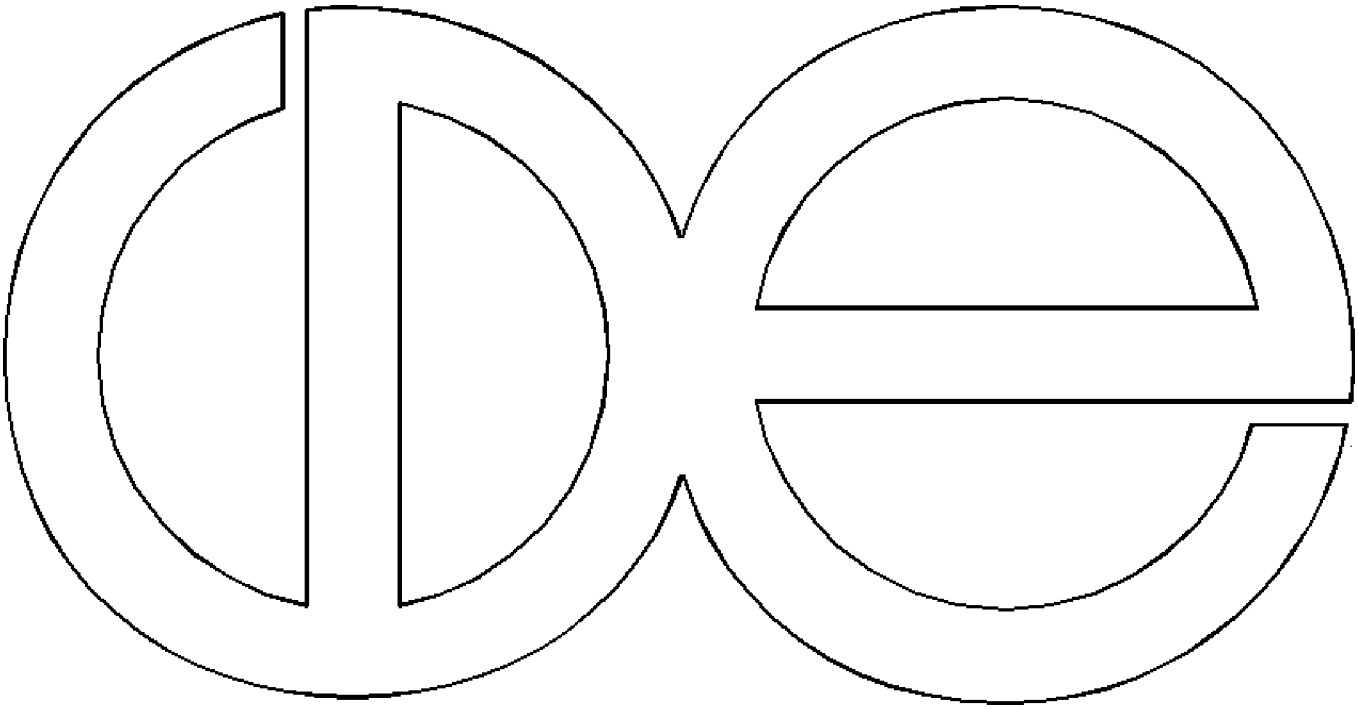
Center for Demography and Ecology

University of Wisconsin-Madison

# The Determinants of Postpartum Amenorrhea: A Multi-State Hazard Approach

**Guido Pinto Aguirre**

**CDE Working Paper No. 96-03**



# THE DETERMINANTS OF POSTPARTUM AMENORRHEA:

# A MULTI-STATE HAZARD APPROACH

Guido Pinto Aguirre

# THE DETERMINANTS OF POSTPARTUM AMENORRHEA:

# A MULTI-STATE HAZARD APPROACH

The objective of this paper is to model the duration of postpartum amenorrhea (i.e., waiting time to resumption of menses) through the use of multi-state hazard models. The proposed model recognizes explicitly the existence of five states (fully breastfeeding, partial breastfeeding, weaning, infant mortality, and menses). Thus, the model depends on several hazards, one for each transition taken into account. The model also considers the role of maternal and child nutrition and health, and women's work patterns as strategic elements in determining the outcome of the process. The unit of analysis is a spell, completed or censored, and every woman contributes with multiple observations to the analysis.

Since a multi-state model is a powerful device for investigating the process of resuming postpartum menses, the results obtained from its estimation provide a better empirical representation of the theoretical model, and the inferences made about the relationships being studied are thus more accurate.

This working paper is organized into several sections. Section one reviews the available evidence on the biological and physiological determinants of postpartum amenorrhea. Section two reviews previous research in this field. Section three is devoted to describing the data used throughout the study and the general estimation strategy of the multi-state hazard approach. Section four contains the analysis of the empirical results obtained. And finally, section five presents the conclusions of the study.

## 1. THE DETERMINANTS OF POSTPARTUM AMENORRHEA

Four mechanisms related to the resumption of normal menstrual cycles in postpartum women can be identified (Jones, 1989; Jones and Palloni, 1990). Two of them, weaning and infant mortality,

have a direct causal effect. They terminate nursing episodes and, therefore, the hormonal stimulus that child suckling has on the delay of fecund menstrual cycles. The third mechanism is related to the breastfeeding patterns prevalent among those mothers who menstruated while they were still breastfeeding. The fourth causal factor, maternal nutrition and health status, may also modify the hormonal stimulus by delaying the resumption of normal menstrual cycles.

There are two possible paths through which nutrition could affect postpartum amenorrhea. The first pathway, deficient nutrition, may have a direct effect on a woman's reproductive system and cause a delay in the resumption of menses. The second path has an indirect effect, and may work through breastfeeding: a malnourished woman may produce reduced amounts of milk so that her child has to suckle more intensely to obtain adequate nutrition. In this way intense nursing delays the resumption of menses.

The effects of the socioeconomic covariates can be expected to operate through one of these four intermediate factors. The failure to distinguish these intermediate mechanisms in any prospective study might lead to the same kind of problems found in retrospective studies; that is, difficulties in interpreting causality and identifying the underlying mechanisms. However, we also have to consider chance factors, which include capacity to breastfeed, propensity to abort spontaneously, and other aspects of biological nature associated with ovarian activity.

The physiological mechanism underlying these observed relations originates in the hypothalamus, which is located in the basal region of the brain. The woman's hypothalamic nuclei release **gonadotropin-releasing hormone** (GnRH) in a regular, episodic fashion, which in turn triggers the pulsatile and episodic release of **luteinizing hormone** (LH) and **follicle stimulating hormone** (FSL) from the pituitary. This biological process is also known as the **hypothalamic pulse generator** (HPG) (see McNeilly et al., 1985). The release of certain amounts of LH have been found to be crucial for normal ovarian activity and fecund menstrual cycles (Glasier et al., 1984; Knobil,

1980; McNeilly et al., 1985). Recent research has also shown that normal patterns of GnRH and LH release can be disrupted by external factors, such as heavy physical exercise or activity, or psychological stress, in addition to breastfeeding (Genazzani et al.,1991; Loucks, 1990). The exact neurotransmitter signals, which interrupt the release of GnRH and LH during these activities, remain to be completely elucidated. In the case of breastfeeding women during the postpartum period, however, the suckling stimulus may be the primary signal which disrupts the normal pattern of hormonal activity (Tay et al., 1992). However, it seems that the disruption of the HPG neurons is a common characteristic of altered GnRH and LH release, and thus of amenorrhea and fertility (Yen, 1987). This general mechanism represents a direct link between the external environment (i.e., stressors) and the internal regulation of the reproductive system (Jones and Palloni, 1994).

## 2. PREVIOUS RESEARCH

The existing body of literature suggests that there is a consistent relationship between breastfeeding and nutrition, on the one hand, and postpartum amenorrhea, on the other hand, even after controlling for potentially confounding factors. The source of data is very heterogeneous; some of these studies are based on aggregate cross-sectional demographic surveys (e.g., Guz and Hobcraft, 1991; Cleland and Hobcraft, 1985; Bongaarts and Potter, 1983; Tyson and Perez, 1978); others rely on clinical studies (e.g., Howie and McNeilly, 1982; Rodriguez and Diaz, 1988); and finally, a small group of them rely on longitudinal studies, such as the INCAP project in Guatemala, MATLAB project in Bangladesh, NGAGLIK project in Indonesia, and CEBU project in the Philippines (Delgado et al., 1978; Lunn et al., 1984; Jones, 1990; Huffman et al., 1987; Santow, 1987; Kurz, 1993; Popkin et al., 1993; Jones and Palloni, 1990).

Many of these studies are important attempts to investigate the role played by either women's nutritional status or women's breastfeeding behavior on the resumption of menses. Only a few studies have examined the simultaneous effects of breastfeeding patterns (duration, frequency, intensity, and

type of breastfeeding) and nutritional status on the risk of resuming fertility after controlling for factors such as work patterns, health status, infant mortality, age, parity, and education (Kurz et al., 1993; Popkin et al., 1993; Jones and Palloni, 1990).

The effect that breastfeeding has on reducing the risk of resuming menses is well documented throughout the demographic and biological literature. The most important conclusion that can be drawn from these studies is that lactation increases the duration of postpartum amenorrhea and that long-term breastfeeding is statistically 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 at progressively slower rates (Santow, 1987; Corsini, 1979; 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 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., Jones 1990; Delgado et al., 1982; Trussell et al., 1989). As it is known, children over one year are more likely to receive significant amounts of supplementary food (liquid and solid).

It is also well established that the relationship between breastfeeding and postpartum amenorrhea depends heavily on the effectiveness of the nursing stimulus (Tyson and Perez, 1978). In fact, when breastfeeding is prolonged and intensive, 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 nutrition affects women's fertility is also present in different ways in the

demographic and biological literature (e.g., Wringley, 1969; Stein and Susser, 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 (Careal, 1978; Chavez and Martinez, 1973; Warren 1980). Second, women often stop their menstrual cycles at times of famine and starvation (Smith, 1947; Stein and Susser, 1975, 1978; Chowdhury, 1978; Keys et al., 1950; Menken, 1979), or when anorexia nervosa, exercise, or stress are present (Wakeling and DeSouza, 1983; Warren, 1980; Pirke et al., 1985; Pirke et al., 1989). However, there is considerable uncertainty about the effects of nutrition on the female's reproductive system in situations where chronic and mild to moderate malnutrition are prevalent, particularly in developing countries (Prentice et al., 1980; Bongaarts, 1980; Huffman et al., 1978; Huffman et al., 1980; Huffman et al., 1987; Delgado, 1978).

Some authors have suggested 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); other researchers suggest that the effect of maternal nutritional status on postpartum amenorrhea seems to be rather small, that maternal undernutrition would be at best a minor determinant of postpartum amenorrhea (Jones and Palloni, 1995; Delgado et al., 1978; Huffman et al., 1978; Huffman et al., 1987; Bongaarts and Delgado, 1979; Chowdhury, 1978), and that there may exist other factors that mediate the effect of nutrition on postpartum amenorrhea (Lunn et al., 1980; Lunn et al., 1984; Kurz et al., 1993). One of these factors seems to be lactation itself. It has been found that children born to malnourished mothers tend to suckle more frequently and intensively to obtain adequate amounts of breastmilk, thus increasing the inhibition of the ovulatory hormones, which lengthens the amenorrheic interval (Salber et al., 1966; Wray, 1978).

**The INCAP study**

Studies 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 raise their total caloric intake during pregnancy beyond 20,000 calories ('highly supplemented' group) had a significantly shorter durations of postpartum amenorrhea relative to those who consumed less than 10,000 calories during pregnancy ('poorly supplemented' group). They were able to establish that the effect of absolute caloric intake during pregnancy was to reduce the length of postpartum amenorrhea by between 1.0 and 1.4 months. In addition, they also found negative correlations, ranging from a low of -.09 to a high of -.14, between various measures of nutritional status assessed at 3 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 more recent 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 reassumption 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 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 reassumption of menses. Furthermore, they also found that maternal supplementation is a very weak determinant once the effect of child's non-breastmilk energy intake is controlled. 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 conducted with INCAP data are certainly important, their results cannot be easily compared with those obtained from more recent research endeavors. This is because just about all of these 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 off-the-mark 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 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.

## 3. DATA AND METHODS

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. Women entered the study on the basis of their reproductive history, i.e., those women who had at least one child aged less than seven years old were considered in the study. Mothers provided data periodically throughout their pregnancy and postpartum period. In this study we retrieved data for all women who had a delivery between 1 January 1969 and 28 February 1977, and who were followed-up until the survey ended officially (28 February 1977). Under this constraint, 608 women and 1430 birth intervals were selected (Pinto, 1994).

### Characteristics of the sample.

Table 1 reports the composition for the most important covariates for two different samples, a full and a reduced sample. 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 of those 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 when missing values are removed. 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, the resulting (reduced) sample is made up of older and higher parity women.

**The dependent variable.**

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.**

The independent variables included in the analysis can be classified into six groups: breastfeeding, 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.

**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 third covariates are time dependent covariates while the second is fixed.

**1. Breastfeeding status**. In this study, breastfeeding status is represented by four time-dependent covariates: full breastfeeding (Bf1), partial breastfeeding (Bf2), weaning (Bf3), and infant mortality (Bf4). In contrast to other studies, the "effects" of infant mortality on resumption of menses are explicitly included in the models. A strategy to deal with infant mortality shared by all previous studies is to eliminate mothers whose children die before menses. This gets around the problem created because 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). In theory, the "effect" of infant mortality should be similar to that of weaning because since the death of the infant implies complete termination of breastfeeding.

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

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

**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. The effects of child nutritional status are assessed by the so-called height by age indicator, which measures protein-energy deficiency among children.

**1. 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. In this analysis, three categories are considered: BMI less than 20 (some degree of malnutrition), BMI more than 20 (from normal to obese), and a third category to accommodate missing values.

**2. 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.), and high nutritional status (52 cms. and more), and a special category for missing values.

**3. Maternal energy intake**. A third measure of nutritional status consists of information about 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.

**4. Child's nutritional status**. To assess child nutritional status we use a very common indicator, height by age (ht/age). Three categories were defined: poorly nourished, adequately nourished, and one for missing values.

**Health variables**

The data gathered on morbidity consists 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.

**Energy expenditure variable**.

**1. 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' these activities exert on the reproductive system. We expect their effects to be important if the hypothesis is, indeed, correct that high energy expenditures disrupt the functioning of the neuroendocrine system by affecting the normal release of GnRH (Schweiger et al., 1987; Schweiger et al., 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.

**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.

**1. 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.

**2. 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 younger at childbirth, (ii) mothers who were between 20 and 29 years old, and (iii) those who were 30 years or more.

**Socio-economic 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 they do the intermediate variables are imperfectly measured.

**1. Mother's education**. Mother's education was defined as the number of years the woman 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.

**2. Mother and child residence**. Finally, we take advantage of the INCAP study design and include an indicator of residence that distinguish 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).

**Modelling strategy**

The data analyzed in this research are characterized by the presence of right-censored observations. This means that there are women that do not have ending times for the duration of amenorrhea, breastfeeding, partial lactation, infant death, either because they were not present during the survey (lost on follow-up) or because they had not experienced the event in question when the survey ended (termination of follow-up). For some women it is only known that their durations exceeded some particular given value, the exact durations are unknown. Because the longer-duration cases are in general more likely to be censored, these data cannot be analyzed by simply removing

such observations. Thus, the method to be utilized in the analysis must use both censored and uncensored observations. One of these methods in event history analysis is the so-called multi-state hazard model.

The reproductive history of a woman can be treated as a collection of random points over time (i.e., a stochastic process) marked by a sequence of events such as births, infant deaths, initiation and termination of breastfeeding, initiation and termination of amenorrhea postpartum, etc. The entire history is thus made up of a finite number of states, say **k**. Furthermore, we assume that a woman stays at every state **j** a random length of time ($T_1$, $T_2$, ..., $T_k$). These random variables are completely characterized by a probability distribution $f(t_j|\mathbf{x})$, where $t_j$ is a realization of $T_j$. One of the basic concepts is that of the "failure" rate or hazard function, $h(t_j)$. The hazard function is basically the instantaneous probability of "failing" at time $t_j$ given that failure has not occurred before $t_j$. Thus, the hazard function is the rate at which events are completed after duration $t_j$ given that they lasted at least until $t_j$.

**Modelling multi-state hazard rates**

The strategy of modelling multi-state hazard rates relies on the separation of different types of events in a way that every woman starts at some point in time (origin state) and moves to another one (destination state). When a particular event occurs, women are removed from the set risk of all other events. Thus, the structure of the process can be identified by a succession of transitions and states which mark the woman's life course.

In this study women are classified into **five** groups (i.e., states), as can be seen in Figure 1, according to the type of event they experienced at every particular time **t** during the follow-up. The first state (fully breastfeeding) contains all women who initiated lactation after the birth of their children. The second state (partially breastfeeding) is made up of those women who were nursing their children and started to introduce supplemental food in their children's diet. The third state

(weaning) contains all women who stopped breastfeeding their children before menses resumed. The fourth state (infant death) is made up of all women whose children died before menses resumed. Finally, the absorbing state (menses) contains all woman who resumed menses at time **t**.

The transitions in this study are defined as follows. Once a woman gets into state 1 (**full**), she is at risk of ending in either state 2 (**partial**), state 3 (**wean**), state 4 (**death**), or state 5 (**menses**). When a woman introduces supplements (state 2) she is at risk of moving to either state 3 (**wean**), state 4 (**death**), state 5 (**menses**). When a woman terminates breastfeeding her child (state 3) she is at risk of ending in either state 4 (**death**) or state 5 (**menses**). Finally, women can move from state 4 (**death**) to state 5 (**menses**)[1].

The transitions from one state to another are governed by a set of **transition rates** with the following general form

$$h_{jk}(t_0,t_j|Z) = h^0_{jk}(t_0,t_j)\exp(\beta Z)$$

where j is the origin and k is the destination state, $t_0$ is the time since the process started (i.e., time since last birth), $t_j$ is the time spent in state j, $\beta$ is a vector of covariate effects, **Z** is the vector of fixed, and time varying-covariates associated with the mother and her child, and $h^0(.)$ is the origin-destination hazard baseline which depends on $t_j$.

The role of the covariates and the baseline hazard in the expression presented above need to be defined more precisely, because the covariates included in the model have different effects on the hazard in each transition, and the behavior of the baseline hazard (i.e., time dependencies) in each transition may be different.

[1] This model rest on the assumption that there are no bidirectional flows among origin and destination states. For instance, once the woman moves from fully breastfeeding to partial breastfeeding, she does not fully breastfeed her children later.

The vector **Z** may contain different covariates depending on the specific transition being analyzed. The risk of moving from **full** to **partial**, **full** to **wean**, and **partial** to **wean** may depend mostly on (1) the woman's past reproductive history (age and parity), (2) characteristics that may affect suckling stimulus (mother's health status, desire to nurse her child, and mother's work patterns), and (3) the use of alternative sources of food (family income, social and cultural norms, and community features).

A different set of covariates may affect the hazard of transiting from **full** to **menses**, and **partial** to **menses**. These covariates are likely to be parity, age, work patterns, frequency and intensity of nursing, and maternal nutritional and health status. All transitions to **death** may depend on child's health, mother's age and parity, frequency and intensity of nursing, and characteristics of the community that determine the availability of health, and basic services. Finally, transitions from **wean** to **menses**, and **death** to **menses** are basically driven by biological factors. Thus covariates such as age and parity may affect in some way the hazard in these two transitions.

In every transition, the baseline hazard may have a different behavior over time, according to the theoretical model presented early in this paper. The hazard for the transitions from **full** to **partial**, **full** to **menses**, **partial** to **wean**, **partial** to **menses**, **wean** to **menses**, and **death** to **menses** is a non-decreasing function of time, whereas the hazard for all the remaining transitions (**full** to **wean**, **full** to **death**, **partial** to **death**, and **wean** to **death**) follows the shape of a non-increasing function of time.

The use of a multi-state model also makes it possible to address some problems encountered in the analysis of birth history data. First, the multi-state model includes all the states and logical flows in the process of resuming postpartum menses, including infant mortality, so the effects of every determinant are defined with precision. For instance, we are able to distinguish cases when weaning occurs because of death from cases when weaning occurs because of other causes. Second, the multi-

state model treats infant mortality in the most efficient and elegant way possible. It avoids questionable solutions that could produce selection bias, non-independent censoring, or cumbersome definitions of the breastfeeding variables. Third, it allows a stochastic treatment of the different waiting times, which have a clear biological significance. This is an important advance with respect to other works in this field, such as the deterministic model to study the resumption of ovulation proposed by Habicht and colleagues (1985), in which breastfeeding is the only determinant of the velocity of resuming menses. Finally, the multi-state model explains the positive duration dependency found in the two-state model (Pinto, 1995).

## 4. ANALYSIS OF THE RESULTS

First, a baseline or "null" model was estimated on the monthly prospective data. Under this specification, we make the assumption that the waiting times for each transition are governed by a Gompertz distribution (shown in Table 2). Second, several Gompertz hazard models with covariates are estimated for each transition, except for the flow from wean to menses, which is assumed to follow an exponential function. Each model includes only additive effects among its covariates, and the baseline hazard rates are assumed to be either monotonically increasing or decreasing function (shown in Table 3). After testing several specifications, we arrived at a more parsimonious model.

The interpretation of the results reported in these tables is straightforward. Since the hazards for all transitions are estimated simultaneously, they can be analyzed as if they were two-state models, after controlling for all the other risks to which women are exposed. Thus the effects of covariates on the hazard have the same interpretation presented in the analysis of the two-state model (Pinto, 1995).

Table 2 presents the results of modelling the different transitions as Gompertz waiting time random variables with no covariates (i.e., with only a constant term in the hazard function). This table reports the transition counts, the estimated transition rates, and the chi-square values for each

transition. As shown in that table, all the slopes are statistically significant, except for the transition from wean to menses, which is better represented by an exponential function. From these results we can easily confirm the direction of the changes in the hazard rates over time. For instance, the baseline hazard rates from full to partial and partial to wean increase over time (positive slopes). The risks in these transitions reflect the time process to which children are subject: as they become older, their nutritional requirements change and mother's milk becomes a less important component in their diets.

We also can see that risks for the transitions whose destinations are menses increase over time. This fact is driven by the progressive decrease in the levels of hormones that inhibit menses. The estimated rates show that women who experienced infant deaths or wean their children move faster to the menses state than women who move to all the other states. This finding agrees with the results obtained in the two-state model (Pinto, 1995), where weaning and infant deaths are treated as time dependent covariates. Finally, as we can see in this table, the risk of transiting from any possible state to death decreases over time. This tendency just reflects the pattern of mortality in early childhood.

It is also important to note in Table 2 that the low number of events in some transitions restrict the complexity of models that can be estimated. The transitions from **full** to **wean** and from **wean** to **death** only have 10 and 16 cases respectively. On the other extreme the bulk of the cases are concentrated in the transition from full to partial (1,262), followed by the transitions from partial to menses (617), partial to wean (312), and wean to menses (261).

The final model is presented in Table 3. The estimates of this model include the effects of breastfeeding, nutritional, health and background variables, some of which are time dependent, others fixed. Listed across Table 3 are 10 columns of estimates. The first column reports estimates for the transition from fully breastfeeding to partial breastfeeding, the second column is the transition from fully breastfeeding to wean, and so on.

As we can see in Table 3, after controlling for all the relevant covariates in the model, the

duration dependencies in each transition remain significantly different from zero and in the proper direction. The fact that a positive duration was found in the transitions from **full** to **menses**, and **partial** to **menses** means that the risk of resuming menses associated with these two breastfeeding variables increases, net of all the other factors. These results support the notion of the existence of an underlying "aging" process in the inhibitory mechanism that leads to resumption of ovulation, regardless of breastfeeding status (McNeilly et al., 1985). The model also reveals the existence of an important duration dependency when women transit from **partial** breastfeeding to **wean**. The "aging" effect is again relevant to this transition.

According to the estimates in Table 3, the transition from **full(1)** to **partial(2)** is mainly determined by breastfeeding variables, mother's health status, work patterns, and community characteristics. It is important to remember that the decision to introduce supplements into the child's diet is always culturally conditioned. It is also affected by the family's income, and the ability and willingness of the mother to continue nursing. For instance, illness or the type of work women in which are engaged could motivate them to introduce supplements. The results show that women who are healthier and younger tend to wean more slowly than those with health problems or those who are older. The effects of work are somewhat counterintuitive. For instance, the results suggest that women who work outside of the home move less quickly to supplements, while housewives are more likely to introduce supplements earlier.

Mother's age and child health are relevant to the transition from **full(1)** to child's **death(4)**. In fact, the risk of moving to the death state increases for old women and mothers with an ill child when compared to young women and mothers with a healthy child. This means that among those children who died during the longitudinal study, the healthier children tended to die later than the more frail ones.

The risk of moving from **full(1)** to **menses(5)** is primarily determined by mother's age, parity,

education, frequency of breastfeeding, maternal nutrition, and child's health. These effects are statistically significant. The estimates show that high parity women, young women, and women with no education tend to resume menses faster than their low parity, old, and more educated counterparts. Also, those women who nurse on demand resumed menses slower than those who nurse more infrequently; and mothers with low nutrition (measured by mother's head circumference) tend to have a delayed resumption of menses when compared to those with better nutrition, after controlling for all the other variables.

The transition from **partial(2)** to **wean(3)** is mainly driven by mother's age, parity, type of community, mother's supplement, and child's nutritional status. According to the results in Table 3, those children who are poorly nourished are weaned earlier, similar results were found in other study (Jones and Palloni, 1990). Not surprisingly, high parity and young women tend to wean their children before they resume menses.

The transition from **partial(2)** to **menses(5)** has particular importance, because it resembles the transition from amenorrhea to menses in the two-state model, purged of all other determinant or causal mechanisms that lead to menses (i.e., weaning and infant death). One important finding is that women's work (energy expenditure) is a powerful inhibitor of menses, even after controlling for breastfeeding patterns and nutritional status. In fact, strenuous work (i.e., agricultural and heavy manual work) reduces considerably the risk of resuming ovulation.

Breastfeeding variables are significant in determining the resumption of menses among those mothers who are partially breastfeeding. Women who breastfeed their children fewer times a day resume menses faster and those who breastfeed on demand have a delayed resumption of menses. Similarly, low intensity of lactation (measured by high amounts of supplement given to the child) increases the likelihood of resuming menses.

In this transition, mother's BMI is highly significant and in the expected direction. Mothers

with a BMI greater than 20 resume ovulation faster than those with a lower BMI. By the same token, mothers who receive high amounts of supplements resume menses faster. We observe also that the fixed covariates are significant after controlling for breastfeeding, nutritional, and health variables.

The transition from **wean(3)** to **death(4)** is determined mainly by child's health status. This means that those children who have health problems are more likely to die once they are weaned. Infants who die may have experienced a period of poor health, and they may have been weaned later precisely because of their health. In this case resumption of ovulation is more a response to weaning rather than to the termination of the inhibitory process triggered by the death of the child.

Finally, in the transitions from **wean(3)** to **menses(5)** and from **death(4)** to **menses(5)** no covariates have significant effect on the hazard of resuming menses. This result was certainly expected because wean and death are the direct causal mechanism that lead to menses; that is, once the child is weaned or the child dies, biological factors take over the process. It is important to realize that this fact also remains hidden in the two-state model.

One important result that we can extract from this analysis is that the inhibiting effects of maternal nutritional status on menses is reinforced when the women are fully (1) or partially (2) breastfeeding, but not to the same extent. These differential effects could be associated with the fact that suckling stimulus may be stronger for malnourished mothers than for well nourished mothers (Lunn et al., 1981).

## 5. CONCLUSIONS

The major conclusion of this study is that the use of a multi-state model provides an improved picture of the resumption-of-menses process than that obtained from a two-state model (Pinto, 1995). Furthermore, the results from the multi-state hazard model are consistent with those found in the two-state model.

The results obtained from the multi-state model are in general agreement with those found

in demographic studies and with the physiological evidence suggesting that resumption of ovulation, and thus resumption of menses, depends on a particular hormonal balance, which in turn depends on suckling stimulus, energy expenditure, and woman's nutritional status (Jones and Palloni, 1990; Huffman et al., 1987; Popkin et al., 1993).

The multi-state model confirms the importance of breastfeeding variables and infant mortality in the resumption of menses. They also suggest that the inclusion of infant death is of nontrivial significance.

The effects of nutritional status (measured by mother's BMI) are stronger, significant, and in the expected direction, after controlling for breastfeeding and woman's work patterns.

The effects of woman's work pattern and the implied level of energy expenditure on the resumption of menses are significant and in the expected direction. However, they operate only when women are fully and/or partially breastfeeding.

The significance of the duration dependencies in the two-state model are not an artifact of the data but the result of active breastfeeding in certain transitions. That is, the multi-state model shows the presence of changes over time in the effects of partial and full breastfeeding (relative to weaning and infant death). This suggests the existence of an "aging" effect.

Finally, the results obtained in this paper show that the multi-state model represents the process being studied more accurately than the two-state model (reduced form) in the following sense: In the two-state model, every woman is assigned to the same risk set, no matter how different their biological propensities to resume menses are. For instance, women whose children died are more likely to resume menses than those who are fully breastfeeding. Since these facts are "invisible" in the two-state model, it treats women that have different propensities and pathways to reach menses state equally. Instead, the multi-state modelling strategy treats them differently, accounting for aggregated or state-specific heterogeneity.

## REFERENCES


Allison, P.D. 1982. Discrete time methods for the analysis of event histories, pp. 61-98. In : S. Leinhardt (ed.), *Sociological Methodology*. San Francisco: Jossey-Bass.

Blossfeld, H.P., A. Hamerle and K.U. Mayer. 1989. *Event History Analysis: Statistical Theory and Application in the Social Sciences*. Hillsdale: Erlbaum.

Bongaarts, J. 1983. The proximate determinants of natural marital fertility, pp. 103-138. In: R.A. Bulatao and R.D. Lee (eds.), *Determinants of Fertility in Developing Countries. Vol. 1. Supply and Demand for Children*. New York: Academic Press.

Bongaarts, J. and Delgado, H. 1979. Effects of nutritional status on fertility in rural Guatemala. In: H. Leridon and J. Menken (eds.), *Natural Fertility: Patterns and Determinants of Natural Fertility*, pp. 109-132. Liege, Belgium: Ordina Editions.

Bongaarts, J. and Potter, R.G. 1983. *Fertility, Biology, and Behaviror: An Analysis of the Proximate Determinants*. New York: Academic Press.

Bracher, M.D., and G. Santow. 1982. Breast-feeding in central Java. *Population Studies* 36:413-429.

Bullen, B.A., Skrinar, G.S., Beitins, I.Z., Von Mering, B., Turnbull, B.A., and McArthur, J.W. 1985. Induction of menstrual disorders by strenuous exercise in untrained women. *New England Journal of Medicine* 312:1349-1353.

Cantrelle, P. and Ferry, B. 1979. Approach to natural fertility in contemporary populations. In: H. Leridon and J. Menken (eds.), *Natural Fertility: Patterns and Determinants of Natural Fertility*, pp. 315-368. Liege, Belgium: Ordina Editions.

Cantrelle, P. and Leridon, H. 1971. Breastfeeding, mortality in childhood and fertility in a rural zone of Senegal. *Population Studies* 25(3):505-533.

Carael, M. 1978. Relations between birth intervals and nutrition in three Central African Populations (Zaire). In: W.H. Mosley (ed.), *Nutrition and Human Reproduction*, pp. 365-384. New York: Plenum Press.

Chavez, A. and Martinez, C. 1973. Nutrition and development of infants from poor rural areas. III. Maternal nutrition and its consequences on fertility. *Nutrition Reports International*, 7:1-8.

Chen, L.C., Ahmed, S. Gesche, M. and Mosley W.H. 1974. A prospective study of birth interval dynamics in rural Bangladesh. *Population Studies* 28(2):278-297.

Chowdhury, A.K.M.A. 1978. Effect of maternal nutrition on fertility in rural Bangladesh. In: W.H. Mosley (ed.), *Nutrition and Human Reproduction*, pp. 401-410. New York: Plenum Press.

Corsini, C.A. 1979. If fertility reducing-effect of lactation really substantial? In: H. Leridon and J. Menken (eds.), *Natural Fertility: Patterns and Determinants of Natural Fertility*, pp. 195-215. Liege, Belgium: Ordina Editions.

Cox, D.R. and V. Isham. 1980. *Point Processes*. New York: Chapman and Hall.

Delgado, H., A. Lechtig, R. Martorell, E. Brineman, and R. E. Klein. 1978. Nutrition, lactation, and postpartum amenorrhea. *American Journal of Clinical Nutrition* 31:322-327.

Delgado, H.L., R. Martorell, and R.E. Klein. 1982. Nutrition, lactation, and birth interval components in rural Guatemala. *American Journal of Clinical Nutrition* 35:1468-1476.

Diaz, S. 1989. Determinants of lactational amenorrhea. *International Journal of Gynecology & Obstetrics*, Suppl. 1:83-89.

Ellison, P.T., N.R. Peacock, and C. Lager. 1989. Ecology and ovarian function among Lese women of the Ituri Forest, Zaire. *American Journal of Physical Anthropology* 78:519-526.

Frisch, R.E. 1975. Demographic implications of the biological determinants of female fecudnity. *Social Biology* 22:17-22.

Frisch. R.E. 1978. Population, food intake, and fertility. *Science* 199:22-30.

Genazzani, A.R., F. Petraglia, A.D. Genazzani, G. Amato, G. D'Ambrogio, S. Angioni, and B. Bidzinska. 1991. Perspectives on stress-induced amenorrhea, pp. 321-326. In: A.R. Genazzani, C. Nappi, F. Petraglia, and E. Martignoni (eds.), *Stress and Related Disorders from Adaptation to Dysfunction*. Carnforth, U.K.: Parthenon Publishing.

Glasier, A., A.S. McNeilly, and P.W. Howie. 1984. Pulsatile secretion of LH in relation to the resumption of ovarian activity post partum.*Clinical Endocrinology* 20:415-426.

Guz, D. and Hobcraft, J. 1991. Breastfeeding and fertility: A comparative analysis. *Population Studies* 45:91-108.

Habicht, J.-P, J. Davanzo, W.P. Butz, and L. Meyers. 1985. The contraceptive role of breastfeeding. *Population Studies* 39:213-232.

Heckman, J.J. and B. Singer. 1984. A method for minimizing the impact of distributional assumptions in econometric models for duration data. *Econometrica* 52:271-320.

Heckman, J.J. and J.R. Walker. 1987. Using goodness of fit and other criteria to choose among competing duration models, pp. 247-307. In : C. Clogg (ed.), *Sociological Methodology*. Washington: American Sociological Association.

Howie, P.W., and A.S. McNeilly. 1982. Effect of breast-feeding patterns on human birth intervals. *Journal of Reproduction and Fertility* 65:545-557.

Huffman, S.L., Chowdhury, A.K.M.A., Chakraborty, J., and Mosley, W.H. 1978. Nutrition and post-partum amenorrhoea in rural Bangladesh. *Population Studies* 32(2):251-260.

Huffman, S.L., Chowdhury, A.K.M.A., Chakraborty, J., and Simpson, N.K. 1980. Breast-feeding patterns in rural Bangladesh. *American Journal of Clinical Nutrition* 33:144-154.

Huffman, S.L., K. Ford, H.A. Allen, and P. Streble. 1987. Nutrition and fertility in Bangladesh: breastfeeding and post partum amenorrhoea. *Population Studies* 41:447-462.

Jain, A., Hsu, T.C., Freedman, R., and Chang, M.C. 1970. Demgraphic aspects of lactation and postpartum amenorrhea. *Demography* 7:255-271.

James, W.P.T., A. Ferro-Luzzi, and J.C. Waterlow. 1988. Definition of chronic energy deficiency in adults. *European Journal of Clinical Nutrition* 42:969-981.

John, A.M., Menken, J.A., Chowdhury, A.K.M.A. 1987. The effects of breastfeeding and nutrition on fecundability in rural Bangladesh: A hazards-model analysis. *Population Studies* 41:433-446.

Jones, R.E. 1988. A hazards model analysis of breastfeeding variables and maternal age on return to menses postpartum in rural Indonesian women. *Human Biology* 60:853-871.

Jones, R.E. 1989. Breast-feeding and post-partum amenorrhoea in Indonesia. *Journal of Biosocial Science* 21:83-100.

Jones, R.E. 1990. The effect of initiation of child supplementation on resumption of post-partum menstruation. *Journal of Biosocial Science* 22:173-189.

Jones, R.E., and A. Palloni. 1990. Effects of infant mortality and weaning on the onset of postpartum menstruation: Hazard model analysis. CDE Working Paper 90-06. University of Wisconsin, Madison.

Jones, R.E., and A. Palloni. 1994. Investigating the Determinants of Postpartum Amenorrhea Using a Multistate Hazards Model Approach. *Annals of the N.Y. Academy of Sciences* 709:227-230.

Kalbfleisch, J.D. and R.L. Prentice. 1980. *The statistical analysis of failure time data.* New York: Wiley.

Keys, A., Brozed, J., Henschel, A., Mickelsen, O., and Taylor, H.L. 1950. *The Biology of Human Starvation*. Minneapolis, Minnesota: The University of Minnesota Press.

Knobil, E. 1980. The neuroendocrine control of the menstrual cycle. *Recent Progress in Hormone Research* 36:53-88.

Knodel, J. 1978. The influence of child mortality in a natural fertility setting: An analysis of German Villages. In: H. Leridon and J. Menken (eds.), *Natural Fertility: Patterns and Determinants of Natural Fertility*, pp. 273-284. Liege, Belgium: Ordina Editions.

Kurz, Kathleen. 1990. Evaluating the effects of maternal and child nutrition on length of lactational amenorrhea among Guatemalan women. PhD dissertation, unpublished. Cornell University.

Kurz, K.M., J.-P. Habitch, K.M. Rasmussen, and S.J. Schwager. 1993. Effects of maternal nutritional status and maternal energy supplementation on length of postpartum amenorrhea among Guatemalan women. *American Journal of Clinical Nutrition* 58:636-640.

Lawless, J.F. 1982. *Statistical Models and Methods for Lifetime Data.* New York: John Wiley and Sons.

London, D. 1988. *Survival Models and their Estimation*. Connecticut: ACTEX Publications.

Loucks, A.B. 1990. Effects of exercise training on the menstrual cycle: existence and mechanisms. *Medicine and Science in Sports and Exercise* 22:275-280.

Lunn, P.G. 1985. Maternal nutrition and lactational infertility: the baby in the driving seat, pp. 41-53. In: J. Dobbing (ed.), *Maternal Nutrition and Lactational Infertility*. New York: Raven Press.

Lunn, P.G., Prentice, A.M., Austin, S., Whitehead, R.G. 1980. Influence of maternal diet on plasma-prolactin levels during lactation. *Lancet* 1:623-625.

Lunn, P.G., M. Atkinson, and A.M. Prentice. 1981. Maternal nutrition and lactational amenorrhoea. *Lancet* 1:1428-1429.

Menken, J. 1979. Introduction. In: H. Leridon and J. Menken (eds.), *Natural Fertility: Patterns and Determinants of Natural Fertility*, pp. 1-12. Liege, Belgium: Ordina Editions.

Mejia, Victor. 1972. *Caracteristicas Economicas y Socioculturales de Cuatro Aldeas Ladinas de Guatemala.* Guatemala: Instituto Indigenista Nacional.

McNeilly, A.S., A. Glasier, and P.W. Howie. 1985. Endocrine control of lactational infertility, pp.1-16. In: J. Dobbing (ed.), *Maternal Nutrition and Lactational Infertility*. New York: Raven Press.

Naidus, A.N., J. Neela and N.P. Rao. 1991. *Nutrition News.* National Institute of Nutrition, vol 12, No. 2.

Palloni, A. and A. Sorensen. 1990. Methods for the Analysis of Event History Data: A Didactic Overview, pp. 291-323. In: P.B. Baltes, D.L. Featherman and R.M. Lerner (eds.), *Life-span Development and Behavior*, volume 10. New Jersey: Lawrence Erlbaum Associates Publishers.

Pinto Aguirre, G. 1994. Breastfeeding patterns, nutrition and postpartum amenorrhea in Guatemalan women: a multi-state hazard approach. Ph.D. dissertation. Department of Sociology. University of Wisconsin-Madison.

Pinto Aguirre, G. 1995. A Longitudinal Analysis of the Risk of Resumption of Menses in Guatemala. CDE Working Paper No. 94-23. Center for Demography and Ecology, University of Wisconsin-Madison.

Pirke, K.M., Schweiger, U., Lemmel, W., Drieg, J.C., and Berger, M. 1985. The influence of dieting on the menstrual cycle of healthy young women. *Journal of Clinical Endocrinology and Metabolism* 60:1174-1179.

Pirke, K.M., Schweiger, U., Strowitzki, T., Tuschl, R.J., Laessle, R.G., Broocks, A., Huber, B., Middendorf, R. 1989. Dieting causes mentrual irregularities in normal weight young women through impairment of episodic luteinizing hormone secretion. *Fertility and Sterility* 51(2):263-268.

Popkin, B.M., Guilkey, D.K., Akin, J.S., Adair, L.S. Udry, J.R., and Flieger, W. 1993. Nutrition, lactation, and airth spacing in Filipino women. *Demography* 30(3):333-352.

Potter, R.G., New, M.L., Wyon, J.B., and Gordon, J.E. 1965. Applications of field studies to research on the physiology of human reproduction: Lactation and it's effects upon birth intervals in eleven Punjab villages, India. *Journal of Chronic Diseases* 18:1125-1140.

Prema, K., A.N. Naidu, S. Neelakumari and P. Ramalakshi. 1981. Nutrition-fertility interaction in lactating women of low income groups. *British Journal of Nutrition* 45, 461.

Prentice, A.M., Roberts, S.B., Watkinson, M., Whitehead, R.G., Paul, A.A., Prentice, A., Watkinson, A.A. 1980. Dietary supplementation of Gambian nursing mothers and lactational performance. *Lancet* 2:886-888.

Rodriguez, G., and S. Diaz. 1988. Breastfeeding and the length of post-partum amenorrhea: a hazards model approach. Seminar on Biomedical and Demographic Determinants of Human Reproduction. July 4-8, Baltimore, Maryland.

Rosetta, L. 1989. Breast feeding and post-partum amenorrhea in Serere women of Senegal. Ann. *Human Biology* 16:311-320.

Salber, E.J., Feinleib, M., and Macmahon, B. 1966. The duration of postpartum amenorrhea. *American Journal of Epidemiology* 82:347-358.

Santow, G. 1987. Reassessing the contraceptive effect of breastfeeding. *Population Studies* 41:147-160.

Schweiger, U., Schwingenschloegel, M., Laessle, R., Schweiger, M., Pfister, K., Pirke, M., and Hoehl, C. 1987. Diet-induced menstrual irregularities: Effects of lage and weight loss. *Fertility and Sterility* 48:746-751.

Schweiger, U., Tuschl, R.J., Laessle, R.G., Broocks, A., and Pirke, K.M. 1989. Consequences of dieting and exercise on menstrual function in normal weight young women. In: K.M. Pirke, W. Wuttke, and U. Schweiger (eds.). *The Menstrual Cycle and Its Disorders: Influences of Nutrition, Exercise and Neurotransmitters*, pp. 142-149. Berlin: Springer-Verlag.

Short, R.V. 1984. Breast feeding. *Scientific American* 250:35-42.

Srinivasan, K., K.B. Pathak and A. Pandey. 1988. Duration of post-partum amenorrhea in Orissa: A hazard-model analysis. Carolina Population Center, Papers, No. 88-28. University of North Carolina, Chapel Hill.

Stein, Z. and Susser, M. 1975. Fertility, fecundity, famine: Food relations in the Dutch famine 1944-45 have a causal relation to fertility and probably to fecundity. *Human Biology* 47:131-154.

Stein, Z. and Susser, M. 1978. Famine and fertility. In: W.H. Mosley (ed.), *Nutrition and Human Reproduction*, pp. 123-145. New York: Plenum Press.

Tay, C.C.K., A.F. Glasier, and A.S. McNeilly. 1992. The 24 h pattern of pulsatile luteinizing hormone, follicle stimulating hormone and prolactin release during the first 8 weeks of lactational amenorrhea in breastfeeding women. *Human Reproduction* 7:951-958.

Trussell, J., van de Walle, E., and van de Walle, F. 1989. Norms and behaviour in Burkinabe fertility. *Population Studies*, 43:429-454.

Tuma, N.B. and M.T. Hannan. 1984. *Social Dynamics: Models and Methods*. Orlando: Academic Press.

Tuma, N.B., M.T. Hannan and L.P. Groeneveld. 1979. Dynamic Analysis of Event Histories. *American Journal of Sociology* 84(4):820-854.

Tyson, J.E. and Perez, A. 1978. The maintenance of infecundity in postpartum women. In: W.H. Mosley (ed.), *Nutrition and Human Reproduction*, pp. 11-28. New York: Plenum Press.

Van Ginneken, J.K. 1977. The chance of conception during lactation. *Journal of Biosocial Science*, Supplement 4:41-54.

Van Ginneken, J.K. 1978. The impact of prolonged breast-feeding on birth intervals and on postpartum amenorrhea. In: W.H. Mosley (ed.), *Nutrition and Human Reproduction*, pp. 179-195. New York: Plenum Press.

Warren, M.P. 1980. The effects of exercise on pubertal progression and reproductive function in girls. *Journal of Clinical Endocrinology and Metabolism* 51:1150-1156.

Wood, J.W. 1994. *Dynamics of Human Reproduction: Biology, Biometry, Demography*. New York: Aldine de Gruyter.

Wood, J.W., Lai, D., Johnson, P.L., Campbell, K.L., and Maslar, I.A. 1985. Lactation and birth spacing in Highland New Guinea. *Journal of Biosocial Science*, Supplement 9:159-173.

Wray, J.D. 1978. Maternal nutrition, breast-feeding and infant survival. In: W.H. Mosley (ed.), *Nutrition and Human Reproduction*, pp. 197-230. New York: Plenum Press.

Yen, S.S.C. 1987. Reproductive strategy in women: neuroendocrine basis of endogenous contraception, pp. 231-250. In: Rolland, R., Heineman, M.J., Naaktegboren, N., Schoeinaker, J., Vemer, H., and Willemsen, W.N.P., (eds.). New York: Elsevier Science Publishers.

**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**
Estimated effects (*) for multistate hazard models.

| Transitions | Coeff. | Slope | Est. rate | Chi-Sq. | N |
|---|---|---|---|---|---|
| Full (1)-> Partial (2) | -1.604 | 0.023 | 0.220 | 9.22 | 1262 |
| | (0.00) | (0.00) | | (0.00) | |
| Full (1)-> Wean (3) | -5.017 | -0.718 | 0.002 | 12.31 | 10 |
| | (0.00) | (0.00) | | (0.00) | |
| Full (1)-> Death (4) | -3.242 | -0.7175 | 0.010 | 72.65 | 59 |
| | (0.00) | (0.00) | | (0.00) | |
| Full (1)-> Menses (5) | -5.070 | 0.130 | 0.012 | 25.93 | 68 |
| | (0.00) | (0.00) | | (0.00) | |
| Partial (2)-> Wean (3) | -4.698 | 0.101 | 0.029 | 142.36 | 312 |
| | (0.00) | (0.00) | | (0.00) | |
| Partial (2)-> Death (4) | -5.078 | -0.102 | 0.003 | 6.70 | 29 |
| | (0.00) | (0.00) | | (0.01) | |
| Partial (2)-> Menses (5) | -3.800 | 0.084 | 0.057 | 185.71 | 617 |
| | (0.00) | (0.00) | | (0.00) | |
| Wean (3)-> Death (4) | -1.596 | -0.172 | 0.026 | 16.46 | 16 |
| | (0.00) | (0.00) | | (0.00) | |
| Wean (3)-> Menses (5) | -0.945 | 0.007 | 0.429 | 0.43 | 261 |
| | (0.00) | (0.51) | | (0.51) | |
| Death (4)-> Menses (5) | -0.889 | 0.050 | 0.529 | 5.89 | 101 |
| | (0.00) | (0.01) | | (0.02) | |

(*) p-values are shown in parentheses
The duration structure is defined as a Gompertz function.
Full: fully breastfeeding
Partial: partially breastfeeding (supplement introduced)
Wean: child weaned before menses
Death: child dies before menses
Menses: resumption of menses

**Table 3**
Covariate effects (*) for multistate hazard models.

| Covariates | Transitions | | | | |
|---|---|---|---|---|---|
| | 1->2 | 1->3 | 1->4 | 1->5 | 2->3 |
| Constant | -0.173 | -5.017 | -3.139 | -3.738 | -4.504 |
| | (0.30) | (0.00) | (0.00) | (0.00) | (0.00) |
| Slope | 0.078 | -0.718 | -0.742 | 0.178 | 0.102 |
| | (0.00) | (0.02) | (0.00) | (0.00) | (0.00) |
| BACKGROUND VARIABLES | | | | | |
| One child (par1) | 0.091 | | | 0.234 | 0.206 |
| | (0.34) | | | (0.48) | (0.30) |
| Four or more children (par2) | 0.067 | | | -1.086 | 0.268 |
| | (0.38) | | | (0.01) | (0.08) |
| 13-19 years (age1) | -0.016 | | 0.687 | 0.589 | 0.555 |
| | (0.88) | | (0.06) | (0.08) | (0.01) |
| 30-49 years (age3) | 0.105 | | 0.856 | 0.333 | -0.206 |
| | (0.14) | | (0.00) | (0.41) | (0.14) |
| 0 or 1 year (educ1) | -0.040 | | | -1.147 | -0.184 |
| | (0.55) | | | (0.00) | (0.19) |
| Atole village | 0.429 | | | 0.461 | -0.278 |
| | (0.00) | | | (0.07) | (0.03) |
| BREASTFEEDING VARIABLES | | | | | |
| Low frequency (8-11 times) | 0.198 | | -5.459 | -0.333 | -0.453 |
| | (0.04) | | (0.24) | (0.44) | (0.02) |
| High frequency (12+ times) | -0.316 | | -1.818 | -1.326 | -0.632 |
| | (0.00) | | (0.09) | (0.03) | (0.00) |
| High supplement (child) | 0.300 | | | | 0.336 |
| (Low intensity nursing) | (0.00) | | | | (0.11) |
| MOTHER'S NUTRITION | | | | | |
| BMI>20 | | | | | |
| High supplement (mother) | 0.089 | | | | -0.402 |
| (high nutrition) | (0.44) | | | | (0.06) |
| CHILD'S NUTRITION | | | | | |
| Poorly nourished | | | | | 0.411 |
| | | | | | (0.04) |
| MOTHER'S HEALTH | | | | | |
| No morbidity (healthy) | -0.331 | | | | |
| | (0.00) | | | | |
| CHILD'S HEALTH | | | | | |
| No morbidity (healthy) | -0.104 | | -2.524 | -1.218 | -0.027 |
| | (0.24) | | (0.00) | (0.00) | (0.85) |
| ENERGY EXPENDITURE | | | | | |
| Agric. and manual workers(work2) | -0.222 | | | | |
| | (0.09) | | | | |
| Merchants, skilled workers(work3) | -0.184 | | | | |
| | (0.02) | | | | |
| log L | -2691 | -67 | -209 | -324 | -1315 |
| Chi Square | 960 | 12 | 241 | 91 | 206 |
| df | 22 | 1 | 8 | 13 | 19 |
| N | 1262 | 10 | 59 | 68 | 312 |

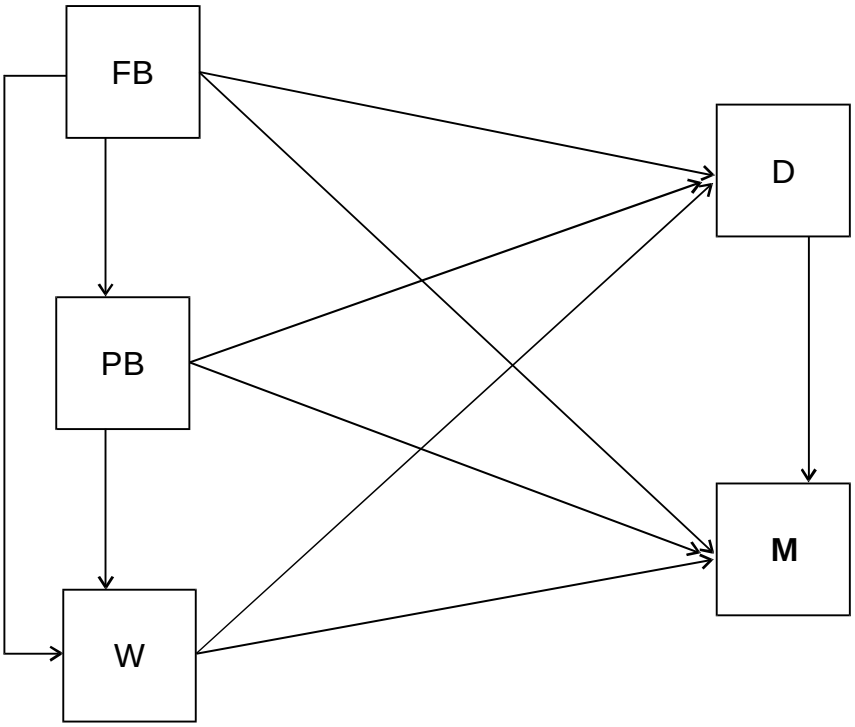
(*) p-values are shown in parentheses

**Table 3** (continued)
Covariate effects (*) for multistate hazard models.

| Covariates | Transitions | | | | |
|---|---|---|---|---|---|
| | 2->4 | 2->5 | 3->4 | 3->5 | 4->5 |
| Constant | -5.078 | -3.884 | -1.596 | -0.773 | -0.889 |
| | (0.00) | (0.00) | (0.00) | (0.00) | (0.00) |
| Slope | -0.102 | 0.122 | -0.172 | | 0.050 |
| | (0.02) | (0.00) | (0.00) | | (0.01) |
| BACKGROUND VARIABLES | | | | | |
| One child (par1) | | 0.284 | | -0.089 | |
| | | (0.03) | | (0.69) | |
| Four or more children (par2) | | -0.252 | | 0.188 | |
| | | (0.02) | | (0.26) | |
| 13-19 years (age1) | | 0.411 | | 0.019 | |
| | | (0.01) | | (0.94) | |
| 30-49 years (age3) | | -0.107 | | -0.257 | |
| | | (0.29) | | (0.07) | |
| 0 or 1 year (educ1) | | -0.359 | | -0.053 | |
| | | (0.00) | | (0.74) | |
| Atole village | | 0.329 | | | |
| | | (0.00) | | | |
| BREASTFEEDING VARIABLES | | | | | |
| Low frequency (8-11 times) | | -0.370 | | | |
| | | (0.00) | | | |
| High frequency (12+ times) | | -0.894 | | | |
| | | (0.00) | | | |
| High supplement (child) | | 0.246 | | | |
| (Low intensity nursing) | | (0.05) | | | |
| MOTHER'S NUTRITION | | | | | |
| BMI>20 | | 0.260 | | | |
| | | (0.02) | | | |
| High supplement (mother) | | 0.383 | | | |
| (high nutrition) | | (0.00) | | | |
| CHILD'S NUTRITION | | | | | |
| Poorly nourished | | 0.226 | | | |
| | | (0.04) | | | |
| MOTHER'S HEALTH | | | | | |
| No morbidity (healthy) | | 0.016 | | | |
| | | (0.91) | | | |
| CHILD'S HEALTH | | | | | |
| No morbidity (healthy) | | -0.100 | | | |
| | | (0.30) | | | |
| ENERGY EXPENDITURE | | | | | |
| Agric. and manual workers(work2) | | -0.587 | | | |
| | | (0.00) | | | |
| Merchants, skilled workers(work3) | | 0.217 | | | |
| | | (0.04) | | | |
| log L | -197 | -2174 | -66 | -478 | -162 |
| Chi Square | 7 | 418 | 16 | 8 | 6 |
| df | 1 | 26 | 1 | 6 | 1 |
| N | 29 | 617 | 16 | 261 | 101 |

(*) p-values are shown in parentheses

Figure 1: A multiple state model of transition to menses.




FB = Fully Breastfeeding PB = Partially Breastfeeding W = Weaned D = Infant Death M = Menses

Center for Demography and Ecology
University of Wisconsin
1180 Observatory Drive Rm. 4412
Madison, WI 53706-1393
U.S.A.
608/262-2182
FAX 608/262-8400
email: name@ssc.wisc.edu