THE CONTRACEPTIVE ROLE OF BREASTFEEDING

Jean-Pierre Habicht, Julie DaVanzo, William P. Butz, Linda Meyers

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This Note should be of interest to demographers and biomedical scientists studying the determinants of postpartum amenorrhea and the mechanisms by which breastfeeding affects it.

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SUMMARY

We review (1) neurohormonal mechanisms by which breastfeeding postpones the return of ovulation and menstruation after birth, and (2) various statistical procedures used to analyze this effect in human populations. This review reveals that the biology and the statistical procedures are incompatible. We propose a statistical approach, compatible with present knowledge of physiology, that differentiates between ovulatory-inhibiting mechanisms at birth and the weakening of these inhibitions thereafter, so that it is possible to investigate the effects on these mechanisms due to breastfeeding and to other determinants such as mother's age. An empirical test with typical recall data indicates that full breastfeeding postpones ovulation longer than does supplemented breastfeeding, and that both have stronger contraceptive effects than has been previously thought.
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I. INTRODUCTION

Empirical research has established that breastfeeding postpones the return of ovulation after birth (Perez et al., 1972; Perez et al., 1971); that is, it lengthens postpartum anovulation, during which a woman cannot conceive. It also postpones the return of menstruation after birth—postpartum amenorrhea (reviewed in Masnick, 1979, and McCann et al., 1981). Full (unsupplemented) breastfeeding extends anovulation and amenorrhea longer than does supplemented breastfeeding (Chen et al., 1974; Jain et al., 1970). Hence, birthspacing is lengthened and fertility reduced in many populations where women practice intensive and lengthy breastfeeding. Both empirical estimates (Rosa, 1978; Jain, Hermalin, and Sun, 1979) and simulations of birth interval dynamics (Bongaarts and Menken, 1983) suggest that, in less developed countries, this contraceptive effect of breastfeeding is substantial. In these populations, breastfeeding may well delay or prevent more births than do all contraceptives supplied by family planning programs (Rosa, 1978).

It is partly for this reason that recent declines in breastfeeding in cities and some rural areas of many less developed countries (McCann et al., 1981) have caused public health concern.¹ Unless compensatory increases in use of other contraceptives occur, women who breastfeed less will experience shorter birthspacing and higher fertility, with adverse consequences for their own health and that of their children (DaVanzo, Butz, and Habicht, 1983). Accurate estimates of the contraceptive role of breastfeeding are required to gauge these effects and to plan the delivery of modern contraceptives.

This article reviews present knowledge about the physiology that links breastfeeding to the durations of postpartum anovulation and amenorrhea. We then relate this knowledge to the statistical procedures that have been used to estimate the nature of these linkages at the

¹Breastfeeding declines are also viewed with alarm because they deprive infants of nutrition and immunological protection where adequate breastmilk substitutes are unavailable or improperly used.
population level. The underlying physiology and these statistical investigations, which have proceeded contemporaneously but independently, are shown to be incompatible. We suggest a statistical procedure that makes use of the most commonly available data—mothers' reports of their breastfeeding practices and of the time when they first menstruated after a birth—and is consistent with physiological mechanisms as now understood. An illustration of this procedure suggests that the contraceptive effect of breastfeeding, especially unsupplemented breastfeeding, may be considerably greater than previous studies have indicated.
II. PHYSIOLOGY OF LACTATIONAL ANOVULATION AND AMENORRHEA

Prolactin and Anovulation

The physiological mediator of the association between breastfeeding and postpartum anovulation appears to be the anterior pituitary hormone prolactin. The principal evidence is that prolactin levels are higher in women who are breastfeeding than in women who are not (Delvoye and Robyn, 1980; Baird et al., 1979; Bonnar et al., 1975), especially in women who are also amenorrheic or anovulatory (Howie et al., 1981; Duchen and McNeilly, 1980; Gross and Eastman, 1979). Fully breastfeeding women also appear to have higher prolactin levels than those who are partially breastfeeding (Howie et al., 1981; Duchen and McNeilly, 1980), and they have longer postpartum amenorrhea (Prema and Ravindramath, 1982; Perez et al., 1971). Furthermore, elevated prolactin levels in the absence of breastfeeding or recent parturition are associated with amenorrhea, a condition that is ameliorated when prolactin is lowered by drug therapy (reviewed in McNeilly, 1979).

Prolactin levels rise during pregnancy (Jaffe et al., 1973). After parturition, they fall more rapidly for those who do not breastfeed than for those who do (Bonnar et al., 1975; Jaffe et al., 1973; Rolland et al., 1975). In the absence of breastfeeding, prolactin levels return to pre-pregnancy levels by three weeks postpartum (Bonnar et al., 1975). A similar rapid decrease to nonpregnant levels has been observed following weaning (Rolland et al., 1975; Tyson et al., 1978). In breastfeeding women, basal prolactin levels may be elevated as long as 18 to 21 months postpartum (Delvoye and Robyn, 1980). The prolactin level usually surges acutely within an hour following a breastfeeding episode (Gross and Eastman, 1979; McNeilly, 1977). This surge has been reported to be blunted or absent beyond 90 days of lactation, but these findings were probably an artifact caused by the greater variability in timing and magnitude of the prolactin response to suckling when lactation is prolonged (Gross and Eastman, 1979).
The maintenance of high prolactin levels appears to be related primarily to nipple stimulation because this activity, even in the absence of lactation, causes prolactin to rise (McNeilly, 1977). In addition, suckling of denervated or anesthetized nipples does not result in prolactin surges. Finally, induction of lactation, presumably through increased prolactin production, has been reported following nipple stimulation or suckling in men, nulliparous women, and postmenopausal women. All these findings imply that a neurogenic stimulus from the nipple promotes prolactin secretion and increased prolactin levels in the blood. Nipple stimulation is thought to have this effect by blocking the release of dopamine, which otherwise inhibits prolactin release (reviewed in Ben-Jonathan, 1980).

There are as yet no studies of the levels of prolactin below which ovulation resumes. Two mechanisms have been proposed to explain prolactin's anovulatory effect. The first, better-enunciated, hypothesis is that prolactin acts to change the sensitivity of the hypothalamus to the feedback effects of estrogen (McNeilly, 1979). This may occur if prolactin inhibits the normal estrogen-induced release of luteinizing hormone-releasing hormone from the hypothalamus (reviewed in Howie and McNeilly, 1979). This inhibition would prevent the episodic release and the surge of luteinizing hormone required for ovulation.

The other proposed mechanism is a direct inhibiting effect of prolactin on the ovaries' response to gonadotropins (luteinizing hormone and follicle-stimulating hormone) (Bonnar et al., 1975; McNeilly, 1979; Howie and McNeilly, 1979). Research testing this hypothesized mechanism has provided conflicting results. The evidence suggests that the ovaries are refractory to induction of ovulation at least during the first three weeks postpartum (McNeilly, 1979), but that could be due to factors other than prolactin (Zarate et al., 1974). Beyond this time, the refractoriness can be overcome by administration of exogenous gonadotropins (McNeilly, 1979). If this second mechanism exists, it may be operative only in the early puerperium.

In summary, research suggests that the suckling stimulus of breastfeeding, through elevated prolactin levels, is the major determinant of prolonged anovulation accompanying lactation.¹

¹The relationship between the suckling stimulus of breastfeeding
Anovulation and Amenorrhea

Most statistical studies of the contraceptive role of breastfeeding (reviewed below) use the duration of postpartum amenorrhea as a proxy variable for the duration of postpartum anovulation. A woman can easily observe menstruation, but she cannot directly tell the timing of ovulation, which is therefore rarely recorded in datasets. Ovulation is, however, the phenomenon of real interest; ovulation, not menstruation, makes a woman able to conceive, and the physiological evidence reviewed above links lactation directly to ovulation. Ovulation precedes menstruation by about 9 days (Jain et al., 1979). However, some first postpartum menstruations precede ovulation; these are called anovulatory menstruations. Because of them, the durations of postpartum anovulation and amenorrhea are not perfectly correlated.

Table 1 presents the probabilities that a first postpartum menstruation is ovulatory, by period postpartum and breastfeeding status, as reported by Perez et al. (1971, Table 12). Menstruations occurring in the first month postpartum are very likely to be anovulatory. Particular observations of first menstruation were more (up to a probability of 0.93) or less likely to be ovulatory, depending on the time postpartum and the woman’s breastfeeding status. In general, a first menstruation is less likely to be ovulatory the closer it follows a birth and the more fully the woman is breastfeeding.

If the timing of the first ovulation was independent of the appearance of a proceeding anovulatory menstruation, one could weight the menstruation according to the probabilities in Table 1 without biasing the data relative to the timing of ovulation. That breastfeeding has a higher correlation with birth intervals than with reported menstruation (Jain, Hermalin, and Sun, 1979) can be taken as evidence for timing of ovulation. This independence of timing of ovulation is more likely to be true for postpartum bleeding, which is and the continued maintenance of lactation is probably also mediated through prolactin (Howie et al., 1981; Duchen and McNeilly, 1980; McNeilly, 1977; Howie and McNeilly, 1979). Thus a common mechanism links duration of postpartum anovulation to the physiological maintenance of lactation, but this relationship is not important for the following discussion about the contraceptive effect of breastfeeding.
Table 1

PROBABILITY THAT A FIRST POSTPARTUM MENSTRUATION IS OVULATORY, BY PERIOD POSTPARTUM AND BREASTFEEDING STATUS

<table>
<thead>
<tr>
<th>Period Postpartum</th>
<th>Breastfeeding Status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Full</td>
</tr>
<tr>
<td>0-&lt;1 month</td>
<td>0.00</td>
</tr>
<tr>
<td>1-2 months</td>
<td>0.00</td>
</tr>
<tr>
<td>&gt; 2 months</td>
<td>0.58</td>
</tr>
</tbody>
</table>

SOURCE: Probabilities are the frequencies reported by Perez et al. (1971).

the most likely cause of reported onsets of menstruation during the first weeks after giving birth. However, true menstruations that are anovulatory may presage an earlier return of postpartum ovulation than if there is no anovulatory menstruation at all. In this case, one should add on to the duration of postpartum amenorrhea the remaining time to ovulation. Although a mean estimate (38.2 days) is known (Perez et al., 1971), this remaining time may not be independent of duration since birth or of intensity of breastfeeding; those relationships are not known.
III. A MODEL OF THE RELATIONSHIP BETWEEN BREASTFEEDING AND THE DURATION OF POSTPARTUM ANOVULATION

Figure 1 depicts a model based on the current knowledge of blood hormone levels, breastfeeding, and the duration of postpartum anovulation. At birth \((t = 0)\), prolactin hormone \((H)\) is at a higher level, \(I\), than at conception. The prolactin level falls more quickly when no breastfeeding occurs (slope \(n\)) than when partial (supplemented) breastfeeding occurs (slope \(p\)). Prolactin falls least rapidly during full (unsupplemented) breastfeeding (slope \(f\)).\(^1\) That is, we hypothesize that \(n < p < f < 0\). When the prolactin level falls below a certain threshold, \(V\), ovulation resumes.

The figure embodies a simplifying hypothesis that the prolactin level declines with slope \(n\) when no breastfeeding is taking place, no matter what the woman's previous breastfeeding history. Likewise the slopes \(f\) and \(p\), referring to full and partial breastfeeding, are independent of previous breastfeeding experience. Although the slope for each segment of prolactin decline is assumed to be independent of previous breastfeeding patterns, the duration of postpartum anovulation is not. This length of time depends on how long following the birth the woman full breastfed, partial breastfed, and did not breastfeed, because the prolactin level is pictured as declining at different rates during each type of breastfeeding. A woman who does not breastfeed will ovulate sooner after a birth (at time \(t\)\(^n\) in the figure) because her prolactin level declines at rate \(n\) and falls most quickly to the ovulatory threshold, \(V\). A woman who first full breastfeeds then partial breastfeeds will experience a longer period of postpartum anovulation (to time \(t\)\(^p\) in the figure), because her prolactin level declines first at the slowest rate, \(f\), and then at an intermediate rate, \(p\).

\(^1\) The model can be easily generalized to include any number of gradations of full and partial breastfeeding, differentiated by the frequency, intensity, and duration of suckling, or their proxies.
Legend:

- **H** = Level of hormone (presumably prolactin) in blood, or level of other factor that suppresses ovulation and promotes lactation

- **I** = Initial level of **H** at birth

- **V** = **H** threshold that allows ovulation to resume

- **n** = Slope of decline in **H** during nonbreastfeeding

- **p** = Slope of decline in **H** during partial breastfeeding

- **f** = Slope of decline in **H** during full breastfeeding

- **t_V** = Duration of postpartum anovulation

**Fig. 1** — Theoretical model of relationships among breastfeeding, blood hormone levels, and duration of postpartum anovulation
When prolactin falls below a threshold even lower than V, lactation ceases if it has not been previously stopped for other reasons unrelated to blood hormone levels. Restriction or cessation of lactation before its physiological limit can occur for many reasons—physiological and behavioral, voluntary and involuntary. In this model, the relationships between breastfeeding and postpartum anovulation are independent of these reasons, and neither the physiological maximum duration of lactation nor the change in prolactin levels after ovulation is germane. They are therefore not depicted in Fig. 1.

Most physiological studies report that prolactin levels decline at exponential rates, rather than linearly as pictured in Fig. 1. The vertical axis in Fig. 1 could accordingly be scaled as the natural logarithm of the prolactin level. In fact, Fig. 1 can represent any physiological or endocrine influence that is related to breastfeeding and causes the resumption of ovulation when some threshold is crossed, as long as the ratios of changes accompanying full, partial and no breastfeeding are constant over time. Thus, physiological tests of the model described in this review should not be restricted to studies of prolactin hormonal levels.
IV. IMPLICATIONS OF THE MODEL FOR DATA ANALYSIS

This section derives the model's implications for data analysis. In light of these implications, we then review the statistical techniques that have been used to describe the breastfeeding-anovulation relationship in population data.

The level, \( V \), of the hormone at the time of first postpartum ovulation (\( t_{V_i} \)) can be read directly from Fig. 1 or described equivalently in algebraic form. For a woman who was full breastfeeding when ovulation resumed (\( i = f \)),

\[
V = I + f \cdot t_{V_f}.
\]  

For a woman who was partial breastfeeding when ovulation resumed (\( i = p \)),

\[
V = I + f \cdot t_f + p \cdot (t_{V_p} - t_f),
\]

where \( (t_{V_p} - t_f) \) equals the length of partial breastfeeding up to the onset of ovulation. This expression applies whether the woman had ever full breastfed this child (\( t_f > 0 \)) or not (\( t_f = 0 \)).

Finally, for a woman who was not breastfeeding at all when ovulation resumed (\( i = n \)),

\[
V = I + f \cdot t_f + p \cdot t_p + n \cdot (t_{V_n} - t_f - t_p),
\]

where \( (t_{V_n} - t_f - t_p) \) equals the length of time the woman did not breastfeed to the onset of ovulation. Equation 3 applies whether the woman had ever full breastfed (\( t_f > 0 \)) or partial breastfed (\( t_p > 0 \)), or neither (\( t_f = 0 \) and \( t_p = 0 \)). As before, we hypothesize that \( n < p < f < 0 \) and that \( I > V > 0 \).¹

¹ Equations 1-3 are special cases of the general situation in which
Equations (1)-(3) are denominated in units of prolactin or other relevant physiological processes. However, most data lack this information but do describe the pattern of breastfeeding activities that directly affect these physiological processes. Solving each of these expressions for \( t_V \), the duration of postpartum anovulation, yields equations that describe the dependence of this duration on the lengths of full and partial breastfeeding:

\[
\begin{align*}
\frac{t_V}{f} &= -(I - V)/f = \alpha_1 > 0 \\
\frac{t_V}{p} &= -(I-V)/p + (1 - (f/p))t_f = \alpha_2 + \beta_2 t_f > 0 \\
\frac{t_V}{n} &= -(I - V)/n + (1 - (f/n))t_f + (1 - (p/n))t_p = \alpha_3 + \beta_3 t_f + \gamma_3 t_p > 0 \cdot
\end{align*}
\]

(4) \hspace{1cm} (5) \hspace{1cm} (6)

To see the correspondence between Eqs. (1)-(6) and Fig. 1, note that a woman who never full breastfed, but ovulated while partial breastfeeding, is equivalently described by the line in Fig. 1 that reaches hormone level \( V \) at time \( t_V \), and by Eqs. (2) and (5) with \( t_f = p_f \) = 0. Similarly, women who resumed ovulation when not breastfeeding are described by Eqs. (3) and (6). For such a woman who partial breastfed but never full breastfed, setting \( t_f = 0 \) in these expressions describes

\( m \) different categories of breastfeeding frequency and intensity can be distinguished:

\[
V = I - \sum b_i \cdot t_i - b_m \cdot (t_V - \Sigma t_i).
\]

Here the \( m \) different slopes \( b_i (i = 1, \ldots, m) \) correspond to the \( m \) categories of breastfeeding. Each slope applies for a particular length of time, \( t_i \), when that type of breastfeeding is being conducted. Any succession of feeding regimes (e.g., more intensive to less intensive breastfeeding and then back again) can be described by this general equation. The final slope, \( b_m \), corresponds to the type of breastfeeding when ovulation resumes; this may be the fullest type, no breastfeeding at all, or any intermediate category. The duration of this last category is simply the time to ovulation minus the summed lengths of breastfeeding of all previous types.
the line in Fig. 1 that reaches level V at time \( t_{V_{n2}} \). For a woman who
full breastfed but never partial breastfed and resumed ovulation when
not breastfeeding, setting \( t_p = 0 \) in Eqs. (3) and (6) describes the line
that reaches level V at time \( t_{V_{n3}} \). And for a woman who never breastfed
at all, setting \( t_f = t_p = 0 \) in Eqs. (3) and (6) yields the formula for
the line in Fig. 1 that reaches level V at time \( t_{V_{n1}} \).

Inspection of Eqs. (4)-(6) reveals three characteristics of the
breastfeeding-ovulation relationship that are important for evaluating
past attempts to estimate this relationship with population-level data:

(1) Only the durations of the breastfeeding states (e.g., full and
partial) that ended before ovulation resumed are included among the
variables on the right-hand side of Eqs. (4)-(6). The duration of the
breastfeeding state that was current when ovulation began does not
appear as a right-hand variable; that is, the length of full
breastfeeding does not appear in Eq. (4), and the length of partial
breastfeeding is not in Eq. (5).

(2) Counting the hormonal difference \( I - V \) as a single parameter,
each of the Eqs. (4)-(6) contains one more underlying physiological
parameter than it contains empirical coefficients that are estimable
with data on durations of breastfeeding and anovulation. For example,
Eq. (4) contains physiological parameters \( I - V \) and \( f \), but only one
estimable coefficient, \( \alpha_1 \). Similarly, Eq. (5) contains physiological
parameters \( I - V \), \( f \), and \( p \), but only estimable coefficients \( \alpha_2 \) and \( \beta_2 \).
Therefore only ratios of the underlying physiological parameters can be
estimated with data on durations of breastfeeding and anovulation. In
Eq. (6), for example, one can estimate the value of \( (I - V)/n \), of \( f/n \),
and of \( p/n \), but not of the four parameters, \( I - V \), \( f \), \( p \), and \( n \),
separately.

(3) The intercept coefficients, \( \alpha_1 = -(I - V)/f \), \( \alpha_2 = -(I - V)/p \),
\( \alpha_3 = -(I - V)/n \), differ, depending on the type of breastfeeding
(including non-breastfeeding) being conducted when ovulation began. The
full breastfeeding coefficients--\( \beta_2 = 1 - (f/p) \) and \( \beta_3 = 1 - (f/n) \)--
differ similarly. Therefore, to know what is being estimated in terms
of the underlying physiological parameters, the relationships between
breastfeeding and postpartum anovulation must be examined within
subsamples defined by the type of breastfeeding activity current at the
time ovulation resumed. Such separate estimation is not straightforward. It would produce unbiased estimates of the intercept and slope coefficients only if the distributions of I - V, f, p, and n were the same in each subsample. Unfortunately, this cannot be the case. For example, women who are fully breastfeeding when ovulation resumes will tend to have shorter durations of postpartum anovulation than women who ceased full breastfeeding before their ovulation resumed. The former women will be in this category precisely because they have smaller (I - V) (see Fig. 1) and/or larger (f/n) than women who first ovulate after ceasing full breastfeeding. In effect, women are self-selected into subsamples based on the magnitudes of the variables whose associations are the subject of investigation. Hence, the parameters (I - V), f, p, and n will differ systematically across the subsamples.

These three implications are derived from a model that is consistent with current understanding of the physiological mechanisms linking breastfeeding to the duration of postpartum anovulation. We now review alternative statistical methods that have been used to estimate the link between breastfeeding and anovulation at the population level. In general, these methods treat the phenomenon of interest either as a duration (length of postpartum anovulation) or as a probability (that ovulation will resume before a certain time postpartum). The two prominent methods in the "duration" category are regression by the technique of ordinary least squares and presentation of mean anovulation durations by length of breastfeeding. The principal method of the "probability" type is life table analysis. Two others, probit analysis and hazards models, have not to our knowledge been applied in this area, but are briefly discussed as possibilities.

**Ordinary Least Squares Regression Analysis**

Previous applications of least squares regression analysis (Perez et al., 1972; Jain et al., 1970; Jain, Hermalin, and Sun, 1979; Salber, Feinleib, and McMahon, 1966; Jain and Bongaarts, 1981) have used as a regressor the *total* length of breastfeeding, both before and after the resumption of ovulation. This procedure contradicts the first implication of our model. These analyses must implicitly assume either that future events (breastfeeding activity after ovulation) can
influence past events (duration of postpartum anovulation), or that events occurring before ovulation (such as intensity of early suckling) influence the durations of both ovulation and lactation. The first assumption cannot be true. The second could be true, according to our model, only if lactation were to continue to its physiological maximum. The regression studies we have seen neither mention these assumptions nor present evidence concerning their appropriateness.

These least squares regression studies have also, in the same analysis, combined women with different breastfeeding activities at the time of the first postpartum ovulation. This procedure forces the same intercept and slope estimates on parameters that differ according to whether the woman was full breastfeeding, partial breastfeeding, or not breastfeeding at that time. The resulting estimates are, by the third implication of our model, difficult to interpret and impossible to compare across studies.

Mean Anovulatory Durations by Length of Breastfeeding

Instead of performing ordinary-least-squares regression analysis, some investigators have presented means of amenorrhea length by duration of breastfeeding (Cantrelle, Ferry, and Mondot, 1978; Corsini, 1979). As a representation of the underlying physiological relationship, these presentations have the same flaws as the existing least-squares regression studies.

Life Table Analysis

Other investigations have used life-table analysis to describe the probability that amenorrhea will continue beyond successive postpartum intervals, depending on lengths of breastfeeding (Salber, Feinleib, and McMahon, 1966; Potter et al., 1965). This method has the advantage over ordinary least squares regression analysis that it can include data about women who are still amenorrheic at the time of observation. Ordinary life-table analysis, however, presumes implicitly that only the type of breastfeeding activity being practiced at the particular postpartum duration being considered is important in determining the probability of ovulation. Thus, life-table analysis does not correspond to Fig. 1, which presumes that all previous breastfeeding activities
affect the probability of ovulation at any subsequent time. If the model in Fig. 1 is correct, it is very difficult to interpret these life-table analyses meaningfully.

Hazard and Probit Analysis

Life-table methods can be generalized to consider multivariate influences on the probabilities of remaining infertile. This could be done with hazard models (Kalbfleisch and Prentice, 1980) or else with probit analysis, which provides a discrete approximation to the hazard function. We have seen no analyses of anovulation or amenorrhea using these techniques. Like life-table analysis, these methods can include data on women with open postpartum anovulatory or amenorrhea intervals. In contrast to simple life-table analysis, these methods can take account of past breastfeeding activities, making the methods compatible in this regard with the model of Fig. 1. However, these approaches still contradict the third implication of our model: They are subject to the biases that arise from pooling all observations into the same sample or, alternatively, to the other biases that result from separate estimation within self-selected subsamples.

In addition, probit analysis has the disadvantage of being highly sensitive to departures from normality of the data. This technique and other methods that are sensitive to strong assumptions about the stochastic distribution of first ovulations are not attractive choices in analyzing recall data on breastfeeding and amenorrhea, which usually exhibit substantial digit-preference biases.

Other Methods

Several methods to investigate the relationship of breastfeeding to postpartum anovulation do not fall into the above categories. They all require strong assumptions about the stochastic distribution of first ovulations after birth.

Ginsberg (1973) developed a bivariate stochastic model relating duration of postpartum anovulation to the durations of different kinds of breastfeeding. His model assumes that breastfeeding activities follow the sequence of full to partial to none, and that the effect on ovulation of a particular breastfeeding activity is independent of
previous breastfeeding activities. These features are compatible with our model. Furthermore, the Ginsberg model provides an excellent fit to his data. However, that model requires stringent assumptions about the distributions of duration of breastfeeding and the onset of ovulation. Further, its parameters are not related to underlying physiological mechanisms as currently understood, nor is it evident how they are related to the parameters in Fig. 1.

Several reports characterizing the distributions of the duration of postpartum amenorrhea in populations have also considered the effect of breastfeeding on amenorrhea (Potter and Kobrin, 1981; Jilliewicz, 1979; Lesthaeghe and Page, 1980). However, these studies were not directed to eliciting an understanding of the physiological link between breastfeeding and amenorrhea, and the parameters of their proposed equations cannot be related to our model.

Summary of Implications

A simple model that is consistent with the most basic evidence about the physiology that relates breastfeeding to the duration of postpartum anovulation is inconsistent with the least squares, tabular, life table, and other methods used to date to investigate this relationship. If only breastfeeding before ovulation affects the duration of postpartum amenorrhea, these analyses misdefine the variables corresponding to length of breastfeeding, and use aggregated samples that cause coefficient estimates to be biased by the composition of the sample. In addition, most of these analyses fail to consider the problems presented by use of amenorrhea as a proxy indicator for anovulation. These problems render interpretation of findings and comparisons across studies very problematic, in many cases probably impossible.
V. ESTIMATION OF THE PHYSIOLOGICAL PARAMETERs

The Estimation Procedure

We now propose a statistical procedure that is consistent with the three implications just discussed, and that can use population data to estimate the physiological parameters of interest. An illustrative estimation of this model is provided, using typical recall data on lengths of amenorrhea and breastfeeding.

We first divide the sample of observations into three subsamples according to whether ovulation resumed during full breastfeeding, during partial breastfeeding, or when no breastfeeding was taking place. We then run a regression of Eq. (4) on this first subsample, of Eq. (5) on the second, and of Eq. (6) on the third. These three regressions are run simultaneously, with the parameters in Eqs. (4) and (5) constrained to be the following functions of the parameters in Eq. (6):

\[
\begin{align*}
\alpha_1 &= \frac{\alpha_3}{1 - \beta_3}; \\
\alpha_2 &= \frac{\alpha_3}{1 - \gamma_3}; \\
\beta_2 &= \frac{(\beta_3 - \gamma_3)/(1 - \gamma_3)}.
\end{align*}
\]

These constraints derive directly from Eqs. (4)-(6); they therefore constitute information about the theoretical model that should be used in estimating the model's parameters.\(^1\)

Simultaneous estimation of Eqs. (4)-(6) provides a method of satisfying these conditions on the parameters and forces each of the intercept and slope coefficients to take account of all the information in all three subsamples, thereby eliminating the biases that would arise from estimation on any one of these self-selected subsamples by itself (see the second implication of our model in the previous section).\(^2\)

\(^1\)The selection of \(\alpha_1\), \(\alpha_2\), and \(\beta_2\) as secondary parameters, to be defined in terms of the other three, is arbitrary. The theory, as embodied in Eqs. (4)-(6), implies that only three of these six coefficients are independent; any three may be chosen, with the others to be defined in terms of them.

\(^2\) Nonlinear least squares routines to accomplish this constrained
Data, Sample Selection, and Variable Construction

We use data from the Malaysian Family Life Survey (MFLS) (Butz and DaVanzo, 1978). This population-based probability sample survey was conducted in Peninsular Malaysia in 1262 households that each contained at least one ever-married woman less than 50 years old. The data for this analysis are from the Round-1 Female Retrospective Life History questionnaire (MF2), which was answered by 88 percent of the eligible probability sample in August-November 1976 and which includes a complete life history of all of each woman's pregnancies and related events. The data analyzed here exclude 49 intervals after the births of twins and 92 intervals for women who were amenorrheic at the time of the survey. For each of the 5312 live births that remain, information is available on length of full (unsupplemented) breastfeeding, length of partial (supplemented) breastfeeding, and duration of postpartum amenorrhea. We selected from this sample a random subsample of 600 observations for the analyses reported here.\(^3\)

The relationship in the MFLS data between total duration of breastfeeding (TBF) and duration of postpartum amenorrhea (PPA), both in months, is: PPA = 3.3 + 0.4 TBF (t = 47.0), n = 5312, \(R^2 = .30, p < .05\). That relationship is very similar to those reported elsewhere, in some cases from prospectively collected, clinical data. (See, for example, Perez et al., 1971, 1972, for Chile; Van Ginneken, 1978, Fig. 5, p. 190 for Colombia, Thailand, and South Korea; and Jain et al., 1970, 1979, for Taiwan.)

In addition to the statistical biases discussed above, the MFLS data and the data used in other studies share two other sources of potential problems: (1) the existence of anovulatory first

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\(^3\)Excluding these open amenorrheic intervals from the analysis may introduce censoring bias. However, the amount of bias should be very small because less than 2 percent of intervals are affected.

\(^4\)This random subsample is representative of the whole sample in that each of the following statistics is within one standard derivation of the mean values (in parentheses) for the whole sample: months of full breastfeeding (1.41), partial breastfeeding (8.21), and amenorrhea (7.17); proportion of amenorrheas lasting less than 8 days (0.0053) and less than one month (0.020).
menstruations; (2) the fact that time to first postpartum ovulatory menstruation is longer than duration of postpartum anovulation. Furthermore, the MFLS data, like other recall data, are subject to three more possible complications: (3) artifactual recall that breastfeeding and amenorrhea end simultaneously; (4) artifactual recall that either breastfeeding or amenorrhea ends at a preferred-digit duration, such as 6, 12, 18, or 24 months; (5) false reporting of very long durations of breastfeeding or amenorrhea.

Concerning the first problem, Table 2 shows the number of observations in our full sample that fall into the nine breastfeeding-amenorrhea categories of Table 1. The probabilities reported in Table 1 suggest that many of these observed menstruations are unlikely to have been ovulatory. To correct for these discrepancies between ovulation and menstruation, we weight the observations in our data by the proportions in Table 1. First menses that are less likely to have been ovulatory are given smaller weights than the others. Observations with zero probability of being ovulatory (in four of the cells in Table 1 and 2) are entirely deleted from the sample.

Table 2

DURATION OF POSTPARTUM AMENORRHEA BY BREASTFEEDING STATUS
(Numbers of observations in the MFLS sample)

<table>
<thead>
<tr>
<th>Period Postpartum</th>
<th>Breastfeeding Status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Full</td>
</tr>
<tr>
<td>0-&lt;1 month</td>
<td>26</td>
</tr>
<tr>
<td>1-2 months</td>
<td>196</td>
</tr>
<tr>
<td>&gt; 2 months</td>
<td>194</td>
</tr>
</tbody>
</table>
As a result, estimates from the weighted data overstate the duration of anovulation for women with early anovulatory postpartum menstruation. Below we investigate the effects of this source of bias by comparing the results of these weighted data with those using unweighted data and a modified weighted scheme,\(^5\) which continues to exclude probable postpartum bleeding episodes but assigns to all anovulatory menstrual periods after the first month another 38 days before ovulation. This correction probably underestimates the duration of anovulation for short anovulatory periods and overestimates it for long anovulatory periods. The greater the independence of ovulation from previous menstruation, the greater this bias will be.

We solve the second problem by subtracting nine days from the duration of postpartum amenorrhea.\(^6\) We argue below that a priori conceptual criteria and the empirical fit favor ignoring the third and fourth problems listed above. We deal with the fifth by excluding observations with very long reported lengths of amenorhea or of supplemented or unsupplemented breastfeeding.\(^7\) In our opinion, the resulting data set most accurately represents the true distributions of postpartum anovulation and breastfeeding in this Malaysian population. We report estimates of the model with these revised data and then show the sensitivity of these estimates to ignoring the above problems.

\(^5\) This procedure excludes menstruations reported in the first month postpartum because fully breastfeeding women do not menstruate in the first month and other women do so rarely (Perez et al., 1971). This procedure adds to the duration of all other postpartum amenorrheas the product of 38 days times one minus the probability in Table 1.

\(^6\) Perez et al. (1972). We subtract the nine days after the data are weighted and assigned to the three subsamples. This procedure and that of footnote 5 are combined. Thus, for example, the mean duration of postpartum anovulation for nonbreastfeeding women who menstruate at 1.5 months = .83 (1.5 months - 9 days) + (1 - .83) (38 days) = Average of durations of postpartum anovulation of women with ovulatory and anovulatory menstruations.

\(^7\) We exclude observations in which lengths of full breastfeeding, partial breastfeeding, and amenorrhea exceed the 95th percentile. These limits are 8 months, 28 months, and 24 months, respectively.
Illustrative Estimates of Model Based on Physiological Knowledge

Table 3 reports nonlinear least squares estimates of the following physiological parameters in our model: I - V, the initial hormone level at birth minus the threshold level permitting ovulation; f, the slope of hormonal change during partial breastfeeding; and p, the slope of hormonal change during full breastfeeding. All are expressed relative

<table>
<thead>
<tr>
<th>Parameter in Equation 6</th>
<th>Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>-(I - V)/n = α₃</td>
<td>1.237 (5.06)</td>
</tr>
<tr>
<td>-f/n = β₃ - 1</td>
<td>0.189 (3.91)</td>
</tr>
<tr>
<td>-p/n = γ₃ - 1</td>
<td>-0.187 (-5.10)</td>
</tr>
</tbody>
</table>

NOTE: These estimates are based on a random sample of 600 observations on duration of postpartum amenorrhea. The values in this sample are within 95-percent confidence limits of values for the complete sample of 5312 observations with completed durations of amenorrhea for the means of duration of full and partial breastfeeding and of amenorrhea, and for the proportions of amenorrhea shorter than one week and one month. These 600 observations are then weighted as per Table 1 (intervals probably ending in anovulatory menstruation are discarded). Finally, we discarded observations with durations for full or partial breastfeeding or of amenorrhea that are longer than the 95th percentile for each variable.
to n, the fall in hormonal level when no breastfeeding occurs. Since n
is negative, all estimates in Table 3 are multiplied by -1 to make the
signs consistent with those depicted in Fig. 1. Each of the parameter
estimates is highly statistically significant (p < 0.01). However, the
standard error of the regression is ± 3.6 months, indicating
considerable individual variation about the coefficient estimates.

The rise in hormonal level due to pregnancy and birth, I - V, is
positive. The slope of hormonal change (n) associated with no
breastfeeding is more negative than the slope of hormonal change (p)
associated with partial breastfeeding. And the latter is more negative
than the slope (f) associated with full breastfeeding. Each of these
results is consistent with the model of Fig. 1.

The estimate of (-f/n) is positive; hence the estimate of f is
positive. This finding, which is completely robust to the corrections
for possible data biases that are reported below, is a considerable
surprise. In terms of the model, it means that the line in Fig. 1 that
depicts hormonal change during full breastfeeding should have a small
positive slope, rather than the negative slope pictured. In practical
terms, this finding implies that full breastfeeding may be a highly
effective contraceptive, at the population level, because the level of H
can obviously not fall to the critical level, V, while it is still
rising. Nevertheless, the estimate of (-f/n) for individual women
contains negative values because of variance around the regression line;
hence, particular women might resume ovulation while full breastfeeding.
In fact, this was the case for 2.3 percent of the observations in our
sample, when the probability of anovulatory menstruation is taken into
account. This heterogeneity among women in their suppression of
ovulation by breastfeeding is thus expressed in our model by the
variance around the regression line. We have attempted more direct
modeling of heterogeneity as it relates to the parameters themselves (I,
V, n, f, and p), but have not found a way that was compatible both with
simultaneous solution of the equations and with the physiology of
postpartum anovulation.
For discussion, it is convenient to treat $n$, the slope of hormonal change when no breastfeeding occurs, as numeraire ($= -1$), thereby denominating the other parameters in $n$-units. At birth, the hormonal level is elevated 1.24 $n$-units above the critical level, $V$, at which ovulation can occur. This implies that without initiation of breastfeeding, ovulatory menstruation will resume in 1.24 months. If the woman full breastfeeds, her hormonal level rises at a rate of 0.19 $n$-units per month, implying that the anovulatory period is lengthened by 1.19 months for every month of full breastfeeding for a woman no longer breastfeeding when menstruation resumes (from Eq. (6)). While the woman partial breastfeeds, the level falls at a slope of 0.19, implying that partial breastfeeding from the baby's birth delays ovulatory menstruation to 6.61 months, compared with an anovulatory period of 1.24 months when no breastfeeding occurs.

The absence of interaction terms between lengths of full and partial breastfeeding in this regression results from our assumption that the slope of hormonal change during no breastfeeding is independent of the prior breastfeeding history. Similarly, the slope during partial breastfeeding is independent of whether and for how long full breastfeeding previously occurred. Nevertheless, the predicted time to first ovulation does depend on the entire breastfeeding history. For example, a woman who full breastfeeds for 2 months and then partial breastfeeds until ovulation or after is expected to experience postpartum anovulation for 10.6 months, compared with 6.6 months for a woman who only partial breastfeeds from the baby's birth.

A nonlinearity not depicted in Fig. 1 was sought in the analyses, that of an initial increase in $H$, above and beyond I of Fig. 1, if any breastfeeding occurred at all. Such an effect was indeed found among women who had ceased breastfeeding before the reappearance of menstruation, which of course included those who never breastfed (Habicht et al., 1981), but disappeared when the data of all the women were pooled as described above. We nevertheless believe that this effect may ultimately prove to be correct.

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8 6.61 months = $1.237/0.187$.
9 10.64 months = $(1.237/0.187) + (((1 + 0.189) - (1-0.187))/0.187) \cdot 2$. 

Whether the slopes (f/n) and (p/n) are linear and noninteractive as hypothesized in Fig. 1 can be tested in a more general fashion by adding curvilinear and interaction terms to the regression model (not reported). This yielded some statistically significant terms and a slightly better fit to the data of the pooled sample.\textsuperscript{10} However, some of the estimates from the nonlinear interactive model were physiologically contradictory. Because these contradictions are explained by the absence of controls such as maternal age and parity,\textsuperscript{11} we believe that the simple model reported in Table 3 is more likely to prove correct. In any case, the implications of the more complex model for the contraceptive effects of breastfeeding are very similar to the implications of the simple model.

**Effects of Weighting and of Reporting Biases**

Table 4 shows the effects on parameter estimates and overall goodness of fit of including or excluding data with several possible types of recall errors. Column 3 is identical to Table 3 to facilitate comparisons with the other estimates in the table. The comparisons indicate that our principal findings are very robust to these sample changes, as evidenced in the unchanging signs and significance of parameter estimates, except for (-f/n), when early menstruations are not weighted for proportion of anovulatory first menstruations. It is clear that the estimate of (-f/n) is severely biased when anovulatory menstruations are treated as if they were ovulatory (column 1).

\textsuperscript{10} The results showed a peaking of H are compatible with a report that prolactin peaks within the first three months of breastfeeding (Lunn et al., 1980). However, that reported finding could also be explained by a shift from full to partial breastfeeding.

\textsuperscript{11} For instance, we show below that partial breastfeeding may postpone ovulation more in older than younger women. Because older women in Malaysia tend to partial breastfeed longer, the slope for partial breastfeeding decreases with length of partial breastfeeding when age is not taken into account. This causes an apparent non-linearity in the effect of partial breastfeeding on duration of anovulation.
Table 4
Analyses Including and Excluding Data with Possible Recall Errors
(t-statistics in parentheses)

<table>
<thead>
<tr>
<th>Variable (Parameter in Equation 6)</th>
<th>No Weighting or Exclusions [(1)]</th>
<th>No Further Exclusions [(2)]</th>
<th>Weighted for Probability of Ovulatory Menstruation</th>
<th>Duration of postpartum anovulation imputed from all first menstruations that occurred after the first month post partum [(6)]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Months of anovulation if never breastfed [{1-V}/n]</td>
<td>1.052 ((3.12))</td>
<td>1.025 ((3.38))</td>
<td>1.237 ((5.06))</td>
<td>1.359 ((4.04))</td>
</tr>
<tr>
<td>Slope of H while full breastfeeding slope while not breastfeeding [-f/n]</td>
<td>-0.145 ((-3.00))</td>
<td>0.204 ((3.08))</td>
<td>0.189 ((3.91))</td>
<td>0.373 ((2.62))</td>
</tr>
<tr>
<td>Slope of H while partial breastfeeding slope while not breastfeeding [-p/n]</td>
<td>-0.164 ((-3.15))</td>
<td>-0.182 ((-3.49))</td>
<td>-0.187 ((-5.10))</td>
<td>-0.315 ((-4.17))</td>
</tr>
<tr>
<td>Standard error of the regression</td>
<td>6.1</td>
<td>5.5</td>
<td>3.6</td>
<td>4.6</td>
</tr>
<tr>
<td>Degrees of Freedom</td>
<td>597</td>
<td>557</td>
<td>494</td>
<td>308</td>
</tr>
</tbody>
</table>

NOTE: The sample for these estimates is described in the note to Table 3.
None of the coefficients are significant in column 6, which not only assumes the same probabilities of anovulatory menstruations as do columns 2-5, but also assumes that anovulatory menstruation precedes ovulation by 38 days independently of the time since birth or of the intensity of breastfeeding. Inspection of "anovulation" residuals plotted against duration of breastfeeding indicates that the difference between column 6 and column 3 is that the 38-day correction introduces nonlinearities in the data, resulting in equations that underestimate durations of menstruation selectively for long periods after birth, whether breastfeeding is or is not occurring at the time of menstruation. These menstruations are most likely to be ovulatory, so this linear noninteractive equation based on the corrected data of column 6 is an incorrect model. This contrasts with the much better fit of data to the same linear noninteractive model, but using data corrections that are based on the assumption that the timing of anovulatory menstruation has no (or little) influence on the timing of ovulation in columns 2-5.

If one then proceeds to exclude data with probable errors in recall (columns 3-5), the estimates of duration of postpartum amenorrhea in the absence of breastfeeding increase, most markedly in column 5. That regression, however, shows little improvement in the error variance; this is why these data were retained in Table 3.

The digit-preferred data excluded in column 4 are well above the mean values in the sample. The digit-preferred amenorrhea lengths are 6, 12, and 18 months, the latter two being long durations that are found predominantly in women who are partially breastfeeding. Hence, omitting these observations would bias the regression estimates. However, if women who report digit-preferred intervals tend to report the preferred digit that is closest to the actual duration, little bias to the parameter estimates will result from retaining these digit-preferred observations, because these estimates then approximate least-squares estimates on six-month midpoints. For these reasons we retain the digit-preferred data in our analyses in Tables 3 and 5.
Among the samples that incorporate the weighting procedure, we prefer the one that omits observations with very long reported durations of breastfeeding or amenorrhea. The principal reason for this choice is that this sample fits the model considerably better, as evidenced by the much lower error variance of the regression reported in column 3.

Investigating an Additional Determinant of Postpartum Anovulation: Mother's Age

Previous studies have shown the duration of postpartum amenorrhea to be positively associated with mother's age (Chen et al., 1974; Jain et al., 1970; Salber, Feinleib, and McMahon, 1966; Potter et al., 1965; Kaur et al., 1976; Fisik, Erdal, and Poyraz, 1981). This relationship also emerges in these data. Figure 2 shows mean residuals from the regression in Table 3, plotted against mother's age. The positive slope of a regression line through these residuals, from age 20 up, shows that durations of postpartum anovulation tend to lengthen with age, controlling for lengths of full and partial breastfeeding.12 The statistical model we propose can be used to investigate the source of this relationship. In general, it might arise from either physiological or behavioral changes associated with age. Any physiological change related to lactation would presumably alter the statistical relationships between anovulation and lengths of both full and partial breastfeeding. A physiological change unrelated to lactation would instead alter the intercept coefficient.

The other possible causes of the relationship between age and duration of anovulation shown in Fig. 2 are behavioral. Differences in the frequency and intensity of breastfeeding, especially partial

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12The mean residual is not zero because we have used a nonlinear estimation technique. Under age 20, the anovulatory intervals are very much longer (p < 0.01) than would be predicted from the data on older mothers. The actual mean durations for mothers less than 18 years old and for mothers aged 19 and 20 are circled in Fig. 2. Also shown (with carets) are the predicted durations from the regression on the sample of women aged 20 and above. Therefore, the mechanism relating age to anovulation may be different for young (<20 years of age) and older mothers. The difference could be due to adolescent subfecundity. Therefore, we investigate the age-anovulation relationship for mothers over 20 years of age.
Fig. 2—Anovulation regression residuals by mother's age groups (points are differences between actual and predicted values of duration of anovulation from parameter estimates in Table 3)
breastfeeding, could well be systematically related to cohort differences and maternal age. Hence, any behavioral causes of the positive age-anovulation relationship in Fig. 2 should alter the partial breastfeeding coefficient and possibly the full breastfeeding coefficient; the intercept coefficient should be unaffected.

We investigate these possibilities by adding to the regressors of our basic model interactions of mother's age with the intercept and with lengths of full and partial breastfeeding. Columns 1-3 of Table 5 show the estimates from regressions that are like the regression in Table 3 but also include interactions of mother's age with the intercept, with full breastfeeding, or with partial breastfeeding.\textsuperscript{13}

These three regressions are the only ones, of the seven regressions possible with combinations of the three age interactions, in which all the coefficients are statistically significant. (When all age interactions were included, or when age interactions with the intercept and partial breastfeeding were included together, none of the coefficients were statistically significant. The age interaction with full breastfeeding was never significant when any other age interactions were included. However, the age interactions with the intercept and with partial breastfeeding were each statistically significant when the age interaction with full breastfeeding was the only other age interaction included.) Even alone, the interaction with full breastfeeding (in column 2) is much less significant than are the interactions in columns 1 and 3. We conclude that effect of age on the duration of postpartum amenorrhea is unlikely to be mediated by an effect of age on full breastfeeding's influence.

In contrast to the results for full breastfeeding, the estimates in column 3 suggest that mother's age significantly affects the association between partial breastfeeding and the duration of anovulation. A 20-year-old woman who partial breastfeeds her child from birth will, on average, first ovulate at 12.7 months postpartum,\textsuperscript{14} while mothers older

\textsuperscript{13} Strong multicollinearity among these three interaction variables prevents estimation of their coefficients simultaneously in a single regression.

\textsuperscript{14} 12.74 months = 2.192/(0.404 - [0.0116 x 20]).
Table 5
ASSOCIATIONS BETWEEN MOTHER'S AGE AND DURATION OF POSTPARTUM ANOVULATION, FOR WOMEN MORE THAN 20 YEARS OLD
(t-statistics in parentheses)

<table>
<thead>
<tr>
<th>Variable</th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Constant</td>
<td>0.482</td>
<td>1.427</td>
<td>2.192</td>
</tr>
<tr>
<td></td>
<td>(2.10)</td>
<td>(4.42)</td>
<td>(8.02)</td>
</tr>
<tr>
<td>b. Full breastfeeding (months)</td>
<td>0.0869</td>
<td>0.184</td>
<td>0.385</td>
</tr>
<tr>
<td></td>
<td>(1.98)</td>
<td>(3.48)</td>
<td>(4.50)</td>
</tr>
<tr>
<td>c. Partial breastfeeding (months)</td>
<td>-0.0899</td>
<td>-0.190</td>
<td>-0.404</td>
</tr>
<tr>
<td></td>
<td>(-2.12)</td>
<td>(-4.49)</td>
<td>(-7.24)</td>
</tr>
<tr>
<td>a'. Mother's age</td>
<td>0.0675</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(7.41)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b'. Full breastfeeding x mother's age</td>
<td></td>
<td>0.0193</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.79)</td>
<td></td>
</tr>
<tr>
<td>c'. Partial breastfeeding x mother's age</td>
<td></td>
<td></td>
<td>0.0116</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(6.38)</td>
</tr>
<tr>
<td>Standard error of regression</td>
<td>3.31</td>
<td>3.51</td>
<td>3.32</td>
</tr>
<tr>
<td>Degrees of freedom</td>
<td>405</td>
<td>405</td>
<td>405</td>
</tr>
</tbody>
</table>

NOTE: The sample for these estimates is described in the note to Table 3. As in Tables 3 and 4, all coefficients are expressed relative to n:

The total effects, taking into account the interactions, are:

\[-(I-V)/n = \alpha_3 = a + a' \cdot \text{Age}\]
\[(-f/n) = \beta_3 - 1 = b + b' \cdot \text{Age}\]
\[(-p/n) = \gamma_3 - 1 = c + c' \cdot \text{Age}.\]

than 34 will generally not ovulate at all while they are still partial breastfeeding. The practice of partial breastfeeding is probably more variable than full breastfeeding in its frequency and intensity of suckling. We are therefore inclined to think that this effect of age would be caused by cohort changes in breastfeeding practices rather than by a direct physiological effect of age; otherwise we would also have
by a direct physiological effect of age; otherwise we would also have expected to see an equally significant age effect on the contraceptive influence of full breastfeeding if it is introduced into the regression of column 3. This was not the case.

The column 1 estimates imply that a 40-year-old woman’s anovulatory period lasts nearly 75 percent longer than that of a 20-year-old, whatever their common breastfeeding practices are. For example, a 20-year-old non-breastfeeding mother would resume ovulation at 1.83 months \((0.482 + 0.0675 \times 20 \text{ years})\), while a 40-year-old would resume ovulation 1.35 months later.\(^{15}\) This relationship is likely to be induced physiologically because it exists independently of whether and how long breastfeeding is practiced.\(^{16}\)

Because of multicollinearity among the explanatory variables in this sample, the above sample cannot distinguish unambiguously between the physiological effect of age on postpartum anovulation that is suggested in column 1 of Table 5, and the probable behavioral effect indicated in column 3. However, the results for mothers who never breastfed support the physiological explanation: For the 89 such mothers in the sample analyzed for Table 5, the increment to the intercept is \(+0.100 \pm 0.050\) \((p < .05)\) per year of maternal age.

For women who breastfed and stopped before resuming menstruation the corresponding results are similar: an increment of \(+0.104 \pm 0.057\) \((p < .05)\) in the intercept per year of maternal age for the 147 such women. This contrasts with the statistically insignificant Partial breastfeeding \(\times\) Mother’s age term for these same women of \(+0.0091 \pm 0.0069\), which does not support the behavioral hypothesis. Similar analyses of never-breastfeeding women and those who ceased breastfeeding before menstruation in the total sample of 5312 live births revealed even higher levels of statistical significance for the test of the physiological hypothesis and no statistical significance for the test of

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\(^{15}\) This effect of age would correspond to an increase in prolactin levels at birth such as that described for nutrition in malnourished mothers (see Lunn et al., 1980, and Lunn et al., 1981).

\(^{16}\) The coefficients for full and partial breastfeeding in column 1 of Table 5 are quite different from those in columns 2 and 3 and in Table 3. However, the implied relationships between lengths of full and partial breastfeeding and between these and the duration of anovulation are similar across these regressions.
the behavioral hypothesis. No other subgroup of mothers (e.g., those corresponding to Eqs. (1) and (2)) permits such unambiguous testing as the contrast provided by never-breastfeeding mothers and mothers who stopped breastfeeding before menstruating. We conclude that the prolongation of postpartum anovulation with age is due to the physiological process that either raises the initial inhibitory influence (I) or depresses the threshold (V) where ovulation occurs.
VI. SUMMARY AND IMPLICATIONS

The statistical procedures used to date to analyze the effect of breastfeeding on the duration of anovulation are incompatible with present knowledge about the neurohormonal mediating mechanisms that link breastfeeding to postpartum anovulation. An estimating equation compatible with current physiological knowledge is derived here and is applied to recall data from the Malaysian Family Life Survey. The results suggest that a month of full breastfeeding prolongs anovulation on average by more than a month, and even partial breastfeeding has a strong prolonging effect. These findings are in marked contrast to the usual results in the literature (for example, Perez et al., 1971, 1972; Van Ginneken, 1978; Jain et al., 1970, 1979), which indicate that a month of breastfeeding is associated with less than two weeks of additional anovulation. Hence, breastfeeding, especially when unsupplemented, appears to be a much more effective contraceptive than previously thought.

Discrepancies in previous estimates of the contraceptive effect of breastfeeding can be illuminated by our findings. For instance, the regression coefficient of breastfeeding on postpartum amenorrhea in a study of Chile (Perez et al., 1971) was 1.5 times that in a study of Taiwan (Jain et al., 1970). According to our model, this discrepancy is due to the fact that women in Taiwan breastfeed for 15 months on the average, while in Chile the average is four months. Because breastfeeding after amenorrhea ends does not affect its length, much of the sample variation in breastfeeding duration in Taiwan is irrelevant, and we contend that this is why a smaller slope coefficient (0.4) results. In Chile, the short average breastfeeding duration of four months means that many women begin menstruating after they stop breastfeeding. Variations in breastfeeding length are predictive of amenorrhea duration for these women; hence a larger slope coefficient (0.6).
A further implication of our synthesis of physiology and analyses of survey data is that breastfeeding declines that take the form of women abandoning breastfeeding altogether will have larger effects on birthspacing and fertility than declines from long to medium durations of breastfeeding.

This model can be extended to investigate the effects of determinants of postpartum anovulation other than breastfeeding, and to examine the effects of a greater variety of breastfeeding practices. It will then be possible to distinguish the effect of a determinant on the ovulation-inhibiting process at parturition from its effect on the process thereafter.

These distinctions not only permit insights that can be checked directly in endocrine-physiological investigations, but they can also indicate which determinants are probably mediated by differences in breastfeeding practices and which by physiological changes independent of breastfeeding practices.

While such investigations are proceeding, conclusions now in the literature about physiological and behavioral determinants of postpartum anovulation, postpartum amenorrhea, interbirth intervals, and fertility must be examined for possible false inferences introduced by the statistical methods used.

NOTE ADDED IN PRESS

Recent studies (reviewed by A. S. McNeilly in J. Dobbing (ed.), *Maternal Nutrition and Lactational Infertility*, Academic Press, London, 1985, in press) suggest that one should envisage an expansion of the model in Fig. 1 to one with three thresholds: one for the onset of menstruation before ovulation (anovulatory menstruation), one for ovulation (as in Fig. 1), and one for the successful establishment of pregnancy. These three events appear to be affected by breastfeeding through the same mechanism. This mechanism inhibits follicular maturation and luteal development in the ovary. As the inhibition is reduced, follicles tend to start cyclic incomplete maturation, and release sufficient estrogen to permit development of the uterine
endometrium followed by menstruation—an anovulatory menstruation. As the inhibition is further reduced, follicular maturation proceeds to ovulation but luteal development is still inhibited, thus preventing the establishment of pregnancy. Finally, as inhibition decreases even more, full follicular and luteal maturation permit ovulation and pregnancy.

This expansion of the Fig. 1 model has implications for estimating ovulation and fertility on the basis of the reappearance of menstruation. It means, for instance, that the period from reappearance of menstruation to that of ovulation depends on breastfeeding intensity during that interval. This is compatible with our findings in this paper that the onset of ovulatory menstruation cannot be modelled by inserting a constant interval after the onset of anovulatory menstruation. It also explains why anovulatory ovulation is more common in breastfeeding women than in nonbreastfeeding women. In the nonbreastfeeding woman, inhibition of follicular maturation falls rapidly. In that case, the first follicle that matures enough to permit menstruation will have proceeded through ovulation—so that the first menstruation will be ovulatory. In contrast, the first follicle of the breastfeeding woman that is just sufficiently disinhibited to permit menstruation will not proceed to ovulation, and anovulatory menstruation will be the result.
REFERENCES


