The Effects of Breast-feeding and the Pace of Childbearing on Early Childhood Mortality in Mexico[1]

ALBERTO PALLONI,[2] GUIDO PINTO AGUIRRE,[2] & SANTIAGO LASTIRI[3]

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Using data from Mexico's Demographic and Health Survey, the authors examine the effects of breast-feeding and the pace of childbearing on early childhood mortality in a sample of 2 665 children born between 1982 and 1986. From a family planning perspective, they seek to assess the impact that changes in childbearing patterns and associated changes in breast-feeding patterns may have on infant and childhood mortality. This is done by integrating breast-feeding models with variables influencing the pace of childbearing.

The analysis indicates that the effects of breast-feeding on infant mortality were strong, consistently negative, and statistically significant until at least the sixth month of life, after which they were considerably diluted. Among the other variables analyzed, the effects of a following conception on mortality were found to be very strong among infants 3–5 months old; and maternal age at delivery appeared to have some importance among infants 1–2 months old and among children in the second year of life.

To examine likely interactions between family planning, breast-feeding, and mortality, an integrated model was applied that simultaneously considered these and a range of other variables. The results suggest that the effects of improved birth spacing and maternal age at delivery associated with family planning are powerful enough to cause 20–40% reductions in infant mortality. However, the effects of reduced breast-feeding associated with family planning are great enough to offset more than 60% of these benefits. Limitations of the model make it necessary to emphasize that all of these conclusions need to be accepted with caution.

This article examines the results of our analyses of early childhood mortality in Mexico using data collected by Mexico's Demographic and Health Survey (DHS) during the period February–August 1987. The presentation has two aims: first, to assess the effects of breast-feeding and the pace of childbearing on early childhood mortality; and second, to evaluate the impact that changes in the pace of childbearing and associated changes in breast-feeding may have on infant and childhood mortality. This latter evaluation involves assessment of the potential survival benefits likely to accrue from policies aimed at reducing fertility.

Such an evaluation requires integration of breast-feeding models with variables influencing the pace of childbearing. This integration is necessary if one is to resolve in a satisfactory manner a debate that has not yet been conclusively closed and that concerns the role of family planning in improving the survival status of infants and young children. Over the past five years or so this debate has pitted those who believe in the influential effects of family planning on childhood survival against those who hold that fam-

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[2]Center for Demography and Ecology, University of Wisconsin, Madison, Wisconsin, United States of America.

[3]Mexican Health Foundation (Fundación Mexicana para la Salud), Mexico City, Mexico.

ily planning has only marginal benefits or none at all in terms of reducing early childhood mortality (*1–6*).[4]

For the most part, this debate has been conducted in the absence of well-defined models and without the support provided by consistent estimates. Since breast-feeding and the pace of childbearing are mortality determinants that not only tightly (and inversely) relate to each other but also interact, producing offsetting effects, disputes based on conjectures and not on well-defined models are pointless. In this article we make use of a simplified model and implement estimation procedures that enable us to obtain consistent estimates of effects. We then integrate these estimates to generate gross and net effects of the pace of childbearing on child survival.

Although we do introduce other variables (such as the mother's education and area of residence) throughout our analyses, we focus on their effects only to the extent that they serve as efficient controls for removing the impact of confounding factors from the estimates of interest to us.

We apply conventional binary choice and hazard models to estimate the effects of various mortality determinants (indicators of the pace of childbearing and breast-feeding). Since the data are affected by reporting errors and selectivity problems, we also implement procedures designed to minimize such problems' impact on our estimates.

The article is divided into four sections, this introduction being the first. The second section provides a brief description of the data; the third section sets forth the models to be estimated and the analysis of early child mortality; and the fourth section presents an assessment of the gross and net effects of the pace of childbearing on infant and childhood mortality.

DESCRIPTION OF THE DATA

The Mexican DHS consists of some 10 310 retrospective interviews with women 15–49 years of age. In our study we utilized reports on the birth histories of 2 665 children born between 1982 and 1986 (all of them with information on breast-feeding) and a total of 6 705 children born between 1975 and 1986 (the oldest cohorts of which did not provide information on breast-feeding). The most important characteristics of the sample of children that we use in the main part of our presentation—a sample born between 1 and 5 years before the survey—have been described elsewhere (*7*). Those characteristics can be briefly summarized as follows:

- The profile for the entire sample with respect to the conditional probabilities of dying within the first 5 years of life was as expected, with the conditional probability of dying during the first month of life exceeding all others.
- Mortality during the first year of life was slightly higher among those born prior to the fifth year before the survey, as should be expected in a context of generally declining mortality.
- Sharp mortality differentials were associated with the mother's education and area of residence. In all cases the conditional probabilities of dying were higher among the less educated and among those living in rural areas. The proportionate educational differences were more salient within the age segments 1–3 months, 12–24 months, and 24–60 months, whereas those associated with area of residence were larger within the age seg-

[4]See also Hobcraft JH. "Does family planning save children's lives?" Technical paper presented at the International Conference on Better Health for Women and Children Through Family Planning held at Nairobi, Kenya, on 5–9 October 1987.

ments 0 months, 6–12 months, and 12–24 months.

- Important differentials were also associated with the mother's age at the birth and birth order. Children born to very young or to older mothers experienced higher mortality, and children with relatively high birth orders were exposed to much higher mortality than children with low birth orders.
- By and large, children whose preceding birth interval was short (less than 18 months) had higher mortality at all ages than those with a longer preceding birth interval. These differentials applied to all birth orders except the first child. We will show later that these differentials contract when we control for other variables, and that they are dwarfed by two conditions that change over the life of a child, namely, the duration of breast-feeding and the occurrence of a subsequent conception.

DETERMINANTS OF EARLY CHILDHOOD MORTALITY

A Theoretical Framework

Although it has long been suspected that short birth intervals and lack of adequate lactation could have detrimental effects on the health and survival of infants and young children (*8–15*),[5] it is only recently that we have been able to marshall evidence on a massive scale to support this conjecture. By far the largest contributor of cross-national information illustrating the effects of both length of birth intervals and breast-feeding on infant mortality is the World Fertility Survey (WFS). In studies of single countries (*16–18*) or in studies including many countries in multiple continents (*19–21*), the evidence unequivocally points to uniformly strong effects, particularly on mortality before the sixth month of life.

The findings in these studies have been replicated by others carried out independently of the WFS program in disparate areas of the world (*22, 23*).[6] All tests carried out so far suggest that despite some skepticism about the validity of the findings (*3, 18, 24–26*), they appear to be remarkably robust to errors in reporting and model misspecification (*27, 28*). Furthermore, the evidence generated so far by the Demographic and Health Surveys (DHS) provides additional support to the idea that the estimated effects of breast-feeding and length of birth intervals are quite tangible and real.

Although past and ongoing research have been successful in approximating the direction and order of magnitude of the effects, we have been less successful in identifying the exact mechanisms that generate these effects. Elsewhere (*7*) we have described in great detail the hypothetical mechanisms that mediate between breast-feeding and early childhood mortality on the one hand, and between the length of birth intervals and early childhood mortality on the other. What follows is a very tight summary of the main relations. For the sake of brevity we omit altogether the discussion of traps and fallacies that are common when one is trying to infer the effects of both breast-feeding and pace of childbearing on early childhood mortality.

[5]See also Millman SR. *Breast-feeding in Taiwan: a study of change.* University of Michigan; 1982. (Doctoral dissertation).

[6]See also Pebley A, Davanzo J; *Maternal depletion and child survival in Guatemala and Malaysia;* (paper presented at the Population Association of America meetings held at New Orleans in April 1988) and Muhuri PK, Menken J; *Mortality of children as affected by the gestation and survival of their immediate younger siblings in Matlab, Bangladesh;* (1992, unpublished manuscript).

The Effects of Breast-feeding on Early Childhood Mortality

Clinical and epidemiologic studies have shown mother's milk has at least three properties that help to protect the health of infants (*12, 29*). First, breast milk appears to meet the nutritional requirements for the normal growth of an infant for at least 6 months (*8*). Consumed in sufficient quantities, it provides protection against malnutrition syndromes such as kwashiorkor and marasmus (*9*). The absence of breast-feeding is related to excess incidence of certain conditions, such as diarrhea and gastrointestinal infections, that are exacerbated by malnutrition (*34, 35*). Second, breast milk contains proteins that enhance immunocompetence and serve to forestall infections of the intestinal tract. And third, breast milk is a sterile fluid containing substances that prevent the growth of bacteria, making it an extremely hygienic product.

Some of the benefits accruing from these properties, particularly the first two, gradually diminish as the infant's nutritional requirements increase. Researchers have argued that the relative advantage in terms of immunocompetence and nutritional status of fully breast-fed children decreases rapidly after the sixth month of life (*9, 17, 36*). Also, the importance of each of these properties for the health of the child depends on conditions that raise or lower the child's exposure to deleterious factors that can be neutralized by mother's milk.[7] In particular, we expect that the effects of breast-feeding would be stronger under conditions that (a) maximize exposure to infections, (b) reduce the opportunities available for adequate supplementation, and (c) diminish the opportunities for choosing hygienic practices in the preparation of foods and handling of the child. More generally, we expect the effects of breast-feeding to drop sharply with age, waning to insignificance beyond the first 12 months of life, and to be stronger within disadvantaged social groups.

The Effects of Birth Intervals on Early Childhood Mortality

At the outset we need to distinguish between the effects of the preceding birth interval (the birth-to-conception interval that is closed by conception of the index child) and the effects of a following conception and following birth interval (the birth-to-conception interval that is closed by conception of another child following the index child). Although the bulk of the literature is devoted to the former, both are important; and at least in some cases the effects of the latter overwhelm those of the former (*19, 23*).

Regarding the effects of the preceding birth interval, studies conducted in the United States of America (*37*) and Great Britain (*16*) found higher mortality among children conceived soon after a birth. Surveys carried out in Punjabi villages in India, in East and West Africa, and in two Latin American countries (Colombia and Ecuador) have confirmed this relation (*38–40*). In addition, cross-national and single-country studies carried out with data from the WFS and DHS have provided a formidable mass of evidence supporting the argument that short birth intervals indeed have powerful deleterious effects on children's health and increase childhood mortality (*18, 20, 21, 23, 25, 31, 32, 41*).[8]

[7]Millman SR. *Breast-feeding in Taiwan: a study of change.* University of Michigan; 1982. (Doctoral dissertation).

[8]See also Palloni A, Ubomba-Jaswa P, Gaigbe-Togbe V. *Breastfeeding, pace of childbearing, and early childhood mortality in Sub-Saharan Africa.* 1992. (Final report submitted to Demographic and Health Surveys).

But what are the mechanisms involved? First, a short birth-to-conception interval can retard fetal growth, and this can result in low birth-weight and increased death risks due to endogenous causes (*15, 16*). Thus, in some cases a short length of gestation is itself a potential result of a short birth-to-conception interval. Second, short birth-to-conception intervals impair the mother's endocrine function and exert deleterious effects on breast milk production, reducing the supply of breast milk for the child that closes the interval (the "index" child). These two mechanisms are linked to maternal depletion and to its effects on the viability of the fetus and the feasibility of lactation. Third, short birth intervals lead to pressure on the demand for material resources and maternal care and exacerbate competition among siblings. And fourth, children born in rapid succession will increase crowding and make more feasible the transmission of childhood diseases, such as measles, whose virulence and case fatality rates are magnified through secondary transmission, frequently jeopardizing the chances of the youngest in a group of siblings (*43*).

If preceding birth intervals of substandard length aggravate maternal depletion and tend to derail the gestation process, then the effects on infant mortality should be important shortly after birth but should subside thereafter. If, however, maternal depletion affects the supply of breast milk, then the effects of such depletion ought to linger for as long as breast milk's protective properties last. Finally, if the effects of a short preceding birth interval depend on competition and crowding, these effects should be enduring and should last for a good fraction of early childhood.

In our previous investigations we have emphasized that the deleterious effects of preceding birth intervals could pale if compared with the effects of following conceptions (*19, 23, 30, 31*). This indeed appears to be the case in Latin America, although in other areas of the world the magnitude of the effects of previous birth interval is larger than in Latin America.

The mechanisms through which the timing of the following conception influences the mortality risks of the index child have been identified but never successfully confirmed (*17, 21, 23, 37, 39, 40*). A new conception is likely to prompt termination of breast-feeding, if breast-feeding is taking place at all. This may occur partly as a result of the inhibitory effects of pregnancy-related estrogens that preclude normal secretion of prolactin, or it may be due to fatigue and hormonal changes caused by the pregnancy itself. Alternatively, it could be the outcome of a behavioral choice supported by social customs, whereby the first signs of a new pregnancy are met with the immediate cessation of lactation (*44*). If, in addition, the pregnancy is successful and terminates in a live birth, there will be increased competition for resources and maternal care, as well as higher levels of crowding.

When the mechanism through which the timing of a following conception affects mortality is lactation, we should expect that the effects on mortality of the index child will be strongest before the sixth month. In contrast, if the main mechanism is competition or crowding, we should expect the effects to be sustained into early childhood and to be stronger among index children whose following sibling survives than among those whose following sibling dies during early infancy.

Formulation and Estimation of Alternative Models

This section deals with the nature of the models to be estimated and the strategies used to reduce or eliminate biases.

Discrete Models for Mortality in Early Childhood

We first formulated a simple model for the odds of dying in the following age segments (intervals defined in completed months): 0, 1–2, 3–5, 6–11, 12–23, and 24–59. The logistic models involved describe the logarithm of the conditional odds of dying in arbitrary age segments, $(x_j, x_j + n_j)$ of length n_j as:

$$\log (Q(x_j, x_j + n_j)/(1 - Q(x_j, x_j + n_j))) = \beta(x_j)Z(x_j) \quad (1)$$

where $Q(x_j, x_j + n_j)$ is the conditional probability of dying in the age segment $(x_j, x_j + n_j)$, $\beta(x_j)$ is a vector of effects in the age segment, and $Z(x_j)$ is a vector of covariates defined for the same age segment. This model can be estimated by maximum likelihood procedures that yield consistent and minimum variance estimates for each of the age segments that we care to identify.

The set of definitions for the independent variables appears in Annex 1. The variables representing maternal education, area of residence, age at delivery, and birth order; the length of the preceding birth interval; and the survival of the preceding child are fixed covariates, whereas the variables breast-feeding and following conception are time-dependent, since they may change as the index child moves from one age segment to another. For example, a child may have breast-feeding (Bf) = 1 for the age segment 3–6 and Bf = 0 for the segments 6–12, 12–24, and 24–60 if the child was weaned before the sixth month of life.

Estimates were obtained for the sample of all children born 1–5 years before the survey was carried out, since information on breast-feeding was available only for these children. Table 1 displays information about the frequency of observations, deaths, and censored cases with respect to children in each of the age segments considered. Table 2(a) shows the values of the estimated parameters, standard errors, and the log-likelihood function at the point of convergence. It should be borne in mind that the accuracy of the estimates is compromised by small sample sizes and, particularly, by the small number of events. Despite this shortcoming, however, several features stand out; these are described in the section that follows.

Analysis of Results

The first salient feature that should be noted in the Table 2 figures is that the effects of breast-feeding are consistently negative as well as strong and statistically significant, at least until the sixth month, after which they are considerably diluted. A second feature is that the effects of a following conception are very strong and in the expected direction in the 3–5 segment, after which the coefficient becomes statistically insignificant. A third feature is that the effects of maternal age, birth order, preceding birth interval, and survival of previous child are somewhat erratic and rarely statistically significant, although they are almost always properly signed. Only maternal age appeared to have some importance during the age segment 1–2 and the second year of life.

These results are consistent with those obtained from the Mexican World Fertility Survey (WFS) (*23*). Both data sets show important effects of breast-feeding and a following conception, while neither one uncovers noticeable effects of maternal age and birth order. The places where discrepancies occur between the Mexico DHS and Mexico WFS data involve the effects of preceding birth intervals and survival of a previous child. Both variables were of some relevance in the WFS data but of negligible significance in the DHS.

In order to evaluate whether these discrepancies were due to the smaller size

Table 1. Basic frequencies by age segments (sample of children born 1–5 years before survey).

	Age segment (in months)					
	0	1–2	3–5	6–11	12–23	24–59
Exposed	2 665	2 578	2 563	2 533	2 516	2 101
Deaths	87	15	30	17	20	10
Censored	—	—	—	—	395*	2 027†
Children whose age at death equals breast-feeding duration	19	7	10	6	6	1

*Censored cases are all those children surviving to the date of the survey who were born between 12 and 23 months before the survey.

†Censored cases are all those children surviving to the date of the survey who were born between 24 and 59 months before the survey.

of the DHS sample, we reestimated our models using a longer window of time, and instead of having the sample include only children born within the 1–5 year window, defined before, we also had it include all those born between 1 and 10 years before the survey.[9]

The new estimates (*8*) were, with some minor exceptions, virtually identical to those shown in Table 2(a). Indeed, the smaller sample provides conservative estimates of the effects of a following conception and to some extent of maternal age. The remaining estimates did not differ from those derived from the larger sample.

An alternative strategy for increasing the stability of the estimates is to consolidate the age segments into wider ones, with a view to including a larger number of events. To accomplish this we consolidated the second and third age segments into one (1–5 months) and the fifth and sixth age segments into another (12–59 months) and reestimated the model using the smaller sample of 2 665 children. As shown elsewhere (*8*), hardly any of the inferences drawn from Table 2(a) were altered. In particular, the effects of breast-feeding were properly signed and statistically significant before the sixth month; the effects of a following conception behaved as expected only before the sixth month; and all the other intermediate variables affecting mortality (birth order, preceding birth interval, survival of previous child, and maternal age at delivery) were of lesser importance.

The Magnitude of Breast-feeding Effects and Some Alternative Interpretations

How powerful are the effects of breast-feeding? The estimated effects appearing in Table 2 are expressed in a logit scale, but they can also be expressed as relative risks. Indeed, the log odds of dying in small age segments with low levels of mortality are approximately equal to the average death risk over the age segment. Thus, in most cases we can interpret the exponentiated estimated effect as a relative risk. For example, in the 1–2 age segment the relative mortality risk of a child who never breast-fed or was weaned before the first month was about 13.1 times higher than the risk of a child who breast-fed longer than 1 month; the relative risk in the 3–5 age segment was about 5.8 times higher; and the relative risk in the 6–11 age segment was about 3.2 times higher. These relative risks are close but not identical

[9]Breast-feeding information for children born between 6 and 10 years before the survey was missing; this was captured using a dummy variable for missing information.

Table 2(a). Estimated effects using a logit model and conventional age segments (standard errors in parentheses). See Annex 1 for definitions of main variables in Column 1.

	Age segment (in months)					
	0	1–2	3–5	6–11	12–23	24–59
Age (1)	−.14 (.38)	−1.43 (.73)*	−.05 (.61)	−1.32 (1.05)	−1.90 (.65)*	−.88 (1.34)
Age (2)	−.41 (.44)	−1.24 (.90)	−.35 (.72)	−1.98 (1.17)	−2.00 (.80)*	−.03 (1.38)
Bint (1)	.44 (.36)	6.21 (8.96)	.20 (.58)	1.00 (1.07)	−.83 (1.07)	6.16 (10.30)
Bint (2)	−.47 (.45)	5.93 (8.95)	−1.03 (.79)	.93 (1.11)	1.36 (1.07)	6.37 (10.30)
BO (1)	−.45 (.45)	6.10 (8.96)	−.32 (.70)	.31 (1.59)	−.37 (1.24)	5.63 (10.30)
BO (2)	.37 (.29)	.59 (.81)	.08 (.49)	1.75 (.81)*	.51 (.62)	.55 (.93)
S	.57 (.45)	−6.44 (16.6)	.48 (.78)	−6.07 (10.5)	−.23 (1.04)	.34 (1.12)
Bf	−2.82 (.27)*	−2.57 (.67)*	−1.75 (.46)	−1.16 (.53)	−.83 (.66)	.53 (1.17)
FC	—	.37 (1.09)	1.81 (.44)*	.22 (.67)	.31 (.47)	−.34 (.69)
−2 log likelihood	607	146	283	162	213	107
Degrees of freedom	2 654	2 566	2 551	2 521	2 504	2 089
Cases	2 665	2 578	2 563	2 533	2 516	2 101

*Statistically significant at $p < .01$.

Table 2(b). Effects of following conception before and after controlling for breast-feeding (standard errors in parentheses).

	Age segment (in months)				
	1–2	3–5	6–11	12–23	24–59
Before control	1.17 (1.07)	2.11 (.43)*	.54 (.65)	.46 (.46)	−.43 (.66)
After control	.37 (1.09)	1.81 (.44)*	.22 (.67)	.31 (.47)	−.34 (.69)

*Statistically significant at $p < .01$.

to those obtained with the WFS data (12.8, 7.9, and 2.8, respectively).

From a policy standpoint, relative risks of this magnitude would be unimportant if the prevalence of traditional lactational practices were pervasive. Unfortunately, this is not the case in Mexico. In the DHS sample, for example, about 22% of the children born were never breast-fed, 29% of those surviving to the first month were weaned before the first month, 45% of those surviving to the third month were weaned before the third month, 57% of those surviving to the sixth month were weaned before the sixth month, and fully 79% were weaned before their first birthday.

How much would infant mortality decline if the breast-feeding norm were to wean no earlier than the 12th month? The proportionate decrease in mortality in the interval (y, 12), with $y > 0$, that is attributable to changes in lactation (δ_b) is given by the following expression:

$$\delta_b = \sum_{j=y}^{j=12-w} (1 - \rho_j)\,\eta_j \qquad (2)$$

where the summation is over all age segments contained in the age interval (y, 12), w is the width of the last age segment in the interval, ρ_j is the population-attributable risk due to breast-feeding in the age segment (j, $j + w_j$), and η_j is the fraction of the integrated hazard in the interval (y, 12) accounted for by the integrated hazard in the age segment (j, $j + w_j$). In our illustration we use the following age segments: 1–2, 3–5, and 6–11 so that $j = 1$, 3, and 6 while $w_j =$ 2, 3, and 6, respectively. The corresponding values of ρ_j are .79, .72, and .65;[10] and the values of η_j are .24, .48, and .28 for $j = 1$, 3, and 6, respectively.

Given these values, the estimated reduction in mortality between month 1 and month 12 would be about 71%. Since infant mortality in the sample of children born between 0 and 5 years before the survey was close to .058, the expected level of infant mortality after the change in lactation practices (considering only mortality reduction in the month 1–month 12 period) would be about 68% of the observed one or .0391. It is important to emphasize that this is a *lower* bound for the expected mortality reduction, since we have assumed that the changes in lactation would have no impact whatsoever on mortality during the first month of life.[11]

One could make the argument that the estimates in Table 2 are contaminated by duration heaping with respect to both breast-feeding durations and ages at death.[12] Indeed, the Mexican DHS shows

[10]The estimated population-attributable risks are calculated according to the following expression: $P_e(r - 1)/[P_e(r - 1) + 1]$ where r is the estimated relative risk and P_e is the fraction of the population exhibiting the behavior we would like to suppress (e.g., cessation of breast-feeding prior to the 12th month of life).

[11]This assumption is made to safeguard against potential biases in the effects of breast-feeding for month 0.

[12]The term "duration heaping" refers to the phenomenon whereby reports of duration (breast-feeding, age) tend to coincide with certain selected digits (such as 0, 5, or 8).

a fair amount of heaping in at least one crucial variable, namely, breast-feeding duration (*8*). To minimize the impact of heaping around selected preferred digits, we redefined the age segments in such a way that they were centered around the preferred digits. Only perverse heaping (a distribution of the deviations between observed and true durations with very high variance) would render this solution ineffective.

The results we obtained revealed that the estimates of the effects of breast-feeding become smoother, since the age segments overlap with those used before. As a consequence, the new estimates are different not just because we reduced the impact of heaping but also because they reflect the changing effects of breast-feeding and of a following conception during the first year of life. One way of purging for these changing effects is to recalculate the population-attributable risks and the expected value of infant mortality when weaning only occurs after the first year of life. The results of this exercise reveal that the expected level of infant mortality (assuming no changes during the first month) would be 68% of the observed level, exactly the same figure obtained before. We conclude that correction for heaping has no effects on our estimates.

The Effects of Following Conceptions

How powerful are the effects of a short time interval between the birth of the index child and a following conception? The results shown in Table 2(a) are unambiguous: The bulk of the effects occur before the 6th or 12th month of life and are substantially diluted thereafter. The results displayed in Table 2(b) point to an additional conclusion: Although the effects of a following conception are attenuated after a control for breast-feeding is introduced, they do not disappear and remain properly signed and statistically significant in the age segments where they were originally properly signed and significant.

The latter results, not unlike those obtained elsewhere in Latin America (*19, 23*), should be interpreted as indicating that cessation of lactation is not the only mechanism through which the occurrence of a following conception affects the mortality risks of young children. The age pattern of the effects supports the idea that it is highly unlikely that a following conception could interfere with survival of the index child through the actual birth of the following child. This is because the effects cease to be relevant before the following child is born.

Some qualification is needed, since there is evidence suggesting that the effects might remain important during the 6–12 age segment, where there is a small probability of competition between the index child and the following sibling (*8*). However, when the feasibility of this alternative is examined in more detail we must conclude there is no basis for suspecting that competition with a recently delivered sibling explains the effects observed.

This leaves us with a puzzle: If neither cessation of lactation nor competition with a living younger sibling is sufficient to explain the observed effect, what mechanisms might explain it? One possibility is that breast-feeding diminishes in intensity and becomes more erratic and irregular after a new pregnancy. This could cause a reduction in the protective effects of lactation that we are not able to control with data revealing only breast-feeding duration, not intensity. If this were the case, the effects of a following conception would remain strong even after controlling for breast-feeding. Another possibility is that a following conception initiates a short period of reduced maternal care due to the burdens of a pregnancy that do not necessarily involve curtailment of

breast-feeding. Again, this is a proposition that is not testable with the information available to us.

To evaluate the magnitude of the effects associated with a following conception, we proceeded to calculate population-attributable risks for the age interval 0–11. The mathematical expression used for these calculations was exactly analogous to (2) with two modifications. The first modification was that the relative risk referred to the ratio between mortality among those children whose mothers had a conception before the child reached some age x and mortality among those children whose mothers did not experience such a conception. The second modification was that the behavior to be suppressed was not weaning but a conception following soon after the birth of the index child.

This calculation showed the relative risks in age segments 1–2, 3–5, and 6–11 to be 1.5, 6.1, and 1.25, respectively, whereas the prevalences of the behavior were .02, .04, and .12, respectively. These conditions combined with a reproductive regime whereby the time elapsed between the birth of the index child and the conception of the following child always exceeded 11 months to yield an expected infant mortality rate of about .0543, or only about 6% less than the observed one. The reason for the low magnitude of the effects is, obviously, the low prevalences of very short birth intervals.

Continuous Time Models of Mortality Risks during the First 2 Years of Life

A proportional hazard model assumes that the risk of dying in the small age interval $(x, x + \delta)$ is a multiplicative function of a baseline hazard and a factor expressing net relative risks:

$$\mu(x) = \mu_0(x) \exp[\alpha\, Y(t)] \qquad (3)$$

where $\mu_0(x)$ is a baseline hazard, α is a vector of effects, and $Y(t)$ is a vector of fixed and time-dependent covariates. It is easy to show that when the vector of covariates is identical and when the model is for age segments within which the hazard is small and constant, the effects estimated by a logistic and a hazard model ought to be the same.

A hazard model has two advantages: It does not require assumptions to define age segments within which estimates are to be obtained, and it can handle time-dependent covariates in a parsimonious way. The smallest unit of time in our hazard models is 1 month, and the last month of observation is the 59th.

A disadvantage of the hazard model is that it relies on an assumption of proportionality of hazards which is not always empirically verified. Distortions of the estimates are likely to occur when departures from proportionality are more than trivial. Furthermore, the proportional hazard model is a somewhat cumbersome tool for handling the estimation of higher order interaction effects with duration (age). By contrast, the logit model relies on an assumption of an underlying logistic distribution which is either more easily verified or of little consequence if moderate departures occur. In addition, the logit model enables us to easily retrieve estimated effects that are specific for each age segment, although it does require that the age span of interest be broken down into age segments of variable (and arbitrary) length.

To strike a balance between parsimony and accuracy we estimated alternative models that enabled us to gauge the sensitivity of the estimated effects to slightly different initial conditions. The first column of Table 3 displays the estimated effects for the age segment 0–12 months (exactly) in the sample of children born 0 to 5 months before the survey. The effects of breast-feeding are large and negative but are probably highly influ-

Table 3. Alternative estimates of effects from a continuous time model for survival during the age segment 0–12 months (exactly). Standard errors are shown in parentheses. See Annex 1 for definitions of main variables in column 1.

	Model 1*	Model 2*	Model 3*	Model 4*	Model 5*
Intercept	−3.43 (.40)†	−4.84 (.46)†	−3.43 (.40)	−4.84 (.46)†	−5.96 (.72)†
Slope	−.40 (.04)†	−.33 (.05)†	−.40 (.04)	−.33 (.05)†	−.12 (.08)†
Age (1)	.55 (.38)	.44 (.38)	.55 (.38)	.44 (.38)	1.12 (.62)
Age (2)	.35 (.26)	.16 (.26)	.35 (.26)	.16 (.26)	.50 (.42)
Bint (1)	.61 (.35)	.68 (.35)	.62 (.35)	.68 (.35)†	.73 (.57)
Bint (2)	−.06 (.39)	−.06 (.39)	−.01 (.39)	−.05 (.39)	.15 (.68)
BO (1)	−.11 (.42)	.04 (.42)	−.11 (.42)	.04 (.42)	.28 (.67)
BO (2)	.37 (.28)	.29 (.28)	.37 (.28)	.29 (.28)	.69 (.46)
S	−.25 (.48)	−.21 (.48)	−.25 (.48)	−.21 (.48)	−1.07 (1.09)
Bf	−2.05 (.20)	.01 (.29)	−2.05 (.20)	.06 (.29)	−1.13 (.32)
FC	−.15 (.53)	.21 (.54)	−.15 (.53)	.21 (.54)	−.27 (.56)
−Log likelihood	663	717	663	717	360
Observations	2 528	2 528	2 528	2 528	2 458

*Definitions of models, all of which control for maternal education and rural-urban residence Model 1 Defined for the age segment 0–12 (exactly). Model 2: Defined for the age segment 0–12 (exactly); if child died at 0 months and never breast-fed, duration of breast-feeding was set equal to 0 Model 3: Defined for the age segment 0–12 (exactly), if duration of breast-feeding equalled age at death then duration of breast-feeding was reduced by 1 month. Model 4: Defined for the age segment 0–12 (exactly); if child died at 0 months and never breast-fed, the duration of breast-feeding was set equal to 0; if duration of breast-feeding equalled age at death then duration of breast-feeding was reduced by 1 month Model 5· Defined for the age segment 1–12 (exactly).

†Statistically significant at $p < .01$.

enced by the inverse relation between survival and lactation that prevails during the first months.

To eliminate the bias that accrues when unmeasured conditions jointly affect breast-feeding and survival status during the first month, we estimated a model for the age segment 1–12 months (exactly). The results appear in Column 5. The magnitude of the breast-feeding effects is cut in half, but the coefficient remains properly signed and statistically significant.

To circumvent the same problem, other authors (*29*) have used a more conservative but also riskier solution. It consists of recoding breast-feeding so that it equals 0 (completed months) for all children who died during the first month and who never breast-fed. The resulting estimates after recoding appear in Column 2 of the same table. A comparison of the estimated effects of breast-feeding in Columns 2 and 5 reveals that the recoding strategy unduly contaminates the estimated effects that apply to ages beyond the first month.

Columns 3 and 4 display estimated coefficients for models where we have recoded the duration of breast-feeding to circumvent a reporting problem of some importance. As shown in Table 1, a significant fraction of children who died had a reported duration of breast-feeding equal to their age at death. If this were a reflection of what actually occurred, our logistic and hazard model estimates would be unbiased. But if the duration of breast-feeding were rounded to equal the age at death, our estimates could contain a downward bias. This poses a very serious problem in Demographic and Health Surveys in African countries and exerts a powerful influence on results (*45*).

In order to evaluate the magnitude of this bias, we reestimated models 1 and 2 after reducing by 1 month the duration of breast-feeding for dead children whose reported duration of breast-feeding equalled their age at death. The only exception to the recoding rule is that no change was implemented if the duration of breast-feeding was equal to 3, 6, or 12.

This exception provided a useful way of ensuring that logistic models applied to the recoded data would yield exactly the same results obtained before. In other words, if our recoding accurately redefined durations, it should in no way alter results from the logistic models or alter conclusions drawn from them.[13] Column 3 should be compared with Column 1 and Column 4 with Column 2. The results in these columns show that the recoding makes little difference, and that the estimates are insensitive to the feature revealed by Table 1.

In sum, the effects of breast-feeding in our hazard model are large and statistically significant within the first year of life but also between the first and twelfth months of life. The latter is a conservative estimate little affected by spurious relations that are likely to take place during the first month of life.

The effects of the other variables in the hazard model are less problematic. First, the effects of a following conception are insignificant. This is not surprising in view of the results from the logistic model, which suggest that the effects of a following conception are statistically significant only during the first 6 months of life but are diluted thereafter. The continuous hazard model averages the changing effects over the first year of life, and the net result is a nonsignificant effect. The estimates of other variables do not lead to conclusions different from those obtained with the logistic models.

[13]The estimates obtained with the recoded data ought to yield an upper bound (absolute value) for the effects of breast-feeding. Other reallocations of durations equal to ages at death should lead to smaller (absolute) values of the estimated effects of breast-feeding. In particular, if all durations equal to ages at death were assigned a value of 0, we would obtain a lower bound of the estimated effects of breast-feeding.

CAN FAMILY PLANNING SAVE LIVES?

The results presented above, at least in principle, support the idea that better spacing of children must necessarily lead to higher probabilities of survival. Consider, for example, the expected reduction in infant mortality resulting from elimination of following conceptions that occurred prior to the first year of life of the index child. Provided one takes this as a simple counterfactual assumption—with all the caveats such assumptions require—the results of our models would have been properly interpreted. The same applies to changes in the distribution of births by length of preceding birth intervals. Indeed, even though the effects of the preceding interval are, for the most part, statistically insignificant, they do exert an influence on infant mortality in the expected direction.

It is altogether different, however, to infer from these findings that by enabling individuals to space children better, family planning programs will induce important gains in child survival. This inference is a non sequitur for the following reasons:

First, reproductive control for the purpose of stopping childbearing—an important and in some cases the only component of family planning programs—should have no effects or only marginal effects upon survival. True, early stopping will favorably alter the distribution of births in terms of parity and maternal age at delivery, and should also reduce the proportion of all live births rapidly followed by another conception. However, the effects of higher parity and older maternal age at delivery were shown to be somewhat weak; and so a slight improvement in the distribution of births in terms of parity and maternal age will induce only minor changes in mortality. Also, even though the effects of a poorly spaced fol-

lowing conception are very strong, the decrease in the proportion of following conceptions induced by stopping childbearing should be very small, since the bulk of poor spacing occurs early in a woman's reproductive career, not at the end. To the extent that family planning merely alters the pattern of stopping, its beneficial effects on child survival should be minuscule.

Second, the introduction of some form of birth control for spacing or stopping childbearing will not occur without affecting breast-feeding practices. Indeed, although the mechanisms have not yet been fully clarified, there is a ubiquitous negative relation between intentional use of birth control and lactation practices; that is, women who practice birth control are significantly less likely to breast-feed for long periods or to breast-feed at all (*6, 10, 46*). Whatever the mechanism producing the relation, it should follow that the beneficial effects of spacing on child survival could be offset by some degree of abandonment of a practice that, as we showed before, has considerable impact on childhood mortality. Furthermore, the reduction of breast-feeding should lead naturally, ceteris paribus, to a shortening of birth intervals and will thus tend to offset the direct effects of practicing increased birth control.

These considerations suggest that one cannot infer the ultimate effects of family planning on infant and child survival from the estimates obtained above. The only solution to the problem is to formulate an integrated model, one that simultaneously takes into account all the relations involved.

What should the nature of this model be? Elsewhere we have elaborated a lengthy and cumbersome but exact procedure (*6*) that combines an arbitrary reproductive regime with an arbitrary mortality regime to assess the ultimate effects on mortality attributable to reproductive practices and breast-feeding. What we propose here is a simplified version of that model.

In a nutshell, the simplified model defines alternative birth distributions according to birth order, maternal age at delivery, length of preceding birth interval, survival of preceding child, and timing of following conception. It then applies to each of the resulting cells a distribution by length of breast-feeding. Each of the classes of births thus defined is associated with levels of mortality in the age segments (x, x + n months) contained within, say, the age intervals 0–11 or 0–23 months. The estimated level of infant mortality for a given cohort is calculated via a weighted projection of children who survive birth from age 0 months to age 12 months (exactly) in each of the cells of the Cartesian product. A full description of this simplified model appears elsewhere (*8*).

In applying the model, 12 patterns of behavior were distinguished. These involved two possible patterns of breast-feeding, three of birth spacing, and two of maternal age. These patterns are as follows:

I. Breast-feeding
   A. Traditional breast-feeding: The duration of breast-feeding was longer than 6 months.
   B. Modern breast-feeding: The duration of breast-feeding was 0 completed months.

II. Birth spacing
   A. Short-short spacing of childbearing: The length of the preceding birth interval was less than 19 months, and the following conception occurred before the third month of life of the index child.
   B. Short-long spacing of childbearing: The length of the preceding interval was less than 19 months, and the following conception oc-

curred before the index child's sixth month of life but after the third.

C. Long spacing: The length of the preceding interval was between 19 and 36 months, and the following conception occurred after the sixth month of life of the index child.

III. Maternal age

A. Early fertility: The mother's age at delivery was less than 20 years.

B. Late fertility: The mother's age at delivery was equal to or greater than 20 years but less than 30.

Mortality risks for the first year of life associated with each of the 12 patterns are shown in Table 4. The effects of breast-feeding, birth spacing, and maternal age are easily calculated using the differences between the various cells.[14]

To assess the contribution of the regime of breast-feeding and the reproductive pattern, consider the results displayed in Table 5. Take as a baseline the case when births are distributed evenly across those cells in the two bottom rows of Table 4, thus effectively eliminating the extreme case of short spacing covered by the cells in the top row of the table. The expected value of infant mortality would be about .0498, slightly below the value obtained using the Mexican DHS data.

If only the breast-feeding pattern were modified so that all the index children were breast-fed at least 6 months, the expected level of infant mortality would drop to .0279, a 44% reduction below the baseline level. On the other hand, if instead of changing the breast-feeding pattern one were to change birth spacing and maternal age at delivery so that short-spaced and early births were eliminated, the expected levels of infant mortality would drop to about .0301 and .0382, respectively, representing reductions of 40% and 23% relative to the baseline level. The most optimistic scenario, implying a traditional lactation pattern combined with optimal spacing and maternal age at delivery, would yield an expected infant mortality of about .013, representing a 75% reduction below the baseline level.

Table 4. Levels of infant mortality, by behavior patterns (see text for definitions).

	Timing of fertility			
	Early		Late	
	Breast-feeding		Breast-feeding	
Spacing	Yes	No	Yes	No
Short	.074	.214	.044	.039
Short/long	.049	.122	.029	.077
Long	.021	.053	.013	.034

As noted above, however, the overhaul of the reproductive regime caused by introduction of modern family planning may come at a cost—namely, that women tend to abandon the pattern of traditional breast-feeding at the same time that they remove early and shortly spaced births. Considering this, if all the women altered their reproductive behavior while only half continued adhering to a traditional pattern of breast-feeding, infant mortality would settle in the neighborhood of .0233, about 53% less than the baseline level. Or, if only a small fraction of the women (10%) continued adhering to a traditional pattern of breast-feeding, then infant mortality would get close to .0340, still lower than the baseline level but higher than the levels found for most of the other scenarios considered.

Two policy-related inferences can be drawn from these results. First, the magnitude of breast-feeding's effects is great enough to offset more than 60% of the beneficial changes attributable to im-

[14]Again, note that the effects of breast-feeding are on the conservative side, since we have assumed that all children are breast-fed at birth.

Table 5. Expected levels of infant mortality under different scenarios.

Scenario	Infant mortality
Scenario I: Uniform distribution of births in cells 5–12 of Table 4	.0498
Scenario II: Universal long breast-feeding (no change in timing or spacing)	.0279
Scenario III: Universal late births (no changes in spacing or breast-feeding)	.0382
Scenario IV: Universal long spacing (no changes in timing or breast-feeding)	.0301
Scenario V: Universal long spacing and late timing (no changes in breast-feeding)	.0233
Scenario VI: Universal long spacing, late timing, long breast-feeding	.0125

proved birth spacing and maternal age. Thus, emphasis on the continuation of traditional breast-feeding practices should be incorporated into family planning programs. Second, the effects of improved birth spacing and maternal age alone are powerful enough to potentially reduce infant mortality by around 20–40% below current levels.

These conclusions must be accepted with caution, however. This is because while the estimated impact of breast-feeding may be a lower bound, the estimated impacts for birth spacing and timing are almost surely upper bounds. That is because we have not accounted properly for the correlation of birth spacing and timing over the lifetime of a woman, and also because it is unlikely that we have succeeded in purging for all confounding effects that are associated with but not attributable to birth spacing and timing. Thus, the seemingly ample room for changes estimated from Table 4 is deceiving if not properly qualified.

It should also be remembered that the room for changes would have been as large or even larger had we compared infant mortality levels among the poor and those who are better off, *even while controlling for reproductive behavior.* In this case the policy-relevant conclusion would have invoked changes in the distribution of wealth rather than changes in reproductive regimes. As this indicates, the road toward reduced mortality involves not one set of determinants but several, and it is unlikely that significant changes will occur if policies are narrowly designed to modify a few conditions in isolation from others.

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REFERENCES

1. Bongaarts J. The projection of family composition over the life course with family status life tables. In Bongaarts J, et al, eds. *Family demography.* New York: Oxford University Press; 1987:189–212.
2. Palloni A. Effects of interbirth intervals and breast-feeding on infant and early childhood mortality. In Ruzicka L, Wunsch G, Kane P, eds. *Differential mortality; methodological issues and biosocial factors.* Oxford: Oxford University Press; 1988.

3. Potter J. Does family planning reduce infant mortality? A comment. *Popul Dev Rev* 1988;14(1):179–187.
4. Trussell J. Does family planning reduce infant mortality? An exchange. *Popul Dev Rev* 1988;14(1):171–178.
5. Palloni A, Kephart G. The effects of breast-feeding and contraception on the natural rate of increase: are there compensating effects? *Popul Stud* 1989;43: 455–478.
6. Palloni A, Pinto G. Family planning and infant and child survival. In: *Volume 1: Proceedings of the IUSSP General Population Conference*. Liege: International Union for the Scientific Study of the Population; 1989.
7. Palloni A, Pinto G, Lastiri S. *Reproductive regimes and early childhood mortality in Mexico*. Madison: Center for Demography and Ecology, University of Wisconsin; 1993. (Working paper 93-14.)
8. Wray JD. Maternal nutrition, breast-feeding, and infant survival. In: Mosley WH, ed. *Nutrition and human reproduction*. New York: Plenum; 1978.
9. Morley D. *Pediatric priorities in the developing world*. Boston: Butterworths; 1973.
10. Millman SR. Breast-feeding and infant mortality: untangling the complex web of causality. *Sociol Q* 1985;26:65–79.
11. Knodel J, Kinter H. The impact of breast-feeding patterns on the biometric analysis of infant mortality. *Demography* 1977; 14:391–409.
12. Plank S, Milanesi L. Infant feeding and infant mortality in rural Chile. *Bull World Health Organ* 1973;48:203–210.
13. Eastman NJ. The effect of interval between births on maternal and fetal outlook. *Am J Obstet Gynecol* 1944;47:445–446.
14. Fedrick J, Adelstein P. Influence of pregnancy spacing on the outcome of pregnancy. *Br Med J* 1973;4:753–756.
15. Gray RH. Birth intervals, postpartum sexual abstinence, and child health. In: Page H, Lesthaeghe R, eds. *Child-spacing in tropical Africa: traditions and change*. New York: Academic Press; 1981.
16. Cleland JG, Sathar ZA. The effects of birth-spacing on infant mortality in Pakistan. *Popul Stud* 1984;38(3):401–418.
17. Palloni A, Tienda M. The effects of breast-feeding and pace of childbearing on mortality at early ages. *Demography* 1986;23: 31–53.
18. Retherford R, Choe MK, Thapa S, Gubhajn BB. To what extent does breast-feeding explain birth-interval effects on early childhood mortality? *Demography* 1989;26: 439–451.
19. Hobcraft J, McDonald JW, Rutstein SO. Child-spacing effects on infant and early child mortality. *Popul Index* 1983;49(4): 585–618.
20. Hobcraft JH, McDonald JW, Rutstein SO. Demographic determinants of infant and early child mortality: a comparative analysis. *Popul Stud* 1985;39(3):363–385.
21. Palloni A, Millman S. Effects of interbirth intervals and breast-feeding on infant and early childhood mortality. *Popul Stud* 1986;40:215–236.
22. Pebley A, Stupp P. Reproductive patterns and child mortality in Guatemala. *Demography* 1987;24(1):43–60.
23. DaVanzo J, Butz W, Habicht JP. How biological and behavioral influences on mortality in Malaysia vary during the first year of life. *Popul Stud* 1983;73:381–402.
24. Miller JE. Is the relationship between birth intervals and perinatal mortality spurious? Evidence from Hungary and Sweden. *Popul Stud* 1989;43:479–495.
25. Miller J, Trussell J, Pebley A, Vaughn B. Birth spacing and child mortality in Bangladesh and the Philippines. *Demography* 1992;29:305–318.
26. Kuate Defo B, Palloni A. *Determinants of infant and early childhood mortality in Cameroon*. Madison, Wisconsin: Center for Demography and Ecology, University of Wisconsin; 1992. (Working series paper 92-23).
27. Lantz P, Partin M, Palloni A. Using retrospective surveys for estimating the effects of breast-feeding and childspacing on infant and child mortality. *Popul Stud* 1992;46:121–139.
28. Hobcraft JH. Fertility patterns and child survival: a comparative analysis. *Popul Bull United Nations*. 1992;33:1–31.
29. Jelliffe DB, Jelliffe ERP. *Human milk in the modern world*. Oxford: Oxford University Press; 1978.
30. Puffer R, Serrano C. *Patterns of mortality in childhood*. Washington, DC: Pan American Health Organization; 1973. (Scientific publication 262).
31. Ajello CA. *A review of the information regarding the relationship between infant feeding*

practices and infant morbidity: an epidemiological perspective. Baltimore: Johns Hopkins; 1982.

32. Cantrelle P, Leridon H. Breast-feeding, mortality in childhood and fertility in a rural zone of Senegal. *Popul Stud* 1971; 25(3):505–533.
33. Wray JD. Population pressure on families: family size and childspacing. *Rep Popul/ Fam Plann* 1971;9:403–458.
34. Wyon JB, Gordon JE. A long-term prospective-type field study of population dynamics in Punjab, India. In Kiser CV, ed. *Research on family planning*. Princeton: Princeton University Press; 1962.
35. Wolfers D, Scrimshaw S. Child survival and intervals between pregnancies in Guayaquil, Ecuador. *Popul Stud* 1975;29(3): 479–496.
36. Wray JD, Aguirre AL. Protein calorie malnutrition in Candelaria, Colombia: 1, prevalence: social and demographic causal factors. *J Trop Pediatr* 1969;15:76–98.
37. Boerma JT, Sommerfelt AE, Bicego G. Child anthropometry in cross sectional surveys in developing countries: an assessment of the survival bias. *Am J Epidemiol* 1992; 135(4):438–449.
38. Boerma JT, Bicego GT. Preceding birth intervals and child survival: searching for pathways of influence. *Stud Fam Plann* 1992;23(4):1183–1203.
39. Aaby P. Malnutrition and overcrowding—exposure in severe measles infection: a review of communities studies. *Rev Infect Dis* 1988;10(2):451.
40. Harfouche JK. The importance of breastfeeding. *J Trop Pediatr* 1970;16:135–175.
41. Gómez de León J, Potter J. Modelling the inverse association between breastfeeding and contraceptive use. *Popul Stud* 43(1).

Annex 1. Definitions of Main Variables

Maternal age (Age 1, Age 2):

Age (1) = 1 if maternal age is between 15 and 20 years
Age (2) = 1 if maternal age is > 34 years

Preceding birth intervals (Bint 1, Bint 2):

Bint (1) = 1 if b ≤ 18 months
Bint (2) = 1 if 18 months < L ≤ 36 months
L is the time in months from delivery of the preceding child to birth of the index child.

Birth order (BO 1, BO 2):

BO (1) = 1 if the birth order was 1
BO (2) = 1 if the birth order was 4 or higher

Survival of previous child (S):

S = 1 if the death of the previous child occurred before the (estimated) date of conception of the index child.

Breast-feeding (Bf) and following conception (FC) (conventional):

	Age Segment					
	0 months	1–2 months	3–5 months	6–11 months	12–23 months	24–59 months
Bf = 1 if	ever breast-fed	D > 1	D > 3	D > 6	D > 12	D > 24
FC = 1 if	—	I ≤ 1	I ≤ 3	I ≤ 6	I ≤ 12	I ≤ 24

D = reported breast-feeding duration
I = interval between birth of index child and estimated date of conception of following child

Breast-feeding (Bf) and following conception (FC) (unconventional):

	Age Segment					
	0 months	1–2 months	3–5 months	6–11 months	12–23 months	24–59 months
Bf1 = 1 if	ever breast-fed	D ≤ 0	D ≤ 0	D ≤ 0	D ≤ 0	D ≤ 0
Bf2 = 1 if	—	D = 1	1 ≤ D < 3	1 ≤ D < 3	1 ≤ D < 3	1 ≤ D < 3
Bf3 = 1 if	—	—	—	3 ≤ D < 6	3 ≤ D < 6	3 ≤ D < 6
Bf4 = 1 if	—	—	—	—	6 ≤ D < 12	6 ≤ D < 12
Bf5 = 1 if	—	—	—	—	—	12 ≤ D < 24
FC1 = 1 if	—	I < 1	I < 1	I < 1	—	—
FC2 = 1 if	—	—	1 ≤ I < 3	1 ≤ I < 3	—	—
FC3 = 1 if	—	—	—	3 ≤ I < 6	—	—
FC4 = 1 if	—	—	—	—	—	—

For the age segments 12–23 months and 24–59 months the following variables were defined:

For 12–23:

FC1 = 1 if I < 6 months and conception results in birth of an infant dying at 0 months.
FC2 = 1 if I < 6 months and conception results in birth of a child who survives to the date of the survey.
FC3 = 1 if 6 months ≤ I < 12 months.

For 24–59:

FC1 = 1 if I < 6 months and conception results in birth of an infant dying at 0 months.
FC2 = 1 if I < 6 months and conception results in birth of a child who survives to the date of the survey.
FC3 = 1 if 6 months ≤ I < 12 months and conception results in birth of an infant who dies at 0 months.
FC4 = 1 if 6 months ≤ I < 12 months and conception results in birth of a child who survives to the date of the survey.
FC5 = 1 if 12 months ≤ I < 24 months.