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A Longitudinal Analysis of the Risk of Resumption of Fecundity in Guatemala

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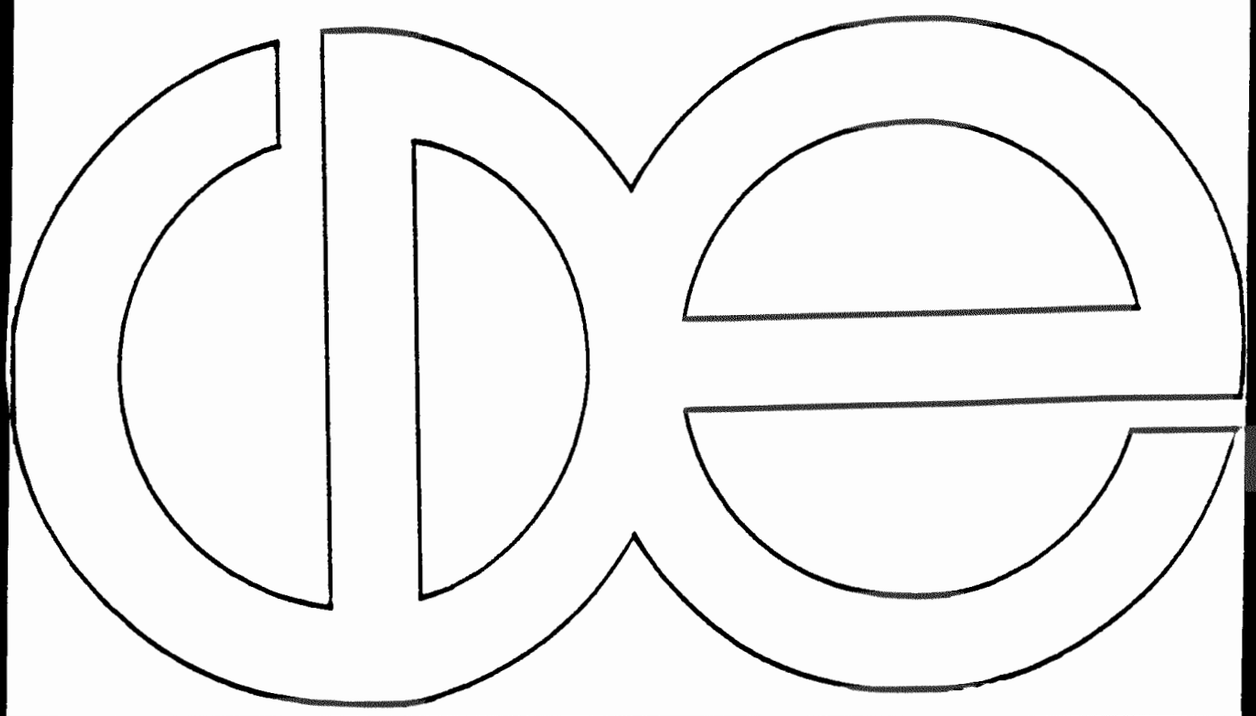

Introduction

The purpose of this paper is to model the resumption of postpartum fecundity[1], or equivalently, the duration of postpartum amenorrhea, through the use of hazard models. The hazard of resuming menses is modeled as a function of time and various fixed and time dependent covariates.

Throughout this study the reproductive history of a woman is considered a sequential process over time marked by different events such as births, infant deaths, nursing episodes, menses, illness spells, malnutrition episodes, supplemental intakes, etc. That is, a woman's reproductive history is taken as a collection of random points over time (i.e., stochastic process) defined by such events. The model presented in this study recognizes explicitly the existence of only two states: amenorrhea and menses. A woman moves to the amenorrheic state after experiencing a menstrual spell. Thus, the attrition process of the women under study is only the history of transitions between these two states.

One important difference with similar studies is that we explicitly include the **direct effects** of infant mortality. In the past, the most common practice was to eliminate from the sample all infant deaths that occurred before menses. This solution, however, may lead to inconsistent estimates and, furthermore, negates the possibility of assessing the direct effects of infant mortality on resumption of menses.

First, we estimate various piecewise exponential hazard models with fixed and time dependent covariates on the monthly prospective resumption-of-menses data from rural Guatemalan women. After experimenting with several time segments, we select five that appear to capture in

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[1] Direct measurements of postpartum anovulatory periods are not available in large scale surveys because of the difficulty in observing and detecting ovulation. Instead we are forced to rely on a more well defined and observable event: the resumption of menses after birth. Thus, in this paper resumption of postpartum menses is used as an indicator of resumption of postpartum fecundity.

a parsimonious way the duration dependence of the risk of resuming menses after birth.

Second, for comparative purposes and to model the duration dependency under a parametric specification, we also estimate various Gompertz hazard models with fixed and time varying covariates.

Finally, we estimate a two-state model with heterogeneity by using non-parametric specification of the error term with two points of support with a Gompertz baseline. All these models are estimated for the full sample (1417 birth intervals).

We rely on the results from the piecewise exponential and Gompertz hazard models to provide empirical support for the claims we make about the relationships involved and the universe of estimation.

The Determinants of Postpartum Amenorrhea

In general, four mechanisms related to the resumption of normal menstrual cycles in postpartum women can be identified (Jones, 1989). 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 behavior prevalent among those mothers who begin menstruating while they are 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. A 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 consists of an indirect effect, and may work through the patterns of 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 problems found in retrospective studies: that is, difficulties in interpreting causality and in identifying 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 the 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 releases **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 signal which interrupts the release of GnRH and LH during these activities remains 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).

SOURCE OF 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. Women entered the study on the basis of their reproductive history, i.e., those who had at least one child aged less than seven years old. Periodically mothers provided data throughout their pregnancy and postpartum period. In this research 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). Under this constraint 608 women and their 1430 birth intervals were selected (Pinto, 1994).

Characteristics of the sample

Since measures for breastfeeding (number of nursing episodes a day) and mother's nutritional status have missing values for some women, we defined specific categories for missing values, so that the full sample (1417 birth intervals) is considered in the analyses.

Table 1 reports basic descriptive statistics for some of the covariates used in the analysis of the INCAP Longitudinal Survey for two different sample sizes. It also reports information about censoring of the dependent variable, duration of postpartum amenorrhea[2].

According to the figures presented in this table, about 26 percent of the birth intervals in the full sample contain censored information on duration of postpartum amenorrhea and about 30 percent in the reduced sample. We can also see that 60 percent of the birth intervals in the

[2] The dependent variable is defined as follows: (1) the number of months 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 between birth and the first menstrual episode when menses occurred beyond the third month after delivery, or (3) the number of months between a birth and the subsequent conception when menses never resumed. The first two cases are considered failures while the last one is a censored case (Pinto, 1994).

complete sample come from a relatively young group of women. However, when missing values for breastfeeding and nutrition are removed from this sample, the percentage of birth intervals produced by older women increases from 37 to 42. In the same way, 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 above mentioned table. These results mean that when missing values are dropped the composition of the sample changes and, in particular, the reduced sample is now made up of older and higher parity women.

Methods of Analysis

In this section we present some basic concepts of survival models as well as the general strategy utilized to model the data at hand.

The length of time being analyzed in this paper is the duration of the amenorrheic period (i.e., the time elapsed from birth to the first postpartum menstrual episode), also known as the waiting time to next menses, which is represented by a random variable $T(\geq 0)$. This random variable measures the time it takes a woman to transit from one state to another, in this case from amenorrhea to menses. Thus the state space consists of only two states: amenorrhea and menstruation, and the attrition process is just a set of transitions from the amenorrheic state to the menses state. The use of hazard models allows us to introduce both fixed and time-dependent covariates[3].

The data analyzed in this study are characterized by the presence of right-censored observations, i.e., there are women who do not have ending times for the duration of amenorrhea, either because they were not available for the follow-up visits during the surveys (lost to follow-

[3] Hazard rate model makes assumptions about changes in the hazard across individuals (population heterogeneity) and changes over time (duration dependence). A distribution whose hazard slopes upward is said to have a **positive duration dependence**. For such distributions, the likelihood of failure at time t, conditional upon duration up to time t, is increasing in t. The opposite case is that of decreasing hazard or **negative duration dependence**.

up) or because the survey ended before they experienced a menstrual episode (termination of follow-up). In the latter case, it is only known that for some women their duration of postpartum amenorrhea exceeded some particular given value; but the exact durations are unknown.

Because longer-duration amenorrheic periods are in general more likely to be censored, these data cannot be analyzed by simply removing these censored observations. Therefore, the methods to be utilized must adequately use both censored and uncensored observations in order to obtain unbiased estimates. One such method is the so-called hazard model[4].

Suppose the duration T is completely characterized by a continuous probability distribution f(t), where t is a realization of T. One of the basic concepts in event history analysis is that of "failure" rate or hazard function[5], which is defined as follows:

$$h(t) = \lim_{\Delta t \to 0^+} \frac{P(t \le T \le t + \Delta t \mid T > t)}{\Delta t}$$

The hazard function is basically the instantaneous probability of "failing" (i.e., resuming menses) at time t, given that failure (i.e., resuming menses) has not occurred before t. Thus, the hazard function is the rate at which amenorrheic episodes are completed after duration t given that

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[4] An alternative way of modeling the time to resumption of menses would be through the use of simple binary models. There are some basic differences between these two models. First, the hazard approach models the logarithm of the incidence rate of resuming menses, while the logistic models the logarithm of the odds of resuming menses. Second, the logistic model ignores the amount of time elapsed before the outcome or censoring occurred; the hazard model accounts explicitly for the duration of time between events.

[5] The probability distribution f(t) is the **unconditional** density of failure at time t (that is, the density of failure at time t given **only** that the quantity existed at t=0), while the hazard rate at time t is defined as a **conditional** density function, conditional on survival. The hazard rate, also called the force of mortality, is a particular case of the **instantaneous transition rates** in a continuous-time, discrete-state stochastic processes scheme.

they lasted at least until t[6]. The hazard rate can take any non-negative value between zero and ∞.

a) Piecewise exponential hazard models

The piecewise exponential model is a generalization of the simple exponential model by allowing the parameters to be piecewise constant functions of time[7].

The underlying logic is simple. Instead of analyzing the entire duration t, we use a stepwise approach. The idea is to divide the duration t into a set of discrete time intervals, say, k disjoint time intervals, and assume the hazard is constant in each one. Then the piecewise exponential hazard model can be expressed as follows:

$$h(t|Z(t))=\exp(b_j Z(t)),\ t_{j-1}<t\leq t_j,\ j=1,2,...,k$$

where $\mathbf{b_j}$ is the vector of coefficients in the interval j and $\mathbf{Z(t)}$ is the vector of covariates.

Under this specification the hazard rate and the effects of the covariates change across the time intervals but are fixed within each interval. That is, there is a separate exponential hazard rate for each piece of the time scale[8](Tuma, Hannan and Groeneveld, 1979).

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[6] From the demographic point of view, the hazard rate is just an age-specific rate for a very small time interval.

[7] The simple exponential model can be written as follows: $h(t|\mathbf{x})=h_0(t)\exp(\beta\mathbf{x})$
This model assumes that covariates have proportional effects and that the model parameters do not vary with time; that is, the hazard rate is constant over time. The choice of this functional form for effects of explanatory variables is a simple way to ensure that the hazard is non-negative at all **x**.

[8] When the time intervals are very short, the piecewise exponential approach is essentially nonparametric; that is, no assumption is made about the dependence of the hazard on time.

b) Gompertz models

The Gompertz model specifies time variation in the hazard rate as a log linear function of time and can be written as

$$h(t|Z(t)) = \exp(aZ(t) + bt)$$

This model is a generalization of the simple exponential model by including the coefficient **b** for t, and the covariates **Z(t)**. Positive values of **b** are the result of a monotonically increasing hazard rate with time; negative values result from a monotonically decreasing hazard rate with time (Tuma and Hannan, 1984).

c) Heterogeneity

The problem of heterogeneity in hazard models can be thought of as the result of incomplete specification, and its presence is likely to produce faulty inferences about the process being studied. Failure to control for unobservables could produce wrong estimates and misleading conclusions (Pinto, 1994). In this particular case, we are interested in the effects that the existence of unobserved person-specific characteristics (i.e., unobserved individual differences) may have on the rate at which women experience resumption of menses[9].

In general, research dealing with unmeasured heterogeneity assumes that unobservables can be captured by a random variable θ, which does not change over time (Heckman and Singer, 1984) and has some specific distribution function. It is also assumed that θ is independent of the initial state of the process. Under these very general conditions the hazard can be written as:

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[9] In general, two types of effects can be identified. On the one hand, the omission of some relevant covariates (i.e., characteristics) accounting for some specific effects may misidentify the structure of the time dependence of the risk; that is, it leads to an erroneous identification of the type of duration. On the other hand, the existence of unmeasured heterogeneity leads to biased estimates of the coefficients of the covariates in the model, even if they are not correlated to the omitted ones (Palloni and Sorensen, 1990).

$$h(t|Z(t),\theta)=\exp[\gamma_0+\beta Z(t)+\frac{\gamma_1(t^k-1)}{k}+\frac{\gamma_2(t^l-1)}{l}+c\theta]$$

In this expression t is the length of the postpartum amenorrhea, Z(t) is a vector that contains the observed variables, β is the vector of coefficients, θ is an unobserved component (i.e., person-specific unobserved heterogeneity component), γ_0, γ_1, γ_2, k, l, and c (i.e., loading factor) are parameters of the model to be estimated along with β, where k<l. By setting k=1 and l=0 we specify a Gompertz hazard baseline (Heckman and Singer, 1984; Heckman and Walker, 1987).

Analysis of the Results

Several piecewise exponential and Gompertz nested hazard models are fitted for the full sample. Each model only includes additive effects. These hazard models are estimated using woman's age, parity, place of residence (atole or fresco village), woman's education, type of occupation, and frequency of breastfeeding as fixed covariates, and the time of introduction of supplementation, time of weaning, time of infant death, intensity of breastfeeding, maternal nutrition, child nutrition, and measures of health for the mother and children as time dependent covariates[10].

Piecewise exponential hazard models

Tables 2 and 3 present the estimates of the covariate effects on the transition to menses, their p-values[11], and the log-likelihood and chi-square statistic for four alternative nested

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[10] Although socioeconomic characteristics of the woman can be expected to influence resumption of menses primarily through breastfeeding, nutrition, and health covariates, they are included in the model to determine whether there is any residual impact of menses. Thus, the models include information on mother's education.

[11] The p-value is the observed significance level that would just barely permit the rejection of H_0 (i.e., β=0) against H_1 (i.e.,β≠0), given the **t** value calculated from the sample. In other words, it is the probability of observing a **t** at least as extreme as the **t** calculated from the sample under the assumption that the null hypothesis is true. A low p-value (near to zero) indicates a high level of statistical significance and vice versa.

piecewise hazard models (models 1-4).

To determine the duration structure of the piecewise model, we started by estimating the hazard of resuming menses for every single month and then we collapsed them into segments where the hazard appeared to be constant. After experimenting with different time intervals we selected a five-segment model. The estimates reported correspond to the following time intervals: 0-5, 6-9, 10-13, 14-17, and 18 and more, in completed months.

The first model represents the baseline model with only segments, where the omitted time interval corresponds to the interval from zero to five months. The second model controls for parity, mother's age, education, occupational status, and place of residence, using 8 fixed covariates (their definitions are in Table 1).

The third model controls additionally for breastfeeding variables (breastfeeding status, frequency of breastfeeding, and intensity of nursing), mother's nutritional status, child's nutritional status, mother's health status, and child's health status[12].

In this model, breastfeeding status is represented by four time-dependent covariates: full breastfeeding (Bf1), partial breastfeeding (Bf2), weaning (Bf3), and infant mortality (Bf4). Contrary to other studies, the direct effects of infant mortality on resumption of menses are considered explicitly. In the past literature, one of the most common strategies to deal with infant mortality was to eliminate from the sample infant deaths that occurred before menses (Rodriguez and Diaz, 1988; Huffman et al., 1987; Delgado et al., 1982).

The second breastfeeding covariate is represented by three fixed dummy variables: low frequency (1-7 times a day), medium frequency (8-11 times a day), and high frequency (12 or more). The third breastfeeding covariate, child's clinical supplementation, is considered an indicator

[12] Models with various interactions among the independent variables were fitted but since they did not lead to significantly better fit, they are not include in the present analysis.

for intensity of breastfeeding (Kurz et al., 1993). This measurement, however, is a more general proxy for patterns of breastfeeding and at the same time is an indicator of child's nutritional status, whose effect may work through breastfeeding.

In order to assess the independent effects of a woman's nutritional status this model introduces three covariates: body mass index (BMI), mother's head circumference, and woman's clinical supplementation. The first and the third covariates are time dependent covariates while the second is fixed. Seven dummy variables are used to measure these effects. Finally, a more parsimonious model is fitted (model 4) by dropping child's nutrition and mother's health[13].

As we can see in Table 2, the improvement of fit is considerable when we move from model 1 to models 3 and 4. In fact, the global chi-square increases from 624 to 1,606 and 1,594, respectively.

The duration structure of the piecewise hazard models presented in Table 2 show that the risk of resuming menses increases systematically with time, even after controlling for all the relevant variables. In fact, the estimated effects of the time segments are highly significant and tend to increase over time in all the models estimated (models 1-4). That is, as time goes by the hazard of resumption of menses increases. These results may be associated with three different factors (Jones and Palloni, 1990). First, the model may have unmeasured covariates, whose effects are mixed with the time structure of the model. Second, some of the variables in the models may be poorly measured, especially those capturing breastfeeding patterns and/or mother's nutrition and health status. Third, there may exist a natural decrease over time in the levels of the hormones that inhibit menses, which are independent of breastfeeding behavior, maternal nutritional status, and/or work patterns.

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[13] In all these models the coefficients for the omitted and the "missing values" categories are not shown in the tables in order to reduce table size.

Most of the fixed covariates are significant even after controlling for breastfeeding, and nutritional and health variables. Age and parity are only introduced as control factors of the underlying process. As we can see in Table 2, all the parity levels have significant independent effects on the risk of resuming menses after childbirth. High parity levels are associated with longer durations of amenorrhea and vice versa. A similar relationship was also found in other studies (Jones and Palloni, 1994; Huffman et al., 1987). Such a relationship could be reflecting the effect of some biological factors, unrelated to either lactation or nutrition, which may be present in the hormonal mechanism responsible for menstruation and ovulation. Individual and random variations may also be present; for instance, some women, particularly those who give birth for the first time, are subject to irregular menstrual cycles.

The age of the mother at child's birth was found to be not significant, although it behaves in the expected direction; that is, older mothers have lower risks of resuming menses. This result is, however, consistent since parity and age are frequently correlated. Other studies have also found that older ages decrease the risk of earlier return of menses (Jones, 1988; Huffman et al., 1987).

The work pattern variable (Table 2, model 4) is used as a proxy for energy-demanding activities. They show that the risk of resuming menses in women who are engaged in agricultural activities or heavy work is 35 percent lower than the risk in women who do not work outside the home (housewives), after controlling for breastfeeding behavior and nutritional status. This effect is highly significant. However, when women work in skilled occupations or as merchants, the effect on resumption of menses postpartum is not significant but in the right direction, that is, the risk of returning menses increases when compared with housewives.

Women who work in agricultural activities or have heavy workloads expend considerable amounts of energy, while women who work in skilled jobs, trade activities, or as housewives tend to expend less energy during the postpartum period. One possible explanation of this result is that

a housewife may get help from her older children or other relatives during this period. In this way some strenuous physical activities that may stress women's bodies, such as cleaning, carrying water and woodfire, washing clothes, etc., are deflected towards other members of the family during this period (Mejia, 1972).

These results clearly support the hypothesis that strenuous physical activities have a direct effect on ovulation through changes of the GnRH cycle and subsequent LH suppression, similar to that found for breastfeeding, which prevents normal ovarian activity and normal menstrual cycles (Prema et al., 1981; Lunn et al., 1981; Rosetta, 1989; Ellison et al., 1989; Jones and Palloni, 1994).

Finally, the village of residence was found to have a significant effect on the risk of resumption of menses, after controlling for all the relevant variables in the model. As we can see in Table 2, model 4, the coefficient associated with women living in an Atole village is positive and highly significant. This means that the risk of resuming menses for women in an Atole village is about 27 percent higher than the risk experienced by women in a Fresco village. The mechanisms underlying this effect are not clear. However, this result could be expected on the basis of underlying mechanisms about nutrition.

In all the estimated piecewise models in Table 2 the independent effects of breastfeeding status (fully breastfeeding, partial breastfeeding, weaning, and infant mortality) are highly significant and in the expected direction, after controlling for the 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 times higher than when the child is fully breastfeeding (reference group). Whereas when the child is weaned the risk increases by about 11 times, controlling for all the variables in the model. In a similar way, the introduction of supplements accelerates the resumption of menses by only 2.2 times.

These results show clearly that early infant deaths have a more appreciable effect on fecundity, and thus on fertility, than weaning (or 'voluntary' termination of breastfeeding).

Given the underlying neuroendocrine mechanisms mentioned above, it is very unlikely that infant death may set in motion any particular hormonal reaction that could accelerate ovulation. This large difference in their effects on resumption of menses relies rather on the fact that an infant death means the immediate termination of suckling while weaning may not signify the same abrupt cessation of lactation, although women reported so.

The frequency of breastfeeding variables also has a significant effect on the dependent variable and 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). In fact, a high frequency of nursing reduces the risk of resuming menses after childbirth in any given time interval by about 45 percent (model 4), controlling for all the variables considered in the model. Similarly, mothers who nursed their children 8-11 times a day also experienced a delay in the resumption of menstruation when compared to those in the reference group.

In short, the higher the frequency of nursing episodes per day, the lower the risk of earlier resumption of menses, and vice versa. Therefore, the frequency of breastfeeding is inversely related to the hazard of resuming postpartum fecundity.

The effect of children's clinical supplementation, an indicator of the intensity of breastfeeding (Kurz et al., 1993), on the hazard rate is barely significant and in the proper the direction. That is, high amounts of supplement (i.e., low intensity of suckling) increase the risk of resuming menses by about 21 percent, compared to low amounts of supplement (model 4).

A higher intensity and number of nursing episodes per day means that the suckling by the infant stimulates more frequently the neural receptors located in the breast nipples, thus setting off a chain of hormonal secretions that prompts the release of endogenous opioids. These hormones in turn inhibit ovulation by decreasing the release of LH needed for normal ovarian activity and normal menstrual cycles. But as the frequency and intensity of lactation decrease, the average levels of endorphins slow down, and once it falls below certain threshold (which varies from one woman to another) ovulation, and therefore menstruation, resumes (Habitch et al., 1985).

It has been found that a suckling frequency of more than 5 times a day and a duration of 10 minutes per feeding each day were sufficient to sustain total suppression of ovarian activity for almost one year (Howie and McNeilly, 1982). These authors believe that such suppression could continue if high frequent and intense suckling are maintained.

Some of the indicators for woman's nutritional status do not show significant effects on the resumption of menses, although they are in the expected direction (Table 2, model 3). We can see that the measure of BMI, which assesses **chronic energy deficiency**, has an effect that is not statistically significant. The indicator for **long term nutritional status**, measured by mother's head circumference, shows that women with a low nutritional level tend to have a slower resumption of menses than those better off; the risk of resuming menses is approximately 16 percent lower.

Finally, the third indicator of nutrition, mother's clinical supplementation, which measures the **current nutritional status**, has a significant effect on the hazard and is also in the expected direction. In fact, the risk of resuming menses increases for mothers with high supplement intake by about 29 percent over the risk of mothers with low intake.

These results may be indicating that malnutrition becomes relevant to fertility and fecundity when women experience extreme situations of under or overnutrition. Chronic malnutrition, rather than famine, is very common among women in developing countries. Thus, dietary intakes that are

less than those recommended by some health organizations are generally enough to maintain body weight, so that a sudden improvement in the dietary intake, through supplemental programs, improves the women's current nutritional status.

These empirical results support the hypothesis that malnutrition has a similar, if not the same, effect as suckling stimulus. It disrupts the pulse generator neurons in the hypothalamus by releasing endogenous opioids (i.e., beta-endorphin), which suppress the release of GnRh and LH required for normal menstrual cycles (McNeilly et al, 1985).

Since breastfeeding patterns provide Guatemalan women with the means of extending their birth intervals, mother's nutritional status may be a simple mechanism through which this duration is extended even further. Lunn (1985) proposed that malnutrition may be acting through the reduction of milk supply so that children of the more poorly nourished women have to be breastfed more frequently to satisfy their nutritional requirements.

Huffman and collaborators (1987) found that poor maternal nutrition delays the resumption of menses postpartum in Bangladesh. However, in Huffman's analysis it is not clear whether maternal health and nutrition act either independently or jointly with breastfeeding patterns.

Contrary to the results we obtain in this research, another reanalysis of the Guatemalan data by Kurz (1990) 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. The argument is that supplementation of mothers increases the risk of resuming menses by improving their nutritional status as well as through a reduction of lactation. Kurz (1990) also found that maternal nutritional status was not a significant predictor of the duration of postpartum amenorrhea when expressed as BMI or as subscapular skinfold[14].

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[14] K. Kurz uses a sample of about 300 women, for whom information was collected mostly during the period 1973-1977.

Model 3 shows that the effects of children's nutritional status (height by age) and mother's health status on the risk of resumption of menses are not significant. On the other hand, we also observe that child's health status is significant and in the expected direction; that is, mothers whose children are healthier tend to resume menses later than mothers with ill children. The risk for those women decreases by about 20 percent.

Maternal and child morbidity have never been reported to be a significant determinant of postpartum menses (Kurz, 1990). However, morbidity may affect the risk of resuming menses in two ways, by either increasing or decreasing the amount of suckling stimulus, depending on the severity and duration of the illness spell.

On the one hand, the woman could be ill and stop her activities, but not be ill enough to jeopardize the production of milk and stop breastfeeding; her child might increase the frequency of breastfeeding because the mother is more available. Thus, the risk of resumption of menses is reduced by mother's morbidity spells. On the other hand, the woman could be sick enough so that the production of milk is jeopardized and she does not feel like letting the child suckle, in which case nursing could be reduced enough to increase the risk of resuming menses.

A third possibility may help to explain the sign of the coefficient found in this study. Chronic maternal morbidity may lead to poor nutritional status. If they are in fact correlated, greater maternal morbidity could explain part of the negative relationship between maternal nutritional status and the risk of resuming menses. If that is the case, maternal morbidity would have a positive relationship with the duration of postpartum amenorrhea.

Finally, the effect of child's morbidity follows similar pathways. On the one hand, a child may desire to breastfeed more often if the illness caused dehydration or if the child is hungry because feeding was stopped during the illness spell. On the other hand, a serious illness may produce anorexia; thus the child may breastfeed less often or even stop breastfeeding, which may

trigger the resumption of menses.

Gompertz hazard models

A second set of estimates was obtained for a Gompertz duration structure. This specification was selected based upon the results obtained in other studies (Jones and Palloni, 1994), the results observed previously with the piecewise exponential specification, and the shape of the hazard function obtained from life tables analysis[15].

The same models used in the previous section were fitted under a Gompertz specification. Therefore, the coefficients of both types of models are in a way comparable.

Table 3 shows similar results to those obtained under a piecewise exponential specification. That is, the parametization of the duration structure has no effects on the value of the estimates. This may be explained by the fact that the piecewise exponential model very strongly suggests a monotonically increasing risk of resuming menses.

The duration structure of the estimated models (models 1-4) show that the risk of resuming menses increases over time, even after controlling for all the variables specified in the model. In fact, the coefficient of the slope of the model is positive and highly significant. Again, we can resort to the same possible explanations given in the previous section to account for the significant effect of the slope on the risk of resuming menses.

The effects of the background variables are about the same as those found in the piecewise exponential model. Parity has a significant effect on the resumption of postpartum menses, with similar relative risks. The effect of age is still not significant but in the expected direction; that is, older women have lower relative risk of returning menses than younger women. Because these

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[15] From estimates obtained through the product limit procedure, we used plots to determine the shape of the hazard function. By using the fact that the hazard function is the derivative of the cumulative hazard, H(t), we found monotonically decreasing hazards functions for the covariates included in the analysis.

variables are correlated, they are included in the models as control factors of the underlying process being studied.

The negative effect of agricultural and manual work is again in agreement with our theoretical model, in which women engaged in hard work activities experience lower risks of resuming postpartum fecundity, once we control for breastfeeding patterns and women's nutritional status.

The community variable has a positive and significant effect on menses. Women living in Atole villages have delayed resumption of menses compared to those living in Fresco villages.

As in the piecewise exponential model, by far the strongest effects on ovulation are associated with the death of the child. In Model 4 we can see that the risk of resuming menses when the child dies is 41 times higher than the risk when the child is fully breastfeeding. In a similar way, weaning and partial breastfeeding also increase the risk of resuming menses 13 and 3 times respectively. All these effects are highly significant.

In the same model, we can observe that high and medium frequency of breastfeeding reduce the risk of resuming menses when compared to low frequency of breastfeeding.

Mother's nutritional status indicators are not significant at all, except for that associated with mother's clinical supplementation (Table 3, Model 4). When women intake high amounts of supplement (either atole or fresco), the risk of resuming menses is 36 percent higher than the risk experienced by those women who intake low amounts of supplement. It seems that the ingestion of food, rich in protein and calories, may be reducing the inhibitory effects of chronic malnutrition on ovulation, at least in the short term.

Again, we observe in Model 4 that mothers with healthy children have a significantly lower risk of resuming menses than those with ill children.

Two-State Model with Heterogeneity

In all the models specified up to now we have assumed that all covariates involved in the process of returning to the postpartum menstruating state are measured. However, the presence of unobserved population heterogeneity is very likely in this type of study. In fact, some of the results reported above indicate the existence of unmeasured factors which are confounding the effects of the actually measured variables[16].

There are two possible sources of unmeasured heterogeneity in this study, both related to the existence of unobserved person-specific characteristics. The first one rests on the measurements about intensity, frequency, and supplementation of breastfeeding used in the models. Poor measures of these dimensions of breastfeeding could produce a bias of the estimates of lactation behavior and timing to supplementation, which in turn produces an upward bias of the estimated baseline, that is, produces a positive duration dependency.

In general, mother's social status is strongly associated with infant feeding patterns in two different ways. On the one hand, it may modify certain attitudes and norms toward breastfeeding and nursing. On the other hand, social position defines in a certain way the access and availability of resources that provide alternatives to lactation. However, the results reported above show that a control for a proxy for social status (i.e., education) does not change the shape of the hazard, and that education remains significant even after controlling for breastfeeding behavior, nutritional status, and energy expenditure.

The second source of heterogeneity is woman's fecundity. Its effect over the underlying biological process is clear. With the passage of time, the more fecund women are selected out of

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[16] The event history techniques used until now do not account for the existence of unobserved person-specific characteristics (i.e., unobserved heterogeneity). The lack of control for unobserved factors generally leads to bias estimates and thus to misleading conclusions (Heckman and Singer, 1984; Heckman and Walker, 1987).

the sample as they resume menses (proxy for ovulation) sooner and leave behind the less fecund women, whose waiting times to resumption of menses are longer, no matter the kind of breast-feeding pattern they show.

However, in every model a positive duration dependency was found and this result may be attributable to unmeasured factors rather than to the presence of a natural trend in the reduction of the levels of inhibitory hormones, which may be independent of breastfeeding behavior, nutritional level, or energy expenditure.

To test the presence of omitted covariates in the specifications presented up to now, we use a Gompertz hazard model with nonparametric heterogeneity[17] (Heckman and Walker, 1987).

Table 4 reports estimates from parametric hazard models that contrast the effects of controlling for a non-parametric representation of heterogeneity (with two points of support; that is, the sample is divided into two groups) along with their standard errors. If the estimated effects change from one model to another, the inferences made about them earlier in this paper are not robust; otherwise they receive further confirmation.

As we can see in the Table 4, the estimated effects of various covariates show significant changes, after controlling for unmeasured heterogeneity. That is, the presence of unmeasured covariates is very strong.

First, there are significant changes in the size of the coefficients associated with the slope of the model, parity, age, mother's occupation, breastfeeding patterns (especially for partial breastfeeding and frequency of breastfeeding), and maternal nutrition. All the other estimated effects seem to be robust and insensitive to the presence of unmeasured heterogeneity (i.e., their changes are statistically insignificant).

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[17] The selection of a nonparametric specification over a parametric one is based on the fact that the results obtained with the latter are very sensitive to the distribution imposed on the unknown covariates.

We observe that the estimated effect of the slope changes from 0.075 to 0.125. In other words, there is a faster return to the fecund state over time when controlling for unmeasured heterogeneity than when unmeasured covariates are not accounted for. This can be explained by the fact that there are some individual characteristics among women in the sample not taken into account that increase their risk of moving from the amenorrheic to the menses state. Thus, the actual risk of resuming menses over time is higher than it was thought before.

Other significant changes in the estimated effects are those associated with mother's occupation: from 0.069 to 0.254 for merchant and skilled workers; frequency of breastfeeding: from -0.089 to -0.287 for medium frequency and from -0.509 to -0.782 for high frequency; mother's nutritional status: from 0.063 to 0.143 for BMI (high nutrition) and from -0.142 to -0.223 for Mhc (low nutrition). All these new estimates are more consistent with our theoretical model and reinforce the direction of the relationship. In a similar way, we may conclude that the presence of unmeasured covariates in our model with no heterogeneity produces biases in the estimated effect of the measured covariates. That is, when some unmeasured covariates are not explicitly taken into account the risk of resuming menses increases or decreases, not because there is an actual change in the respective risks, but because there are changes in the subpopulation weights.

Conclusions

The results obtained for the two-state model are in general agreement with those found in other socio-demographic studies and accord with the biological evidence which suggests that resumption of menses, and thus ovulation, depends on a particular type of hormonal balance dependent on the suckling stimulus, energy expenditure, and women's nutritional status (Jones and Palloni, 1994; Huffman et al, 1987).

From these results we can conclude that: (1) the major determinants of the time to first menstruation after childbirth are still breastfeeding variables. In fact, all breastfeeding factors are

highly significant even after introducing the background covariates, nutritional and health variables. These changes suggest that the role of the breastfeeding variables in the resumption of menses mediates the effect of other covariates. By far the strongest effect is that of infant mortality, because the inhibiting effects of suckling on menstruation are terminated immediately with the death of the child; its effects should be equivalent to those of weaning.

With all other factors being equal, women who nurse their children less than 12 times a day increase their risk of early resumption of menses in comparison with those who nurse more frequently and more intensively. Therefore, it is clear that breastfeeding and amenorrhea are highly correlated and that lactation suppresses ovarian activity, and thus menstruation.

(2) Despite the importance of maternal nutrition in the determination of the amenorrheic period, we found that the effect of this variable is not significant in some cases (for instance, when measured by BMI) but it is when measured by supplementation and mother's head circumference. It seems that its effect is partially absorbed by breastfeeding variables. Another explanation is that its effect is confounded in some way with other factors because women with different risks and propensities all pooled together in the two-state model; thus we are not modeling the actual process adequately (Jones and Palloni, 1990).

Mother's health status was not found to be significant in all the models estimated. One interpretation is that the indicator being used is an unsatisfactory measure of health characteristics of the woman.

(3) Patterns of work were found to be important determinants of resumption of postpartum menses. Maternal work patterns play a significant role in suppressing postpartum fecundity and fertility. Its effects remain significant and in the expected direction after controlling for breastfeeding behavior and nutritional status. These results are similar to findings for another study from Indonesia (Jones and Palloni, 1994).

(4) The socioeconomic covariates included (mother's education and town of residence) are all highly significant, according to the respective test of significance. The fact that increasing education increases the risk of resuming menstruation earlier may reflect both cultural practices and the economic condition of the woman, such as socioeconomic status and family income (not considered among the socioeconomic variables). In general, for the Guatemalan case, women with low education tend to have the worst economic and nutritional conditions.

Among the demographic covariates, parity was significant in the determination of resumption of menses. Increased parity reduces substantially the risk of resuming menses, after controlling for all the other factors.

The direction of the relation between age of the mother at birth and duration of amenorrhea is consistent with the results obtained in other studies; however these effects are not significant.

(6) The model that controls for heterogeneity indicates the presence of unobserved covariates in the specifications considered throughout this study. When unobservables are controlled for, the significance of breastfeeding variables, mother's occupation, and mother's nutritional status increases and their effects remain in the expected direction.

However, with the data at hand, it is not clear if the Gompertz specification has captured all the sources of heterogeneity. It seems that this model does not deal adequately with the second source of heterogeneity described above (i.e., the fecundity argument).

In summary, the two-state model representation of our theoretical model shows that net of other factors, the risk of resuming menses increases over time and that it depends strongly on patterns of breastfeeding and women's work, and less so on maternal nutritional status.

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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 N	Full Sample %	Reduced Sample N	Reduced Sample %
PARITY					
One child	(Par1)	250	17.6	104	13.7
2 or 3 children	(Par2)	421	29.7	226	29.8
4 or more	(Par3)	746	52.6	428	56.5
MOTHER'S AGE					
13-19 years	(Age1)	168	11.9	72	9.5
20-29 years	(Age2)	722	51.0	371	48.9
30-49 years	(Age3)	527	37.2	315	41.6
MOTHER'S EDUCATION					
0 or 1 year	(Educ1)	394	27.8	220	29.0
2 or more	(Educ2)	791	55.8	462	60.9
Missing	(Educ3)	232	16.4	76	10.1
MOTHER'S OCCUPATION					
Houseviwes	(Work1)	839	59.2	482	63.6
Agric.and manual workers	(Work2)	74	5.2	36	4.7
Merchants, skilled workers	(Work3)	264	18.6	163	21.5
Missing	(Work4)	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)	(Freq1)	198	14.0	198	26.1
Medium (8-11 times/day)	(Freq2)	345	24.3	345	45.5
High (12 + times/day)	(Freq3)	215	15.2	215	28.4
Missing	(Freq4)	659	46.5	-	-
MOTHER'S NUTRITIONAL STATUS (HEAD CIRCUMFERENCE)					
Low (00.00-50.19 cms.)	(Mhc1)	247	17.4	158	20.8
Medium: (50.20-51.04 cms.)	(Mhc2)	249	17.6	172	22.7
High: (51.05 cms. +)	(Mhc3)	499	35.2	341	45.0
Missing	(Mhc5)	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)
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)
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)
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.

Covariates	Model 1	Model 2	Model 3	Model 4
Constant	-3.778	-3.880	-4.362	-4.225
	(0.00)	(0.00)	(0.00)	(0.00)
Slope	0.098	0.114	0.081	0.080
	(0.00)	(0.00)	(0.00)	(0.00)
BACKGROUND VARIABLES				
One child (par1)		0.203	0.266	0.275
		(0.04)	(0.01)	(0.01)
Four or more children (par2)		-0.112	-0.190	-0.189
		(0.19)	(0.03)	(0.03)
13-19 years (age1)		0.413	0.168	0.146
		(0.00)	(0.14)	(0.20)
30-49 years (age3)		-0.059	-0.117	-0.120
		(0.45)	(0.13)	(0.12)
0 or 1 year (educ1)		-0.337	-0.273	-0.272
		(0.00)	(0.00)	(0.00)
Agric. and manual workers(work2)		-0.497	-0.647	-0.631
		(0.00)	(0.00)	(0.00)
Merchants,skilled workers(work3)		0.085	0.016	0.038
		(0.31)	(0.85)	(0.63)
Atole village		0.305	0.227	0.227
		(0.00)	(0.00)	(0.00)
BREASTFEEDING VARIABLES				
Partial (Bf2)			0.953	0.950
			(0.00)	(0.00)
Weaning (Bf3)			2.540	2.548
			(0.00)	(0.00)
Infant death (Bf4)			3.734	3.717
			(0.00)	(0.00)
Medium frequency (8-11 times)			-0.091	-0.085
			(0.41)	(0.44)
High frequency (12+ times)			-0.530	-0.530
			(0.00)	(0.00)
High supplement (child) (Low intensity nursing)			0.154	0.158
			(0.15)	(0.13)

* p-values are shown in parentheses

Table 3 (continued)
Covariate effects* for the transition to menses in two-state Gompertz hazard models.

Covariates	Model 1	Model 2	Model 3	Model 4
MOTHER'S NUTRITION				
Bmi>20			0.065	0.072
			(0.52)	(0.47)
Mhc<50.19 (low nutrition)**			-0.165	-0.140
			(0.09)	(0.15)
50.20 < Mhc < 51.04 (medium)			-0.062	-0.051
			(0.52)	(0.59)
High supplement (mother)			0.313	0.311
(high nutrition)			(0.00)	(0.00)
CHILD'S NUTRITION				
Poorly nourished			0.178	
			(0.06)	
MOTHER'S HEALTH				
No morbidity (healthy)			0.106	
			(0.39)	
CHILD'S HEALTH				
No morbidity (healthy)			-0.249	-0.247
			(0.00)	(0.00)
log L	-3728	-3662	-3223	-3229
Chi Square	515	646	1525	1513
df	1	11	32	28
N	1417	1417	1417	1417

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

Table 4
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)
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)
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) (Low intensity nursing)	0.170	0.181
	(0.12)	(0.13)

* standard errors are shown in parentheses

Table 4 (continued)
Covariate effects* for the transition to menses in two-state Gompertz hazard models without and with heterogeneity

Covariates	without heter.	with heter.
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)
High supplement (mother) (high nutrition)	0.302	0.308
	(0.11)	(0.12)
CHILD'S HEALTH		
No morbidity (healthy)	-0.277	-0.238
	(0.09)	(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.)

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