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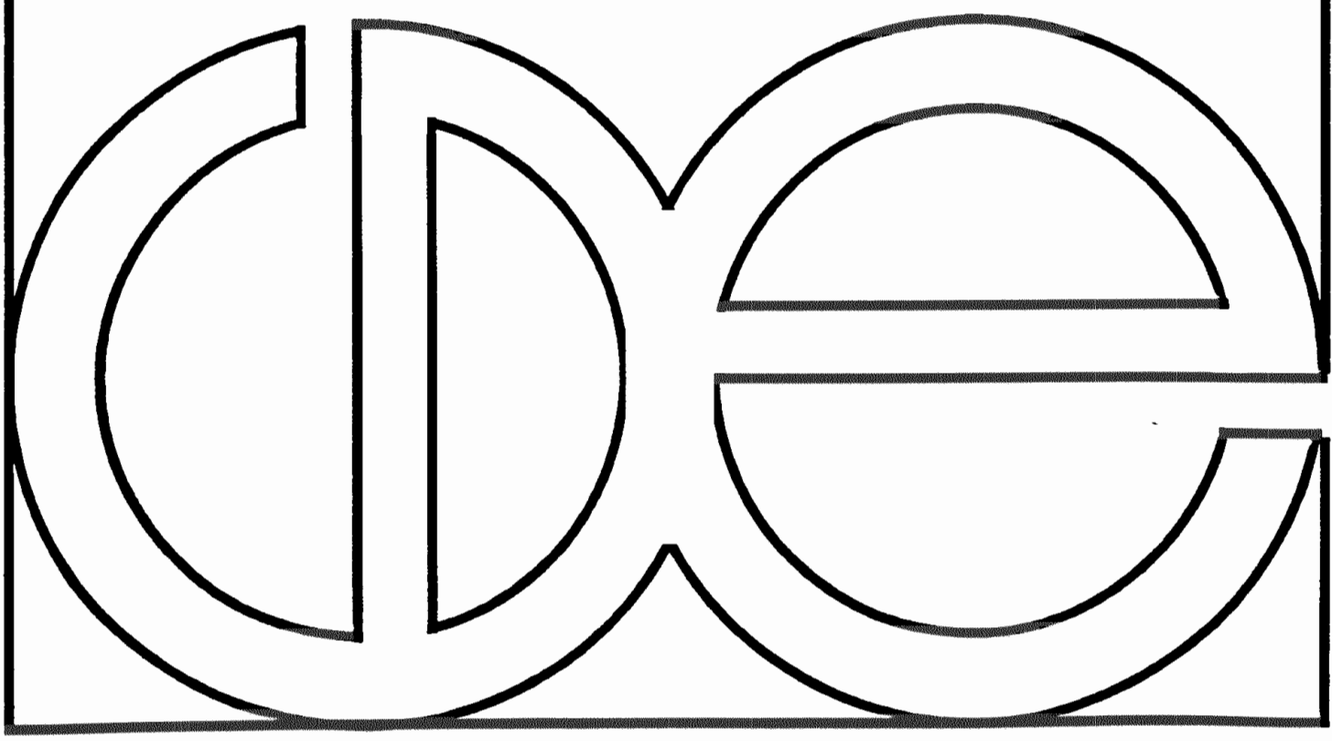

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1. Introduction

This paper examines the results of our analyses of early child mortality in Mexico with data collected by the DHS during the period February-August, 1987. For comparative purposes we also utilize selected results from the WFS survey carried out in the mid-1970s. Our research encompasses two goals. **First**, to assess the effects of pace of childbearing and breastfeeding on early child mortality. **Second**, to evaluate the impact that changes in pace of childbearing and associated changes in patterns of breastfeeding may have on infant and child mortality. Part of this goal is to evaluate the potential survival benefits that are likely to accrue from policies aimed at reducing fertility. Although throughout our analyses we introduce other variables (such as mother's education and region of residence), we focus on their effects only insofar as they are efficient controls for confounding factors on the estimates of interest to us.

We apply conventional statistical techniques to estimate the effects of various determinants on mortality. In particular, we utilize binary choice models and discrete hazard models to evaluate the magnitude of the effects of indicators of pace of childbearing and breastfeeding. Since the data are potentially affected by reporting errors and by selectivity problems, we implement a series of procedures designed to minimize their impact on our estimates and to generate a range of possible values for the most strategic estimates.

An important part of our research is the integration of models involving breastfeeding and variables measuring 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. This concerns the role of family planning in improving survival status of infants and young children. In the past five years or so the debate has pitted

those who believe in the influential effects of family planning against those who hold that family planning has only marginal or no benefits at all to offer in terms of gains in early child survival (Bongaarts, 1987; Palloni, 1988; Potter, 1988; Trussell, 1988; Hobcraft, 1987; Palloni and Kephart, 1989; Palloni and Pinto, 1989). For the most part, however, this debate has been conducted in the absence of well-specified models and without the support provided by consistent estimates. Since breastfeeding and pace of childbearing are determinants of mortality that not only are tightly (and inversely) related to each other but that also interact producing offsetting effects, disputes based on conjectures and not in well-specified models are pointless. In this article we propose a simplified framework and implement estimation procedures that enable us to obtain consistent estimates of effects. We then integrate these estimates to generate net and gross effects of pace of childbearing on child survival.

The paper is divided into four sections of which this introduction is the first. The second section presents a brief description of the data and evaluates its main characteristics. The third section contains the analysis of early child mortality and the fourth section is devoted to an assessment of the net and gross effects of pace of childbearing on infant and child mortality.

2. Description of the Data.

The Mexican DHS consists of some 10,310 retrospective interviews of women aged 15-49. In our study we utilize the reports on the birth histories of 2,665 children born between 1982 and 1986 and of 6,705 children born between 1975 and 1986. The most important characteristics of the sample of births that we use in our paper--a sample of children born between 1 and 5 years before the survey-- appear in Table 1. To provide more robust information on descriptive parameters of interest, this section utilizes the

sample of children born between 1 and 10 years before the survey. Although in the remainder of the paper we concentrate on mortality determinants on the smaller sample - which has information on breastfeeding - we show that the larger sample leads to identical inferences.

Figure A1 (see Appendix 1) shows the conditional probabilities of dying within age segments in the first 5 years of life for the entire sample. The profile of these figures is as expected, with the conditional probability of dying during the first month of life exceeding all others. Figure A2 indicates that mortality during the first year of life is slightly higher among those born. Figures A3 and A4 reveal sharp differentials by education and area of residence of the mother. In all cases the conditional probabilities of dying are higher among the less educated and among those living in rural areas. The proportionate educational differences are more salient within the age segments 1-3, 12-24 and 24-60, whereas those associated with area of residence are larger at 0, 6-12 and 12-24. Figures A5 and A6 illustrate differentials by mother's age at birth and by birth order of the child (parity of the mother at birth). These figures confirm the existence of differentials that have been verified elsewhere: namely, children of very young or older mothers experience higher mortality and higher order births are exposed to much higher mortality than lower birth orders.

Finally, Figures A7 through A11 illustrate the effects of parity and length of preceding interval. By and large, children whose preceding interval is a short one (less than 18 months) have higher mortality than those who follow a longer birth interval within all age segments. Further, they also demonstrate that such differentials apply to all birth orders except the first child. However, 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, length of breastfeeding and the occurrence of a following conception.

To what extent do the aforementioned differentials persist in the face of multivariate controls? This is the question that we set to answer in the following sections.

3. The Determinants of Early Childhood Mortality.

3.1 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 (Wray, 1978; Morley, 1973; Millman, 1982; Ajello, 1984; Knodel and Kinter, 1977; Plank and Milanesi, 1973, Wray, 1978; Eastman, 1944; Federick and Adelstein, 1973; Gray, 1981), it is only recently that we have been able to marshall evidence on a massive scale to support the conjecture. By far the largest contributor of cross-national information illustrating the effects of both length of birth intervals and breastfeeding on infant mortality is the World Fertility Survey. In studies of single countries (Cleland and Sathar, 1984; Palloni and Tienda, 1986; Retherford et al., 1989) or in studies including many countries in multiple continents (Hobcraft et al., 1983 and 1985; Palloni and Millman, 1986), 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 (Pebley and Davanzo, 1988; Pebley and Stupp, 1987; Davanzo et al., 1983; 1984; Muhuri and Menke, 1992). All tests carried out so far suggest that despite some skepticism about the validity of the findings (Potter, 1988; Retherford et al., 1989; Miller, 1989; Miller et al., 1992; Kuate Defo and Palloni, 1992) they appear

to be remarkably robust to errors in reporting and model misspecification (Lantz et al., 1992; Hobcraft, 1992). Furthermore, the evidence generated so far by the Demographic and Health Surveys (DHS) provides additional support to the idea that the estimated effects of breastfeeding 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 such effects. In sections 3.1.1 and 3.1.3 below we describe alternative hypothetical mechanisms that mediate the relation between breastfeeding and early childhood mortality on the one hand and between length of birth intervals and early childhood mortality on the other.

3.1.1. The effects of breastfeeding on early childhood mortality.

Clinical and epidemiological studies have shown that mother's milk has at least three properties which help protect the health of infants (Jelliffe and Jelliffe, 1978; Plank and Milanesi, 1973). **First**, breast milk appears to meet the nutritional requirements for the normal growth of an infant for at least six months (Wray, 1978). Consumed in sufficient quantities, it provides protection against malnutrition syndromes, such as kwashiorkor and marasmus (Morley, 1973). The absence of breastfeeding is related to excess incidence of some diseases, such as diarrhea and gastrointestinal infections that are exacerbated by malnutrition (Puffer and Serrano, 1973; Ajello, 1982). Although many substitutes contain a significant fraction of basic nutrients for the normal growth of a child, none are as rich or complete as mother's milk. **Second**, breastmilk contains proteins that enhance immunocompetence and, in particular, serve to forestall infections of the intestinal tract. **Third**, and finally, mother's milk is a sterile fluid containing substances that prevent the growth of bacteria and make breast milk a highly hygienic

product.

Some of the benefits accruing from these properties, particularly the first two, gradually diminish as the nutritional requirements of the infant increase. Researchers have argued that the relative advantage in terms of immunocompetence and nutritional status of fully breastfed children decrease rapidly after the sixth month of life (Cantrelle and Leridon, 1971; Gray, 1981; Wray, 1978). The importance of each of these properties for the health of the child depends on conditions that heighten or lower the child's exposure to deleterious factors that can be neutralized by mother's milk (Millman, 1982). In particular, we expect that the effects of breastfeeding 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 breastfeeding to drop sharply with age and to wane to insignificance beyond the first 12 months of life, and to be stronger within disadvantaged social groups.

3.1.2. Pitfalls in the estimation of effects of breastfeeding.

Estimating the direction and magnitude of the effects of breastfeeding is a fairly difficult enterprise since statistical inferences are by and large marred by a series of poorly understood difficulties.

i) Spurious Relations.

One of the most important but usually overlooked pitfalls is the failure to control for associated factors. Birth order and mother's age at the birth of the index child are two of the most important factors that simultaneously exert an influence on the length of lactation and on mortality risks (Federeci and Terrenato, 1980). Other characteristics

of the mother, such as education, place of residence, and household income are similarly related to both conditions of interest and ought to be controlled for to avoid sizeable biases. In general, important consideration should be given to the fact that lactation is a behavioral choice influenced by conditions that have a more than moderate impact on child mortality. For example, adoption of modern attitudes in traditional societies could go hand in hand with increased use of health care facilities and gradual abandonment of traditional breastfeeding practices (Potter, 1988). If the model does not include suitable controls for the factors that affect both behavioral choices, the effects of breastfeeding could be downwardly biased.[1]

A thornier problem is posed by the relation between mother's behavior, episodes of illness, and child mortality. Although malnourished children tend to suckle more intensely and, **ceteris paribus**, more frequently, children who become ill are more likely to suckle less intensely or less frequently. In addition, in some societies women tend to interrupt lactation as soon as the child is affected by a disease. In both cases the consequences are the same: an exogenous episode of illness leads to a modification of the behavior (of the child or the mother) which is consequential for the continuity of breastfeeding. Since an episode of illness heightens mortality risks, part of the association between breastfeeding and mortality is an artifact induced by the occurrence of illness. Elimination of this problem is difficult when the protocol of the study does not include continuous information on illnesses.[2]

ii) Simultaneity.

A second set of problems arises due to inappropriate representation of the causal relations involved. Breastfeeding initiation is thought to be dependent on the health status of the child at birth. Thus, healthier children are more likely to initiate lactation

and set in motion the hormonal mechanisms that at least ensure short term duration of lactation. Children who are too sick or too weak to nurse immediately after birth are less likely to ever be breastfed. Since the mortality risks of the latter are higher than those of the former, an association between breastfeeding and mortality will be observed, but it reflects a causal process that reverses the causal direction of the relations: it is ill-health that affects lactation, not the other way around.

This simultaneity problem is not easily corrected. One possibility is to estimate models that predict mortality above age 0, that is at ages where the effects of pre-existing health are less likely to overwhelm breastfeeding choices. The other solution is to control for measures of health at birth. Although, due to lack of adequate information, in our research we use only the first strategy, we rely on results from other research that seem to indicate that both strategies are equally efficient even if used in isolation from each other (Kuate Defo and Palloni, 1992).

3.1.3. The effects of birth intervals on early child mortality.

At the outset we need to distinguish between the effects of the preceding birth interval, or the birth-to-conception interval that is closed by the conception of the index child, and the effects of the following conception, or the birth-to-conception interval that is closed by the conception of another child following the index child. Although the bulk of the literature is devoted to the former, both are important and in some cases at least, the effects of the latter overwhelm those of the former (Palloni and Millman, 1986; Palloni and Tienda, 1986). We begin by investigating the nature of the effects of preceding birth interval.

Results obtained in a number of surveys in developed and developing countries have repeatedly confirmed a relation between preceding birth interval and infant

mortality. Studies conducted in the United States (Wray, 1971) and Great Britain (Federick and Adelstein, 1973), showed higher mortality among children conceived shortly following a birth. Surveys carried out in Punjabi villages in India, in East and West Africa, and in some countries in Latin America (Colombia and Ecuador) confirmed this relation (Wyon and Gordon, 1962; Wolfers and Scrimshaw, 1975; Wray and Aguirre, 1969). Cross-national and single country studies carried out with 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 mortality (Hobcraft et al., 1983; Palloni and Millman, 1986; Cleland and Sathar, 1984; Lantz et al., 1992; Hobcraft, 1992; Pebley and Stupp, 1987; Pebley and Da Vanzo, 1988 Retherford et al., 1989; Boerma et al., 1991; Boerma and Bicego, 1992). But what are the mechanisms involved?

First, a short birth-to-conception interval can retard fetal growth, resulting in low birth weight and increased death risks due to endogenous causes (Federick and Adelstein, 1973; Eastman, 1944). It is important to note that, according to this hypothetical mechanism, a short length of gestation is itself a potential result of a short birth-to-conception interval. **Second,** short birth-to-conception intervals may impair endocrine function and exert deleterious effects on breastmilk production, reducing its supply for the child that closes the interval (the 'index' child). This mechanism is likely to have a cumulative impact and be stronger for births of higher parity. 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 more young children exerting pressure on the demand for material resources and maternal care, thus exacerbating competition. In addition, 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 (Aaby, 1988).

If preceding birth intervals of substandard length aggravate maternal depletion and derail the process of gestation, the effects on infant mortality should be important shortly after birth but subside thereafter. If, however, maternal depletion affects the supply of breastmilk, its effects ought to linger for as long as breastmilk's protective properties last. Finally, if the effects of preceding birth interval depend on competition and crowding, they should be enduring and 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 (Palloni and Millman, 1986; Palloni, 1986; Palloni and Tienda, 1986; Lantz et al., 1992; Kuate Defo and Palloni, 1992). This is indeed the case for Latin American countries, although in other areas of the world the magnitude of the effects are very significant also.

The mechanisms through which the timing of the following conception influences the mortality risks of the index child have been identified but never verified successfully (Wolfers and Scrimshaw, 1975; Wray and Aguirre, 1969; Wray, 1971; Gray, 1981; Palloni and Millman, 1986; Hobcraft et al., 1983). A new conception is likely to prompt termination of breastfeeding, if this 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 may be due to fatigue 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 (Harfouche, 1970). In either case, all that is needed for the mechanism to be activated is that a new conception take place. If, in addition, the pregnancy is successful and terminates in a birth another mechanism may begin to operate. This involves competition for resources and for maternal care, and increases in crowding.

We expect that the effects on mortality of the index child will be strongest before the sixth month if the mechanism through which the timing of a following conception affects mortality is lactation. In contrast, if the mechanism is competition or crowding we would 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 early during infancy.

3.1.4. Pitfalls in the estimation of effects of birth intervals.

As in the case of breastfeeding, the estimation of effects of birth intervals requires special caution to eliminate the possibility of artifacts, spuriousness, and simultaneity biases.

i) Artifacts and spuriousness.

The first interpretational problem of some significance is whether the observed effects of length of preceding interval are a result of an artifact induced by short gestation (Miller, 1989). Indeed, short gestation is a powerful predictor of mortality and since births of short gestation constitute an important fraction of very short birth intervals, they contribute to exaggeration of the association between birth intervals and infant mortality. However, in several studies that have addressed this issue in developing countries it has been found that the impact of prematurity on the observed

associations is small (Wolfers and Scrimshaw, 1975; Hobcraft et al., 1985; Pebley and Stupp, 1987). It is also very important to keep in mind that short gestation itself may be a result of short birth-to-conception intervals. To the extent that this is the case, a control for prematurity--the most obvious solution to the problem we describe--would lead to underestimates of the **gross** effects of birth intervals.

The **second** difficulty is that there exists the possibility that the association between birth intervals (preceding and following) and infant and early childhood mortality is due to the spurious association between birth spacing and mortality generated by characteristics that lead both to better spacing and better access and use of health care (Potter, 1988). This is a plausible conjecture in countries where contraception is indeed used for spacing purposes (as opposed to stopping) and where contraceptive failure ends up closing a long birth interval. It is by no means obvious that these two regularities are verified in developing countries. And even if they were, it is not at all clear that the same artifact could be producing the strong patterns of effects that we have observed when the provision of health care and family planning services varies so widely (Hobcraft, 1992).

A problem for the estimation of the effects of preceding birth intervals that is both more complex and less tractable is related to unmeasured family effects. In non-contraceptive societies the early death of the child born immediately before the index child could trigger another conception either through a volitional mechanism (the so-called replacement behavior) or through biological ones (the cessation of breastfeeding and the consequent resumption of ovulation). As some conditions increasing mortality risks are shared by children born in the same family unit, there will be an association between the survival status of the index child and the immediately preceding one. If

some or all of the common conditions are not controlled for, it would appear as if it is the short birth interval that increases the mortality risks. There are several solutions to this problem which are implemented in this research. Although none of them is ideal, they are all useful in protecting against serious biases .

ii) Simultaneity.

Finally, the estimates of the magnitude of the effects of a following conception are themselves subject to potential simultaneity biases. In fact, if couples attempt to replace a lost index child or if its death accelerates resumption of ovulation, the resulting short birth-to-conception interval is, naturally, the outcome of the death, not the reverse. In this case the length of the following-birth interval is endogenous, and failure to deal with the resulting simultaneity problem will lead to serious exaggeration of the estimated effects.

3.2. Formulation and Estimation of Alternative Models.

In this section we evaluate the nature of the models to be estimated and the type of strategies we have chosen to reduce or eliminate the problems discussed above.

3.2.1. Discrete models for mortality in early childhood.

We first formulate simple models for the odds of dying in the following age segments (in exact months): 0, 1-3, 3-6, 6-12, 12-24 and 24-60. The logistic models describe the logarithm of the conditional odds of dying in arbitrary age segments, (x_j,x_j+n_j) of length n_j as:

$$\ln ((Q(x_j,x_j+n_j)/ (1-Q(x_j, x_j+n_j))) = \beta(x_j)Z(x_j) \qquad (1)$$

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.

To simplify interpretation and to introduce a more flexible parameterization of effects, we categorize all the independent variables. The set of definitions for the independent variables appears in Appendix 2. Note that these variables include fixed and time-dependent covariates. Thus the variables representing mother's education, region of residence, mother's age at birth, birth order, length of preceding interval, survival of preceding child are fixed covariates, whereas the variable for breastfeeding 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 BF=1 for the age segment 3-6 and BF=0 for segments 6-12, 12-24 and 24-60 if he is weaned before the 6th month of life.

Estimates were obtained for the sample of all children born 1-5 years before the survey was carried out, since it is only for these children that information on breastfeeding is available. Table 1 displays information about the frequency of observations, deaths and censored cases for each of the age segments considered here. Table 2 (panel *A*) displays 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.

3.2.2. *Analysis of results.*

The **first** salient feature in Table 2 is that the effects of breastfeeding are consistently negative, strong and statistically significant at least until the sixth month,

after which they are considerably diluted. The **second** feature is that the effects of following conception are very strong and in the expected direction in the segment 3-5 but not thereafter when the coefficient becomes statistically insignificant. The **third** feature is that the effects of mother's age, birth order, preceding birth interval and survival of previous child are somewhat erratic and rarely statistically significant although they are always properly signed. Only mother's age appears 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 WFS (Palloni and Millman, 1986). Both data sets produce important effects of breastfeeding and of following conception and neither one uncovers noticeable effects of mother's age and birth order. The discrepancies between Mexico DHS and Mexico WFS are in the effects of preceding birth intervals and of survival of previous child. Both variables were of some relevance in WFS but are of negligible significance in DHS. In order to evaluate whether the discrepancies are due to the smaller DHS sample, we have re-estimated our models using a longer window of time, and instead of including in the sample only children born within the 1-5 years window defined before, we have included all those born between 1 and 10 years before the survey.[3]

The new estimates appearing in Table 3 (Panel *A*) should be examined with a view to identifying discrepancies in the estimated effects of all variables excluding breastfeeding. With some differences to be highlighted shortly, the main features of this table are virtually identical to those in Table 2. First, the effects of mother's age, length of preceding birth interval, and birth order though properly signed are statistically insignificant. In addition to being significant in the age segments 1-2 and 12-23, mother's age attains significant in the age segment 6-11. Second, the effects of survival of

previous child are only relevant during the first age segment but not thereafter. Finally, the effects of following conception remain relatively large and statistically significant up to the end of the first year of life. In summary, one could argue that the smaller sample provides conservative estimates of the effects of a following conception and to some extent of the estimates of mother's age. The rest of the estimates are no different from those derived from the larger sample.

An alternative strategy to increase the stability of the estimates is to consolidate the age segments into wider ones, with a view to including a larger number of events. The estimates appearing in Table 4 (panel *A*) correspond to the consolidation of the second and third age segments (1-5) and the fifth and sixth age segments (12-59). Note that hardly any of the inferences drawn from Table 2 are altered. In particular, the effects of breastfeeding are properly signed and statistically significant before the sixth month, the effects of following conception behave 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 age of mother at birth) are of lesser importance. Here again, the one exception to this regularity is the effects of age of mother on mortality risks for the age segment 12-59, which turn out to be negative and statistically significant. In all likelihood, the modified estimates for this age segment reflect the effects within the age segment 12-23 where we already observed the same behavior (see Table 2, panel *A*, columns 5 and 6).

3.2.3. The magnitude of breastfeeding effects and some alternative interpretations.

How powerful are the effects of breastfeeding?

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 age segment 1-2 the relative risk of a child who never breastfed or who was weaned before the first month is about 13.1 times higher than the risk of a child who breastfeeds longer than 1 month; the relative risk in the age segment 3-5 is about 5.8 times higher and the relative risk in the age segment 6-11 is 3.2 times higher. These relative risks are close but not identical to those obtained with WFS data (which are, respectively, 2.8, 7.9 and 2.8).

From a policy point of view relative risks of this magnitude would be unimportant if the prevalence of traditional lactational practices is pervasive. But this is not the case in the case of Mexico. In the DHS sample about 22 percent of the children ever born are never breastfed, 29 percent of those surviving to the first month are weaned before the first month, 45 percent of those surviving to the third month are weaned before the third month, 57 percent of those surviving to the 6th month are weaned before attaining their sixth month of life and, finally, fully 79 percent are weaned before their first birthday. How much would infant mortality decline if the norm of breastfeeding 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:

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

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 breastfeeding 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 values of ρ_j are .79, .72 and .65.[4] Finally, the values of μ_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 percent. Since infant mortality in the sample of children born between 0 and 5 years before the survey is close to .058, the expected level of infant mortality after the change in lactation practices will be about 68 percent of the observed one (.0391). It is important to emphasize that this is a **lower** bound for the expected reduction since we have assumed that the changes in lactation have no impact whatsoever on mortality during the first month of life.[5]

The argument could be made that the estimates in Tables 2 (and 3 and 4) are contaminated by heaping of both breastfeeding durations and ages at death. Indeed, as revealed by Figure 1, the data do contain a fair amount of heaping in at least one crucial variable, namely, duration of breastfeeding. To minimize the impact of heaping around preferred digits we redefine the age segments in such a way that they are centered around the preferred digits. Only perverse heaping (a very high variance or thick-tailed distribution of the deviations between observed and true durations) would render this solution ineffective. The results are displayed in Table 5 (panel *A*). To be sure, the estimates of the effects of breastfeeding have been smoothed since the age segments overlap with those used before. As a consequence the estimates in Table 5 are different not just because we reduce the impact of heaping but also because they reflect the **changing effects** of breastfeeding and of following conception during the first year of life. One way of purging for the latter 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 percent of the observed level, exactly the same figure obtained before. We conclude that correction for heaping has no effects on our estimates.

3.2.4. The strength of the effects of following conceptions and alternative interpretations.

How powerful are the effects of a short time interval between the birth of the index child and the following conception?

The results displayed in tables 2 through 5 (panel *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 the tables, panel *B*, point to another conclusion: although after introduction of a control for breastfeeding the effects of a following conception are attenuated, they do not disappear and remain properly signed and statistically significant in the age segments where they were originally so. This result is not unlike those obtained elsewhere in Latin America (Palloni and Millman, 1986; Palloni and Tienda, 1986) and 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 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. However, a qualification is necessary since Table 3 does support the idea that effects could remain important during the age segment 6-12 where there is a small but nevertheless non-zero probability of competition between the index child and the following birth. But when

the feasibility of this alternative is examined in more detail (see below, section 3.2.5) we must conclude that there is no basis to suspect that competition with a new sibling explains the effects we observe. 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 are the mechanisms producing it? One possibility is that breastfeeding 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 breastfeeding duration only (but not intensity). If this were the case, the effects of following conception would remain strong even after controlling for breastfeeding. An alternative 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 breastfeeding. But this is an untestable proposition with the information available to us.

To evaluate the magnitude of the effects associated with a following conception we proceed to calculate population attributable risks for the age interval 0-11. The expression we use for these calculations is exactly analogous to (3) above except for two modifications. The first is that the relative risk must refer now to the ratio of mortality of children whose mother has a conception before some age x to the mortality of children whose mother does not experience such a conception. The second is that the behavior to be suppressed is not weaning, but a rapid conception following the birth of the index child. The relative risks in age segments 1-2, 3-5 and 6-11 are 1.5, 6.1 and 1.25 respectively, whereas the prevalence of the behavior is .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

exceed 11 months to yield an expected infant mortality rate of about .0543, or only about 6 percent less than observed. The reason for the low magnitude of the effects is, obviously, the very low prevalence of very short birth intervals.

3.2.5. Do effects depend on the timing of the relevant event?

One of the hypotheses we formulated before was that the effects of either cessation of breastfeeding or the occurrence of a following conception may depend on the timing of the event. Thus, for example, the effects of cessation of breastfeeding are stronger immediately after weaning and that they taper off and disappear thereafter. This could occur as a result of pure selection also: children who are more frail and more vulnerable experience a sharper increase of mortality soon after weaning occurs. This changes the average level of frailty among those children who are exposed (who were weaned at the same time as the less frail). The consequence is to compress the excess mortality of children who stopped breastfeeding relative to those who are still lactating. A similar argument applies to the effects of a following conception.

In addition, the timing of the following conception may have different impact according to whether or not the conception ends in a living birth which survives. Thus, for example, while a conception may have negative effects as a result of cessation of breastfeeding, its deleterious impact may be further enhanced by the birth of a child who competes for maternal care and other resources.

To test these conjectures we estimated models utilizing dummy variables for breastfeeding that took into account the timing of the pertinent event (cessation of lactation). New dummy variables for the presence of a following conception enabled us not only to consider the timing of the event (following conception) but also to account for the occurrence of a live birth and for its survival within the first year of life of the

index child. The definitions of the variables and the results obtained from the restricted and unrestricted sample of children are displayed in Table 6 (panels *A* and *B*). If the conjecture regarding the effects of breastfeeding were accurate, the difference between the estimates associated with the variables Bf1, Bf2, Bf3, Bf4 and Bf5 should be statistically significant. A cursory examination of the table reveals that this is not the case and that indeed it suffices to estimate a single parameter (with possible interactions with time) to completely account for the effects of lactation.

The conjectures regarding the effect of timing of the following conception receive mixed support. In fact, examination of panel *B* shows that occurrence of a following conception has an immediate effect on mortality and that these effects are reduced as time goes by. Thus, for example, in the age segment 6-11, the variable FC2 has a statistically significant effect but not FC1. But in the age segment 3-5, just the opposite occurs. Furthermore, the **differences** between the regression coefficients are not statistically significant. Thus, the data do not lend support to the idea that either the timing of cessation of breastfeeding or of the following conception makes any difference above and beyond the main effects described before.

3.3. Continuous time models of mortality risks during the first two 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 $_0(x)$ 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 are identical and when the model is for age segments within which the hazard is small and

index child. The definitions of the variables and the results obtained from the restricted and unrestricted sample of children are displayed in Table 6 (panels *A* and *B*). If the conjecture regarding the effects of breastfeeding were accurate, the difference between the estimates associated with the variables Bf1, Bf2, Bf3, Bf4 and Bf5 should be statistically significant. A cursory examination of the table reveals that this is not the case and that indeed it suffices to estimate a single parameter (with possible interactions with time) to completely account for the effects of lactation.

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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 one 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 somewhat cumbersome as a tool to handle 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 flexibly retrieve estimated effects **specific** for each age segment, but requires that we break down the age-span of interest 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 7 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 breastfeeding are large and negative but are probably highly influenced 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 breastfeeding and survival status during the first month, we estimate a model for the age segment 1-12 months (exactly). The results appear in column 5. The

magnitude of the breastfeeding effects is cut in half but the coefficient remains properly signed and statistically significant. To circumvent the same problem, other authors (Miller et al., 1992) have used a more conservative but also riskier solution. It consists of recoding breastfeeding so that it equals 0 (completed months) for all children who died during the first month and who never breastfed. The resulting estimates after recoding appear in column 2 of the same table. A comparison of the estimated effects of breastfeeding in columns 2 and 5 reveal that the recoding strategy unduly contaminates the estimated effects that apply to ages beyond the first month.

Columns 3 and 4 display estimated coefficient for models where we have recoded the duration of breastfeeding to circumvent a reporting problem of some importance. As shown in Table 1, a significant fraction of children who die have reported duration of breastfeeding equal to their age at death. If this were a reflection of actual practice, our logistic and hazard model estimates would be unbiased. But if the duration of breastfeeding were rounded to equal the ages at death, our estimates could contain some downward bias. This is a serious problem in DHS for African countries and exerts a powerful influence on results (Palloni, Ubomba-Jaswa and Gaigbe-Togbe, 1992). In order to approximately gauge the magnitude of this bias, we reestimated models 1 and 2 after reducing by one month the duration of breastfeeding for dead children whose reported duration equals their age at death. The only exception to the recoding rule is that no change was implemented if the duration of breastfeeding was equal to 3, 6 or 12. The exception is useful to ensure that **logistic** models applied to the recoded data would yield exactly the same results obtained before. In other words, if our recoding accurately redefines durations, it should in no way alter results from the logistic models or alter conclusions drawn from them.[6] Column 3 should be compared with column 1 and

column 4 with column 2. A cursory look at the results shows that the recoding makes little difference and that results are insensitive to the feature revealed by Table 1.

In sum, the effects of breastfeeding retrieved by our hazard model are large and statistically significant within the first year of life but also between the first and 12th month of life. The latter effect 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 following conception are insignificant. This is not surprising in view of the results from the logistic model. These suggest that the effects of following conception are statistically significant only during the first sixth month 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 non significant effect. The estimates of other variables do not lead to conclusions that are different from those we obtained from the logistic models.

4. Can Family Planning Save Lives?

The results that we presented above do, in principle at least, support the idea that better spacing of children must necessarily lead to higher probabilities of surviving. Just consider, for example, the **expected reduction** in infant mortality that would result from the elimination of following conceptions occurring prior to the first year of life of the index child. Provided that one takes this scenario as a simple counterfactual--with all the caveats that counterfactuals 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 intervals. Indeed, even though the effects of preceding interval were, for the most part, statistically insignificant, they do exert an influence on infant mortality

in the expected direction since the coefficients are properly signed.

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. For reasons that we explore below this inference is a **non sequitur.**

First, reproductive control for the purpose of stopping childbearing--an important and in some cases the only component of family planning programs--should have only marginal effects on survival if any at all. Early stopping will alter favorably the distribution of births by age of mother at birth and by parity and should also reduce the proportion of all children who are rapidly followed by another conception. However, the effects of higher parity and older age of mother at birth were shown to be somewhat weak, and a slight improvement in the distribution of births by age and parity will induce only minor changes in mortality. And although the effects of a poorly spaced following conception are very strong, the decrease in the proportion of following conceptions induced by stopping should be very small since the bulk of poor spacing occurs early in the reproductive career of a woman, not at the end. To the extent that family planning 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 for stopping will not occur without affecting breastfeeding 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: women who practice birth control are significantly less likely to breastfeed for long periods or to breastfeed at all (Millman, 1985; Palloni and Kephart, 1989; Gomez de Leon and Potter, 1989).

Whatever the mechanism producing the relation, it should follow from it 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 mortality. Furthermore, note that the reduction of breastfeeding should naturally lead, **ceteris paribus**, to a shortening of birth intervals and will thus offset the direct effects of increased practice of birth control.

These considerations suggest that one cannot infer the ultimate effects of family planning on infant and child survival from the estimates obtained in Sections 3.2 and 3.3. The only solution to the problem is to formulate an **integrated** model 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 (Palloni and Kephart, 1989) that combines an arbitrary reproductive regime with an arbitrary mortality regime to assess the ultimate effects on mortality attributable to reproductive practices and breastfeeding. What we propose here is a simplified version of this model. In a nutshell, the simplified model consists of defining alternative birth distributions according to birth order, mother's age at birth, length of preceding interval, survival of preceding child, and timing of following conception. We then apply to each of the resulting cells a distribution by length of breastfeeding. Each of the classes of births defined by the Cartesian product of the joint distribution of births and the distribution by length of breastfeeding is associated with levels of mortality in the age segments (in months) (x, x+n) contained within, say, the age intervals (in months) 0-11 or 0-23. The estimated level of infant mortality for a given cohort is calculated via a weighted projection that survives births from age 0 to age 12 (exactly) in each of the cells of the Cartesian product.

Let us assume that a reproductive regime of, say, type i is mixed with a pattern of lactation, say j. Let us also assume that mortality during the first year is broken down into n segments and that within each of these segments the integrated force of mortality is given by $\varepsilon_x(i*j)$ for x=1,n. In this notation, i*j denotes the Cartesian product of the classification of births by birth order, age of mother at birth, length of preceding interval, timing of following conception, survival of preceding child and length of lactation. If these variables are dichotomized, we will have 2^6 classes for each of the age segments. Infant survival for those in a particular class, (i_0, j_0), depends on the value of $\varepsilon_x(i_0,j_0)$. The latter is in turn calculated according to the estimated effects retrieved in our models.

To implement the model we introduce some simplifications. First, we do not consider the effects of parity or of survival of the previous siblings, since none of the corresponding effects are relevant in our models. Second, we apply calculations to the average composition of the population by education of mother and area of residence rather than performing separate calculations or taking into account differentials in the effects of variables across educational levels and areas of residence. The errors that derive from these simplifications are trivial since the corresponding interaction effects are of little significance at least in the DHS sample. Third, the association between breastfeeding and pace of childbearing on the one hand, and the correlation between the length of birth intervals experienced by a given woman, on the other, are not empirically estimated. Instead we assume that a) any pattern of breastfeeding can be mixed with any pattern of reproduction, and b) following and preceding intervals are not necessarily related. As a result of these two simplifications we will not be able to produce a single value of infant mortality to characterize a scenario but a range of values. Fourth, we assume that all children are breastfed at birth and that the minimum duration of

breastfeeding (in completed months) is 0. This simplification enable us to set a lower bound for the effects of breastfeeding that is uncontaminated by the possible association between health status at birth and breastfeeding initiation at month 0. Finally, we distinguish 12 patterns of behaviors: two possible patterns for breastfeeding, three for spacing and two for timing. The following are the basic patterns of behavior:

a) traditional breastfeeding: the duration of breastfeeding is longer than 6 months;

b) modern breastfeeding: the duration of breastfeeding is 0 completed month;

c) short-short spacing of childbearing: the length of the preceding birth interval is less than 19 months and the following conception occurs before the third month of the index child;

d) short-long spacing of childbearing: the length of the preceding interval is less than 19 months and the following conception occurs before the sixth but after the third month of age of the index
child.

e) long spacing: the length of the preceding interval is between 19 and 36 months and the following conception occurs after the sixth month of life of the index child;

f) early fertility: the age of mother at birth is less than 20;

g) late fertility: the age of the mother at birth is greater than 20 but less than 30.

Mortality risks for the first year of life associated with each of the 12 patterns are displayed in Table 8. The effects of breastfeeding, age of mother, and spacing are easily calculated by taking differences between the various cells.[7]

To assess the contribution of the regime of breastfeeding and the reproductive pattern, consider the results displayed in Table 9. Take as a baseline the case when births are distributed evenly across cells 5 through 12 in Table 8, thus effectively

eliminating from consideration the extreme case of short spacing described in the first four cells of Table 8. The expected value of infant mortality would be about .0498, slightly below the observed value in Mexico DHS. If only the breastfeeding pattern is modified so that all births are breastfed at least 6 months, the expected level of infant mortality drops to .0279, a 44 percent reduction from the baseline level. If, instead of breastfeeding one were to change the spacing regime and the ages at birth so that shortly-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 percent relative to the baseline level. The most optimistic scenario, implying a traditional lactation pattern combined with optimal spacing and timing of births, would yield an expected value of infant mortality of about .0125, representing a 75 percent reduction relative to the baseline level.

But, as noted before, the overhaul of the reproductive regime caused by the introduction of modern family planning may come at a cost, namely, that women tend to abandon the pattern of traditional breastfeeding at the same time that they remove early and shortly spaced births. If all women alter their reproductive behavior and only half of the women continue to adhere to a traditional pattern of breastfeeding, the levels of infant mortality would settle in the neighborhood of .0233, about 53 percent less than the baseline levels. But if only a small fraction of women resisted changes in breastfeeding, the levels of infant mortality would get close to .0340, still lower than the baseline but relatively higher than most of the scenarios we have considered.

There are two policy-relevant inferences that can be drawn from these results. First, the magnitude of the effects of breastfeeding is high enough to offset more than 60 percent of the beneficial changes attributable to spacing and timing of births. Thus,

emphasis on the continuation of traditional breastfeeding practices should be incorporated in family planning programs. Second, the effects of spacing and timing of births alone are powerful enough to make possible reductions in infant mortality of around 20 to 40 percent from current levels.

These conclusions must be taken with caution, however. This is because while the estimated impact of breastfeeding may be a lower bound, those for spacing and timing are almost surely upper bounds. This is due to the fact that we have not accounted properly for the correlation of 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 spacing and timing of births. Thus, the seemingly ample room for changes that we estimate from Table 9 is deceiving if not properly qualified. It should be remembered that the possibilities for changes are even greater if we compare infant mortality levels among the poor and those who are better off, regardless of reproductive behavior. In this case the policy relevant conclusion should have invoked changes in the distribution of wealth rather than changes in reproductive regimes. The road toward reduced mortality is not one that involves only 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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1. It has been shown elsewhere that, at least in some areas of the world, the magnitude of the biases due to this source are likely to be inconsequential. See Lantz, Partin, and Palloni, 1992.

2. In a recent study, however, it has been shown that when a control for pre-existing illnesses is introduced, the estimates of the effects of breastfeeding change only slightly. See Kuate Defo and Palloni, 1992.

3. Breastfeeding information for children born between six and ten years before the survey is missing and was captured using a dummy variable for missing information.

4. 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 breastfeeding prior to the 12th month of life).

5. This assumption is made to safeguard against potential biases in the effects of breastfeeding for month 0. See discussion of potential biases in section 3.1.2(ii).

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

7. Note that the effects of breastfeeding are on the conservative side since we have assumed that all children are breastfed at birth.

Table 1: Basic Frequencies by Age Segments
(Sample of Children Born 1-5 Years Before Survey)

	Age Segments					
	0	**1-2**	**3-5**	**6-11**	**12-23**	**24-59**
Exposed	2665	2578	2563	2533	2516	2101
Deaths	87	15	30	17	20	10
Censored	--	--	--	--	395	2027
Children whose age at death equals breastfeeding duration	19	7	10	6	6	1

Table 2. Panel A: Estimated Effects of Using a Logit Model and Conventional Age Segments[a]

	Age Segments					
	0	1-2	3-5	6-11	12-23	24-59
Age (2)	- .14 (.38)	-1.43 (.73)*	-.05 (.61)	-1.32 (1.05)	-1.90 (.65)*	- .88 (1.34)
Age (3)	- .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 (2)	- .45 (.45)	6.10 (8.96)	- .32 (.70)	.31 (1.59)	-.37 (1.24)	5.63 (10.30)
BO (3)	.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	2654	2566	2551	2521	2504	2089
Cases	2665	2578	2563	2533	2516	2101

[a]Standard errors in parentheses
*Statistically significant at $p < .01$

Table 2. Panel B: Effects of Following Conception Before and After Controlling for Breastfeeding[b]

	Age Segment				
	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)

[b]Standard errors in parentheses
*Statistically significant at $p < .01$

Table 3, Panel A: Estimated Effects of Using a Logit Model and Conventional Age Segments: Sample of Births Born 1-10 Years Before Survey[a]

	Age Segments					
	0	1-2	3-5	6-11	12-23	24-59
Age (2)	- .14 (.23)	- .33 (.43)	- .61 (.38)	-.88 (.37)*	-1.01 (.42)*	- .48 (.64)
Age (3)	- .38 (.27)	- .40 (.51)	- .46 (.44)	-.98 (.44)*	-1.33 (.49)*	- .04 (.69)
Bint (1)	.11 (.23)	1.37 (.72)*	.27 (.41)	.80 (.54)	.56 (.49)	.71 (.75)
Bint (2)	- .50 (.28)	.61 (.81)	- .68 (.53)	.64 (.57)	.51 (.53)	.89 (.77)
BO (2)	- .33 (.28)	.36 (.79)	- .18 (.49)	.16 (.63)	-.53 (.65)	.20 (.93)
BO (3)	.21 (.18)	.37 (.40)	.12 (.33)	.32 (.32)	.76 (.33)*	.07 (.47)
S	.56 (.27)*	- .17 (.73)	.24 (.53)	-.27 (.60)	.21 (.53)	- .73 (1.02)
Bf	-2.77 (.28)*	2.39 (.65)*	-1.60 (.45)	-.46 (.52)	-.59 (.64)	-1.26 (1.10)
FC	--	.71 (.74)	1.50 (.34)	.93 (.30)	.37 (.29)	.33 (.39)
-2 log likelihood	1671	522	674	688	619	354
Degrees of freedom	6693	6490	6443	6379	6314	5862
Cases	6705	6503	6456	6392	6326	5875

[a]Standard error in parentheses
*Statistically significant at $p < .01$

Table 3, Panel B: Effects of Following Conception Before and After Controlling for Breastfeeding: Sample of Births Born 1-10 Years Before Survey[b]

	Age Segment				
	1-2	3-5	6-11	12-23	24-59
Before control	.79 (.73)	1.57 (.34)*	.96 (.29)*	.40 (.29)	.25 (.39)
After control	.71 (.74)	1.50 (.34)*	.93 (.30)*	.37 (.29)	.33 (.39)

[b]Standard error in parentheses
*Statistically significant at $p < .01$

Table 4, Panel A: Estimated Effects of Using a Logit Model and Consolidated Age Segments[a]

	Age Segments		
	1-5	6-11	12-59
Age (2)	- .50 (.44)	-1.32 (1.05)	-1.77 (.59)*
Age (3)	- .64 (.54)	-1.98 (1.17)	-1.59 (.67)*
Bint (1)	.63 (.56)	1.00 (1.07)	1.44 (1.04)
Bint (2)	- .23 (.66)	.93 (1.11)	1.96 (1.04)
BO (2)	.27 (.64)	.31 (1.59)	.34 (1.18)
BO (3)	.22 (.41)	1.75 (.81)*	.68 (.51)
S	- .08 (.76)	-6.07 (10.5)	.11 (.75)
Bf	-1.49 (.34)*	-1.16 (.53)*	- .33 (.49)
FC	1.33 (.53)*	.22 (.67)	.48 (.39)
-2 log likelihood	397	162	289
Degrees of freedom	1866	2521	2505
Cases	2578	2533	2516

[a]Standard error in parentheses
*Statistically significant at $p < .01$

Table 4, Panel B: Effects of Following Conception Before and After Controlling for Breastfeeding[b]

	Age Segment		
	1-5	6-11	12-59
Before control	1.90 (.51)*	.54 (.65)	.55 (.39)
After control	1.33 (.53)*	.22 (.67)	.48 (.39)

[b]Standard errors in parentheses
*Statistically significant at $p < .01$

Table 5, Panel A: Estimated Effects of Using a Logit Model and Conventional Age Segments[a]

	Age Segments			
	1-4	3-7	8-14	15-59
Age (2)	- .90 (.48)*	- .024(.60)	-2.16 (.76)*	-1.27 (.80)
Age (3)	- .77 (.57)	- .49 (.70)	-2.63 (.88)*	- .72 (.86)
Bint (1)	.30 (.58)	.33 (.57)	1.00 (1.06)	1.05 (1.06)
Bint (2)	- .46 (.70)	- .67 (.72)	1.32 (1.08)	1.38 (1.07)
BO (2)	- .01 (.68)	- .25 (.69)	- .13 (1.28)	.01 (1.31)
BO (3)	.15 (.47)	.24 (.45)	1.34 (.69)*	.43 (.61)
S	.15 (.78)	.31 (.76)	-6.35 (10.56)	.64 (.78)
Bf	-1.88 (.37)*	-1.38 (.40)*	-1.125 (.48)*	.10 (.55)
FC	.89 (.57)	1.72 (.43)*	.48 (.48)	.26 (.48)
Log likelihood	335	314	216	205
Degrees of freedom	2566	2551	2518	2409
Observations	2378	2563	2530	2421

[a]Standard error in parentheses
*Statistically significant at $p < .01$

Table 5, Panel B: Effects of Following Conception Before and After Controlling for Breastfeeding[b]

	Age Segment			
	1-4	3-7	8-14	13-23
Before control	1.39 (.55)*	1.98 (.42)*	.82 (.45)	.24 (.47)
After control	.89 (.57)	1.72 (.43)*	.48 (.48)	.26 (.48)

[b]Standard error in parentheses
*Statistically siginificant at $p < .01$

Table 6: Effects of Breastfeeding and Following Conception With Dependence on the Timing of Cessation of Lactation and Following Conception[a]

Panel A: Sample of children born 1-5 years before survey

	Age Segment			
	3-5	6-11	12-23	24-59
Bf1	1.21 (.45)*	.91 (.70)	.55 (.64)	-5.83 (8.4)
Bf2	1.88 (.60)	.99 (.81)	1.18 (.65)	.90 (.92)
Bf3	--	.58 (1.10)	-5.21 (3.38)	.58 (1.15)
Bf4	--	--	.28 (.80)	-6.44 (11.2)
Bf5	--	--	--	-6.59 (9.7)
FC1	2.49 (.60)*	-6.43 (16.4)	-5.35 (26.0)	-4.98 (44.0)
FC2	1.69 (.58)*	.84 (.68)	- .10 (.78)	.48 (.92)
FC3	--	--	.30 (.55)	-6.55 (55.9)
FC4	--	--	--	- .31 (.90)
FC5	—	—	—	-1.20 (1.11)

[a] Standard error in parentheses
* Statistically significant at $p < .01$

Panel B: Sample of children born 1-10 years before survey

	Age Segments[b,c]			
	3-5	6-11	12-23	24-59
Bf1	1.62 (.51)*	.13 (.67)	.45 (.61)	-5.84 (9.41)
Bf2	1.42 (.45)*	.33 (.79)	1.00 (.62)	.92 (.87)
Bf3	--	.29 (1.06)	-4.40 (5.86)	.79 (1.12)
Bf4	--	--	.02 (.79)	-6.22 (12.24)
Bf5	--	--	--	-6.70 (10.40)
FC1	1.16 (.44)*	.71 (.48)	-4.79 (12.9)	-6.28 (35.6)
FC2	.84 (.59)	1.07 (.34)*	- .28 (.53)	.38 (.58)
FC3	--	--	.51 (.33)	-6.15 (42.0)
FC4	--	--	--	- .13 (.65)
FC5	--	—	—	.20 (.42)

[b] Standard error in parentheses
[c] The effects of breastfeeding were obtained by including a dummy variable for missing cases to capture all those born 5 to 10 years before the survey
* Significant at $p < .01$

Table 7: Alternative Estimates of Effects from a Continuous Time Model for Survival During the Age Segment 0-12 months (exactly)[a]

	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 (2)	.55 (.38)	.44 (.38)	.55 (.38)	.44 (.38)	1.12 (.62)
Age (3)	.35 (.26)	.16 (.26)	.35 (.26)	.16 (.26)	.50 (.42)
Bint (2)	.61 (.35)	.68 (.35)	.62 (.35)	.68 (.35)*	.73 (.57)
Bint (3)	- .06 (.39)	- .06 (.39)	- .01 (.39)	- .05 (.39)	.15 (.68)
BO (2)	- .11 (.42)	.04 (.42)	- .11 (.42)	.04 (.42)	.28 (.67)
BO (3)	.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	2528	2528	2528	2528	2458

Definitions of models:

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 and never breastfed, duration of breastfeeding was set equal to 0

Model 3: Defined for the age segment 0-12 (exactly). If duration of breastfeeding equals age at death then duration of breastfeeding was reduced by 1 month

Model 4: Defined for the age segment 0-12 (exactly). If child died at 0 and never breastfed, the duration of breastfeeding was set equal to 0. If duration of breastfeeding equals age at death then duration of breastfeeding was reduced by 1 month

Model 5: Defined for the age segment 1-12 (exactly).

All models control for education of mother and rural-urban residence

[a] Standard error in parentheses

* Statistically significant at $p < .01$

Table 8: Levels of Infant Mortality by Behavioral Patterns

Spacing	Timing of Fertility			
	Early Breastfeeding		Late Breastfeeding	
	Yes	No	Yes	No
Short	.074	.214	.044	.139
Short/Long	.049	.122	.029	.077
Long	.021	.053	.013	.034

Table 9: Expected levels of infant mortality under different scenarios

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Scenario I: Uniform Distribution of Births in Cells 5-12 of Table 8
Infant mortality = .0498

Scenario II: Universal Long Breastfeeding (no change in timing or spacing)
Infant mortality = .0279

Scenario III: Universal Late Births (no changes in spacing or breastfeeding)
Infant mortality = .0382

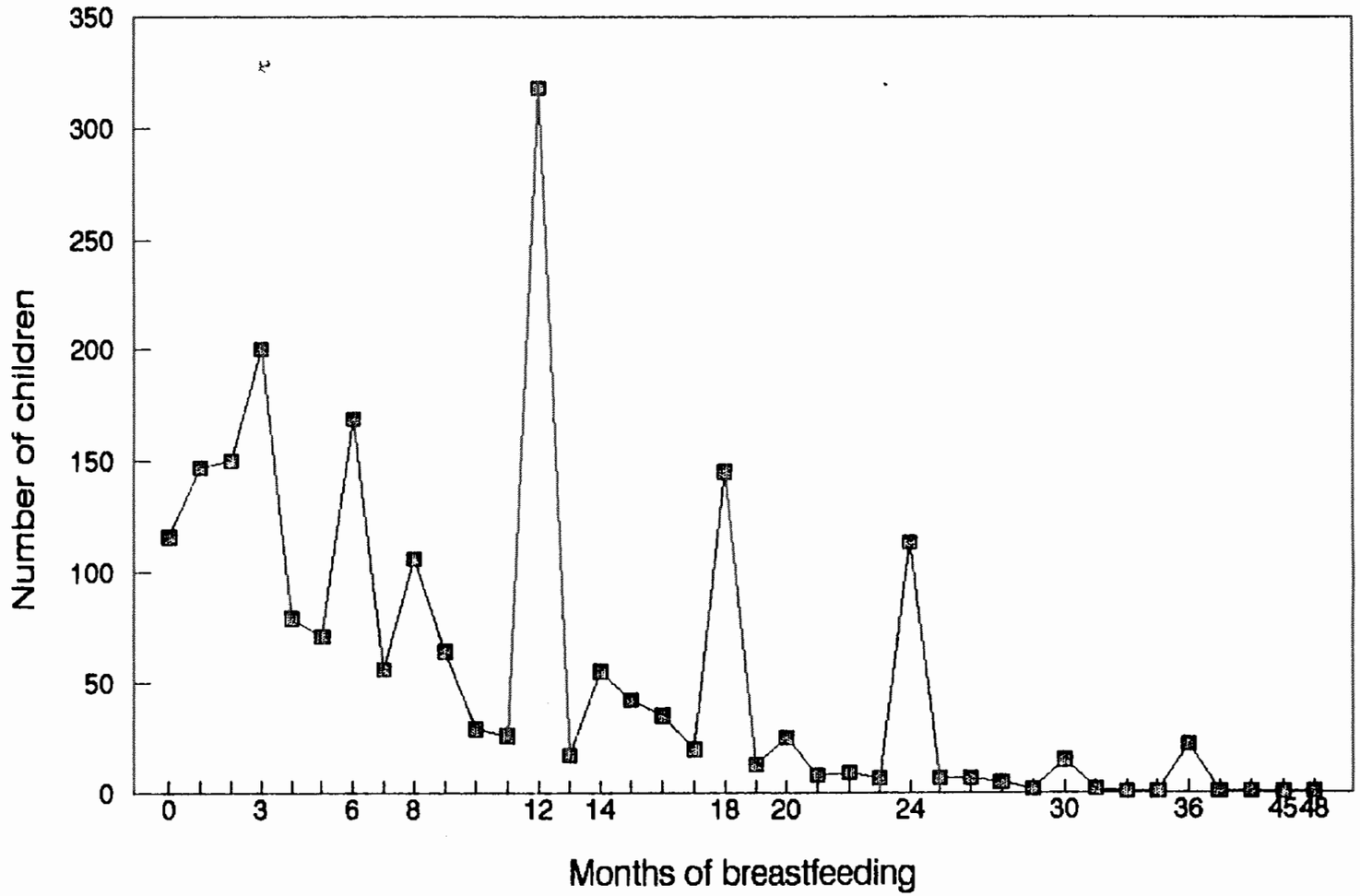
Scenario IV: Universal Long Spacing (no changes in timing or breastfeeding)
Infant mortality = .0301

Scenario V: Universal Long Spacing and Late Timing (no changes in breastfeeding)
Infant mortality = .0233

Scenario VI: Universal Long Spacing, Late Timing, Long Breastfeeding
Infant mortality = .0125

Figure 1. Distribution of children by duration of breastfeeding

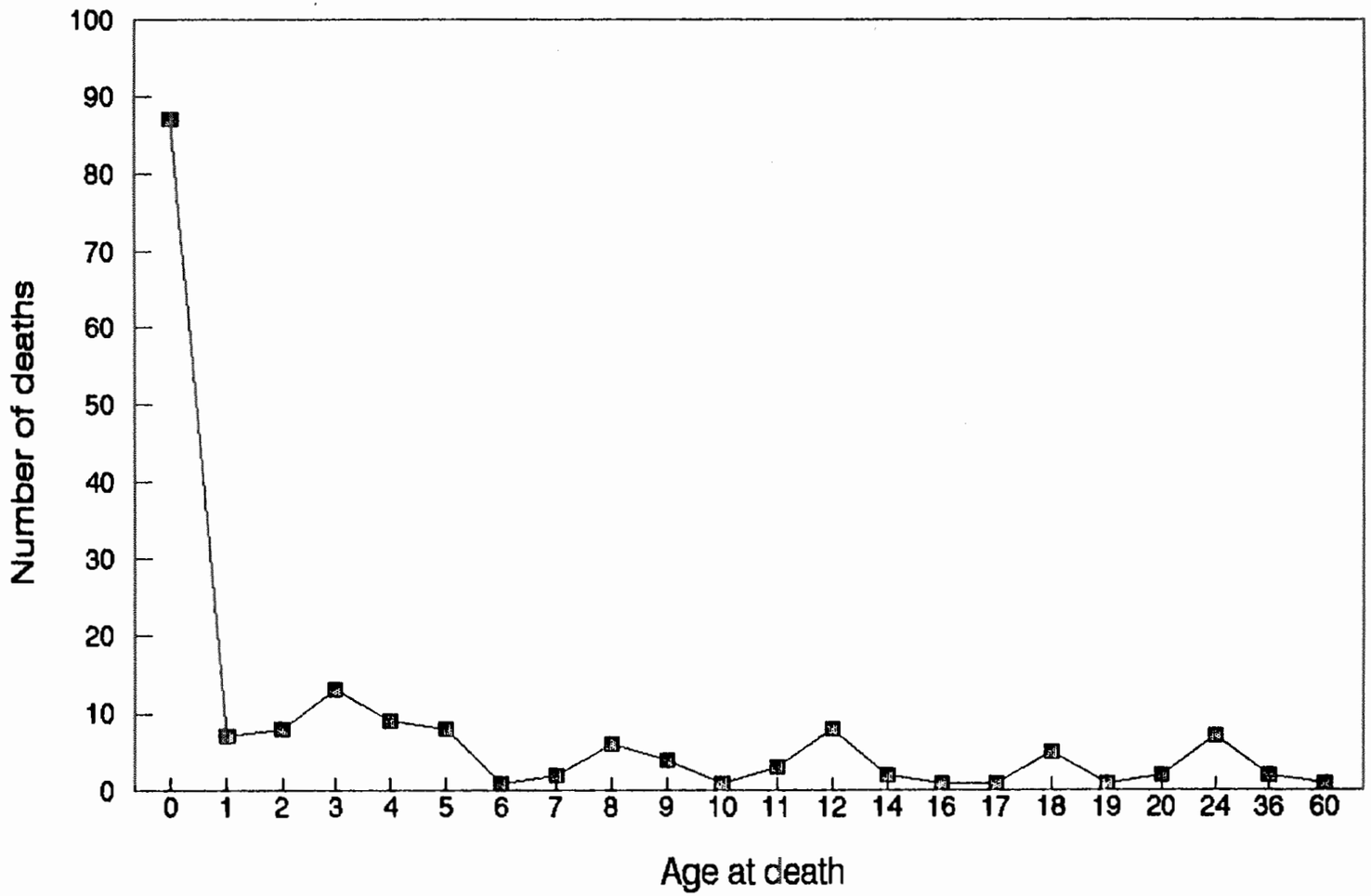
Mexico, Demographic and Health Survey 1987



Sample of children born 1 to 5 years before the survey

Figure 2. Distribution of children by age of death

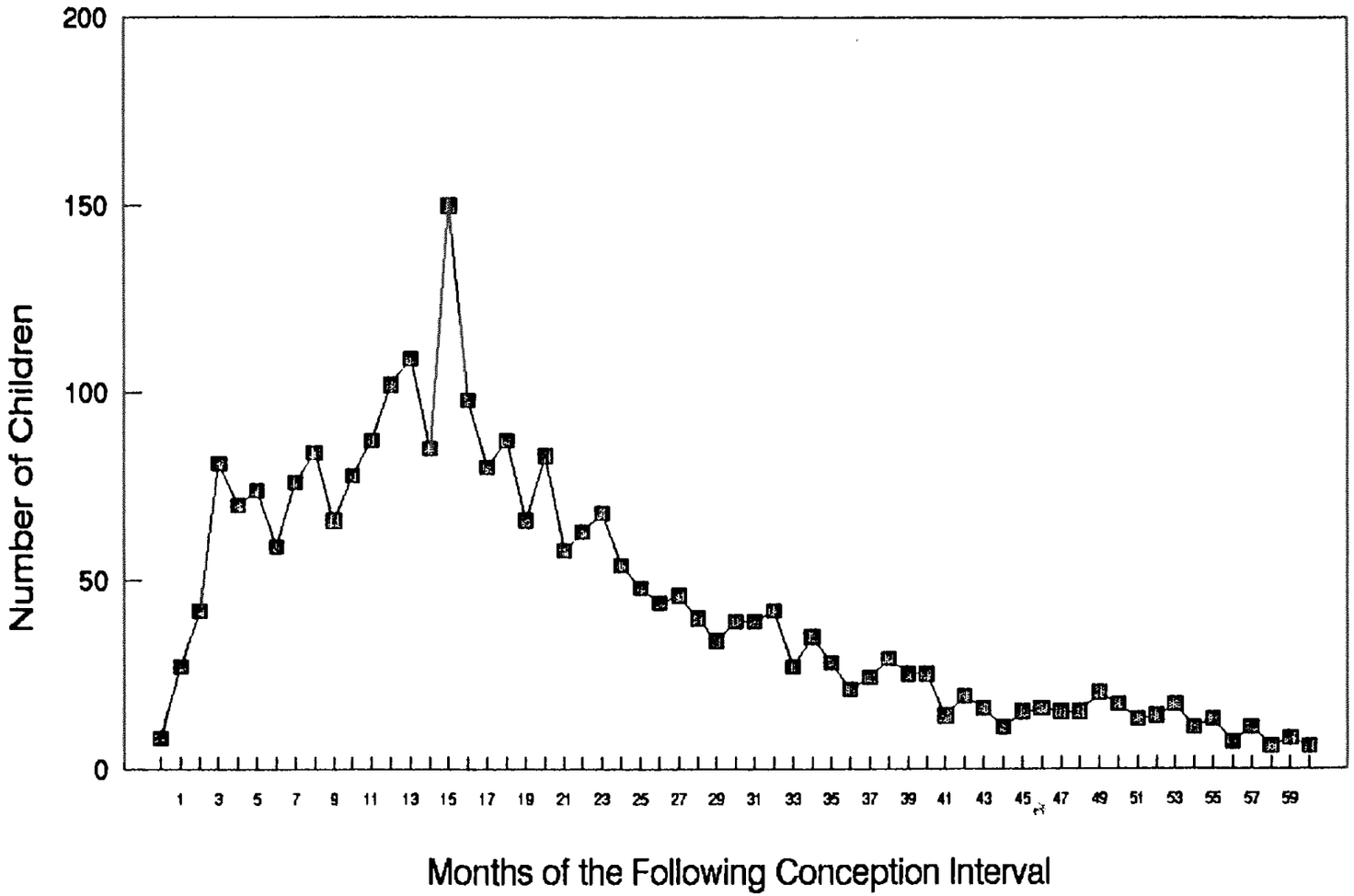
Mexico, Demographic and Health Survey 1987



Sample of children born 1 to 5 years before the survey

Figure 3. Distribution of Children by Following Conception Interval

Mexico, Demographic and Health Survey 1987

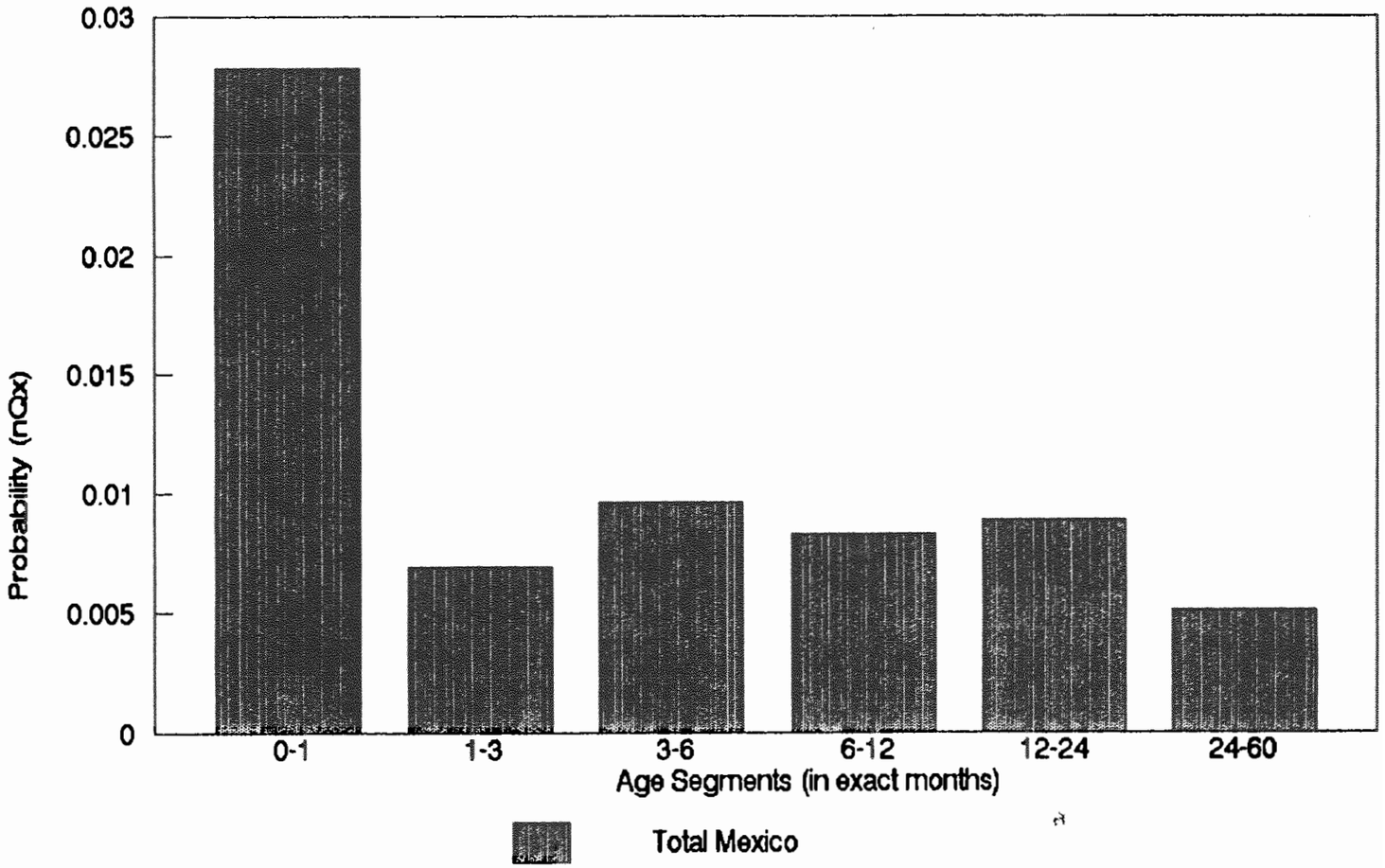


Sample of children born 1 to 5 years before the survey

Appendix 1

Figure A1. Conditional Probability of Dying

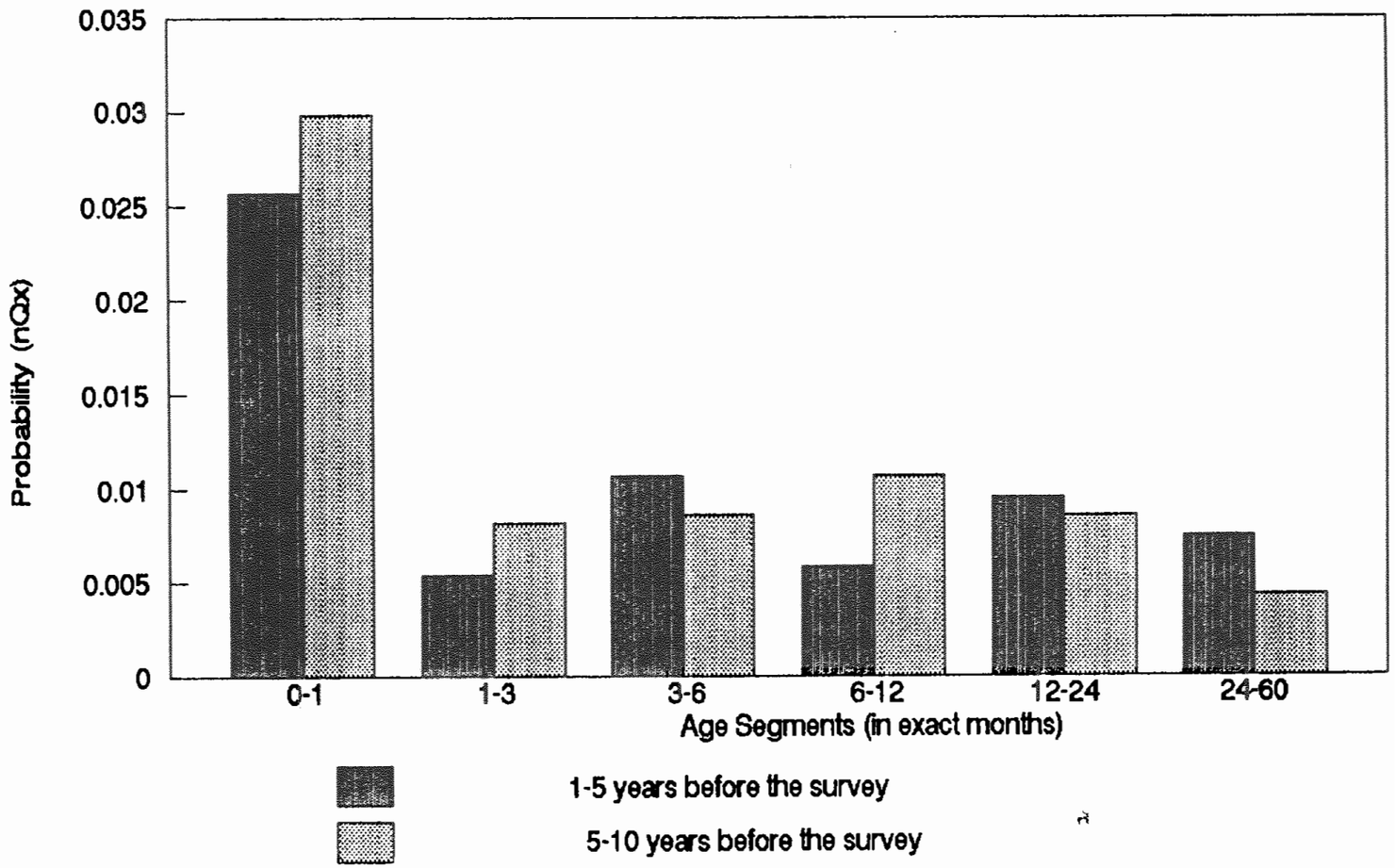
Mexico, Demographic and Health Survey, 1987



Sample of children born 1 to 10 years before the survey

Figure A2. Conditional Probability of Dying

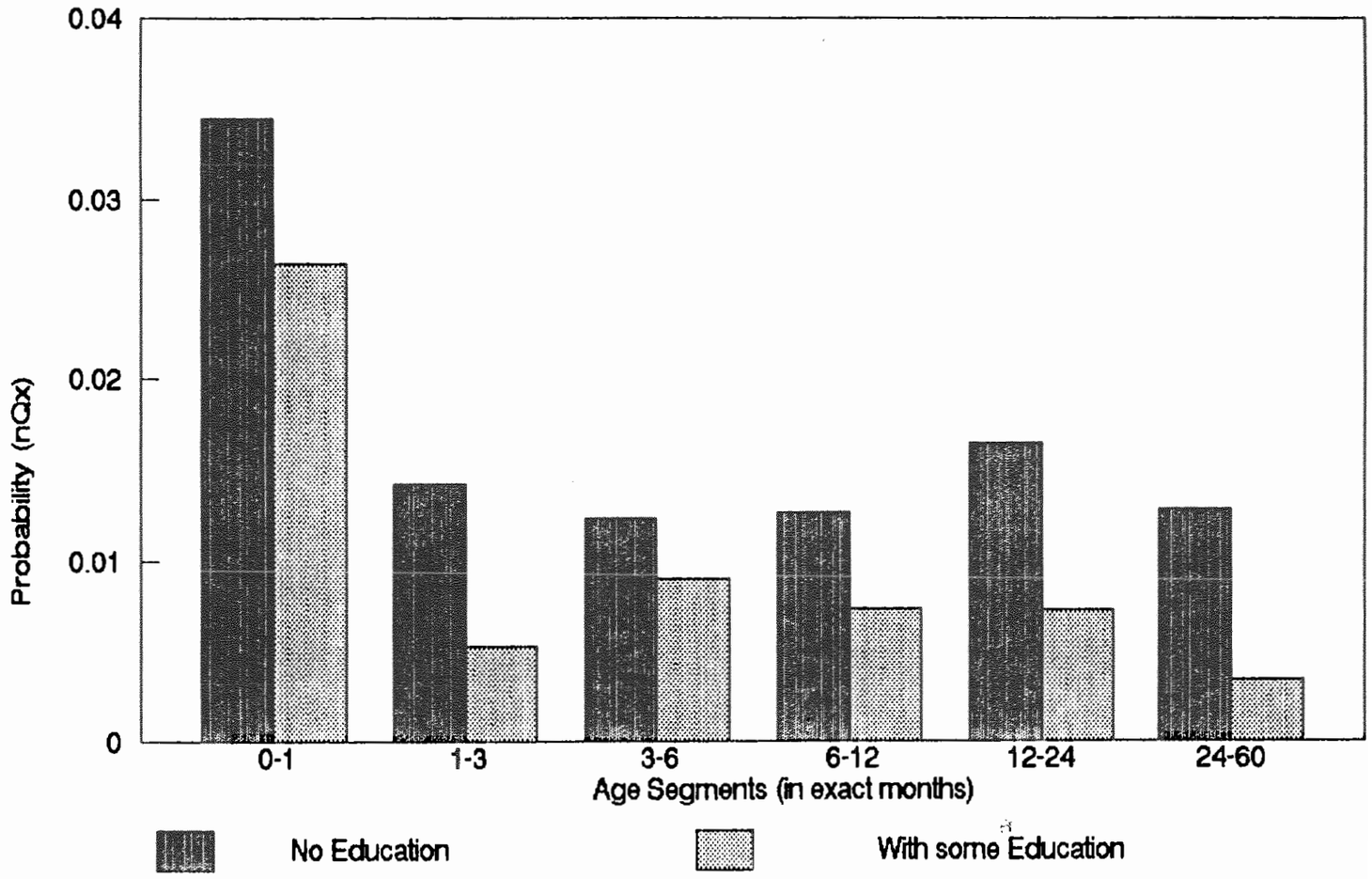
Mexico, Demographic and Health Survey, 1987



Sample of children born 1 to 10 years before the survey

Figure A3. Conditional Probability of Dying by Mother's Education

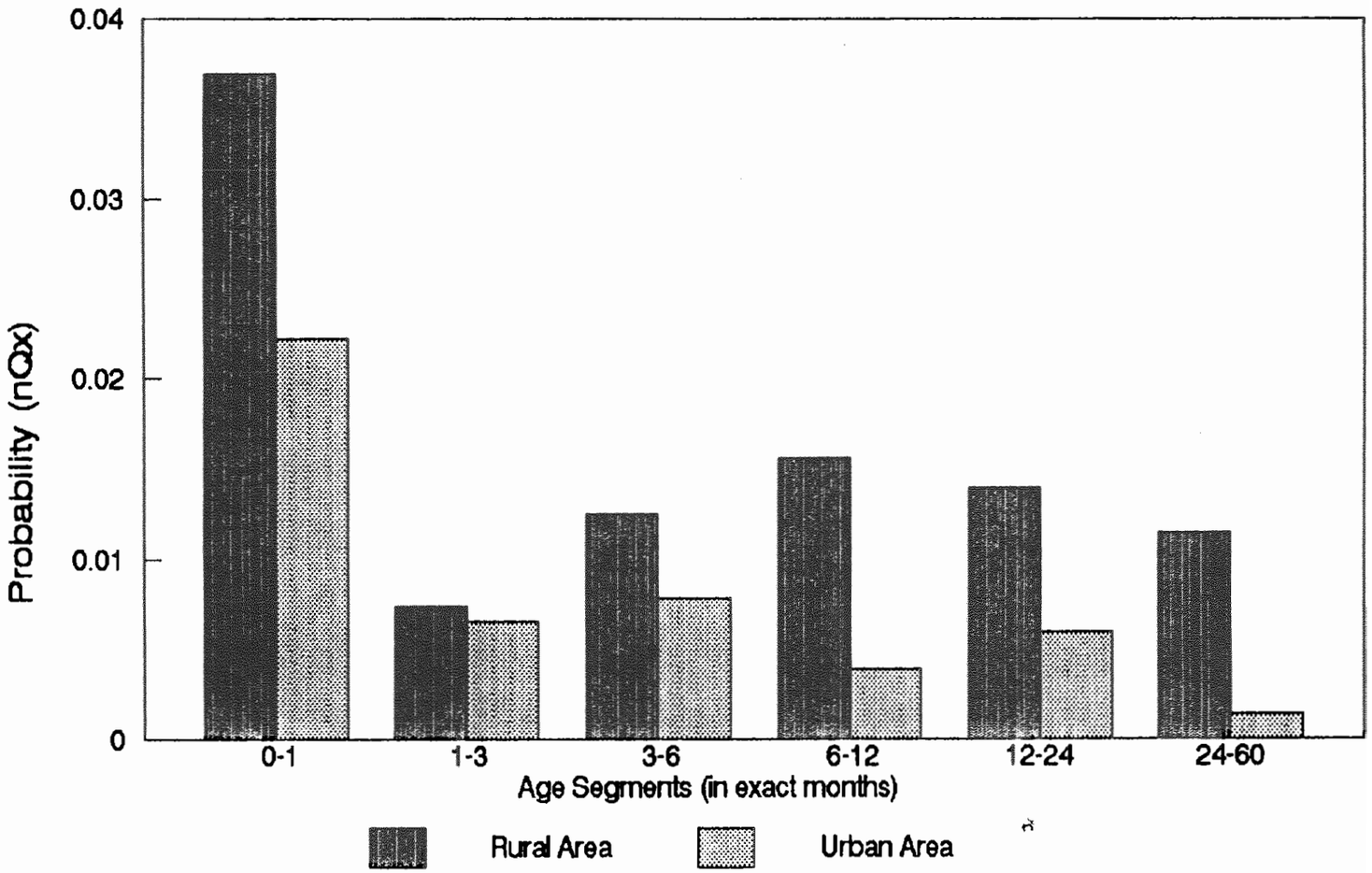
Mexico, Demographic and Health Survey, 1987



Sample of children born 1 to 10 years before the survey

Figure A4. Conditional Probability of Dying by Mother's Residence

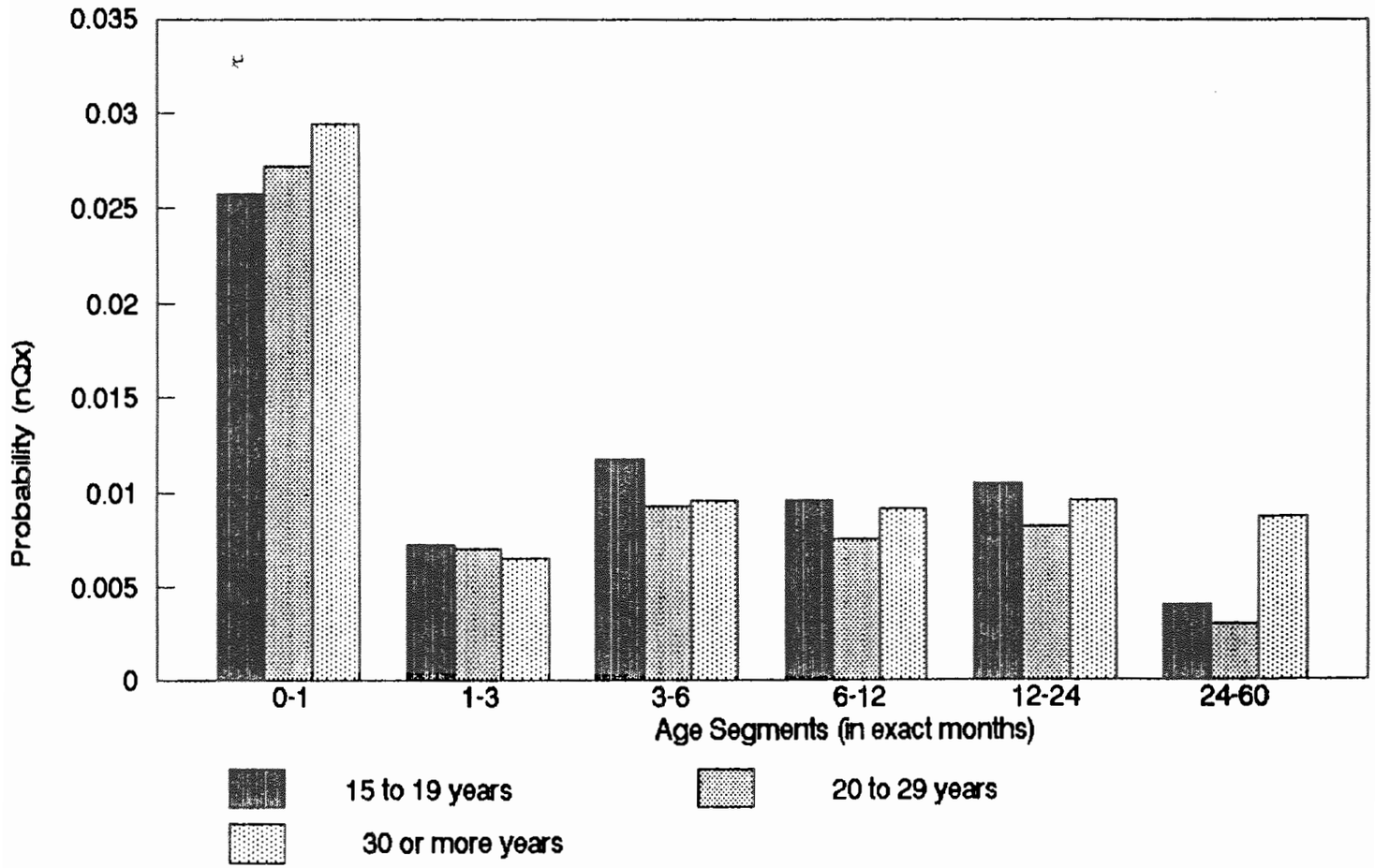
Mexico, Demographic and Health Survey, 1987



Sample of children born 1 to 10 years before the survey

Figure A5. Conditional Probability of Dying by Mother's Age

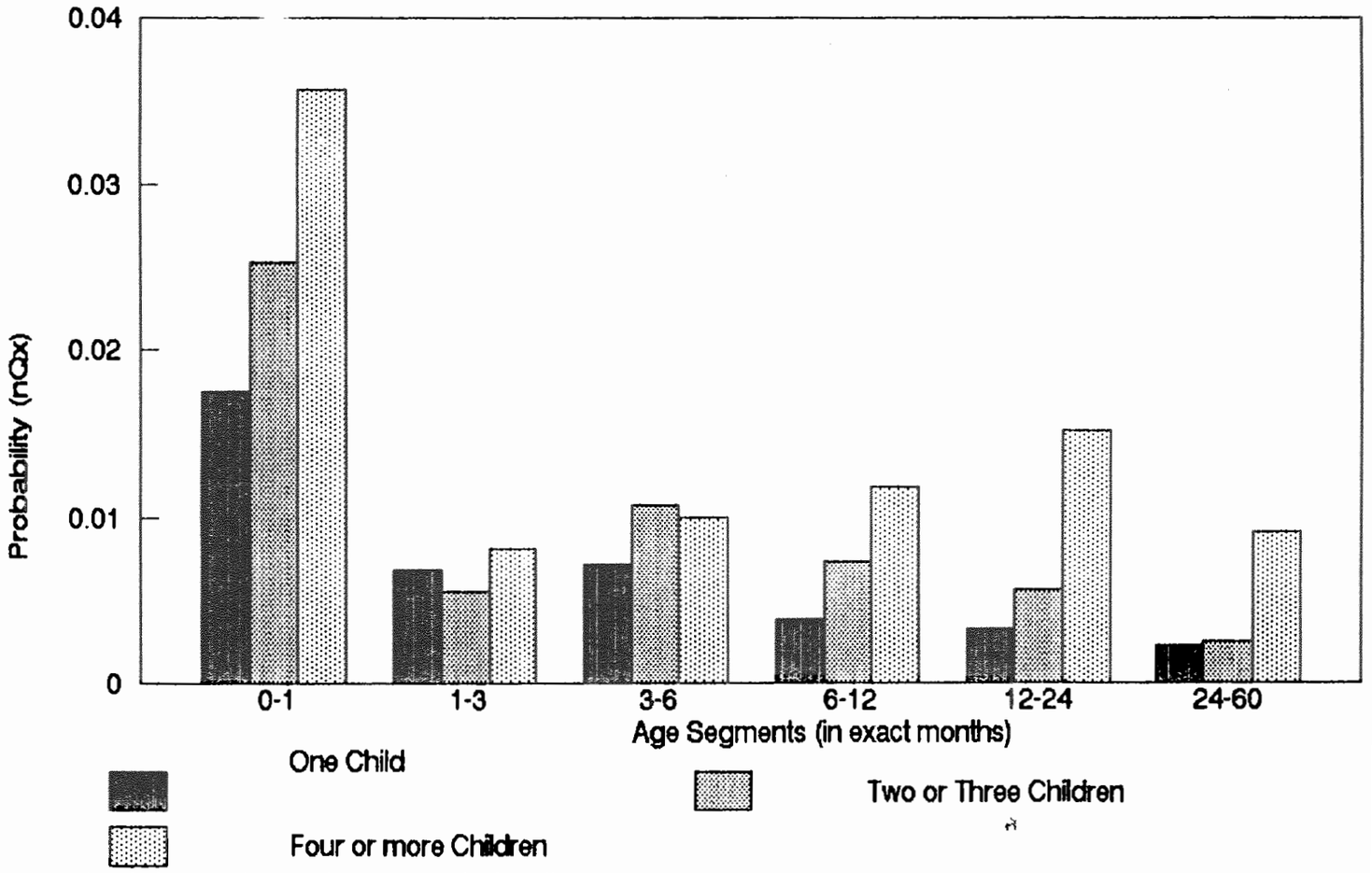
Mexico, Demographic and Health Survey, 1987



Sample of children born 1 to 10 years before the survey

Figure A6. Conditional Probability of Dying by Mother's Parity

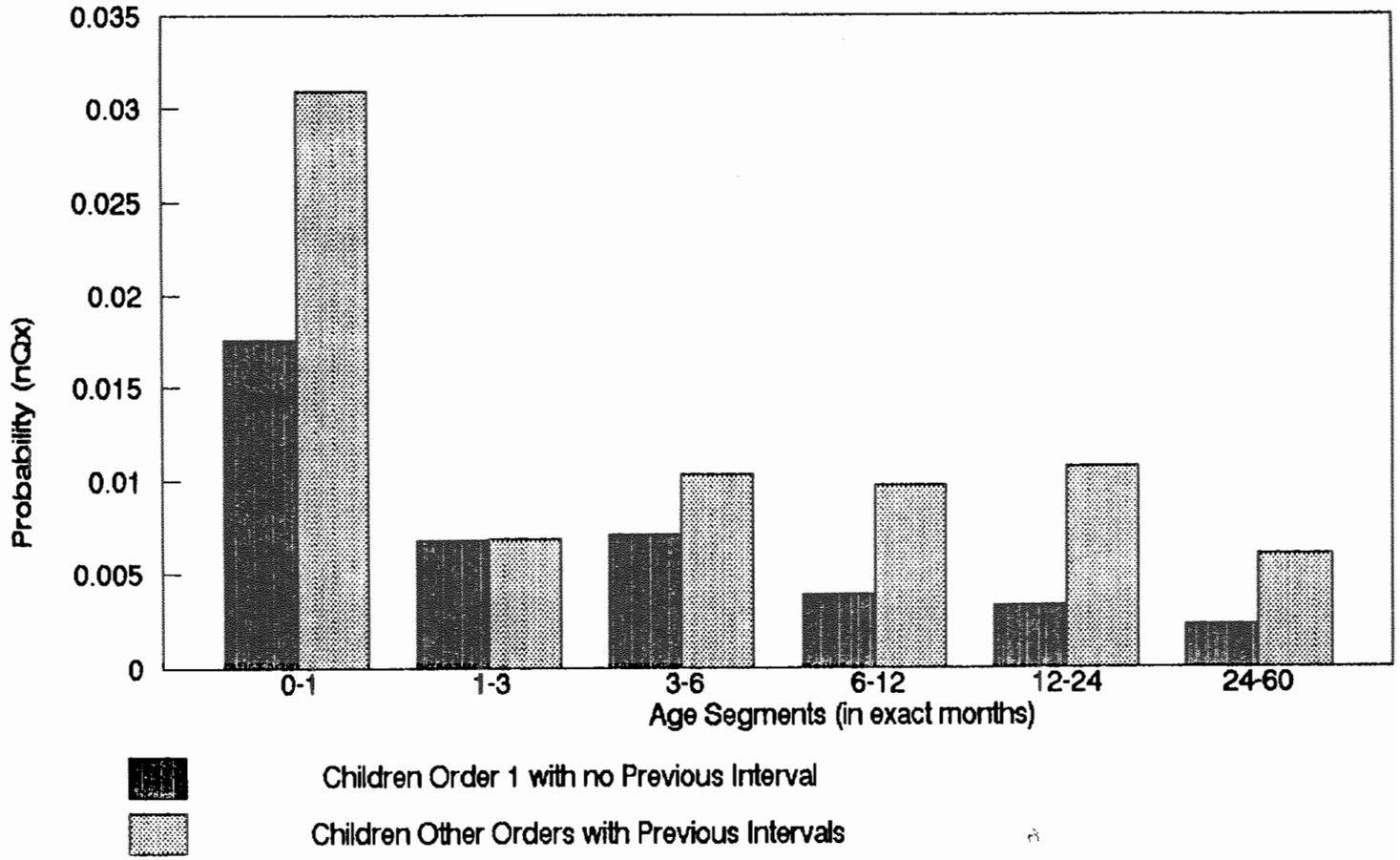
Mexico, Demographic and Health Survey, 1987



Sample of children born 1 to 10 years before the survey

Figure A7. Conditional Probability of Dying by Birth Order and Interval

Mexico, Demographic and Health Survey, 1987

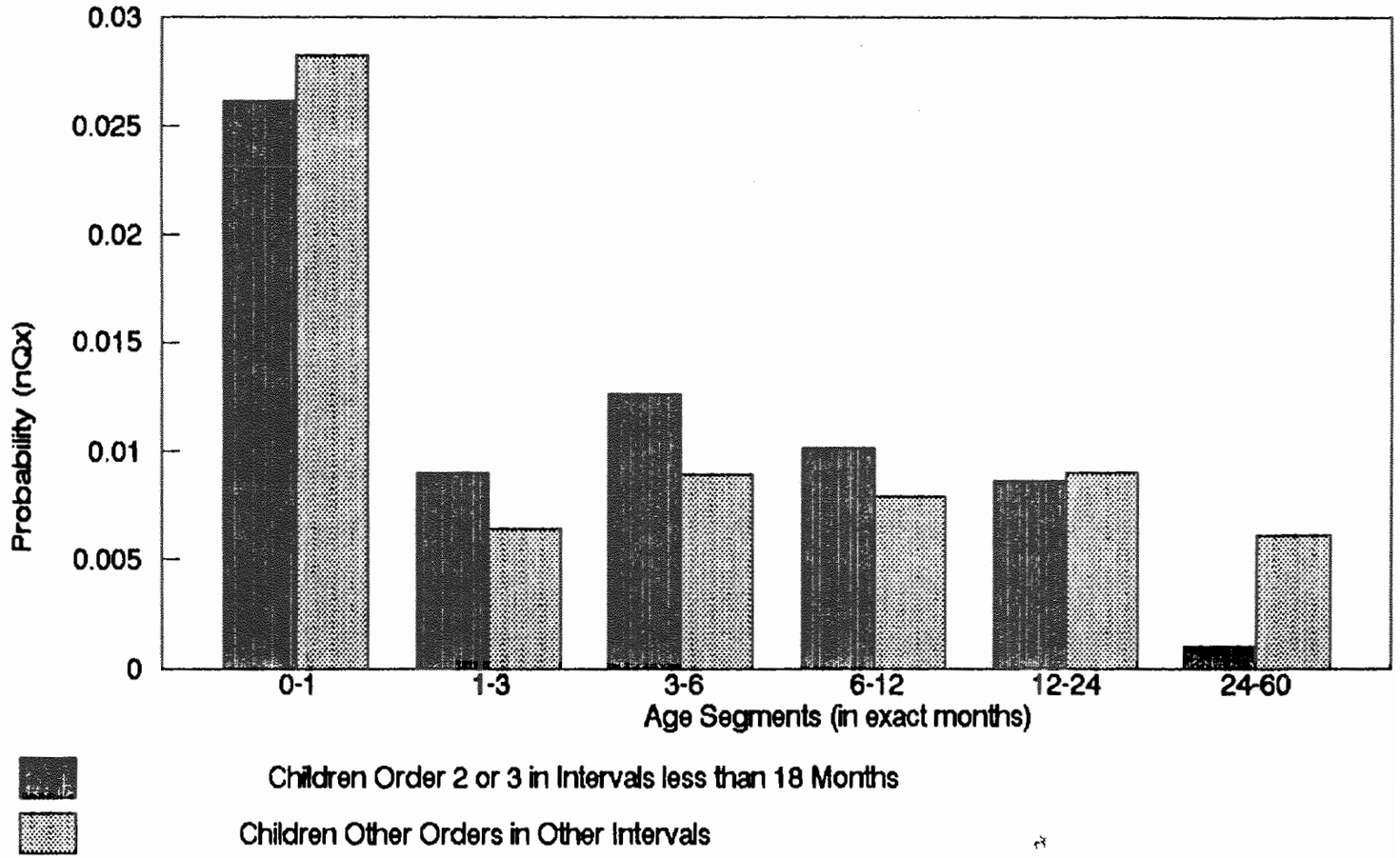


Interval refers to previous conception interval

Sample of children born 1 to 10 years before the survey

Figure A8. Conditional Probability of Dying by Birth Order and Interval

Mexico, Demographic and Health Survey, 1987

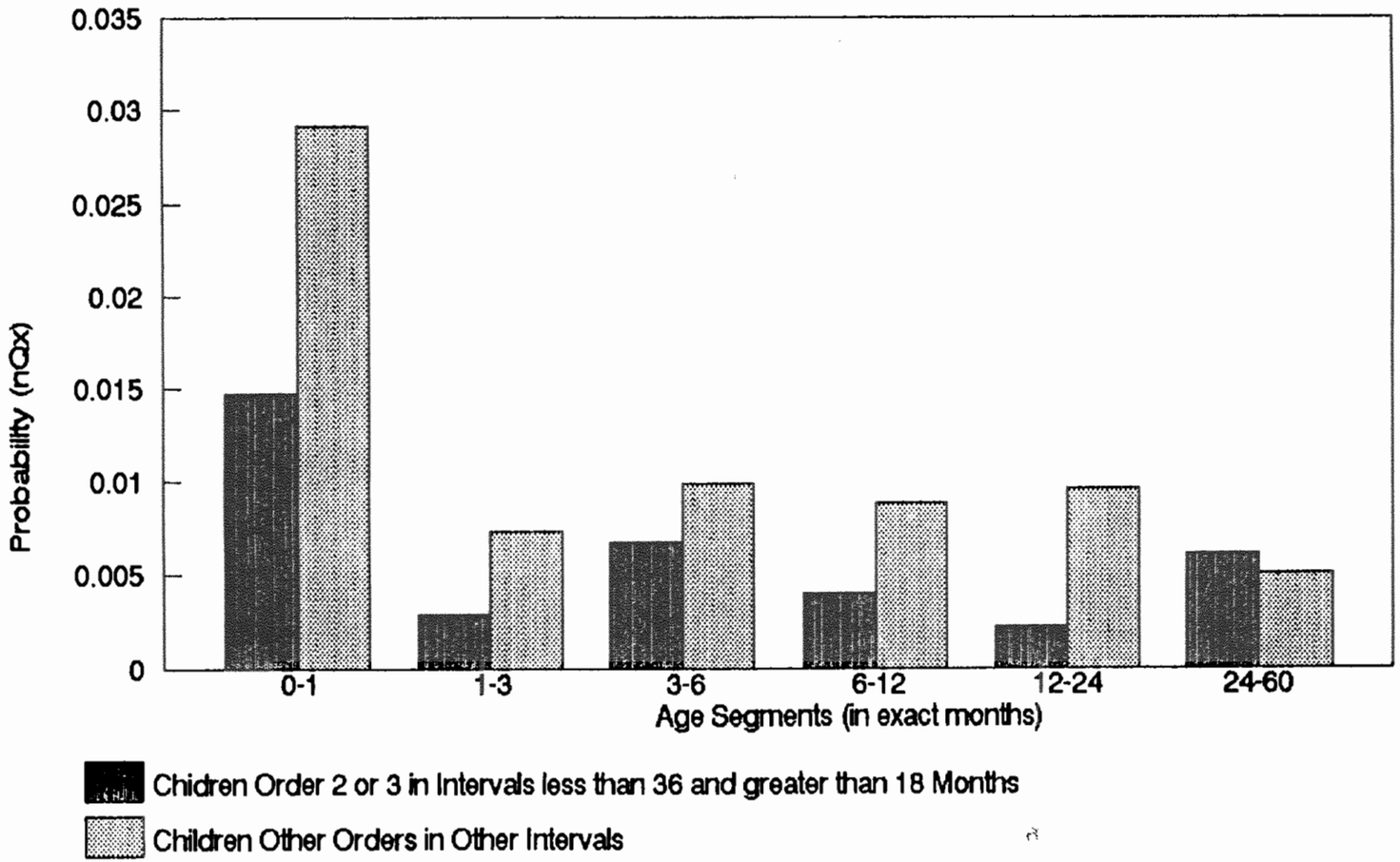


Interval refers to previous conception interval

Sample of children born 1 to 10 years before the survey

Figure A9. Conditional Probability of Dying by Birth Order and Interval

Mexico, Demographic and Health Survey, 1987

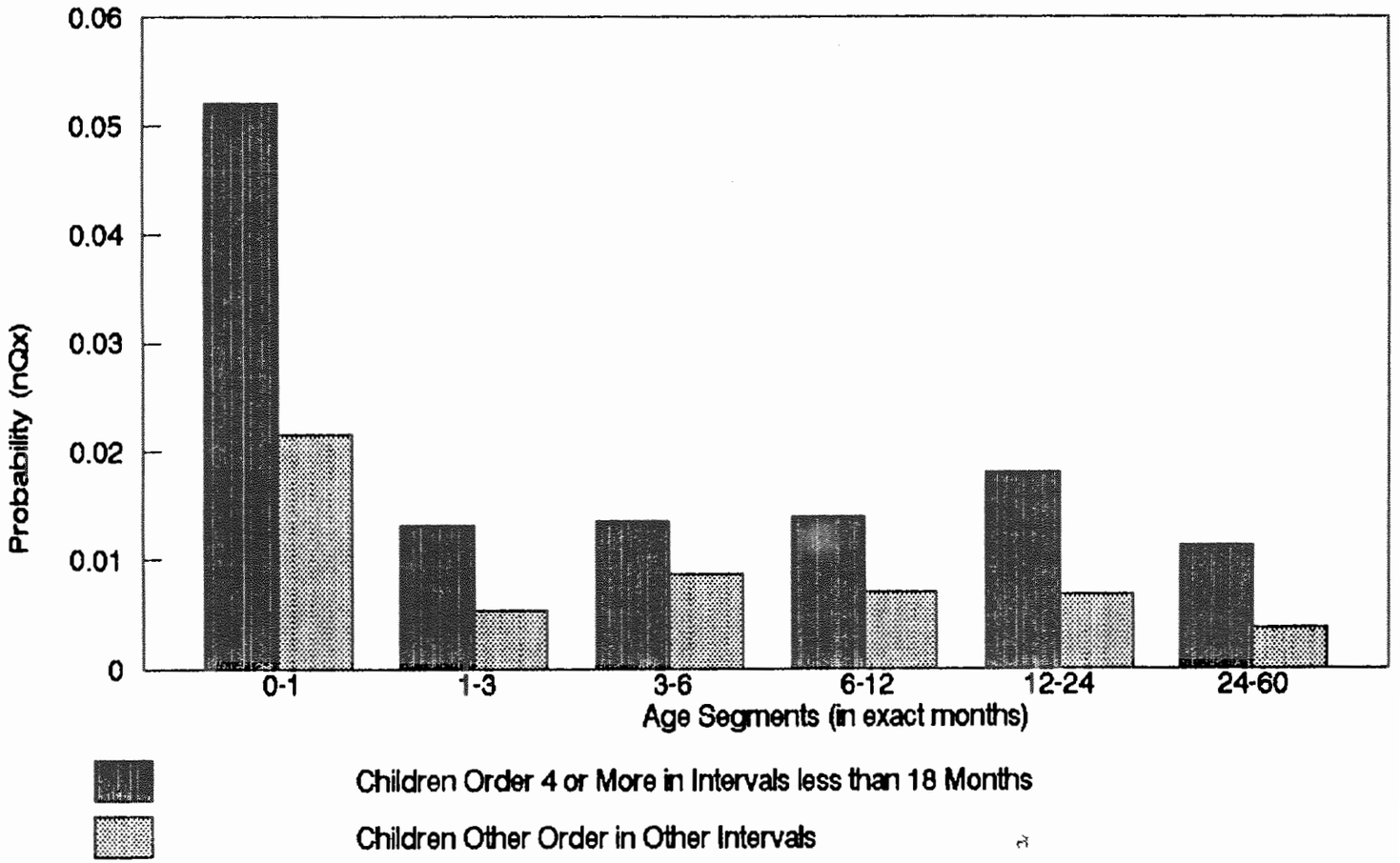


Interval refers to previous conception interval

Sample of children born 1 to 10 years before the survey

Figure A10. Conditional Probability of Dying by Birth Order and Interval

Mexico, Demographic and Health Survey, 1987

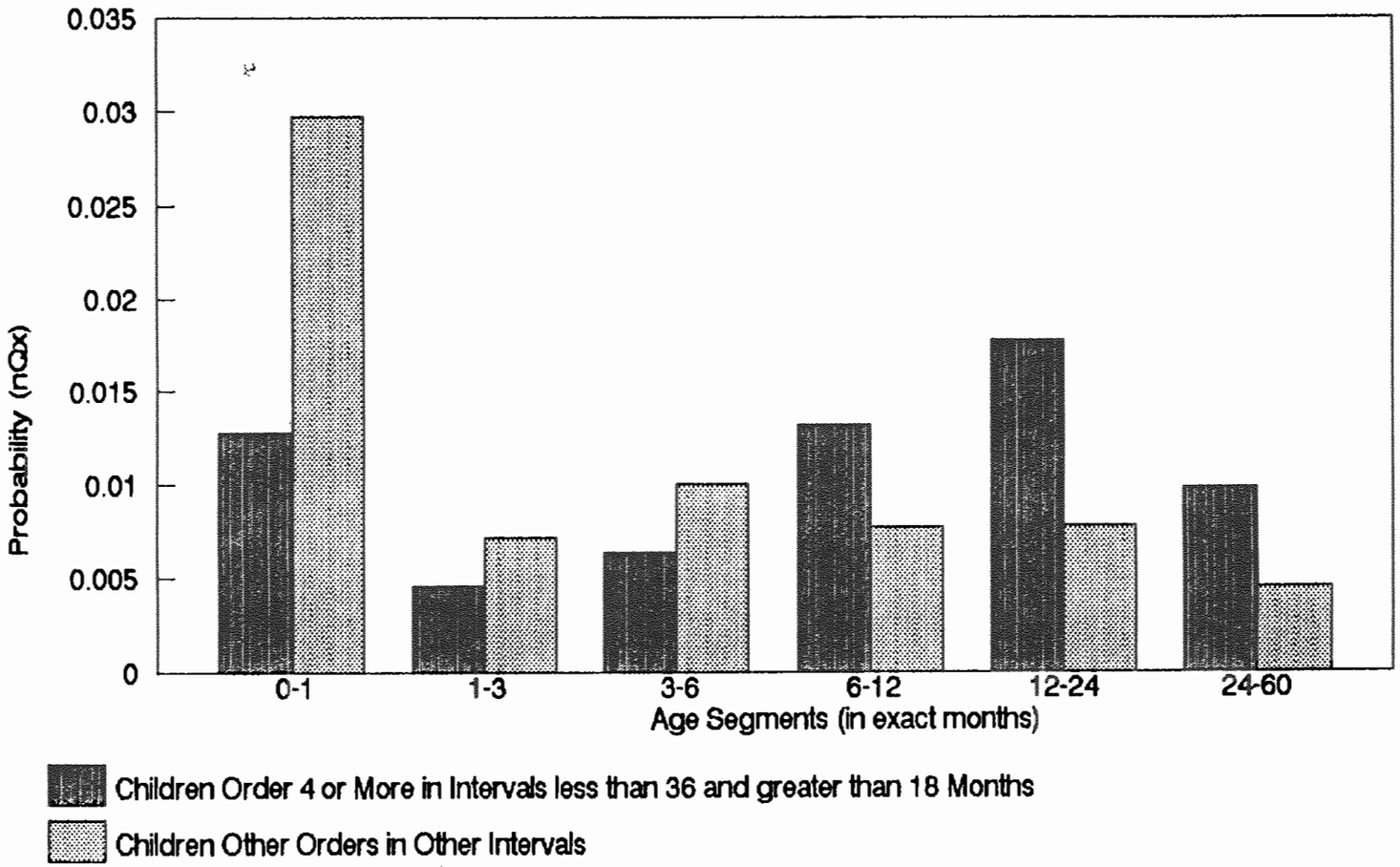


Interval refers to previous conception interval

Sample of children born 1 to 10 years before the survey

Figure A11. Conditional Probability of Dying by Birth Order and Interval

Mexico, Demographic and Health Survey, 1987



Interval refers to previous conception interval

Sample of children born 1 to 10 years before the survey

Appendix 2

Appendix 2: Definition of Main Variables

Preceding Birth Interval:

Bint (2) = 1 if $b \leq 18$
Bint (2) = 1 if $18 < L \leq 36$
L is the length of the birth-to-birth (of index child) interval

Birth Order:

BO(2) = 1 if birth order is 1
BO(3) = 1 if birth order is 4 or higher

Survival of Previous Child:

$\leq$ = 1 if the death of the previous child occurs before the (estimated) date of conception of the index child

Breastfeeding and following conception (conventional)

	Age Segment					
	0	1-2	3-5	6-11	12-23	24-59
B = 1 if	ever breastfed	$D > 1$	$D > 3$	$D > 6$	$D > 12$	$D > 24$
FC = 1 if	--	$I \leq 1$	$I \leq 3$	$I \leq 6$	$I \leq 12$	$I \leq 24$

D = reported breastfeeding duration

I = time elapsed (in months) between date of birth of index child and estimated date of conception of following child

Breastfeeding and following conception (unconventional)

	Age Segment					
	0	1-2	3-5	6-11	12-23	24-59
B1 = 1 if	ever breastfed	$D \leq 0$	$D \leq 0$	$D \leq 0$	$D \leq$	$D \leq 0$
B2 = 1 if	--	$D = 1$	$1 \leq D < 3$	$1 \leq D < 3$	$1 \leq D < 3$	$1 \leq D < 3$
B3 = 1 if	--	--	--	$3 \leq D < 6$	$3 \leq D < 6$	$3 \leq D < 6$
B4 = 1 if	--	--	--	--	$6 \leq D < 12$	$6 \leq D < 12$
B5 = 1 if	--	--	--	--	--	$12 \leq D < 24$
FC1 = 1 if	--	$I < 1$	$I < 1$	$I < 1$	--	--
FC2 = 1 if	--	--	$1 \leq I < 3$	$1 \leq I < 3$	--	--
FC3 = 1 if	--	--	--	$3 \leq I < 6$	--	--
FC4 = 1 if	--	--	--	--	--	--

Appendix 2 (continued)

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

For 12-23:

FC1 = 1 if $I < 6$ and conception results in a birth who dies at 0
FC2 = 1 if $I < 6$ and conception results in a birth who survives
FC3 = 1 if $6 \leq I < 12$

For 24-59:

FC1 = 1 if $I < 6$ and conception results in a birth who dies at 0
FC2 = 1 if $I < 6$ and conception results in a birth who survives
FC3 = 1 if $6 \leq I < 12$ and conception results in a birth who dies at 0
FC4 = 1 if $6 \leq I < 12$ and conception results in a birth who survives
FC5 = 1 if $12 \leq I < 24$

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