Maternal Nutrition and Lactational Infertility: A Review

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Ample data exist to show that lactation prolongs postpartum amenorrhoea and provides some degree of protection against pregnancy (1,2). There are, however, marked variations in the duration of lactational infertility among women of different countries and women in different communities in the same country (1,2). Investigations undertaken during the past two decades have shown that a large proportion of the observed variations are attributable to differences in the duration of lactation and breastfeeding practices among communities (1). An association between maternal nutritional status and duration of lactational amenorrhoea has also been observed in some of the studies (1).

Before reviewing the literature, it might be worthwhile to consider whether delay in return of fertility in undernourished women could constitute a beneficial evolutionary adaptation. Famine and acute starvation are dramatic short-term events that result in acute undernutrition. There are data showing that such drastic reduction in food intake and consequent acute undernutrition are associated with amenorrhoea and reduction in fertility in women (3). With improvement in the food supply, rapid improvement in nutritional status and fertility occur. There are reports of a rebound increase in total marital fertility rates after famine (3). Given the complex socio-economic and political milieu in which famines arise it is unlikely that anyone could document, investigate, and segregate the contribution of undernutrition per se, as well as the psychological factors, to the observed reduction in fertility during famine. But undoubtedly, temporary short-term relative infertility associated with acute starvation is a mechanism by which the breastfed infant might continue to receive breast milk and benefit. Avoidance of the extra nutritional stress of pregnancy could be considered as protective adaptation for the mother.

Chronic mild and moderate undernutrition of the kind commonly seen among poorer segments of the population in developing countries is quite a different kind of a problem. Food shortages of varying extent have existed for hundreds of years and are likely to continue to exist, at least for a few more decades. Many nutritionists believe that these population groups might have become "adapted" to the low level of dietary intake, because there is no deterioration in maternal nutrition during pregnancy and lactation or with increasing age and parity (4,5). Prolonged lactation is the rule among undernourished women in developing countries. The
mean duration of lactation ranges from 18 to 24 months and the interbirth interval is approximately 30 to 36 months. As the dietary intake does not change, prolongation of the interbirth interval by a few more months is unlikely to result in any improvement in maternal nutritional status. Undue prolongation of the interbirth interval is likely to be counterproductive in terms of species survival, because in such situations infant and childhood mortality rates are likely to be high and have to be “compensated” by high birth rates. Thus, theoretical considerations suggest that it is unlikely that chronic mild maternal undernutrition will be associated with marked reduction in fertility. Reduction in fertility is likely to be obvious among the more severely undernourished women and is likely to be of relatively small magnitude, except in the rare situations when massive shifts in nutritional status occur.

PARAMETERS USED FOR ASSESSMENT OF NUTRITIONAL STATUS AND FERTILITY

Before attempting to look for an association between maternal nutritional status and fertility during lactation, it is necessary to define the parameters that have been used for assessment of maternal nutritional status and fertility during lactation, and possible fallacies and problems in using various parameters, especially when attempts are made to look for an association between these two variables.

Parameters for Assessment of Maternal Nutritional Status

Differences in socio-economic status are used in many demographic studies as a method of distinguishing undernourished and well-nourished population groups in different countries. Economic status and purchasing power do give some indication with regard to the probable nutritional status in any community. But in studies attempting to relate breastfeeding and return of maternal fertility to maternal nutritional status, comparisons among economic groups may not be useful, because variations in breastfeeding patterns, times of introduction of supplements, and duration of lactation act as confounding variables.

Dietary intake has often been used as an indicator of nutritional status. The type of data from which dietary intakes have been computed range from community averages of food consumption, household dietary surveys, and careful dietary surveys of food consumed by the individual. Information on community averages or even household dietary surveys may not provide any information on the dietary intake among lactating women, the segment of the population that is to be investigated. Individual dietary surveys are tedious and time consuming, and they do not take into account possible day-to-day variations in dietary intakes. Additional dietary intake in the form of supplements administered with or without actual data on increase in total dietary intake has also been used as an indicator of changes in nutritional status. In an environment of poverty, supplementary food given to the mother is often shared by the children, and it is difficult to assess the actual increment in maternal dietary intake due to food supplements.
Maternal body weight has been one of the most widely used parameters for assessment of maternal nutritional status. Changes in body weight do indicate changes in nutritional status in women, but we still do not know to what extent variation in body weight represents variation in nutritional status in adult women. Under these circumstances, body weight may not be a sensitive index for the assessment of maternal nutritional status in relation to return of fertility.

Parameters of Assessment of Return of Fertility During Lactation

Ideally, return of ovulation should be the parameter used for assessment of return of fertility during lactation. However, methods for diagnosis of return of ovulation are difficult to use in community-based studies. Basal body-temperature charts are difficult to keep; periodic blood and urine collections on a longitudinal basis are costly and difficult and require good laboratory facilities. Therefore, specific methods of diagnosing return of ovulation are used only in small-scale studies investigating endocrine profiles during lactation.

Duration of lactational amenorrhoea is the most widely used index for assessment of return of fertility. It is well recognized that ovulation might precede menstruation and that the first few menstrual cycles might be anovulatory in some women; but by and large, return of menstruation represents the most readily measurable method of return of fertility during lactation.

Interpregnancy or interbirth interval is yet another commonly used parameter, especially by demographers for measurement of return of fertility during lactation. This index takes into account both occurrence of ovulation during lactational amenorrhoea and anovulatory cycles after return of menstruation. In many communities, contraceptive use and cultural taboos regarding intercourse during lactation modify interpregnancy intervals. An estimate of the prevalence of either or both these factors, at least at the community level, is an essential prerequisite before information on interbirth interval can be used as an index for measurement of fertility during lactation.

REVIEW OF LITERATURE

Stimulated by the early observations on the association between maternal nutritional status and lactational infertility, investigators from a wide variety of specialities, ranging from demographers and sociologists to nutritionists, physiologists, and endocrinologists, have undertaken studies on the possible role of maternal nutritional status in determining duration of lactational infertility. Broadly, the studies fall into two categories: (a) large-scale community-based information gathered by demographers and sociologists, and (b) detailed physiological investigations of a small number of women undertaken by nutritionists, physiologists, and endocrinologists.

Epidemiological findings demonstrate trends in communities and have the advantage of covering large segments of population and providing information from several countries. However, because of the difficulties of extensive population
coverage, detailed information on dietary intake, nutritional status, suckling patterns, sexual taboos, and contraceptive use during lactation may not be accurately collected. Sophisticated analytical techniques and mathematical models are often used to predict fertility trends, but because of the lack of detailed information on vital factors, these predictions may not be readily translatable into the real-life situation of communities.

The in-depth physiological studies can take all these details into account, but the very small size of the sample and the obvious difficulties in choosing the right sample and testing the effect of the correct intervention for the appropriate duration render interpretation of data from these studies a rather difficult exercise. For obvious reasons, enormous fallacies can creep in if these findings from small selected samples are extrapolated to large-population groups with varying characteristics.

HISTORICAL STUDIES

Over the past century, there was a tremendous improvement in the nutritional status of women from poorer segments of the population in Europe and North America. Attempts have been made to make use of the available information on interbirth interval from historical records and to see whether the alteration in maternal nutritional status over centuries may have resulted in any change in interbirth intervals. Hutterites in the United States who breastfeed their infants and do not use any contraception are taken as representing mid-twentieth-century well-nourished population groups. Available data suggest that, unlike age at menarche and age at menopause, there has not been any alteration in interbirth intervals over the last two centuries (6) (Table 1). However, over centuries there have been many alterations in ways of life, infant-feeding practices, and sexual mores. In the absence of information on these factors, which are major determinants of fertility during lactation, and of any records about nutritional status of women in earlier centuries, it may not be valid to draw conclusions concerning the possible effect of maternal nutritional status on interbirth intervals from these historical studies.

<table>
<thead>
<tr>
<th>Population</th>
<th>Mean birth interval (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mid-18th century, Canada</td>
<td>~20–24</td>
</tr>
<tr>
<td>Mid-19th century</td>
<td></td>
</tr>
<tr>
<td>U.K. (labouring class)</td>
<td>20–24</td>
</tr>
<tr>
<td>Ile de France</td>
<td>26</td>
</tr>
<tr>
<td>Mid-20th century, Hutterites (U.S.A.)</td>
<td>24</td>
</tr>
</tbody>
</table>

*From ref. 6.
DATA FROM DEMOGRAPHIC SURVEYS

Comparison Between Undernourished and Well-Nourished Communities in Different Countries

Several attempts have been made to compare fertility patterns, duration of lactational amenorrhoea, and interbirth intervals in well-nourished women in developed countries who are not using any contraception with those of poorly nourished women in developing countries. The most widely cited comparison is that between Hutterite women, who have a lifetime average of 9.5 births, and women from Bangladesh, who have only 7.2 births by the end of their child-bearing years (Table 2). The differences are even more striking when compared with the !Kung hunter-gatherers, who have a mean interbirth interval of 5 years and total births between 4 and 5 (6). Contraceptive use is negligible in these population groups. It has been suggested that the difference in fertility among these groups might be attributable to the differences in maternal nutrition. However, available information suggests that differences in the duration of lactation and breastfeeding practices rather than differences in maternal nutritional status might account for most of the observed differences.

From the literature, one could provide several other examples that are in conflict with the Hutterite/Bangladesh comparison. In Amish women, who are ethnically and nutritionally similar to Hutterites, the total marital fertility rate is only 6.3 births. Rich, well-nourished women in Kuwait and Saudi Arabia have fertility rates that do not differ from those observed in Bangladesh (6). Obviously, details of infant feeding and weaning practices and traditional cultural taboos regarding resumption of intercourse during lactation might account for some of the variations and similarities in fertility among different communities. It is therefore unwise to

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Hutterites</th>
<th>Bangladesh</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total general fertility rate (births per woman)</td>
<td>9.5</td>
<td>7.2</td>
</tr>
<tr>
<td>Total marital fertility rate (births per woman)</td>
<td>10.9</td>
<td>6.8</td>
</tr>
<tr>
<td>Mean interbirth interval (months)</td>
<td>24.0</td>
<td>37.4</td>
</tr>
<tr>
<td>Mean duration of lactational amenorrhoea (months)</td>
<td>6.0</td>
<td>17.9</td>
</tr>
<tr>
<td>Median age at marriage (years)</td>
<td>22.0</td>
<td>17.0</td>
</tr>
<tr>
<td>Contraceptive use</td>
<td>Absent</td>
<td>Very low</td>
</tr>
</tbody>
</table>

*From ref. 6.
attempt such cross-country comparisons to obtain information on the possible impact of maternal nutrition on the return of fertility during lactation.

**Comparison Between Socio-Economic Groups in the Same Country**

In most of the developing countries, there is a good correlation between socio-economic status and nutritional status, because poverty and lack of purchasing power are the major factors leading to undernutrition in these countries. Demographers have therefore attempted comparing duration of lactational amenorrhoea and interbirth intervals between richer and poorer segments of the population in the same community. Most of the data indicate that lactational amenorrhoea and interbirth intervals are longer in the poorer segments of population (7). It is, however, likely that most of the observed differences might be attributable to the differences in breastfeeding practices rather than to differences in maternal nutritional status. Since demographers seldom collect information on nutritional status and infant-feeding practice, it would appear that it might be difficult if not impossible to draw conclusions regarding the possible effect of maternal nutritional status on fertility during lactation from existing demographic data on fertility patterns in various socio-economic groups.

**Linkages Between Existing Demographic and Nutrition Surveys**

In many developing countries, large-scale fertility surveys are done periodically. Periodic nutritional surveys or national nutritional monitoring systems exist in many of these countries. An attempt to link these two sets of information to assess the effect of maternal nutritional status on fertility was made by Centrelle and Ferry in Senegal (8) and by Carael (9) in Zaire. Dietary intakes were lower, and duration of lactational amenorrhoea was longer in rural as compared to urban women in both Senegal and Zaire (Tables 3 and 4).

In Zaire, dietary intakes were lower, and lactational amenorrhoea was longer in the highlands (9) compared to tropical forest areas. However, there might be

**TABLE 3. Dietary intake and fertility during lactation in Senegal**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Urban</th>
<th>Periurban</th>
<th>Rural</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unsupplemented lactation (months)</td>
<td>7.4</td>
<td>5.7</td>
<td>6.1</td>
</tr>
<tr>
<td>Total duration of lactation (months)</td>
<td>18.9</td>
<td>19.7</td>
<td>23.2</td>
</tr>
<tr>
<td>Lactational amenorrhoea (months)</td>
<td>12.3</td>
<td>15.4</td>
<td>17.3</td>
</tr>
<tr>
<td>Interbirth interval (months)</td>
<td>32.5</td>
<td>32.6</td>
<td></td>
</tr>
<tr>
<td>Calorie intake (kcal/day)</td>
<td>2495</td>
<td>2257</td>
<td>2068</td>
</tr>
</tbody>
</table>

*From ref. 8.
significant differences in breastfeeding practices and sexual taboos during lactation between tribes, and in the absence of information regarding these aspects, one can only conclude that variations in maternal nutritional status might be among the many factors responsible for the longer duration of lactational amenorrhoea in undernourished highlanders.

SMALL-SCALE PHYSIOLOGICAL STUDIES

Effect of Maternal Body Weight on Duration of Lactational Amenorrhoea

In Guatemala (10,11), Bangladesh (12-14), and Zaire (9), studies have been undertaken to investigate the effect of maternal body weight on the duration of lactational amenorrhoea. In all of these studies, there is a consistent small negative correlation between maternal body weight and the duration of lactational amenorrhoea. In Bangladesh, the duration of lactational amenorrhoea was shorter by 1 month in women with mean plus 1 SD body weight compared with women with mean minus 1 SD body weight (Table 5) (12). For any given duration of lactation, menstruating women were heavier than amenorrhoeic women, but the differences
in body weight among groups seldom exceeded 1 kg (13) (Table 6). These data suggest that the effect of maternal nutritional status on lactational amenorrhoea is demonstrable in community-based studies but is of a relatively small magnitude.

Other investigators, however, have reported changes of larger magnitude in the duration of lactational amenorrhoea with variation in maternal body weight. Data from Zaire showed that for any given duration of lactation, women who were menstruating were heavier by 1 to 4 kg (Table 7). The investigators in India calculated the duration of lactational amenorrhoea for any given length of duration of lactation in women belonging to different body-weight groups (15,16). The duration of lactational amenorrhoea was shorter by 4.9 months in women who weighed more than 55 kg compared to those with body weight less than 40 kg (16) (Tables 8 and 9). Obviously, the two extreme body-weight groups constitute only a small segment of the community, and differences between these two extreme groups contribute very little to variations in duration of lactational amenorrhoea in the community. However, the data demonstrate that large differences in maternal body weight might be associated with substantial alterations in duration of lactational amenorrhoea.

Effect of Food Supplementation on Duration of Lactational Amenorrhoea

Studies investigating the effect of food supplementation to undernourished women on duration of postpartum amenorrhoea and interpregnancy interval might appear

<table>
<thead>
<tr>
<th>Duration of lactation (months)</th>
<th>Amenorrhoeic</th>
<th>Menstruating</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>66</td>
<td>19</td>
</tr>
<tr>
<td>14</td>
<td>172</td>
<td>66</td>
</tr>
<tr>
<td>15</td>
<td>190</td>
<td>79</td>
</tr>
<tr>
<td>16</td>
<td>163</td>
<td>82</td>
</tr>
<tr>
<td>17</td>
<td>130</td>
<td>82</td>
</tr>
<tr>
<td>18</td>
<td>135</td>
<td>93</td>
</tr>
</tbody>
</table>

| Amenorrhoeic | Mean body weight (kg) ± SD | Menstruating | Mean body weight (kg) ± SD |
|--------------|---------------------------|--------------|
| 66           | 40.3 ± 4.47               | 19           | 43.2 ± 3.52               |
| 172          | 40.9 ± 4.55               | 66           | 41.4 ± 4.80               |
| 190          | 41.1 ± 4.19               | 79           | 41.9 ± 4.59               |
| 163          | 40.1 ± 4.26               | 82           | 42.7 ± 3.78               |
| 130          | 40.2 ± 4.44               | 82           | 40.9 ± 4.71               |
| 135          | 40.1 ± 4.40               | 93           | 40.5 ± 4.03               |

*From ref. 14.

<table>
<thead>
<tr>
<th>Duration of lactation (months)</th>
<th>Amenorrhoeic</th>
<th>Menstruating</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–2</td>
<td>53.6 ± 7.6</td>
<td>98</td>
</tr>
<tr>
<td>3–5</td>
<td>52.1 ± 6.0</td>
<td>99</td>
</tr>
<tr>
<td>6–11</td>
<td>51.2 ± 5.6</td>
<td>96</td>
</tr>
<tr>
<td>12–24</td>
<td>51.3 ± 6.9</td>
<td>58</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Body weight (kg)</th>
<th>Amenorrhoeic n</th>
<th>Menstruating n</th>
</tr>
</thead>
<tbody>
<tr>
<td>53.6 ± 7.6</td>
<td>78</td>
<td>56.2 ± 6.1</td>
</tr>
<tr>
<td>52.1 ± 6.0</td>
<td>99</td>
<td>56.5 ± 8.7</td>
</tr>
<tr>
<td>51.2 ± 5.6</td>
<td>96</td>
<td>55.9 ± 5.9</td>
</tr>
<tr>
<td>51.3 ± 6.9</td>
<td>58</td>
<td>52.5 ± 5.4</td>
</tr>
</tbody>
</table>

*From ref. 9.
TABLE 8. Effect of body weight on duration of lactation and lactational amenorrhoea in Indian women*

<table>
<thead>
<tr>
<th>Body weight (kg)</th>
<th>Mean duration of unsupplemented lactation (months)</th>
<th>Mean duration of lactation (months)</th>
<th>Mean duration of lactational amenorrhoea (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;40</td>
<td>9.2</td>
<td>20.9</td>
<td>13.2</td>
</tr>
<tr>
<td>40-44</td>
<td>8.9</td>
<td>19.8</td>
<td>11.4</td>
</tr>
<tr>
<td>45-49</td>
<td>8.6</td>
<td>20.1</td>
<td>10.2</td>
</tr>
<tr>
<td>50-54</td>
<td>8.4</td>
<td>19.8</td>
<td>9.3</td>
</tr>
<tr>
<td>≥55</td>
<td>7.6</td>
<td>16.6</td>
<td>7.5</td>
</tr>
</tbody>
</table>

Body weight versus duration of lactation: duration of lactation = 45.80 – 0.0314 x body weight (r = −0.0566, NS)

Body weight versus duration of lactational amenorrhoea: duration of lactational amenorrhoea = 46.78 – 0.1824 x body weight (r = −0.7251, P< 0.001)

Partial correlation between
(1) lactation and lactational amenorrhoea = 0.6914, P < 0.001 (keeping body weight constant)
(2) body weight and lactation = −0.0250, NS (keeping lactational amenorrhoea constant)
(3) body weight and lactational amenorrhoea = −0.8139, P < 0.001 (keeping duration of lactation constant)

*From ref. 16; 2,042 lactational periods studied.

TABLE 9. Effect of body weight on duration of lactational amenorrhoea in a group of Indian women from a low-income group matched for duration of lactation*

<table>
<thead>
<tr>
<th>Body weight (kg)</th>
<th>n</th>
<th>Mean duration of lactation (months)</th>
<th>Mean duration of lactational amenorrhoea (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>&lt;40</td>
<td>276</td>
<td>21.3</td>
<td>10.61</td>
</tr>
<tr>
<td>40-44</td>
<td>356</td>
<td>22.5</td>
<td>10.32</td>
</tr>
<tr>
<td>45-49</td>
<td>297</td>
<td>20.8</td>
<td>10.76</td>
</tr>
<tr>
<td>50-54</td>
<td>209</td>
<td>20.7</td>
<td>10.58</td>
</tr>
<tr>
<td>≥55</td>
<td>222</td>
<td>20.7</td>
<td>8.53</td>
</tr>
</tbody>
</table>

*From ref. 16.

to be the best method of obtaining reliable information on possible impact of alteration in maternal nutritional status on fertility during lactation. Variations in types of food given, duration of food supplementation, extent of sharing of food supplements among family members, dietary intake prior to and during the supplementation programme, and amount and type of supplements given to infants render comparison and interpretation of data from many of these studies rather difficult. Studies from Guatemala demonstrate a small but consistent reduction in duration of lactational amenorrhoea and interpregnancy interval among women who consumed supplements during pregnancy. The magnitude of reduction was about 1 to...
2 months and varied with the type and amount of food supplements ingested (Table 10) (10).

From Table 10 it would appear that the women who received protein and calorie supplements (Atole) showed the reduction in duration of lactational amenorrhoea, whereas those who received calorie supplements (Fresco) did not. However, it has been suggested that the observed difference might be attributable to the difference in the supplements received by the infants (J-P. Habicht, personal communication). The fact that the women who received supplements were a self-selected group and that there was no information on the type and amount of food supplementation given to breastfed infants prevents the drawing of any definite conclusion about the magnitude of variation in return of fertility in relation to the amount of maternal food supplement.

Chavez and Martinez (17) demonstrated that providing powdered-milk supplements of 64 g/day to Mexican women during pregnancy and 94 g/day during lactation resulted in an increase in maternal body weight of 3 kg and a reduction in duration of lactational amenorrhoea and interpregnancy interval by 6 months (17). Bongaarts (18) noted that new observations presented by Chavez at the Workshop on Nutrition and Reproductive Competency indicated that many of the infants of supplemented mothers also received supplements; so in this study also it is difficult to evaluate whether the observed effects were solely due to maternal food supplementation. Lunn and colleagues (19) have shown that dietary supplements of about 720 kcal/day to undernourished Gambian women result in a reduction of duration of lactational amenorrhoea by 6 months and a reduction in interpregnancy interval of similar magnitude. The fact that all the infants in the Gambian villages were given supplements provided by the intervention project from the third month onwards is a confounding factor in the study also and makes it difficult to assess the effect of maternal dietary supplementation on duration of lactational amenorrhoea. It is reported that in The Gambia, there is a cultural taboo against sexual intercourse during lactation, though these taboos might not be strictly observed. The prevalence of these cultural taboos renders interpretation of data on interpregnancy interval rather difficult. Data from the latter two studies appear to

<table>
<thead>
<tr>
<th>Total caloric intake (calories/day)</th>
<th>Duration of postpartum amenorrhoea (months)</th>
<th>Atole n</th>
<th>Fresco n</th>
<th>Total n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low, &lt; 1,308</td>
<td></td>
<td>14.33</td>
<td>14.38</td>
<td>14.35</td>
</tr>
<tr>
<td>Middle, 1,309–1,630</td>
<td></td>
<td>13.60</td>
<td>14.04</td>
<td>13.78</td>
</tr>
<tr>
<td>High, &gt; 1,631</td>
<td></td>
<td>12.29</td>
<td>14.21</td>
<td>13.04</td>
</tr>
</tbody>
</table>

*From ref. 10; Atole intake contains protein and energy; Fresco intake contains energy only.

**Total caloric intake is the sum of home caloric intake and caloric supplementation during pregnancy.
suggest that sustained, substantial improvement in dietary intake brings about significant reduction in duration of lactational amenorrhoea and interpregnancy interval.

A review of the literature thus suggests that there may be two distinct trends. One set of data suggests that in communities where there is very little variation in dietary intake, there is a small but consistent trend towards reduction in lactational infertility among better nourished groups. Dietary supplementation of 300 calories or less for a relatively short time during pregnancy brings about a similar small reduction in duration of lactational amenorrhoea. However, alterations in duration of lactational amenorrhoea of large magnitude are seen in groups that receive a substantial increment in dietary intake over a long period. These two sets of observations need not represent conflicting pieces of information but might, in fact, be two sides of the same coin. The latter studies demonstrate the magnitude of the biological potential for variations in fertility during lactation in relation to maternal nutritional status, and the former, the expression of this potential under existing conditions in population groups.

**Effect of Nutrition on Fertility: How Is the Effect Mediated?**

If we accept the hypothesis that maternal nutrition does modify the return of fertility during lactation, then the question of how the effect is mediated needs to be answered. Two hypotheses have been put forward to explain this: one assigns a pivotal role to prolactin, the other to oestrogens and body fat.

Prolactin, as the name implies, was thought to play a major role in initiation and maintenance of lactation. More recently, increasing attention has been focused on its role in the regulation of the return of fertility during lactation (20). There are data indicating that a fall in plasma prolactin levels precedes the return of menstruation, in both developed and developing countries. Undernourished women from developing countries have significantly higher plasma prolactin levels, which are sustained for a longer duration, than do women from developed countries (20).

The relative role of frequency of suckling, duration of suckling, intensity of suckling, and night feeds in determining the reported higher plasma prolactin levels in developing countries is still poorly understood. Lunn et al. (19) have shown that in Gambian women, plasma prolactin levels are higher, and the duration of lactational amenorrhoea is longer during the wet season when food is scarce. Food supplementation to these women resulted in a rapid fall in prolactin levels and in an earlier return of menstruation and fertility. Based on these findings, they suggest that a sustained elevation in prolactin levels plays a major role in delaying the return of fertility in undernourished women.

In the early 1970s, Tyson and Perez (21) showed that in some women, milk production is so abundant that the infant has to suck very little to obtain its required milk intake. They had also demonstrated that in such instances a prolactin peak induced by suckling either does not occur at all or is small. Lunn et al. (22) propose that milk secretion might be decreased in undernourished women so that the infant
has to suckle vigorously and/or for a longer duration so that plasma prolactin levels are elevated and result in delay in onset of menstruation. Or as an alternative, they suggest that through mechanisms yet unknown, maternal undernutrition stimulates prolactin production to ensure adequacy of milk secretion in spite of maternal undernutrition (22).

A decade ago, Frisch and Revelle (23) showed that in the United States a critical weight associated with menarche could be defined. Further studies have shown that the "critical weight" in fact represented a critical proportion of body fat to lean body mass, which determined time of menarche and regular menstruation (24). There is no information about the relationship, if any, between body weight, body fat, lean body mass, and age at menarche and resumption of menstruation during lactation in developing countries.

It is well known that oestrogens are stored in body fat and may be released into the circulation. Frisch and co-workers (25) have shown that oestrogen levels were higher in obese rats. Pharmacokinetic studies on contraceptive steroids have demonstrated that these steroids are excreted faster and the steady-state levels of steroids are lower in undernourished women. There was a good correlation among fat-fold thickness, body weight, and steroid levels (26,27). Prema et al. (16) have suggested that a similar situation might exist in relation to endogenously produced steroids, and that for any given level of oestrogen production, the circulating levels of oestrogens might be lower in undernourished women with lower body weight and fat. Low oestrogen levels might stimulate prolactin production. High circulating levels of prolactin inhibit oestrogen production. This cycle leads to sustained high prolactin levels and delay in return of menstruation and fertility during lactation in undernourished women.

It is possible that the prolactin- and the oestrogen-mediated mechanisms might be operating simultaneously. In fact, they may be mutually reinforcing and be responsible for the delay in the return of menstruation and fertility during lactation in undernourished women.

CONCLUSION

The current controversy about the role of maternal nutritional status to some extent resembles the controversy regarding the role of breastfeeding in fertility in the 1950s and 1960s. Data from developed countries were equivocal; those from developing countries indicated that there was a difference of approximately 6 months in the duration of postpartum amenorrhoea and the interpregnancy interval between lactating and non-lactating women. Enthusiasts claim that lactation offers more protection against pregnancy than all the family-planning programmes put together, whereas sceptics believe that lactation is an inefficient and undependable contraceptive (28). Data are now available that clearly demonstrate that giving up breastfeeding in the absence of changes in contraceptive usage is associated with a substantial increase in fertility and birth rates (29,30).

The controversy in relation to the role of maternal nutritional status in lactational infertility today is somewhat similar. Studies have demonstrated that differences in
body weight of over 15 kg (<40–55 kg) or in dietary intake of over 700 kcal/day (<1,500–2,200) result in 4- to 6-month differences in duration of lactational amenorrhoea and interpregnancy interval. Obviously, women representing these two extremes are very few in any community, and marginal shifts in nutritional status do not cause alterations in duration of lactational amenorrhoea by more than 1 month. However, in contrast to the situation in relation to infant-feeding practices, massive changes in dietary intakes or nutritional status of poorer segments of the population in developing countries are unlikely to happen because of socioeconomic and political constraints. Therefore, it is unlikely that any massive changes in fertility during lactation will in practice occur because of changes in maternal nutritional status.

The question is repeatedly asked about the possible impact of the existing food-supplementation programmes on return of fertility during lactation among poorer segments of population. It is well known that food supplements are shared by family members and that the increase in dietary intake of target women is only marginal. Such a marginal increase is unlikely to cause a marked shift in return of fertility during lactation.

It is very desirable to achieve freedom from hunger among poorer segments of population. Food-supplementation programmes contribute to the achievement of this objective. Viewed in this context, the problem that the food given may not reach the “target women” is irrelevant, as food does go to feed hungry individuals. The giver of food is an accepted and respected person in any community. If maternal and child health services and contraceptive care are linked with food-supplementation programmes and services are provided through the same personnel, the acceptance and use of maternal and child health and contraceptive services might improve. Such an integration might result in effective implementation and usage of all the three types of services. Achievement of a significant improvement in maternal and child health, substantial reduction in mortality rates, and a substantial fall in birth rate before the turn of the century might then be feasible.

REFERENCES


COMMENTARY

C. ROBYN

We too did not find any apparent deterioration of the nutritional status of the mother during prolonged lactation. What kind of adaptive mechanism to a low level of dietary intake do you expect in lactating mothers? Reduction in milk yield, reduction in physical activity, etc? When the dietary intake increases, what are the consequences for the lactating mother? More milk, more physical exercise, or increased body weight?

It is obvious that the duration of the lactational amenorrhoea is the most widely used index of assessment of return of fertility. However, I do not agree with you about the
accuracy of this criterion even if, by and large, return of menstruation is the most readily measurable index of returned fertility during lactation. The occurrence of anovulatory bleeding is more frequent than is often realized, and it is variable from population to population.

The birth interval is likely to be more accurate for evaluating the duration of the lactational infertility, provided other factors do not interfere, such as contraceptive practices, sexual abstinence, taboos, polygamy.

Your remarks on the comparison between undernourished and well-nourished communities in different countries are very interesting. Your ideas and those of Dr. Frisch seem to be divergent on this point. Would you please extend your comments by considering data suggesting the influence of non-nutritional factors?

In Kivu, the difference of 1 to 4 kg in body weight between menstruating, lactating mothers reported by Carael (1) was not confirmed by Delvoye et al. (2). In this study, the difference was only of 800 g on average (approximately 1.5%). It may be explained equally well by the nutritional status or by the endocrine balance. The discrepancy between the two studies is likely to be due to different social traditions existing between the two populations investigated.

When food supplements are given to the mother, their nature should be specified, particularly during lactation.

You mention that suckling may be more frequent in the case of undernourished mothers. This is not always so. In our study, urban mothers of Kivu, also better nourished, suckle more frequently than rural mothers of the same region. But the total duration of nursing per 24 hr is longer in rural mothers. There is a significant correlation between prolactin levels and the frequency of nursing but not its duration. The duration of nursing is correlated with the nutritional status of the sucking infant. Thus, at least in the two populations of Kivu as investigated by Dr. Hennart, the frequency of sucking is more a choice of the mother, whereas the duration of sucking depends more on the nutritional status of the infant and perhaps on the amount of milk he can get from the breast.

You mention that low levels of oestrogens decrease prolactin production. According to my own experience and to the literature, high levels of oestrogens stimulate prolactin synthesis and release.


H. L. Vis

“But undoubtedly temporary short-term relative infertility associated with acute starvation is a mechanism by which the breastfed infant might continue to receive breast milk and benefit.” When a mother suffers acute malnutrition, the milk production becomes insuffi-
cient, and the child will also be undernourished and runs the risk of dying (1,2). Consequently, we cannot speak about a protective adaptation.

"Thus, theoretical considerations suggest that it is unlikely that chronic maternal undernutrition will be associated with a marked decrease in fertility." Yet it is associated with a marked decrease in fertility. The duration of reproductive life is diminished (Rose Frisch: Menarche occurs later and menopause starts earlier). The impression that in Third World countries the population growth seems to be important (as indeed it is) might be attributed to the lack of contraception. If the nutritional status were to be improved, the population growth rate would possibly be more elevated than it is now.

Parameters for Assessment of Maternal Nutritional Status

In developing countries, maternal nutritional status depends not only on quantitative but also on qualitative factors. The effect of the qualitative aspect of the diet on fertility is not well studied. What are the consequences on fertility of a lack of protein (kwashiorkor), lack of trace elements (Zn, Cu, Se, etc.), lack of iodine (as in endemic goitre and cretinism), lack of vitamins, etc?

In protein malnutrition, there is an impairment of phenylalanine and tyrosine metabolism, but we do not know what happens to the synthesis of dopamine, which seems to be the prolactin-inhibiting factor (PIF). In areas where endemic goitre and cretinism prevail, there is decreased fertility. Zinc deficiency causes hypogonadism in males. The factors interacting with the mother's nutritional status are given in Fig. C-1, p. 81, this volume.

The main comment on Ramachandran's own work is that the weight of the lactating mother is used as the only parameter to be compared with the duration of lactation and lactational amenorrhoea.

In her own work (Table 8), there seems to be a correlation between the mother's weight and the mean duration of unsupplemented lactation. Heavy mothers have a shorter unsupplemented lactation duration than light ones (3). It is possible that because of the early introduction of weaning food breastfeeding frequency decreases; therefore, it would not be directly the weight, but the change in suckling pattern that makes the difference.

We have discussed the figures of Carael (4) (Table 7) (Ntomba-Havu) in relation to the paper of Rose Frisch. As a matter of fact, Pagezy (5) found a difference of more than 5 to 10 kg between Bantus (Oto) and pygmyoid mothers (Twa) of the Ntomba society that were lactating and menstruating (see Table C-1, p. 83, this volume). So Rose Frisch is right when she claims that if we must consider the weight it has to be in relation to height.


A. S. MCNEILLY

The statement that giving supplements to women caused a small but consistent decrease in duration of postpartum amenorrhoea is supported only by the results from Atole village and not by those from Fresco village (Table 10). It would be important to know the actual range of the durations and whether supplements altered the distribution around the mean for duration of amenorrhoea.

One essential flaw in the oestrogen-prolactin theory as proposed is that in many women lactational amenorrhoea is associated with prolonged periods of absent ovarian function, with levels of oestrogen equivalent to those seen in postmenopausal women. I do not see how the cycle of low oestrogen, high prolactin, etc., might operate. Suckling is the key parameter which maintains prolactin secretion. The comparative effect of oestrogen is very small.

The key issue that remains is what is the reason for the shorter periods of lactational amenorrhoea when maternal body weight is increased by 15 kg and/or dietary intake is increased by 700 kcal/day. The possibility that this is linked directly to the availability of milk and the consequent amount of suckling required by the child for it to receive its necessary daily input has not been fully considered.

D. J. NAISMITH

This review highlights the difficulties involved in unravelling the relationship between nutrition and lactational infertility—the interactions of nutrition of the mother, infant feeding practices (duration of breastfeeding, milk volume, the introduction of supplements, frequency of suckling in satisfied and hungry infants), physiological adaptation, and hormone secretion.

Perhaps there has been too much emphasis on maternal body weight. Would weight for height be a more useful measurement? Changes in body weight are invariably due to alterations in body fat alone and are caused by changes in energy balance. A change in "energy intake," however, is a change in total food intake, and physiological responses may result from a deficiency of nutrients other than "energy." For example (Table 10), dietary supplementation reduced lactational amenorrhoea by 2 months but only (if I remember correctly) when the supplement (Atole) contained protein. An energy supplement (Fresco) had no appreciable effect.

I do not agree with Dr. Ramachandran's theoretical argument about evolution, lactational infertility, and maternal nutrition. The evolution of social and cultural practices associated
with breastfeeding is likely to interfere with any biological process designed to ensure species survival. The social condition (especially urban) makes a major contribution to infant mortality. Prolonged interbirth intervals may not seem advantageous to species survival, but a malnourished mother with reduced resistance to infection is unlikely to contribute to the species for very long.

P. G. LUNN AND R. G. WHITEHEAD

In discussing whether a long delay in the return to fertility after childbirth constitutes a beneficial evolutionary adaptation, Dr. Ramachandran differentiates between severe dietary restriction or starvation, which usually occurs acutely and over relatively short periods, and the mild to moderate chronic undernutrition to which most inhabitants of the developing world are permanently subjected. Amenorrhoea occurs in response to severe malnutrition, whether women are breastfeeding or not, and it is therefore doubtful that this can be an adaptation which has developed to protect the suckling child. Rather it appears to be a mechanism which protects potential mothers from conception when environmental conditions or her nutritional status are extremely poor. In order for an evolutionary trend to develop which would assist the suckling child, one would expect that the child itself would play some role in controlling the mother's metabolism, and this is exactly the situation which seems to occur during suckling-induced lactational amenorrhoea. We cannot agree that prolonged interbirth intervals would be detrimental to species survival, as Dr. Ramachandran suggests, and in fact would consider this phenomenon to be a highly desirable adaptation. If maternal food supply is so short, surely the best chance of species survival (for humans) is to ensure the best possible nutrition and growth of a few children, which clearly requires long-term lactation. The alternative would be to produce more children, many or all of whom might die because of inadequate milk supply and/or poor weaning foods. This latter reproductive strategy is seen mainly in small animals which are capable of producing large litters very frequently. In rats, for example, it is more important for the dam to survive rather than her pups.

Assessment of Nutritional Status

It must be recognized that a true assessment of nutritional status is an extremely difficult task to perform and that it is probable that no single parameter can be assumed to be a reliable index. Moreover, the types of assessments which have been used are in general only concerned with energy status. Very few consider protein and even fewer mineral and vitamin levels, but all nutrients really should be considered to be potentially essential to a successful lactation.

Clearly, measurement of food consumption is the most direct type of assessment, but as Dr. Ramachandran warns, to do this adequately requires a very considerable amount of time and expertise as well as highly cooperative subjects. Even then, it must be appreciated that this approach measures only nutrient intake, and not status, which is influenced by many additional factors and in particular energy expenditure. Consequently, even if in situations where there is a constant supply of food, seasonal variations in work load, in particular at times of crop sowing and harvesting, can markedly affect maternal health. It is also rather
difficult to interpret food intake data in terms of the recommended allowances, as the consumption of most nutrients by inhabitants of developing countries is very much below the generally accepted figures. This inevitably raises questions about possible adaptive mechanisms to their low food intake.

Maternal body weight has also been widely used in this context but really this must be regarded as a poor index. Weight/height or weight/height$^2$ may be better parameters, but even they must be used with considerable caution. Although very low weights may well indicate a very poor nutritional status, small variation within a more normal range may mean very little. The extreme fallibility of maternal weight as an index has been clearly demonstrated in the Gambian study, where lactating women receiving an average 723 kcal/day dietary supplement (46%) were only 1.8 kg heavier than their unsupplemented counterparts and still showed the seasonal changes associated with agricultural labouring (1).

In the frequently quoted study carried out by Huffman et al. in Bangladesh (2), assessment of nutritional status by maternal body weight and by socio-economic status gave differing results; the former did not correlate with duration of amenorrhoea, but the latter did. Which of these is the correct answer is difficult to decide, because although the use of socio-economic measures may give a good indication of maternal status, different socio-economic groups are likely to have different breastfeeding practices which will certainly confound the results.

Nutritional Status and Lactational Infertility

We would agree entirely with Dr. Ramachandran's suggestion that the result of the Gambian study should be considered as demonstrating the potential reproductive capacity of the women studied. However, it must be realized that the dietary supplement, although large in terms of food consumption by non-supplemented women, was still slightly below the values obtained from lactating United Kingdom women, which itself was well below the recommended allowances. We cannot agree with Dr. Ramachandran's argument that because such improvements in food availability are unlikely to occur quickly in most parts of the developing world the effect on fertility and birth rate is of little practical importance. If the aim for the future is to improve food availability up to accepted norms, then the considerable effect on potential birth rates observed in The Gambia must be expected to occur, irrespective of how long such dietary improvements may take. Unless this is appreciated and counteracted, the population can be expected to expand in parallel with food supply, and malnutrition will not be overcome.

Finally, we are not aware of the data suggesting that low levels of oestrogens stimulate prolactin production, but there is ample data indicating the reverse. Despite this criticism of the argument, however, it is still possible for the two theories for maintenance of lactational amenorrhoea to coexist. However, it seems more likely from the literature that the body fat/oestrogen pathway only becomes important under extreme conditions.

G. S. MASNICK

Dr. Ramachandran notes that whereas "temporary short-term relative infertility associated with acute starvation is a mechanism by which the breastfed infant might continue to receive breast milk and benefit... theoretical considerations suggest that it is unlikely that chronic mild maternal undernutrition will be associated with marked reduction in fertility," because "undue prolongation of interbirth interval is likely to be counter-productive in terms of species survival because in such situations infant and childhood mortality rates are likely to be high and have to be 'compensated' by high birth rate" (see chapter by Ramachandran, this volume). An alternative hypothesis would recognize that even areas of chronic low nutrition are punctuated by periods of relative abundance in food resources. During these periods, an increase in food available to both mothers and infants could initiate the reduction in suckling frequency/intensity and signal the resumption of ovulation. The response of adaptation would then be the ability for reproductive events to respond quickly to changes in nutritional intake. Such a view of reproductive events as opportunistic responses to environmental changes would seem to be highly adaptive.

J-P. HABICHT

This review paper examines the evidence for the hypotheses that malnutrition affects lactational infertility. The paper proceeds in a logical progression from examination of measures of the putative cause and its effect, through evidence of association, evidence for confounding due to breastfeeding behaviour, evidence of association when this confounding is taken into account in studies with and without nutritional interventions, and ends in a conclusion that extremes of nutrition do affect lactational infecundity, but in most populations this has a minimal effect on fertility. There is also some discussion about possible mechanisms mediating malnutrition's effect on lactational infecundity.

As would be expected from a paper of this breadth, the discussion of mechanisms linking malnutrition to lactational infecundity is not as well developed as in the endocrinological papers in this series. However, I feel better development should be possible, at least so that one can examine the assumptions necessary to analyze population data on future studies. A simplistic model underlying such an analysis (A), and an examination of the assumptions used in the simplistic model (B) are set out in the appendix by Habicht (this volume, p. 139). One needs this kind of model not only to develop more powerful statistical analysis of these kinds of data but also, and very important, to select a feasible number of measures of mediating variables between the malnutrition and the infecundity so that one can choose between competing hypotheses. The author presents a hypothesis integrating the idea that the resumption of menstruation depends on a certain critical mass of adipose tissue and that it depends on prolactin levels. However, that hypothesis is not sufficiently developed to address the questions which are raised in (B) (p. 139, this volume) or to design studies to test it against alternative hypotheses.
The point made in the review about self-selection and other confounding intervention trials needs clarification, not because it affects the argument in the review but because of the fact that future work will depend on such trials. The author criticizes the Guatemalan study because the women receiving the supplements were a self-selected group and their babies might be receiving supplements not given to the babies of the other women. This has always been my own view also. I would have expected that these issues would be controlled for in future work and that reviewers would examine reports to see whether or not this was so. It was disappointing that these issues were not even raised in the review for the other intervention trials which were reviewed in this paper after the criticism of the Guatemalan study.

Another much less serious issue is the (evolutionary?) argument which I paraphrase as "Mankind would have disappeared if malnutrition impaired fertility." The logic of that paragraph is weak.

J. HOBCRAFT

Dr. Ramachandran asserts there is "no deterioration in maternal nutrition during pregnancy and lactation or with increasing age and parity." I have not read the studies cited, but a number of other studies suggest different conclusions of very low weight gain during pregnancy (1) and of steady weight loss during lactation (2). Older women in Bangladesh are less well nourished from anthropometric measures (3).

Use of interpregnancy or interbirth intervals is clearly inappropriate for "measurement of return of fertility during lactation." Even if we know about postpartum abstinence and contraceptive levels, this does not permit disentanglement, unless both are negligible. Even then, one could only hope to infer the length of amenorrhoea or anovulation inexactly, by making assumptions about fetal loss, and more particularly the mean wait to conception.

Chavez study: Bongaarts (2) notes new observations presented at the Twelfth Workshop on Nutrition and Reproductive Competency by Chavez, which indicated that supplemented mothers whose children were not supplemented had the same mean amenorrhoeic interval as unsupplemented mothers (but does not state whether these unsupplemented mothers' children were supplemented). This substantially changes interpretation of Chavez's results.

Conclusions

1. Lactational amenorrhoea is a fairly effective contraceptive. Suppose a 12-month average length and that 5% of women conceive before menstruation. This would make lactational amenorrhoea more effective than the condom or diaphragm for menstruating United States women and not much less effective than the IUD (4% failures in a year for exposed women). Such analyses can, of course, be complicated by effects of postpartum abstinence: a very effective method, if easily reversible (cf. ref. 4, Table 3.9).

2. The evidence for rising fertility rates in Kenya or Latin America is equivocal at best. There is also very little direct evidence on changes in durations of breastfeeding for these countries. Thus, the last sentence is a gross overstatement. But it is very clear that big changes in duration of lactation could have very substantial fertility effects (4–6). Also,
shortening of the period of amenorrhoea may reduce contraceptive effectiveness of lactational amenorrhoea.

Table 3 suggests astonishing differences in mean wait to conception of 11.2 months for urban and 6.3 months for rural areas. This raises severe doubts about data quality.

Table 8: I would like to see more discussion of the regression results and correlations at the foot of this table, as they seem quite relevant. Also, what about effects of partial breastfeeding in this study? How many women were studied?

Table 9: Did all body-weight groups begin supplementation at the same time? What about duration and frequency of suckling differentials between the groups?


R. E. FRISCH

Harrison et al. (1) showed decreases in adiposity in poorly nourished lactating women in New Guinea. It would be very surprising if poorly nourished women showed no change in nutritional status with increasing parity, since there is an increase in pregnancy wastage with increasing parity, and birth weights are lower in the later parities.

Mild weight loss (10% to 15% of normal) causes primary and secondary amenorrhoea in women who were formerly well nourished. The amenorrhoea is due to hypothalamic dysfunction. The hormonal profile becomes prepubertal. See also Wilmsen (2) on seasonal birth rates, and Chen (3).

Loss of body weight in the range of 10% to 15% of normal is loss of fat (about one-third body fat). Greater losses cause loss of lean body mass in addition to loss of fat.

There are no precise data on birth interval changes in the last two centuries. The Kipley-Kipley data cited by Bongaarts (4) are very poor. The data from Salber et al. (5) show a very short duration of lactational amenorrhoea (4 to 6 months) in well-nourished women in Boston.

It is very important to give weight for height, in relation to duration of lactational amenorrhoea. Women who weighed over 55 kg compared to less than 40 kg: statement needs reversing.
I heartily concur with the conclusion: The linkage of food supplementation and family planning services has been recommended by many public health workers to improve maternal and child health.