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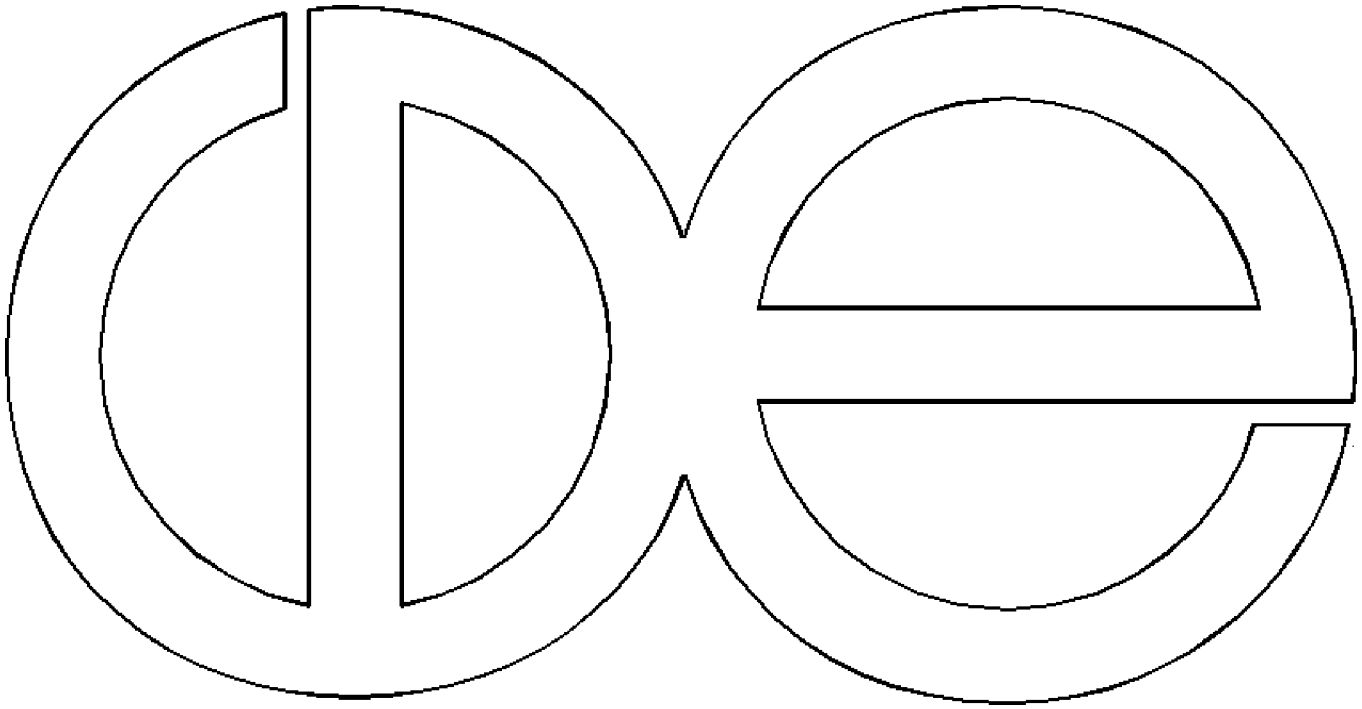

Effects of Lactation on Post-Partum Amenorrhea: Re-estimation Using INCAP Data

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INTRODUCTION

The relationship between postpartum infertility, breastfeeding patterns and women's nutritional status has received a great deal of attention in the literature on biological and demographic determinants of fertility. This preoccupation rests on the fact that longer durations of breastfeeding tend to increase the average birth interval and therefore to reduce a woman's fertility over her life span, particularly in societies where the use of contraceptive methods is not widespread. Similarly, under some social and economic conditions poor maternal nutritional status may also reduce fecundity. The discussion has centered on the magnitude of the inhibitory effects of lactation (e.g., Bracher, 1992; Chen et al., 1974; Knodel, 1978; van Ginneken, 1978; Delgado et al., 1978; Bongaarts and Potter, 1983; Lunn et al., 1981; Huffman et al., 1987; Santow, 1987; Jones, 1988; Rodriguez and Diaz, 1988) but has, by and large, neglected careful evaluation of the extent to which a woman's nutritional status affects ovulatory function (e.g., Chowdhury, 1978; Lunn et al., 1981; Delgado et al., 1982; Huffman et al., 1987; Jones and Palloni, 1994). Only a handful of studies have examined the joint operation of these two factors (e.g., Popkin et al., 1993; Jones and Palloni, 1990; John et al., 1987; Kurz et al., 1993). As a consequence, it is not yet clear whether nursing women's malnutrition affects amenorrhea independently of breastfeeding, or, alternatively, whether women's nutrition affects the amenorrheic period indirectly through breastfeeding behavior. The latter has two possible outcomes: (a) a woman's improved nutrition might mean that the child does not have to nurse as intensely to receive an adequate volume of breastmilk, thus weakening nursing's inhibitory effects, or (b) a woman's malnutrition might mean than the child has to nurse more intensely to receive an adequate volume of breastmilk, thus strengthening nursing's inhibitory effects. In either case it is the child's nutritional needs that determine the direction of causality, while in the former it is maternal malnutrition that affects amenorrhea independently of breastfeeding.

A landmark study of these relations is the INCAP longitudinal study which found a strong

association between breastfeeding, mother's nutritional status, and the duration of postpartum amenorrhea. At the time the INCAP study began in 1968 it was a unique data collection enterprise both because of its intervention research design and the wealth of information that it could retrieve on a number of themes related to nutrition. Today the INCAP study is among a handful of more recent but similar studies which, among other goals, were designed to probe the relations between fecundity, lactation and nutrition. Indeed, the INCAP study stands side by side with influential special surveys conducted as part of MATLAB (Huffman et al., 1987), the NGAGLIK study (Ngaglik Study Team, 1978), and the so-called CEBU study (Cebu Study Team, 1992).

Despite the richness of the information collected by the teams of researchers, the INCAP data on fertility was never analyzed either using all the information gathered or by applying state-of-the-art statistical procedures which, having been developed only recently, benefitted the analysis of the newer data sets. It is important, then, to investigate the extent to which the results obtained from previous analyses, their influence notwithstanding, continue to be valid after inclusion of supplementary information and the deployment of more refined analysis techniques. The purpose of this paper is to re-estimate the effects of lactation and maternal nutrition on timing of menses in the INCAP data using all the relevant information and an array of more suitable estimation procedures and to compare these new estimates with others derived either from previous INCAP studies or from other data sets.

The paper begins with a formulation of the relations underlying the process of resumption of postpartum menstruation.[1] Although most of them remain unknown, elusive or ill-specified, important breakthroughs in the last 15 years have identified some of the mechanisms through which lactation, nutrition, stress, and work patterns produce variability in the length of the anovulatory period (e.g., Knobil, 1980; McNeilly et al., 1985; Short, 1984; Glasier and McNeilly, 1990). We then summarize the results obtained in previous research using the INCAP data and other sources of information. These results are then contrasted with those obtained from a reanalysis of the INCAP data with

supplemental information and more robust statistical techniques.

THE DETERMINANTS OF POSTPARTUM AMENORRHEA

The timing of resumption of postpartum menstruation depends on the status of three intermediate conditions (or external factors) through which all effects of socioeconomic characteristics are expected to operate, and on random conditions (Wood, 1994; Jones and Palloni, 1990; Jones, 1989). Two of these intermediate conditions, weaning and infant mortality, have a direct causal effect as both lead to termination of nursing episodes and thus remove altogether the neurohormonal stimulus that child suckling has on the delay of fecund menstrual cycles. The third mechanism involves the breastfeeding patterns prevalent among mothers who are still breastfeeding when they resume menses and other external factors such as maternal nutrition and health status, which are also suspected to interfere with neurohormonal stimuli and delay the resumption of normal menstrual cycles.

It has been recognized that more intense and frequent breastfeeding episodes generate a hormonal environment inhibiting ovulation, and that this environment may break down when the suckling stimulus diminishes. There are two possible pathways through which maternal nutrition and health status could affect postpartum amenorrhea. First, deficient maternal nutritional status and/or ill health may directly alter the immunological and hormonal status of the woman and thus induce a delay in the resumption of menses (Warner, 1980; Frisch, 1978). Second, maternal nutritional status may exert an indirect effect working through breastfeeding patterns (Lunn, 1985). If malnourished or sick women produce significantly lower amounts of breastmilk, their children's suckling will have to increase in intensity to obtain adequate nutrition. On the other hand, if it is the child who is ill, he/she may not be able to nurse adequately and there will be a decrease in the intensity of the nursing stimulus. As mentioned above, either an increase or decrease in the suckling intensity is the pathway through which breastfeeding patterns alter the neurohormonal environment to affect ovulation.

Finally, resumption of menses is also a function of individual factors not related to the intermediate mechanisms such as capacity to breastfeed, propensity to abort spontaneously, and other pathways associated with ovarian activity.

Anovulation and amenorrhea are neurotransmitter phenomena driven by hypothalamic and pituitary processes. The main force behind the process is the tendency to maintain critical levels and patterns of GnRH (Gonadotropin Releasing Hormone) and LH (Luteinizing Hormone) secretion. Factors that lead to the disruption of normal patterns of GnRH and LH release produce anovulation and amenorrhea. The physiological mechanism that underlies the hypothesized relationship originates in the hypothalamus. The woman's hypothalamic nuclei release GnRH in a regular, episodic fashion and this, in turn, triggers the pulsatile and episodic release of LH and **follicle stimulating hormone** (FSL) from the pituitary. This biological mechanism is also known as the **hypothalamic pulse generator** (HPG) (see McNeilly et al., 1985). Release of LH has been found to be crucial for normal ovarian activity and fecund menstrual cycles (Glasier et al., 1983, 1984; Knobil, 1980; McNeilly et al., 1985).

Recent research has shown that normal patterns of GnRH and LH release can be severely disrupted not only by lactation but also by external factors such as heavy physical exercise or activity and by psychological stress (Warren, 1980; Kaiserauer et al., 1989, Genazzani et al., 1991a, 1991b; Loucks, 1990). However, the exact neurotransmitter signal which interrupts the release of GnRH and LH during these activities remains to be completely elucidated. In contrast, it is better known that the suckling stimulus is the primary signal which disrupts the normal pattern of GnRH activity for breastfeeding women (Tay et al., 1992). It appears that disruption of HPG neurons is a common characteristic of altered GnRH and LH release and thus a common pathway whereby breastfeeding, stress, physical activity and, more generally, other stressors originating in the environment, affect amenorrhea and fertility (Yen, 1987; Jones and Palloni, 1994).

PREVIOUS RESEARCH

Findings about the relationship between breastfeeding, nutritional status and postpartum amenorrhea obtained in previous studies are quite heterogeneous. We first review studies using general data sources and then we discuss the most important findings of studies with INCAP data.

a. General data sources.

The existing body of literature suggests that postpartum amenorrhea is strongly and consistently related to breastfeeding patterns, even after controlling for potentially confounding factors, but that its association with maternal nutritional and health status is more tenuous and less definitive. The sources of data are quite heterogeneous as some studies are based on aggregate cross-sectional demographic surveys (e.g., Guz and Hobcraft, 1991; Bongaarts and Potter, 1983; Tyson and Perez, 1978); others rely on clinical studies (e.g., Howie and McNeilly, 1982; Rodriguez and Diaz, 1988; Diaz, 1989); and finally, a handful of them, including the Gambia project, INCAP, the MATLAB project in Bangladesh, the NGAGLIK project in Indonesia, and the CEBU project in the Philippines, rely on longitudinal protocols (Delgado et al., 1978; Lunn et al., 1980, 1981; Jones, 1990; Bracher and Santow, 1982; Huffman et al., 1987; Santow, 1987; Kurz, 1990; Jones and Palloni, 1990; Popkin et al., 1993). Although in all these cases the goal is to investigate the role played by either women's nutritional status or women's breastfeeding behavior on resumption of menses, few of them have examined the joint effects of breastfeeding patterns (duration, frequency, intensity, and type of breastfeeding) and maternal nutritional status while simultaneously controlling for other intermediate conditions such as work patterns and health status and characteristics such as infant mortality, age, parity, and education (Kurz et al., 1993; Popkin et al., 1993; Jones and Palloni, 1990).

The most important conclusion drawn from these studies is that lactation increases the duration of postpartum amenorrhea and that long-term breastfeeding is consistently associated with long periods of postpartum amenorrhea, ovarian inactivity, and reduced fertility (e.g., Knodel, 1978;

van Ginneken, 1978). In the absence of breastfeeding the average amenorrheic period may last between one and three months; but when nursing is initiated just after childbirth, the duration of amenorrhea increases systematically with the duration of breastfeeding though at progressively slower rates (Santow, 1987; Bongaarts and Potter, 1983). However, long durations of breastfeeding do not always result in longer periods of amenorrhea nor do they provide complete contraceptive protection (van Ginneken, 1977; Cantrelle and Leridon, 1971; Jain et al., 1970; Chen et al., 1974; Potter et al., 1965). Supplementation, by reducing suckling intensity and curtailing high frequency episodes of lactation, seems to be the most important factor that explains why prolonged breastfeeding does not always extend the average time of amenorrhea beyond 18 or 20 months (e.g., Howie and McNeilly, 1982; Jones 1990; Delgado et al., 1982; Trussell et al., 1989). It is relatively well established that the relationship between breastfeeding patterns and postpartum amenorrhea depends heavily on the effectiveness of nursing stimulus (Tyson and Perez, 1978). When breastfeeding is prolonged, frequent, and intense, the average postpartum amenorrhea may last from one to two years (Huffman et al., 1987; Cantrelle and Ferry, 1979; Wood et al., 1985; Jones, 1988; Rodriguez and Diaz, 1988).

The idea that maternal nutrition and health affect women's fertility has appeared frequently in the demographic and biological literature but is supported by weaker and less convincing evidence than that pertaining to the role of breastfeeding (e.g., Warren, 1980; Stein, 1975; Frisch, 1975; Bongaarts and Delgado, 1979; Bullen et al., 1985). The notion that poor maternal nutritional status may decrease a woman's reproductive capacity is based on two empirical facts. First, long durations of postpartum amenorrhea are more often found in populations where women are undernourished than in populations where women are better nourished (Carael, 1978; Chavez and Martinez, 1973; Warren 1980). Second, the evidence suggests that menstrual cycles cease altogether during times of famine and starvation (Stein and Susser, 1975, 1978; Chowdhury, 1978; Keys et al., 1950; Menken, 1979). It has been further noted that delays in resumption of fecund menstrual cycles also accompany

other forms of physiological and psychological stress such as those associated with anorexia nervosa, vigorous exercise, or severe depression (Warren, 1980, 1989; Pirke et al., 1985; Pirke et al., 1989). There is more uncertainty, however, about the effects of nutritional status (and other stressors) on a female's reproductive system in less extreme situations as, for example, when malnutrition is chronic or varies from mild to moderate rather than being acute (Prentice, 1980; Bongaarts, 1983; Huffman et al., 1978; Huffman et al., 1980; Huffman et al., 1987; Delgado et al., 1978).

Some authors argue that maternal nutritional status may have an important, and independent effect on the risk of resuming ovulation after childbirth (Frisch, 1978; John et al., 1987). The majority, however, are more sanguine and suggest that the empirical evidence only reveals direct effects of maternal nutritional status on postpartum amenorrhea that are small and unimportant except under quite extreme conditions (Jones and Palloni, 1990; Delgado et al., 1978; Huffman et al., 1987; Huffman et al., 1978; Bongaarts and Potter, 1983; Bongaarts and Delgado, 1979; Chowdhury, 1978). However, this empirical evidence also indicates that the indirect effects of malnutrition, through selected mediating factors, may be more consequential (Lunn, 1985; Lunn et al., 1981; Kurz et al., 1993). One of these mediating factors is the pattern of lactation itself. Indeed, several studies have found that children born to malnourished mothers tend to suckle more frequently and more intensively to obtain adequate amounts of breastmilk, thus increasing the inhibitory effect of the ovulatory hormones, which lengthens the amenorrheic interval (Salber et al., 1966; Wray, 1978).

b. The INCAP study.

Results based on the INCAP longitudinal study found consistent associations between breastfeeding patterns, mother's nutritional status, and the duration of postpartum amenorrhea. In an early study, Delgado and colleagues (1978) grouped women according to daily caloric intake and breastfeeding duration and found that those with caloric supplementation during pregnancy experienced reduced duration of postpartum amenorrhea. Their study showed that women who were supplemented to increase their total caloric intake during pregnancy by 20,000 calories ('highly supplemented' group) had a significantly shorter postpartum amenorrhea period relative to those who consumed supplements of less than 10,000 calories during pregnancy ('poorly supplemented' group). Furthermore, they were able to establish that the effects of absolute caloric intake during pregnancy was to reduce the length of postpartum amenorrhea to between 1.0 to 1.4 months. In addition, they found negative correlations, ranging from a low of -.09 to a high of -.14, between various measures of nutritional status assessed at three months postpartum--mother's weight, height, head circumference and weight-for-height--and the length of postpartum amenorrhea.

In another study of the INCAP data, Bongaarts and Delgado (1979) grouped women into three categories of nutritional status according to a combined index of anthropometric indicators and again found a consistent and negative relation between this index and the duration of postpartum amenorrhea. The effects of four alternative measures of nutritional status assessed at 3 months postpartum was strongly negative and statistically significant even after controlling for some background characteristics. The average difference in the length of the postpartum amenorrhea period for mothers with high and low nutritional status hovered around 1.6 months.

In later analyses, Delgado and co-workers (1982) investigated the relationship between postpartum amenorrhea, nutrition and lactation with linear regression models. Nutritional status was measured as changes in women's weight during the third and ninth months of the postpartum period.

The results show, here again, that the effect of nutrition on duration of postpartum amenorrhea is statistically significant but rather small in magnitude when compared to the large effects of breastfeeding. The authors found very large effects of length and pattern of breastfeeding: not only was resumption of menses delayed with increased duration of lactation but also with increased intensity of breastfeeding. Thus the probability of remaining amenorrheic 15 months by tercile of frequency of suckling at six, nine, and twelve months was always greater in the higher tercile than in the lower tercile at each period.

Another more recent study carried out by Kurz and colleagues (1993) finds that, when modelled simultaneously with the effects of nutritional status, length of breastfeeding and supplementation are the most important determinants of the timing of resumption of menses. Furthermore, they also find that maternal supplementation is a very weak determinant once child's non-breastmilk energy intake is controlled for. This suggests that maternal caloric intake interferes with anovulation mainly through child nutritional status. Finally, a measure of maternal nutritional status, triceps-skinfold thickness, proved to be only weakly associated with the duration of postpartum amenorrhea.

While past studies with INCAP data are important landmarks, their results cannot be easily compared with those obtained from more recent research endeavors. This is because virtually all past INCAP studies share shortcomings that undermine their robustness and weaken the commonalities with analogous studies. First, all studies with INCAP data utilize procedures (linear models) that do not take full advantage of the time-varying nature of the INCAP study design. This could generate inconsistent and inefficient estimates of effects. For the most part, recent analyses employ a full battery of modelling techniques that are suitable for dynamic phenomena. Second, there is inadequate statistical treatment of the simultaneous effects of the most important factors being considered, namely, maternal nutrition, breastfeeding patterns, mother's work patterns, and selected potential

confounding factors. By and large, INCAP studies proceed to assess effects on a piecemeal basis and with only partial controls for covariates, thus increasing the risk of contamination from unmeasured characteristics. Third, earlier studies did not make use of the full range of information on breastfeeding and maternal nutrition since some of it was collected only after those studies had been concluded. Finally, as in many of the recent studies, the INCAP studies fail to appropriately include mothers whose children died before resumption of menses thus leaving open the possibility of selection biases.

In what follows we re-assess the empirical relationships between postpartum infertility on the one hand, and breastfeeding behavior, maternal nutritional and health status, work patterns, and infant mortality, on the other, while controlling for measured and unmeasured characteristics. We utilize two-state hazard models to incorporate time-varying covariates and time-varying effects. These models are estimated with reconstructed INCAP data fully incorporating all the pertinent information available for the observations. The models we use are well suited to retrieve information on the link between the physiology of postpartum anovulation with its biological and social determinants and to allocate the effects of the latter among intermediate pathways. A non-trivial resource that is available from these models is the ability to correctly deal with child deaths. The most common practice of INCAP studies (and also of most studies based on more recently collected data sets) was to eliminate from the sample all mothers whose child died before resumption of menses. This solution, however, could lead to selection biases and to inconsistent estimates since child's mortality risks, child nutritional status, and maternal nutritional status are correlated with each other.

DEFINITION OF SAMPLES AND VARIABLES.

a. Features of the data.

The data used in this paper come from the Longitudinal Study in Guatemala carried out in a chronically malnourished population, a prospective study of the physical and mental development of

infants in four rural villages. A sample of 755 women was followed longitudinally by regular visits for nearly eight years, between 1969 and 1977. Women entered the study depending on their reproductive history; the main condition was that they had at least one child aged less than seven years old. At regular intervals throughout their pregnancies, and postpartum, mothers provided information about the status of their children and their own conditions. For the purpose of this reassessment we retrieved data for all women who had a delivery between January 1, 1969 and February 28, 1977 and who were followed-up until the latter date (when the survey officially ended). In all, we selected 608 women and their 1430 birth intervals (Pinto, 1994). To handle missing information for some of the variables, such as the number of nursing episodes per day and mother's nutritional status, we define categorical variables that include categories to capture cases with missing values. This enable us to use nearly the full sample (1417 birth intervals)--omitting 13 cases with missing ages.[2]

b. Dependent and independent variables.

The dependent variable (i.e., length of postpartum amenorrhea) is defined as follows: (1) the number of months elapsed between birth and the first incidence of at least two consecutive menstrual episodes within the first three months after delivery, or (2) the number of months elapsed between birth and the first menstrual episode when menses occurred beyond the third month after delivery, or (3) the number of months elapsed between a birth and the subsequent conception when menses never resumed. The first two cases represent the occurrence of the event of interest while the last case corresponds to a censored observation (Pinto, 1994).

The independent variables included in the analysis can be classified into six groups: breastfeeding, maternal and child nutritional status, health status, energy expenditure, and demographic and socioeconomic conditions. All breastfeeding variables (except frequency of breastfeeding), nutritional variables (except mother's head circumference), and health variables are

treated as time-varying covariates. Energy expenditure and all the demographic and socioeconomic variables are fixed covariates. A full description of the variables appears in the Appendix.

MODELLING STRATEGY AND RESULTS

a. Models.

The reproductive history of a woman is considered as a sequential process over time punctuated by a series of events such as births, infant deaths, nursing episodes, menses, illness spells, malnutrition episodes, and supplemental food intakes that occur randomly over time. That is, the woman's reproductive history is taken as a collection of random points over time (i.e., a stochastic process) defined by each one of these events. To analyze the processes involved one can use two different models. The model we use in this paper is defined in a state-space with only two states: one is the origin state represented by amenorrhea and the other is the destination state corresponding to an absorbing state marking the arrival of menstruation. The second model (not included in this paper) involves a more complex state-space consisting of five states: full breastfeeding, partial breastfeeding, weaning, infant mortality and menstruation. In both cases the aim is to retrieve the parameters for the distribution of waiting times to resumption of menses by modelling the corresponding transition risks (Blossfeld et al., 1989; Allison, 1982; Kalbfleisch and Prentice, 1980; Tuma and Hannan, 1984). Both types of hazard models enable us to deal with right censored observations and to model the dependency of the risk of resuming menses on (possibly) changing individual characteristics (covariate structure) and time (duration structure). Very generally, the hazard function for the transition from state j to state k is defined as follows:

$$h_{jk}(t_{jk}|Z) = (h_{ojk}(t_{jk})\exp(\beta_{jk}Z(t_{jk}))$$

where j is the origin and k is the destination state, t_{jk} is the duration elapsed in state j before a transition to k occurs, β_{jk} is a vector of covariate effects (possibly dependent on the states of origin

and destination), $\mathbf{Z}(t_{jk})$ is the vector of fixed and time varying-covariates associated with the mother and her child, and $h_{ojk}(t_{jk})$ is the origin-destination hazard baseline. A more general model includes the presence of time-invariant and independent unmeasured covariates and a flexible functional form for the baseline hazard:

$$h_{jk}(t_{jk}|Z(t_{jk}),\theta) = \exp[\gamma_{0jk} + \beta_{jk}Z(t_{jk}) + \sum_{r=1}^{R} \gamma_{rjk}\frac{t^{\lambda rjk}-1}{\lambda_{rjk}} + c_{jk}\theta]$$

In this expression, t_{jk} is again the length of exposure to the risk of moving from state j to state k, $Z(t_{jk})$ is a vector of covariates, β_{jk} is a vector of coefficients, θ is an unobserved component or covariate (i.e., person-specific unobserved heterogeneity component), and c_{jk} is a loading factor. The baseline hazard now depends on the parameters R and λ_{rjk} (which are generally fixed a priori) and γ_{rjk} (which is generally estimated from the data). In the remainder of this paper we will only discuss the results of the simpler two-state specification.[3]

b. Analysis of Results.

b.1. A two-state model without unmeasured heterogeneity.

Our main representation of the process consists of a two-state model where the origin state is amenorrhea (starting at delivery) and the destination state is reached with resumption of menses. We proceed to estimate a series of models that gradually increase in complexity. In all cases these models are additive and use either a Gompertz or a piecewise exponential representation of the baseline hazard.[4] The estimates of these hazard models are displayed in Table 2 and include the effects of the woman's age, parity, place of residence (atole or fresco village), the woman's education, type of occupation, and frequency of breastfeeding as fixed covariates, and breastfeeding status (introduction of supplementation, weaning, and infant death), intensity of breastfeeding, maternal nutrition, child nutrition, and health measures for the mother and children as time dependent covariates.[5] The first model (model 1) is the baseline model with estimated effects for time intervals

only; these effects are relative to the omitted time interval corresponding to the first five months after birth.[6] The second model (model 2) introduces the effects of parity, mother's age, education, occupational status, and place of residence. The third model (model 3) introduces breastfeeding variables (breastfeeding status, frequency of breastfeeding, and intensity of nursing), mother's and child's nutritional status and, finally, mother's and child's health status. The last model (model 4) is the most parsimonious of all and is obtained after removing some of the non-control variables that proved to have insignificant effects in other models, namely, child's nutritional status and mother's health.[7]

The estimated duration structure of the piecewise hazard models reveals that the risk of resuming menses increases systematically with time, even after controlling for all the relevant variables in the model. In fact, the estimated parameters for each time segment are significantly different from zero and increase over time in all models. According to model 4, for example, the monthly risk of resuming menses increases fivefold (the ratio (exp(-4.20+1.60)/exp(-4.20))), by the time the women enters the eighteenth month postpartum. The fact that the risk of resumption of menses progressively increases over time, **even after controlling for conditions that supposedly reflect the suckling and hormonal environment**, indicates that these conditions are not accurate measures of the underlying physiological mechanisms that drive the development of ovulation. In other words, the time-dependency retrieved by the estimated model reflects the net effects of unmeasured and/or poorly measured characteristics.

In all models, the independent effects of breastfeeding status (full breastfeeding, partial breastfeeding, weaning, and infant mortality) are highly significant and in the expected direction, even after controlling for relevant covariates. Infant mortality has the strongest effect on the hazard of first menses postpartum. For instance in Model 4, when a child dies, the risk of resuming menses is about 39.9 (exp(3.69)) times higher than when the child is fully breastfeeding (reference group). Similarly,

when the child is weaned the risk is about 11 (exp(2.38)) times higher than when full breastfeeding takes place. The difference between these two estimated effects is somewhat counterintuitive but probably explained by the fact that, in practice, actual weaning rarely consists of the sudden withdrawal of mother's milk and termination of suckling as the death of the infant surely does.[8]

The variables representing frequency of breastfeeding also have a significant effect on the dependent variable and is in the expected direction. At each postpartum duration, women who breastfed their children on demand (12 times a day or more) experienced a significant delay in the resumption of menses when compared to women who breastfed between one and seven times a day (baseline group). Indeed, the risk of resumption of menses associated with those women who breastfeed with medium and high frequency is .85 (exp(-.16)) and .55 (exp(-.59)) of the risk for women who breastfeed with low frequency.

The effect of children's clinical supplementation, our indicator of the intensity of breastfeeding (see also Kurz et al., 1993), is also properly signed and statistically significant. Mothers who provide high amounts of supplementation (and therefore lactate at lower intensity of suckling) have a risk of returning to menses 1.21 (exp(.19)-1) as high as those mothers who do not.

The findings regarding breastfeeding patterns and supplementation are highly consistent with the thesis that higher intensity and higher frequency of nursing episodes are at the root of the inhibitory effects of breastfeeding: suckling stimulates the neural receptors located in the breast nipples and GnRH and LH release is disrupted and reduced, thus inhibiting ovulation and the return to normal menstrual cycles. And as the duration, frequency, and intensity of lactation decrease, release of GnRH and LH recovers its normal pulse, frequency, amplitude and level, and menstruation resumes.

Although they are in the expected direction, not all the effects associated with maternal nutritional status turn out to be statistically significant. First, the measure of body mass, BMI, has,

as expected, a positive effect but it does not attain statistical significance. Second, lower mother's head circumference (a sign of long term undernourishment) is associated with reduced risk of resumption of menses compared with those who appear to be better nourished but, here again, the effects are small and statistically insignificant. However, the third indicator of maternal nutrition, mother's clinical supplementation, has a relatively large and statistically significant effect on the hazard and is also in the expected direction.[9] In fact, the risk of resuming menses for mothers with high supplement intake is about 1.29 as large, exp(.251)), as the risk for mothers with lower levels of the supplement. Note that this effect is observed even when controlling for variables through which maternal nutrition may operate, namely, suckling stimulus. This finding goes against the idea that maternal nutrition affects postpartum amenorrhea by altering the intensity of suckling episodes. Note also that the magnitude of the estimated effect is intermediate between the effect of child supplementation and of high frequency of suckling episodes. This supports the hypothesis that maternal malnutrition, even at moderate levels, has non-negligible **direct** effect.

These findings are comparable to those obtained elsewhere. For example, Huffman and collaborators (1987) found that poor maternal nutrition delays the resumption of menses postpartum in Bangladesh. Their analysis, however, only retrieves gross effects of maternal nutrition. Similarly, Popkin and colleagues (1993) found a significant inverse relationship between BMI (maternal body fat) and time to menses. Here again, the measured effects are gross, not net effects. However, our inference that maternal supplementation does not operate through child suckling contradicts the results obtained in another reanalysis of the INCAP data. In fact, Kurz and collaborators (1993) found that maternal energy supplementation (i.e., current nutritional status) was **not** a significant predictor of the duration of postpartum amenorrhea **after** controlling for child's supplementation and breastfeeding.[10]

We argued before that child ill-health and inferior nutritional status could potentially inhibit

ovulation by decreasing the intensity of the suckling stimulus. The results of our models provide support for this idea: the relative risk of resumption of menses is higher when the child's nutritional status is poor, exp(.154), and lower when the child is in good health, exp(-.22). Although this finding is in agreement with the idea that poor child nutrition and ill-health may lead to decreasing suckling intensity, it presents us with two problems that need resolution. First, why should the effects of child health status be statistically significant but not those of child nutritional status? Second, if suckling stimulus were the mediating mechanism for the purported effects of child nutritional and health status, we would expect the effects of the indicators for child's nutritional status to be attenuated after controlling for indicators that proxy for suckling intensity. But this does not take place in our case: results of models (not shown) that include indicators for child health and nutritional status but exclude the indicator of high supplementation, our proxy for suckling intensity, are virtually identical to those in model 4. This means one of two things: either frequency of breastfeeding episodes is a very weak or coarse indicator of suckling intensity, or the mediating mechanism through which child health and child nutrition operates is a different one.

Finally, we come to the examination of the less explored hypothesis about the existence of mechanisms that, responding to external stresses (work, exercise, psychological hardship), could generate a hormonal environment unfavorable to ovulation. As the argument goes, several forms of physical and psychological stress could disrupt the pulse generator neurons in the hypothalamus by triggering the release of endogenous opioids or other hormones which, in turn, suppress the release of GnRH and LH required for normal menstrual cycles (McNeilly et al, 1985; Prema et al., 1981; Lunn et al., 1981; Rosetta, 1989; Ellison et al., 1989). If this were so, we would find delays in resumption of menses among women who are exposed to such stresses. In our model we use women's work pattern as a proxy for energy-demanding activities. Admittedly this is not an ideal indicator to capture the degree of physical stress associated with work activities nor of stress in

general. However, if the associated effects are sufficiently large, we should identify some regularities. The most important finding is that the estimated effects in model 4 indicate that the risk of resuming menses in women who are engaged in agricultural activities is only two thirds (exp(-.444)) of the risk to which women who do not work outside the home (housewives) are exposed. This effect is what remains **after** controlling for breastfeeding behavior and nutritional status and is statistically significant. On the other hand, women who work in skilled occupations or as merchants experience somewhat higher risks but the magnitude of the differences are not statistically significant. Women who work in agricultural activities may have heavy workloads and expend considerable amounts of energy in daily activities while women who work in skilled jobs, trade activities, or as housewives tend to expend less energy during the postpartum period. Housewives may get help from her older children or other relatives during this period thus being able to reduce the load of strenuous physical activities involved in cleaning, carrying water and firewood, and washing clothes (Mejia, 1972).

These results are similar to those found in the Ngaglik Study from Indonesia. In that study, rural Javanese women who worked hard and expended considerable amounts of energy in a rice-based agricultural system had significantly reduced risk of resumption of menses compared to women who only worked as housewives or in less strenuous trading activities after controlling for breastfeeding and nutritional status.

Although these results are consistent with the main hypotheses formulated before, it is important to note that some or all of the observed effects of women's work activity could also be attributable to the contribution of patterns of breastfeeding associated with occupational categories which are only imperfectly or partially reflected in our measures of breastfeeding patterns. Under this interpretation, the observed effects of mother's work is a result not of the influence of energy expenditures but of the associated pattern of lactation that is poorly approximated by the indicators designed to identify it. However, since the results are consistent with those of the Ngaglik study and

because breastfeeding and nutritional variables are controlled for, these results point to work and energy expenditure as being important independent factors in the process of resumption of postpartum fecundity.

b.2. A two-state model with unmeasured heterogeneity.

All models specified up to now assume that all relevant covariates involved in the resumption of menses are measured correctly. However, as indicated before, some of the results we obtain can only be explained by invoking the role of omitted characteristics.[11] In particular, the persistently positive duration dependency found in all models may be attributable to unmeasured factors that increase the risk of resumption of menses. Some of these unmeasured factors may be related to and others may be independent of the main characteristics included in our models. Regardless of which of the two cases applies, their influence on the process may, in all likelihood, lead to biased estimates of the effects of the measured covariates.

Although there are procedures to handle unmeasured heterogeneity, they are subject to two fairly strict constraints. The first is that they are only suited to assess the degree of sensitivity of estimates to the presence of unmeasured covariates not to provide us with best estimates. The second constraint is that they only apply under two fairly restrictive assumptions, namely, that the unmeasured covariates are independent of those included in the model and that they are time-invariant (Trussell and Rodriguez, 1990; Trussell and Richards, 1985; Heckman and Singer, 1982).

To estimate models with unmeasured heterogeneity we adopt procedures suggested by Heckman and Singer (Heckman and Singer, 1982) which are fully implemented in CTM (Continuous Time Models), a program designed to estimate general families of hazard models (Yi, Walker and Honore, 1986). To simplify numerical estimation we modify slightly our model and assume a two-parameter Gompertz function for the baseline hazard instead of a piecewise exponential model.[12] Table 3 displays estimates from two parametric hazard models, one without and the other with

control for unmeasured heterogeneity. The estimates in the first column of the table correspond to a parametric model with no heterogeneity. A comparison of these estimates with those from model 4 in Table 1 reveal, as expected, that they are virtually identical to each other except for those associated with the duration structure of the model. The second column of Table 3 displays the estimates that result after assuming the existence of unmeasured characteristics that partition the sample into two subgroups.[13] Comparing columns 1 and 2 reveals that our estimates are fairly robust to the postulated unmeasured heterogeneity. Note that for the most part estimates are very stable but that some become larger and in some cases they reach statistical significance. This is the case of one of the measures of breastfeeding frequency (medium frequency) and of two of the measures of maternal health (body mass and head circumference). In all other cases, however, stability seems to prevail. To summarize: the test of sensitivity we have just performed suggests that the estimates from model 4 are robust at least to a class of unmeasured characteristics (independent and time-invariant).[14]

b.3. A comparison of results.

Although comparing results obtained from our re-analysis of the INCAP data with those obtained with alternative data sets is admittedly a difficult and delicate exercise due to variability in samples, model specification, and construction of indicators, we proceed to do so since the results could be illuminating. This comparison could not be performed with previous analyses of the INCAP data due to the nature of the statistical procedures that have been applied so far.

The pertinent figures are displayed in Table 4. The first comparison involves estimated risks of resuming menses under a regime of partial breastfeeding (complemented with some liquid or solid foods) relative to full breastfeeding. The first column of Table 4 displays estimated relative risks in the five most recent studies, all of which benefit from the application of hazard analysis. What is remarkable is the closeness of the estimates: in all cases the corresponding effects are statistically significant and cluster within a very small range (1.75-2.38), with our estimates falling somewhat in

between the extremes of the range. The second result in the table is that the effects of the measures of maternal nutritional status, though incomparable because of different metrics, are always statistically significant. This is all the more remarkable considering that in some cases there is control for suckling stimulus and in most cases such control is lacking. Table 4 is far from being the reflection of a rigorous meta-analysis but it strongly suggests that the relations involved are remarkably regular and constant across social and cultural contexts.

b.4. Fertility and the inhibiting effects of lactation, nutrition, and health.

Although the estimates from the simple two-state model are informative, they do not tell us much about the ultimate effects on fertility of changes in lactation or maternal nutritional status. Due to non-linearities embedded in the estimated models, the only way to address this issue properly is through simple though tedious numerical calculations.[15]

In what follows, our goal is to evaluate the **potential** relative effects that lactation choices have on a woman's lifetime fertility--if she chooses to lactate for shorter (longer) periods of time, or to lactate with lower (higher) frequency, or to establish a feeding pattern conducive to less (more) intense suckling. These effects are then compared with effects on lifetime fertility associated with other conditions, such as maternal nutritional status and maternal health status. We assume several subpopulations of mothers who differ **only** in terms of (a) typical length of lactation; (b) frequency of breastfeeding; (c) child supplementation; and (d) mother's nutritional status. For each of these subpopulations, a corresponding unique median length of postpartum amenorrhea can be calculated from the predicted survival functions associated with the estimated effects from Table 2 (model 4). Once the median length of amenorrhea for the subpopulation is known and assumed to prevail throughout the woman's childbearing period, we calculate the expected number of children that a typical woman from the subpopulation will have by the end of her childbearing period. Table 5 displays the main results in the form of proportional reductions (increases) in total fertility associated

with selected categories of the variables involved. For example, when a mother chooses to fully breastfeed up to the sixth month and thereafter shift to a pattern of partial breastfeeding (lactation pattern 2), her fertility will increase by 21 percent relative to what she would have experienced had she chosen to fully lactate past the 18th month (lactation pattern 1). If she chooses to fully lactate up to the sixth month and then terminate lactation (lactation pattern 3), her total fertility will be 48 percent higher than if she had adopted the more traditional pattern. Similarly, if a woman combines high frequency of lactation up to the sixth month with low frequency from the sixth month onward, her lifetime fertility will be 6 percent higher than if she had breastfed with high frequency throughout the first and one half year of life. Analogous interpretations apply to all figures in the remaining panels of the table.[16]

An important feature apparent from Table 5 is that the effects of lactation patterns are by far the strongest and are followed at some distance by those associated with frequency of breastfeeding. The effects of intensity of breastfeeding and maternal nutritional status are fairly similar to each other but both are of relatively limited import. Thus, for example, if a traditional pattern of breastfeeding characterized by long durations and high frequency of breastfeeding is replaced by a more modern pattern with a combination of full and partial but low frequency breastfeeding, one would expect an increase of total fertility of the order of 1.35 (1.21*1.12). The increase that would be experienced if, in addition, high intensity leads to low intensity is 1.41. This means that the increase explained by breastfeeding intensity is only about 3.9 percent of the total increase ((1.41-1.31)/1.41). Note that the ratio of increase in total fertility attributable to potential improvements in maternal nutrition are of equally small magnitude and will probably never exceed 1.05.

The figures calculated above correspond to extreme scenarios whereby **all** women shift from one lactation pattern to another and from one nutritional status to another. A more realistic assessment of potential changes should take into account the fact that not all women would

experience the same changes in nutritional status or lactation behavior. Since different subpopulations will experience staggered transitions, the upward forces on fertility are of lesser magnitude than what is implied by the calculations discussed before.

SUMMARY AND CONCLUSION

The contribution of this paper is to show that application of simple hazard models on an expanded INCAP data set provides estimates of effects on the timing of resumption of menses that are (a) comparable to others discussed in the recent literature and (b) generally consistent with hypotheses relating patterns of lactation, maternal nutritional status, and maternal stressors to processes that accelerate (decelerate) resumption of anovulatory cycles.

A first finding confirms the importance of length of full breastfeeding as an inhibitor of ovulation. These effects are ubiquitous, strong, and translate into important reductions of levels of lifetime fertility. The magnitude of these effects as well as their statistical significance are similar to those reported in different social and cultural contexts. A second finding reveals that recent insistence on frequency and intensity of breastfeeding (McNeilly et al., 1985) is not misplaced and that indeed, frequency and intensity of suckling operate to enhance the inhibiting effects of breastfeeding. A third finding regards the role of women's stress as a factor that delays menses. Although our measurement of stressful activities (pattern of work associated with occupations) is far from perfect and could be contaminated, our models suggest that this factor may have an importance that has not been heretofore recognized. A fourth finding indicates that although the **direct** effects of maternal nutrition are measurable and, in some cases attain statistical significance, they do not translate into more than weak influences on levels of fertility. Furthermore, the **direct** effects of maternal nutrition are only slightly smaller than the **gross** effects, particularly those that obtain after controlling for child's health and nutritional status and suckling stimulus. This suggests that maternal nutrition operates through

mediating mechanisms other than those involving child's need to suckle more intensely.

A negative finding relates to maternal and child health status. We find that there is no evidence to support the idea that mother's or child's health status affects the process of resumption of menses. But in this regard at least, our study is not alone: maternal and child morbidity have never been reported to be a significant determinant of postpartum menses either with or without control for mechanisms that proxy for pathways through which the effects take place (Kurz, 1990). And yet, there is reason to suspect that morbidity may affect the risk of resuming menses by either increasing or decreasing the amount of suckling stimulus, depending on the severity and duration of the illness spell. In this study, however, the gross **and** net effects of maternal and child health are unimportant.

Finally, the paper provides information about model specification; namely, we demonstrate that our results are robust to control for a sub-class of unmeasured covariates. Although the assumptions of the model are restrictive, it is comforting to verify that estimated effects are not vulnerable to the influence of at least a subset of unmeasured characteristics. This confirms a result obtained more recently by other researchers working with analogous procedures on a different data set (Popkin et al., 1993).

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Table 1
Frequency distribution of child-woman records by socio-demographic characteristics and size of the sample. Guatemala, 1967-1976.

Socio demographic Characteristics	Full Sample		Reduced Sample	
	N	%	N	%
PARITY				
One child	250	17.6	104	13.7
2 or 3 children	421	29.7	226	29.8
4 or more	746	52.6	428	56.5
MOTHER'S AGE				
13-19 years	168	11.9	72	9.5
20-29 years	722	51.0	371	48.9
30-49 years	527	37.2	315	41.6
MOTHER'S EDUCATION				
0 or 1 year	394	27.8	220	29.0
2 or more	791	55.8	462	60.9
Missing	232	16.4	76	10.1
MOTHER'S WORK PATTERNS				
Housewives	839	59.2	482	63.6
Agric. and manual workers	74	5.2	36	4.7
Merchants, skilled workers	264	18.6	163	21.5
Missing	240	16.9	77	10.2
TYPE OF SUPPLEMENTATION				
Atole	765	54.0	388	51.2
Fresco	652	46.0	370	48.8
FREQUENCY OF BREASTFEEDING				
Low (1-7 times/day)	198	14.0	198	26.1
Medium (8-11 times/day)	345	24.3	345	45.5
High (12 + times/day)	215	15.2	215	28.4
Missing	659	46.5	—	—
MOTHER'S NUTRITIONAL STATUS (HEAD CIRCUMFERENCE)				
Low: (00.00-50.19 cms.)	247	17.4	158	20.8
Medium: (50.20-51.04 cms.)	249	17.6	172	22.7
High: (51.05 cms. +)	499	35.2	341	45.0
Missing	422	29.8	87	11.5
DURATION OF AMENORRHEA POSTPARTUM				
Censored	370	26.1	229	30.2
Observed	1047	73.9	529	69.8
TOTAL	1417	100.0	758	100.0

Source: INCAP Longitudinal Study.

Table 2
Covariate effects* for the transition to menses in two-state piecewise exponential hazard models.

Covariates	Model 1	Model 2	Model 3	Model 4
Constant	-3.757	-3.793	-4.324	-4.199
	(0.00)	(0.00)	(0.00)	(0.00)
6 -9	0.430	0.446	0.404	0.403
	(0.00)	(0.00)	(0.00)	(0.00)
10-13	1.341	1.393	1.232	1.224
	(0.00)	(0.00)	(0.00)	(0.00)
14-17	1.905	1.993	1.630	1.616
	(0.00)	(0.00)	(0.00)	(0.00)
18 +	1.975	2.161	1.582	1.572
	(0.00)	(0.00)	(0.00)	(0.00)
BACKGROUND VARIABLES				
One child (par1)		0.241	0.301	0.307
		(0.02)	(0.00)	(0.00)
Four or more children (par2)		-0.140	-0.208	-0.206
		(0.09)	(0.01)	(0.02)
13-19 years (age1)		0.355	0.150	0.131
		(0.00)	(0.19)	(0.26)
30-49 years (age3)		-0.060	-0.090	-0.093
		(0.44)	(0.25)	(0.24)
0 or 1 year (educ1)		-0.312	-0.267	-0.265
		(0.00)	(0.00)	(0.00)
Atole village		0.310	0.242	0.241
		(0.00)	(0.00)	(0.00)
BREASTFEEDING VARIABLES				
Partial (Bf2)			0.809	0.803
			(0.00)	(0.00)
Weaning (Bf3)			2.375	2.381
			(0.00)	(0.00)
Infant death (Bf4)			3.706	3.687
			(0.00)	(0.00)
Medium frequency (8-11 times)			-0.166	-0.161
			(0.13)	(0.14)
High frequency (12+ times)			-0.592	-0.594
			(0.00)	(0.00)

* p-values are shown in parentheses

Table 2 (continued)
Covariate effects* for the transition to menses in two-state piecewise exponential hazard models.

Covariates	Model 1	Model 2	Model 3	Model 4
High supplement (child)			0.181	0.191
(Low intensity nursing)			(0.09)	(0.07)
MOTHER'S NUTRITION				
Bmi>20			0.102	0.107
			(0.32)	(0.29)
Mhc<50.19 (low nutrition)**			-0.179	-0.156
			(0.07)	(0.11)
50.20 < Mhc < 51.04 (medium)			-0.081	-0.071
			(0.40)	(0.46)
High supplement (mother)			0.255	0.251
(high nutrition)			(0.01)	(0.01)
CHILD'S NUTRITION				
Poorly nourished			0.154	
			(0.11)	
MOTHER'S HEALTH				
No morbidity (healthy)			0.105	
			(0.40)	
CHILD'S HEALTH				
No morbidity (healthy)			-0.222	-0.220
			(0.01)	(0.01)
ENERGY EXPENDITURE				
Agric. and manual workers(work2)		-0.394	-0.454	-0.444
		(0.01)	(0.00)	(0.00)
Merchants, skilled workers(work3)		0.127	0.069	0.087
		(0.12)	(0.42)	(0.31)
log L	-3673	-3620	-3182	-3188
Chi Square	624	730	1606	1594
df	4	14	35	31
N	1417	1417	1417	1417

* p-values are shown in parentheses
** Mhc= mother's head circumference (in cms.)

Table 3
Covariate effects* for the transition to menses in two-state Gompertz hazard models without and with heterogeneity

Covariates	without heter.	with heter.
Constant	-4.065	-6.618
	(0.23)	(0.34)
Slope	0.075	0.125
	(0.01)	(0.01)
BACKGROUND VARIABLES		
One child (par1)	0.279	0.504
	(0.10)	(0.12)
Four or more children (par3)	-0.189	-0.167
	(0.09)	(0.10)
13-19 years (age1)	0.148	0.068
	(0.11)	(0.13)
30-49 years (age3)	-0.124	-0.054
	(0.08)	(0.10)
0 or 1 year (educ1)	-0.256	-0.294
	(0.08)	(0.09)
BREASTFEEDING VARIABLES		
Partial (Bf2)	0.936	0.696
	(0.15)	(0.15)
Weaning (Bf3)	2.539	2.461
	(0.16)	(0.17)
Infant death (Bf4)	3.718	3.625
	(0.18)	(0.21)
Medium frequency (8-11 times)	-0.089	-0.287
	(0.12)	(0.14)
High frequency (12+ times)	-0.509	-0.782
	(0.13)	(0.15)
High supplement (child)	0.170	0.181
(Low intensity nursing)	(0.12)	(0.13)
MOTHER'S NUTRITION		
Bmi>20	0.063	0.143
	(0.11)	(0.12)
Mhc<50.19 (low nutrition)**	-0.142	-0.223
	(0.10)	(0.12)
50.20 < Mhc < 51.04 (medium)	-0.059	-0.138
	(0.10)	(0.12)

Table 3 (cont'd)
Covariate effects* for the transition to menses in two-state Gompertz hazard models without and with heterogeneity

High supplement (mother)	0.302	0.308
(high nutrition)	(0.11)	(0.12)
CHILD'S HEALTH		
No morbidity (healthy)	-0.277	-0.238
	(0.09)	(0.09)
ENERGY EXPENDITURE		
Agric. and manual workers(work2)	-0.607	-0.610
	(0.12)	(0.15)
Merchants, skilled workers(work3)	0.069	0.254
	(0.09)	(0.11)
Atole village	0.218	0.282
	(0.08)	(0.09)
Factor Loading	—	2.347
		(0.20)
log L	-3301	-3271
N	1417	1417

* standard errors are shown in parentheses
** Mhc= mother's head circumference (in cms.)

Table 4
Comparison of estimated effects of breastfeeding and maternal nutrition

STUDY	BREASTFEEDING[a]	MATERNAL NUTRITION[b]
Bangladesh (MATLAB) (Ford and Huffman, 1993)	1.75 ($p<.01$)	significant at $p<.01$
Phillipines (CEBU) (Popkin et al., 1993)	2.36 ($p<.01$)	significant at $p<.01$
Indonesia (NGKALIK) (Jones and Palloni, 1993)	2.38 ($p<.01$)	significant at $p<.01$
Pacific Islands (Bolwis and Regni, 1993)	1.72 ($p<.01$)	not available

[a] Relative risks associated with partial breastfeeding (relative to full breastfeeding)
[b] Significance of estimated effects of measures of maternal nutrition (head circumference, weight for height, skinfold, weight at onset of pregnancy, supplementation).

Table 5
The effects of lactation and maternal nutrition on lifetime fertility.(+)

	Median Amenorrhea Period (A_i)	Proportionate Increase in TFR (C_i)
Pattern of Lactation		
Full	21.78	—
Full/Partial	14.55	1.21
Full/Wean	8.74	1.48
Total Wean	2.0	1.96
Frequency of Lactation		
High	13.50	—
High/Low	11.75	1.06
Low	10.12	1.12
Intensity of Lactation		
High	11.40	—
Low	10.30	1.04
Maternal Supplementation		
None	11.11	—
Full	9.78	1.04
Nutritional Status (Maternal Circumference)		
Low	12.00	—
High	10.90	1.03

(+) See text for description of procedures and definition of variables.

ENDNOTES

1. Direct measurements of the postpartum anovulatory period are rarely available in large scale surveys due to the difficulty in observing and detecting ovulation. Instead, it is conventionally accepted to rely on a more well defined and observable but seldom perfectly accurate event, the resumption of menses after a birth. In this, as in other studies on the same theme, resumption of postpartum menses is an indicator of return of fecundable ovulation.

2. The number of observations used in previous analyses of these data varies widely. Thus, Delgado et al. (1978) studied 438 births, Delgado et al. (1982) used 806 births, Bongaarts and Delgado (1979) relied on 438 births whereas Kurz and colleagues (1993) used only 339 births. In all prior studies, cases with missing information and births which resulted in an infant death prior to the end of the first year of life were discarded and not included in the analyses.

3. It can be argued that a more appealing representation of the process requires a multi-state model such as the one in Figure 1. The advantage of this representation is that it captures better the various pathways through which a particular background determinant may operate to influence return to menses. For example, the **total** effects of maternal nutritional and health status on the timing of return to menses are a combination of their effects on various sets of transitions: a) from full breastfeeding to menses; b) from full to partial breastfeeding and then to menses; c) from full to partial, from partial to weaning and then to menses; d) from full to weaning and then to menses and, finally, e) from full breastfeeding to infant mortality and then to menses. A two-state model enables us to estimate the total effects but does not help us to reveal the composition of these effects. In other words, the two-state representation is a reduced form model of the process, whereas the multi-state model is the structural model of the process. The disadvantage of a multi-state representation is that it is considerably less parsimonious, it depends on the assumption of independence of risks and, without explicit constraints, requires a larger number of parameters to be fully described. A fuller formulation and estimation of a multi-state models have been pursued elsewhere (Pinto, 1995; Habicht et al., 1985).

4. Although we experimented with alternative functional representations, we will only discuss two formulations of the baseline hazard. The first corresponds to a Gompertz model, that is, $h_o(t)=b_o*\exp(b_1*t)$. This functional specification can be obtained by setting R=1 and $\lambda_{rjk}=1$ in equation (2) (Heckman and Singer, 1984; Heckman and Walker, 1987). The piecewise exponential hazard function is expressed as follows:

$$h_o(t)\} = \begin{matrix} b_o \exp(b_j), & t_{j-1}<t\le t_j \ , \ j=1,2,...,k \\ b_o, & t\le t_o \end{matrix}$$

(see also footnote 6.)

5. We also fitted models with various interactions among the independent variables but their addition did not improve significantly the overall fit of the model.

6. To determine the duration structure of the piecewise exponential model we proceeded as follows: first, we estimated the hazard of resuming menses for every single month during the first 24 months after birth. We then collected together monthly intervals into segments within which the hazards were approximately constant. After some experimentation we settled on a five(month)-segment model: 0-5 (residual category), 6-9, 10-13, 14-17, and 18 and more (in completed months). See footnote 4.

7. To reduce cluttering we do not display the coefficients for the omitted category and the category corresponding to missing values.

8. We also estimated models to test the hypothesis that both weaning and infant death had effects that changed over time. The results (not shown) indicate that this is in fact the case, and that both events exert lesser influence as time goes by. However, the pattern of effects over time is somewhat erratic and the effects, though properly signed, are not always statistically significant.

9. Reinforcing the idea that nutritional supplementation has important influence is the fact that the estimated effects of the *atole/fresco* variable is significant and properly signed.

10. It should be remembered that Kurz uses a sample of about 300 women for whom information was collected mostly during the period 1973-1977.

11. There are two possible sources of unmeasured heterogeneity in this study, and both are related to the existence of unobserved person-specific characteristics. The first one results from the measures of intensity, frequency, and supplementation of breastfeeding. Imperfect measures of these dimensions could bias the estimates of lactational behavior which in turn, could translate (among other things) into a bias in the baseline hazard. Indeed, a positive duration dependency may be a reflection of poor proxies for various dimensions of lactation behavior. The second source of heterogeneity is each woman's innate healthiness and fecundity. Its effect over the underlying biological process is straightforward: with the passage of time the more fecund women are selected out of the sample as they resume ovulation sooner and leave behind the less fecund ones, whose waiting times to resumption of menses are longer and whose risks are lower. The net result of this bias is to impart a downward bias to the baseline hazard and thus to yield the appearance of a decreasing hazard of returning to menses. The fact that the observed duration dependency is positive indicates that the biases due to this second source are of lesser import than those associated with the first source of heterogeneity.

12. Indeed, very little is lost by replacing the piecewise exponential representation and using instead a Gompertz function. With only two (rather than four) parameters, the latter describes well the monotonic increase of the risk of resumption of menses.

13. This formulation corresponds to a model with unmeasured non-parametric heterogeneity with two points of support. More general models assuming a larger number of points of support could not be estimated with the data.

14. The fact that the estimated slope of the Gompertz function is significantly different from 0 suggests that there are unmeasured characteristics (not belonging to the sub-class we are attempting to control for) that imparts a positive duration dependence to the baseline hazard.

15. Simulation can be avoided only by making simplifying assumptions that are somewhat unrealistic and that compromise the integrity of final estimates.

16. The figures in Table 5 were estimated as follows. First, we calculate the joint distribution of women by age, parity, education, work activity, village type, and child health status for each of the time segments used in the estimation (see Table 2). Second, for each segment and cell of the joint distribution we estimate the hazard of return to menses. Combining the joint distribution with the estimated hazard leads to an estimate of the average hazard corresponding to the residual categories of each of the **other** variables considered in the model, namely, those proxying for lactation pattern, frequency, and intensity as well as mothers' nutritional health status. We then add the effects associated with each of these variables to obtain the hazards by pattern of lactation, frequency, intensity, and woman's nutritional status. From the reconstructed integrated hazards we estimate median waiting times to first menstruation for each of the subpopulations induced by these categories, M_i. Finally, this quantity is used to calculate the fertility inhibiting effect of the pattern of lactation,

C_i. This is done according to the expression suggested by Bongaarts, $C_i=20/(18+M_i)$ (See Bongaarts, 1978)

APPENDIX

b.1. Breastfeeding variables

Patterns of breastfeeding are defined according to three different sets of measures: breastfeeding status, which defines the type and duration of lactation; frequency of breastfeeding, which measures the number of times a day a child nurses; and an indicator for intensity of breastfeeding. The first and the third covariates are time-dependent covariates while the second is fixed.

Breastfeeding status. In this study, the child (mother)'s breastfeeding status is represented by a four category time-dependent covariate: full breastfeeding (Bf1), partial breastfeeding (Bf2), weaning (Bf3), and infant mortality (Bf4). The strategy to deal with infant mortality shared by all previous studies is to eliminate mothers whose children die before menses resumes. Dropping mothers who experience an infant death avoids the problem created when variables such as breastfeeding status or breastfeeding frequency are undefined after the death of the child (Rodriguez and Diaz, 1988; Huffman et al., 1987; Delgado et al., 1982; Popkin et al., 1983). In contrast to previous studies, the "effect" of infant mortality should be similar to that of weaning since the death of the infant implies complete termination of breastfeeding.

Frequency of breastfeeding. The second breastfeeding covariate is also time dependent and is represented by four categories: low frequency (1-7 breastfeeding episodes per day), medium frequency (8-11 per day), high frequency (12 or more per day), and a fourth category that collects missing values.

Intensity of breastfeeding. The third breastfeeding covariate, child's clinical supplementation, is an indicator that serves as proxy for intensity of breastfeeding.[1] Admittedly, however, this is a more general indicator that may reflect other characteristics of breastfeeding as well as to stand for child's nutritional status. The indicator is defined as the amount of kilocalories (kcal) per day consumed by

children who attended the INCAP's supplementation centers. Three categories were included: low intake (34.25 kcal. per day or less), high intake (more than 34.25 kcal. per day), and a third category that represents the missing values for this variable.

b.2. Variables for nutritional status.

In order to assess the effects of a woman's nutritional status on resumption of menses, three variables are used: body mass index (BMI), which assesses chronic energy deficiency; mother's head circumference, which measures long term nutritional status; and maternal energy intake, which measures current nutritional status. The first and the third covariates are time-dependent covariates while the second is fixed.

Mother's body mass index. The effects of mother's nutritional status on the resumption of menses in this study is primarily measured by the so-called body mass index, or BMI.[2] In this analysis, we consider three categories: less than 20 (some degree of malnutrition), more than 20 (from normal to obese), and a third category to accommodate missing values.

Mother's head circumference. The second measure of nutritional status, mother's head circumference, is captured by four categories representing low (0-50 cms.), medium (50-51 cms.), high nutritional status (52 cms. and more), and a special category for missing values.

Maternal energy intake. A third measure of nutritional status consists of information about the mother's energy intake from INCAP supplementation. This indicator reflects the amount of supplementation intake and we express it in kilocalories (kcal) per day for mothers who went to INCAP's centers. Three categories were defined: high intake, low intake, and missing values.

Child's nutritional status. To assess child nutritional status we use a very common indicator of child's nutritional status, namely, Z score of height by age (ht/age). Three categories were defined: (Z score ≤ 0) poorly nourished, (Z score > 0) adequately nourished, and one for missing values.[3]

b.3. Health variables

The data gathered on morbidity consist of symptoms of illness that mothers reported for themselves and their children and that correspond to episodes taking place two weeks before the interview. Information is also available on the number of days that women and children experienced some symptoms of illness during the month prior to the survey. Symptoms such as anorexia, apathy or irritability, and fever are considered among the **severity symptoms**. The indicators for mother's and child's health status are the monthly cumulative number of days spent ill. The categorization of these is as follows: no morbidity (zero days of illness), some degree of severity (one or more days of illness). A third category collects missing values.

b.4. Energy expenditure variable

Mother's work patterns. Women's physical labor is captured by identifying work activities they performed on a daily basis. Different types of work activities reflect distinct levels of energy expenditure and, therefore, allow us to differentiate the 'stress-load' they exert on the reproductive system. We expect their effects to be important if the hypothesis that they disrupt the functioning of the neuroendocrine system by affecting the normal release of GnRH is, indeed, correct (Schweiger et al., 1987, 1989). We defined four categories: (i) women who reported no work outside the home (housewives), (ii) agricultural and manual workers, (iii) craftswomen, merchants and skilled workers, and (iv) women who did not provide any information about their occupation. Although the correlation is certainly not perfect, there is a correspondence between these categories and the underlying dimension we seek to tap: the first and the third occupational categories involve non-strenuous work activities and the expenditure of energy is considerably lower when compared to agricultural and heavy manual work, where strenuous physical labor is always present.

b.5. Demographic variables

Age of mother and birth order of child (parity) have important biological and behavioral effects of their own on the menstrual interval. In our analysis, however, we use them as controls for

potentially confounding influences.

Parity. Parity is defined as the number of live births the woman had at the date of the survey, that is, the birth order of the child being studied. The categories are three: (i) women with one child, (ii) women with two or three children, and (iii) women with four or more children.

Mother's age. Mother's age is defined as the last birthday at the time of the birth of the last child. Three groups of women are considered, (i) mothers who were 19 years old or less at childbirth, (ii) mothers who were between 20 and 29 years old, and (iii) those who were 30 years or more.

b.6. Socioeconomic variables

Although socioeconomic characteristics of the woman can be expected to influence resumption of menses primarily through breastfeeding, nutritional and health status, we include them in the model to determine whether they have any residual impact of menses. To the extent that they do, the intermediate variables are imperfectly measured.

Mother's education. Mother's education is defined as the number of years she spent in school. Women were grouped into three categories: (i) women with low or no education (none or one year of formal instruction), (ii) women with a higher education (two or more years in school), (iii) and women with no information about this characteristic.

Mother and child residence. Finally, we take advantage of the INCAP study design and include an indicator of residence that distinguishes mothers who reside in a village where INCAP administered special dietary supplementation (atole villages) from those living in control villages supplied with lower grade supplementation (fresco villages). If supplementation is effective we would expect Atole villages to have, on average, lower waiting times to first ovulation since both maternal nutrition improves and child suckling is reduced.

Table 1 reports basic descriptive statistics for the most important covariates for two different samples: a full and a reduced sample that excludes cases with missing information. The table also

reports information about censoring of the dependent variable, duration of postpartum amenorrhea. According to the figures presented in Table 1, about 26 percent of the birth intervals in the full sample and about 30 percent in the reduced sample contain censored information on duration of postpartum amenorrhea. About 60 percent of the birth intervals in the full sample are from a relatively young group of women but when cases with missing values for breastfeeding and nutrition are removed to generate a reduced sample, the percentage of birth intervals produced by older women increases from 37 to 42. By the same token, birth intervals to mothers of high parity (4 or more children) increase from 53 to 57 percent. Similar changes are observed in the rest of the variables that appear in the table. These results imply that when cases with missing values are dropped, the composition of the sample changes and, in particular, that the resulting (reduced) sample is made up of older and higher parity women.

APPENDIX NOTES

1. The rationale is that among infants who have similar energy requirements, those who have higher intake from non-breastfeeding sources experience lower breast milk intake with the result that the intensity of nursing is reduced and the suckling stimulus to the mother's nipple is lowered, thus favoring an environment that facilitates resumption of menses (Kurz et al., 1993).

2.This index is defined as the ratio of mother's weight to her height squared, that is, kg/m^2. Malnourished women are generally defined as those with a BMI less than 20. This value may be considered a cut-off point of chronic energy deficiency (James et al., 1988).

3. According to WHO guidelines, there are three indicators to measure protein-energy malnutrition: (i) weight by age, (ii) height by age, and (iii) weight by height. The first indicator reflects some aspects of acute and chronic malnutrition; the second one reflects chronic malnutrition influences; the third one reflects acute or current nutritional status. In order to obtain standardized values, the height quotients were transformed into z scores (or standard deviation scores) by subtracting from the individual's value the mean value of the reference population and dividing the entire expression by the standard deviation value of the reference population. In this case, the population of reference is the population under study itself and not the WHO population.

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