (Lindstrom and Berhanu 2000). A fertility response to a nonindex child's death in this context would require that women are cognizant of the contraceptive value of breastfeeding, and willing to accept a higher risk of death for their most recent born in order to accelerate the timing of a next birth. A second plausible explanation for the absence of a significant fertility response to the death of a nonindex child is that replacement fertility has a strong emotional component to it, in which a new birth serves more to compensate for the emotional loss associated with an infant's death than to meet a targeted family size. This explanation fits the cultural practice in Ethiopia of delaying the naming of infants until the child is at least several months of age because of the high risk of infant mortality. As infants age into children with well defined personalities and increased parental bonds, they become less replaceable in an emotional sense.

Coital frequency may also go up after the death of an index child if breastfeeding normally leads to reduced sexual relations between partners, because breastfed infants sleep with their mothers and/or because interest in sexual relations is lower among partners during the breastfeeding period (Brass and Barrett 1978). Both the emotional desire to replace a deceased infant, and a change in sleeping arrangements and sexual attraction following the termination of breastfeeding, may contribute to a rise in the risk of conception following an infant's death. Nevertheless, these effects should be similar across parities, and if anything one might expect them to be stronger at low parities when couples are typically more sexually active. However, the substantially larger effect of an index child's death on the risk of a subsequent conception at parities near desired family size suggests that some component of the response to the death of an

index child is intentional. At the same time, the absence of any evidence of a fertility response to the death of nonindex children suggests that in Ethiopia the effects of replacement fertility on the pace and timing of women's overall reproductive careers are fairly limited.

A shortcoming of this study is that we are not able to test for insurance effects on fertility. In the context of high and variable infant and child mortality rates, having more children than desired is a strategy used by parents to guarantee a minimum number of surviving children. We expect actual experienced mortality to produce a larger fertility response than anticipated mortality. The fact that we do not detect a significant fertility response to the deaths of nonindex children leads us to believe that the desire to insure against future child mortality is not an important component of fertility behavior in Ethiopia. However, fertility goals may still take into account anticipated mortality risks for children in adulthood in an effort to insure for future old age support.

Our results highlight the importance of near universal and prolonged breastfeeding as a proximate determinant of fertility in Ethiopia - any change in breastfeeding patterns that reduces protection against conception, and that is not accompanied by a commensurate increase in contraceptive use, has the potential to significantly raise fertility.

### *Notes*

<sup>1</sup> The models with heterogeneity estimated in this paper use a nonparametric correction term introduced by Heckman and Singer (1984). The advantage of the nonparametric specification is that frequently one has no prior knowledge about the expected parametric form of the unobserved variables. Studies also have shown that model results can be sensitive to the parametric form of heterogeneity chosen (Heckman and Singer 1982; Trussell and Richards 1985:247).

The nonparametric approach developed by Heckman and Singer uses a discrete probability distribution with a step function to describe the mixing distribution of the unobserved effects. The distribution works with a finite number  $k$  of support points at locations  $z_j$ , and is confined to the interval  $[0,1]$  by setting  $z_1 = 0$  and  $z_k = 1$ . The  $k$  support points correspond to latent groups in the population. Associated with each support point is a probability mass  $p_j$  that can be interpreted as the proportion of the population found in a particular latent group.  $\mathbf{z}$  is a  $1 \times k$  vector of location parameters, and  $c_i$  is a factor loading. Each  $c_i z_j$  defines a contrast to the

constant term that indicates the shift in the baseline hazard associated with a particular latent group. In the model without heterogeneity, one support point is assumed ( $z = 0$ ), with all the probability mass located on the constant term. When two points of support are specified ( $z_1 = 0$  and  $z_2 = 1$ ), the parameters  $p$  and  $c_i$  are estimated. When three points of support are specified ( $z_1 = 0$  and  $z_3 = 1$ ),  $z_2$ ,  $p_1$ ,  $p_2$ , and  $c_i$  are estimated (see Heckman and Walker 1987; Trussell and Richards 1985). In general,  $(k-2) + (k-1) + 1$  parameters are estimated for  $k$  points of support.

To estimate the hazard of conception, we used the computer program CTM (for continuous time models). Although CTM permits time-varying regressors, the correction for unobserved heterogeneity is appropriate only for heterogeneity attributable to constant unobserved effects

<sup>2</sup>We estimated multistate models that distinguished nonindex children deaths by the deceased child's birth order, but found no significant effects.

<sup>3</sup>Current and ever use of contraception was asked for married women. Retrospective information on contraceptive use was not collected.

<sup>4</sup>Not all women who reported complete information on the last two births, reported complete birth histories, hence more women were included in the analysis of the last two births than in the analysis of the birth histories.

<sup>5</sup>In hazard regression models of the resumption of postpartum menses, Berhanu and Hogan (1998) included a dummy variable set equal to one if the duration ended in a multiple of six. The estimated coefficient for heaping was negative and significant, indicating a lower than expected hazard, controlling for other factors, and thus a longer than expected duration.

<sup>6</sup>Rounding-up durations of postpartum amenorrhoea increases the likelihood that conceptions which occur after the resumption of menses are incorrectly assigned to the postpartum amenorrheic period and not assigned to the period following the resumption of menses. The net effect of this misreporting is to underestimate both the negative effect of postpartum amenorrhoea on the hazard of conception (because conceptions that did not occur during this period are treated as if they did) and the positive effect of death of an index child on the hazard of conception, net of postpartum amenorrhoea (because conceptions that occurred after the resumption of menses are treated as if they did not occur during this period).

<sup>7</sup>From the generalized form of the conditional hazard, the Weibull, Gompertz, and exponential regression models can be specified by changing the form of the duration dependence term.

<sup>8</sup>Among currently married fecund women interviewed in the 1990 NFFS the mean additional number of children wanted by women with zero living children was 4.54, for women with one living child the mean was 3.70, for women with two living children the mean was 2.76, for women with three living children the mean was 2.28, for women with four living children the mean was 1.64, and for women with five living children the mean additional number of children wanted was 1.17 (CSA 1993:247).

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Table 1. Kaplan-Meier Estimates of the Median Length (Months) of Conception Intervals with and without an Index Child's Death, Ethiopia.

	Conception interval following					
	First birth	Second birth	Third birth	Fourth birth	Fifth birth	Sixth birth
Index child:						
Survives to next conception	22	21	22	24	25	24
Dies in first year	14	15	14	14	12	11
Dies after first year	20	19	16	16	15	-- <sup>a</sup>
All intervals	18	20	22	23	25	24

Note: <sup>a</sup>Not enough cases were available to produce a reliable estimate of the median.  
Source: National Family and Fertility Survey, 1990

Table 2. Variable Definitions for Hazard Model of Conception, Ethiopia.

Variable Label	Description
<b><i>Outcome</i></b>	Conception in the current month
<b><i>Replacement effects</i></b>	
<i>D</i> <sub>1</sub> Death of index child	Index child died before the current month (time-varying).
<i>D</i> <sub>2</sub> Death of nonindex child	A lower parity child died during the current conception interval and before the current month (time-varying).
<i>D</i> <sub>3</sub> Death of a child in a prior interval	A lower parity child died before start of current conception interval (time-invariant).
<b><i>Physiological effects (Time-varying)</i></b>	
Postpartum amenorrhoea	Current month is before the return of menses.
Breastfeeding and not PPA	Current month is after the return of menses, and still breast-feeding in the month prior to the current month.
<b><i>Socio-economic and demographic controls (Time-invariant)</i></b>	
Mother's characteristics	
No school	0 years of school (reference category)
Some primary	1-6 years of school
Some secondary	7 or more years of school
Addis Ababa	Living in Addis Ababa at start of interval (reference category)
Other urban area	Living in other urban area at start of interval
Rural central highlands	Living in rural central highlands at start of interval
Rural southwestern highlands	Living in rural southwestern highlands at start of interval
Rural southern highlands	Living in rural southern highlands at start of interval
Rural east and southeast high.	Living in rural east and southeastern highlands at start of interval
Rural northern highlands	Living in rural northern highlands at start of interval
Rural lowlands	Living in rural lowlands at start of interval
More surviving boys than girls	Mother has more surviving boys than girls at the start of the inter
Age	Age at start of interval
1st birth	Index child is first birth
2nd - 4th birth	Index child is second to fourth birth
5th or higher birth	Index child is fifth or higher birth (reference category)

Table 3. Hazard Ratios and Parameter Estimates for Parity-specific Hazard of Conception, Conception Intervals following First-Sixth Birth, Currently Married Women, Ethiopia.

	Transition to conception following					
	First birth	Second birth	Third birth	Fourth birth	Fifth birth	Sixth birth
<b>Replacement effects</b>						
$D_1$ Death of index child	1.661*	1.714*	1.562*	2.299*	1.917*	1.252
$D_2$ Death of nonindex child	--	.949	.945	1.161	1.788	1.046
$D_3$ Death of a child in a prior interval		1.027	.969	.987	.908	.996
<b>Socio-economic and demographic controls</b>						
Mother's characteristics						
No school (reference)	--	--	--	--	--	--
Some primary	1.100	1.111	1.075	.910	.785	.740
Some secondary	1.201*	.706*	.393*	.600*	.626*	.529
Addis Ababa (reference)						
Other urban area	1.045	1.080	1.039	1.156	1.210	2.010*
Rural central highlands	1.120	1.163	1.170	1.253*	1.326*	.865
Rural southwestern highlands	.988	.996	1.106	1.214	1.112	1.127
Rural southern highlands	.956	1.168	1.158	1.431*	1.258	1.431*
Rural east and southeast high.	1.456*	1.410*	1.487*	1.356*	1.522*	1.708*
Rural northern highlands	.873	1.018	.971	.981	1.198	.432
Rural lowlands	1.063	1.227*	1.254*	.875	1.085	.926
More surviving boys than girls	1.029	.934	1.084	.932	.902	.861
Age	1.000	1.210*	1.214*	.726*	1.663*	1.057
Age <sup>2</sup>	.836*	.786*	.754*	1.302*	1.150	1.311
<b>Heterogeneity parameters</b>						
$c$	.197*	.104*	.134*	.001	.000	.000
$p$	(.844*)					
<b>Duration dependence parameter</b>						
$\gamma$	(.516*)	(.652*)	(.475*)	(.496*)	(.444*)	(.386*)
Constant	(-4.694*)	(-4.882*)	(-4.584*)	(-4.801)	(-4.748)	(-4.575)
Negative log likelihood	35,609					
Number of women	3,405					
Number of intervals	11,700					

Note: (untransformed coefficients in parentheses) \*  $P < .05$ .

Source: National Family and Fertility Survey, 1990

Table 4. Hazard Ratios and Parameter Estimates for Hazard of Conception, Conception Intervals Following Two most Recent Births, Currently Married Women, Ethiopia.

	Without physiological effects	With physiological effects
<b>Replacement effects</b>		
<i>D</i> <sub>1</sub> Death of index child	1.804*	1.462*
<b>Physiological effects</b>		
Postpartum amenorrhoea	--	.196*
Breastfeeding and not PPA	--	.675*
<b>Socio-economic and demographic controls</b>		
Mother's characteristics		
No school (reference)	--	--
Some primary	1.178*	1.051
Some secondary	1.139	.910
Addis Ababa (reference)		
Other urban area	1.151	1.116
Rural central highlands	1.247*	1.287*
Rural southwestern highlands	.863	.999
Rural southern highlands	1.137	1.157
Rural east and southeast high.	1.181	1.096
Rural northern highlands	1.027	1.260*
Rural lowlands	.986	1.065
More surviving boys than girls		
Age	.823*	.842*
Age <sup>2</sup>	.834*	.868*
1st birth	1.154	1.023*
2nd - 4th birth	1.290*	1.269*
5th or higher birth (reference)	--	--
<b>Heterogeneity parameters</b>		
<i>c</i>	.106*	.138*
<i>p</i>	(.843*)	(.851*)
<b>Duration Dependence</b>		
(	(1.100*)	(.661*)
Constant	(-5.681*)	(-5.643)
Negative log likelihood	8,497	8,326
Number of women	3,924	
Number of intervals	5,761	

Note: (untransformed coefficients in parentheses) \*  $P < .05$ .

Source: National Family and Fertility Survey, 1990

Table 5. Estimates of the Volitional Replacement Effect of an Index Child's Death on the Hazard of a Conception or Live Birth.

Study	Country (Year)	Volitional replacement effect /Model – dependent variable /Physiological control variables	Estimated hazards ratio
Lehrer (1984)	Malaysia (1976-1977)	Index child died in the birth interval /Cox regression – hazard of a live birth, fifth birth interval /Number of months index child breastfed	1.46
Park, et al. (1998)	Bangladesh (1991)	Index child died during the fecundable period /Cox regression – hazard of a live birth /Breastfeeding status of the index child during the fecundable period	1.53
Grummer-Strawn et al. (1998)	25 African countries (1986-1992) 5 Asian countries (1987-1991) 14 Latin Am. countries (1986-1992)	Index child died before the next conception (time-varying) /Piecewise exponential – hazard of a live birth /Lag (10 months) breastfeeding (time-varying)	range (0.88-1.83) mean 1.35 range (0.85-2.55) mean 1.46 range (0.98-2.81) mean 1.44
Palloni & Rafalimanana (1999)	7 Latin Am. countries (1986-1990)	Index child died and not breastfeeding <sup>a</sup> /Piecewise exponential - hazard of a live birth /Index child died and still breastfeeding	range (1.26-2.29) mean 1.75
Lindstrom & Kiros	Ethiopia (1990)	Index child died before next conception (time-varying) /Weibull – hazard of a conception leading to a live birth /Postpartum amenorrhoea (time-varying) /Lag (1 month) breastfeeding and not PPA (time-varying)	1.46

Note: <sup>a</sup>The estimate provides an upper bound for replacement effects (Palloni and Rafalimanana 1999:53).