Nutrition–fertility interaction in lactating women of low income groups

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(*Received* 28 *May* 1980 – *Accepted* 27 *November* 1980)

1. Duration of lactational amenorrhoea and interpregnancy interval were related to maternal body-weight in an urban low-income group population of 2250 women.
2. Breast-feeding practices were similar in all the women, none of whom had received any nutritional intervention.
3. In the study group the mean duration of lactation, lactational amenorrhoea and interpregnancy interval (months) were 20·7, 11·2 and 24·2 respectively.
4. Mean duration of lactation was signficantly shorter in women whose body-weights were over 55 kg. Duration of lactational amenorrhoea increased with increasing duration of lactation (r 0·5164).
5. The duration of lactational amenorrhoea showed a progressive fall with increasing body-weight (r −0·8139) even when duration of lactation was kept constant.
6. The mean duration of post-partum amenorrhoea in women who did not lactate remained unaltered irrespective of body-weight.
7. The findings suggest that maternal nutritional status modifies return of menstruation only in lactating women.

Epidemiological and laboratory investigations have confirmed the traditional belief that lactation prolongs post-partum amenorrhoea and provides some extent of protection against pregnancy (Population Reports, 1975; Prema *et al.* 1979). Studies in undernourished women from developing countries have shown that undernutrition does not have any adverse effect on the duration of lactation, quantity or quality of milk produced with respect to proximate principles (Jelliffe & Jelliffe, 1978; Belavady, 1979). There have been a few studies on the effect of maternal nutritional status on the return of menstruation and fertility in lactating women (Chavez *et al.* 1972; Chen *et al.* 1974; Jain *et al.* 1976; Delgado *et al.* 1977). It is, however, difficult to draw any definite conclusion from the available information because in each of the previously-mentioned studies one or more confounding factors, for example differences in socio-economic status, seasonal variations and nutritional interventions, were present which could account at least in part to the reported differences in the return of menstruation. A study was therefore undertaken to evaluate the effect of maternal nutritional status on the return of menstruation and fertility in a homogenous low-income-group population with similar breast-feeding practices who did not receive any nutrition or health intervention.

MATERIAL AND METHODS

Women from an urban low-income group (per capita income 30–90 Rs/month) formed the study group. Only women who could clearly recall events and give accurate history were selected. Detailed information on demographic and obstetric profile was obtained in all cases. Only women who had not used any form of contraception were included in the study. Information on the duration of unsupplemented lactation, total duration of lactation, lactational amenorrhoea and interpregnancy interval was obtained for 4749 lactational periods in 2250 women. Height, weight, mid-arm circumference and skin-fold thickness at triceps were measured in all the women. Harpenden calipers were used for measurement

0007-1145/81/3536-2711 $01.00

Table 1. *Effect of lactation on post-partum amenorrhoea in Indian women from a low-income group*

Group	Lactating	Non-lactating
No. of women studied	2042	208
No. of post-partum periods studied	4463	286
Mean duration of unsupplemented lactation (months)	8·9	—
Mean duration of lactation (months)	20·7	—
Mean duration of post-partum amenorrhoea (months)	11·2	4·6***
Mean interpregnancy interval (months)	24·2	17·3***

*** $P < 0.001$.

of skin-fold thickness. On the basis of body-weight the study population was divided into five groups. The mean duration of lactation, lactational amenorrhoea and interpregnancy interval were calculated separately for each of these groups. Similarly in women who did not lactate, the mean duration of post-partum amenorrhoea and interpregnancy interval was calculated in each of the five weight groups. The correlation between body-weight and post-partum amenorrhoea was tested in both lactating and non-lactating women.

RESULTS

Prolonged and unsupplemented lactation was the usual breast feeding practice in this population group. Babies were breast fed on demand. The mean duration of unsupplemented lactation in this population was 8·9 months. The mean duration of lactation in the population was 20·7 months, the mean duration of lactational amenorrhoea 11·2 months and the mean interpregnancy interval 24·2 months. Among non-lactating women the mean duration or post-partum amenorrhoea was 4·6 months and mean interpregnancy interval 17·3 months. Thus in the women studied lactation resulted in an increase in the relatively infertile period of lactational amenorrhoea by 6 months and a consequent 6 months increase in the mean interpregnancy interval (Table 1).

Duration of lactational amenorrhoea increased with increasing duration of lactation (Table 2). However, prolonged lactation cannot indefinitely postpone resumption of menstruation or return of fertility. Menstruation had in fact returned long before the women stopped breast-feeding; in approximately 20% of the women the advent of next pregnancy was the factor responsible for stopping breast-feeding.

The study population was divided into five groups on the basis of body-weight. There were no significant differences in the age, parity or socioeconomic status between women in those five different groups. The mean duration of unsupplemented lactation, total duration of lactation and lactational amenorrhea were, however, significantly shorter in women whose body-weight was over 55 kg (Table 3).

Since duration of lactation is an important factor influencing duration of lactational amenorrhoea it is possible that shorter duration of lactational amenorrhoea in women weighing more than 55 kg might at least in part be due to shorter duration of lactation. In order to eliminate this factor, mean duration of lactational amenorrhoea was calculated in relation to body-weight among women in whom the mean and frequency distribution of duration of lactation were similar. The sample was chosen by random sampling procedure keeping the proportion of women who had been lactating for various durations i.e. 0–6, 7–12, 13–18, 19–24, > 24 months equalized in different body-weight groups with reference

Table 2. *Effect of duration of lactation on duration of lactational amenorrhoea and interpregnancy interval in Indian women from a low-income group*

(2042 lactational periods studied)

Duration of lactation (months)	Mean duration of lactational amenorrhoea (months)	Mean interpregnancy interval (months)
⩽ 6	5·3	16·3
7–12	6·0	17·0
13–18	10·4	20·0
19–24	12·6	24·6
25–30	14·5	28·4
> 30	15·4	30·4

Lactational amenorrhoea *v.* lactation (r 0·5164, $P < 0·001$); interpregnancy interval *v.* duration of lactation (r 0·4728, $P < 0·001$); interpregnancy interval *v.* lactational amenorrhoea (r 0·5064, $P < 0·001$).

Table 3. *Effect of body-weight on duration of lactation and lactational amenorrhoea in Indian women*

(2042 lactational periods studied)

Body-weight (kg)	Mean duration of unsupplemented lactation (months)	Mean duration of lactation (months)	Mean duration of lactational amenorrhoea (months)
< 40	9·2	20·9	13·2
40–44	8·9	19·8	11·4
45–49	8·6	20·1	10·2
50–54	8·4	19·8	9·3
⩾ 55	7·6	16·6	7·5

Body-weight *v.* duration of lactation:
Duration of lactation = 45·80 − 0·0314 × body-weight (r −0·0566 not significant).
Body-weight *v.* duration of lactational amenorrhoea:
Duration of lactational amenorrhoea = 46·78 − 0·1824 × body-weight (r −0·7251 $P < 0·001$).
Partial correlation between:
Lactation and lactational amenorrhoea = 0·6914 $P < 0·001$ (keeping body-weight constant).
Body-weight and lactation = −0·0250 NS (keeping lactational amenorrhoea constant).
Body-weight and lactational amenorrhoea = −0·8139 $P < 0·001$ (keeping duration of lactation constant).

to distribution of lactational periods in the 45–49 kg groups (Table 4). Duration of lactational amenorrhoea was also calculated in relation to body-weight in women who had been lactating for 13–18 and 19–24 months (Fig. 1). For the same duration of lactation, duration of lactational amenorrhoea showed a progressive fall with increasing body-weight (correlation between body-weight and duration of lactational amenorrhoea r −0·8139). The findings suggest that there is a delay in return of menstruation in lactating women who are undernourished. The mean duration of post-partum amenorrhoea or interpregnancy interval in women who did not lactate remained essentially unaltered irrespective of variations in body-weight (Table 5). The findings suggest the possibility that nutritional status modifies the return of menstruation only in lactating women.

Table 4. *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*

(Mean values and standard deviation; no. of observations given in parentheses)

Body-wt (kg)	Mean duration of lactation (months)		Mean duration of lactational amenorrhoea (months)	
	Mean	SD	Mean	SD
< 40 (276)	21·3	10·61	13·1	11·74
40–44 (356)	22·5	10·32	12·0	8·23
45–49 (297)	20·8	10·76	10·7	9·36
50–54 (209)	20·7	10·58	9·3	10·13
⩾ 55 (222)	20·7	8·53	8·2	7·82

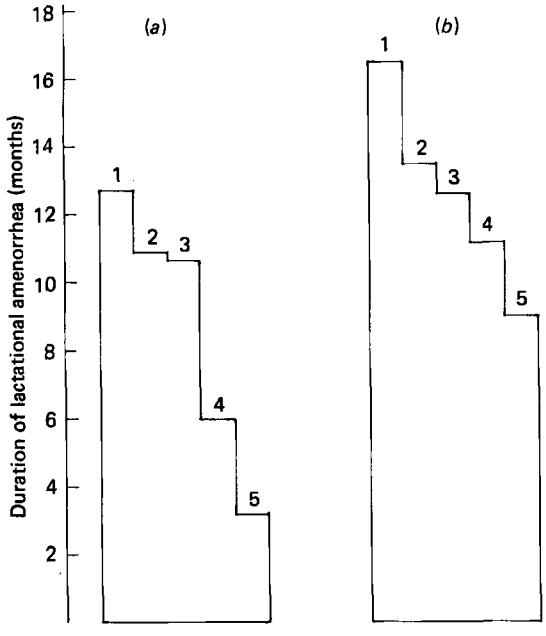

Fig. 1. Effect of body-weight (kg) on duration of lactational amenorrhoea (months) in Indian women from low-income groups. (*a*) Woman lactating for 13–18 months and (*b*) women lactating for 19–24 months. Body-weights (kg) were 1, < 40; 2, 40–44; 3, 45–49; 4, 50–54; 5, ⩾ 55.

DISCUSSION

The clinical impression that obese women do not lactate as well as do their leaner counterparts is supported by the present study. Though there was no correlation between body-weight on the one hand and duration of lactation on the other in the entire group, the mean duration of lactation was significantly lower in women whose body-weight was more than 55 kg as compared to those women with a lower body-weight. The exact mechanism through which this effect is mediated is not clear. All the women studied here

Table 5. *Duration of post-partum amenorrhoea in non-lactating Indian women from a low-income group in relation to body-weight*

(Mean values and standard deviation; no. of observations given in parentheses)

Body-wt (kg)	Mean duration of post-partum amenorrhoea (months)	
	Mean	SD
< 40 (25)	3·9	0·50
40–44 (20)	4·7	0·52
45–49 (32)	4·4	0·59
50–54 (41)	4·9	0·56
⩾ 55 (26)	3·6	0·68

belonged to apparently similar socio-economic and educational groups with similar breast-feeding practices. Therefore the effect could not be due to differences in these factors. It has recently been shown that the pharmacokinetics of exogenous steroids is influenced by body-weight and that heavier women with a higher skinfold thickness excrete the drug more slowly (Prasad *et al.* 1979). It is possible that a similar mechanism might operate with respect to endogenous steroids as well. If this were to be so, for any given level of secretion of endogenous steroids heavier women are likely to have higher and more sustained plasma steroid levels. Oestrogens are known to antagonize the effect of prolactin on milk secretion. It is possible therefore that higher and more sustained circulating levels of oestrogens might at least in part be responsible for the shorter duration of lactation in heavier women. It is well known that there are marked inter-individual and diurnal variations in circulating oestrogen levels. Variations in levels of oestrogen, oestrogen:prolactin and target-organ sensitivity are likely to be important factors which modify the effect of oestrogens on lactation. Because of the multiplicity of factors which influence lactation and the known large variation in each of these factors in normal individuals, it is difficult to verify experimentally the hypothesis that the shorter duration of lactation seen in heavier women might be due to higher or more sustained levels of circulating oestrogens. There had been speculations that return of menstruation during lactation might be delayed in undernourished women (Chavez *et al.* 1972; Chen *et al.* 1974; Jain *et al.* 1976; Delgado *et al.* 1977). It is, however, difficult to draw any definite conclusion from the available information because of confounding factors such as differences in socio economic status (Jain *et al.* 1976) seasonal variations (Chen *et al.* 1974) and nutritional intervention (Chavez *et al.* 1972; Delgado *et al.* 1977) in the study groups. The present study was free of most of the pitfalls noted in the earlier studies. All the women belonged to the low-income group. There was no known or organized nutritional intervention either for the mother or for the infant. The pattern of introduction of supplementary feeds was similar in all the groups. The observation that in such a population for any given duration of lactation, lactational amenorrhoea lasted longer in those women who had lower body-weights as compared to the heavier women was signficant. There was a good correlation between mean duration of lactation amenorrhoea and body-weight (r −0·7251) (Table 3).

Available information on nutrition–fertility interaction suggests that undernutrition delays the onset of menstruation in two situations: at the time of menarche and at the time of re-establishment of menstruation in lactating women. The exact mechanisms by which undernutrition delays menarche and resumption of menstruation in lactating women is not

yet clearly understood. Presumably the effect is mediated through alterations in the hypothalamus–pituitary–ovarian axis.

Plasma prolactin levels in lactating women from developing countries are higher and remain elevated for a year or more unlike the rapid return to normal levels seen in women from developed countries (Delvoye *et al.* 1978; Tyson *et al.* 1978; Lunn *et al.* 1980). In the past it was generally assumed that the persistent elevated prolactin levels in the former group was mainly due to the prolonged, unsupplemented, on-demand breast feeding practices. Some of the more recent investigations indicate that this may not be the sole factor. Lunn *et al.* (1980) reported that in Gambian women maternal dietary intake had a marked effect on plasma prolactin levels. Some results from plasma prolactin levels in a low income group of women in Hyderabad indicate that plasma prolactin levels were higher in undernourished women (K. Prema, unpublished results). It has been suggested (Lunn *et al.* 1980) that the prolonged high prolactin concentation found in undernourished women may ensure milk synthesis when food intake is limited by preferentially channelling nutrients towards milk production. It is also possible that an anabolic effect of prolactin may play a vital role in adaptive mechanisms that prevent deterioration of maternal nutritional status in lactating women subsisting on inadequate diets. It is possible that persistantly elevated prolactin levels might also play an important role in prolongation of the relatively infertile post-partum amenorrhoea and prevent early advent of next pregnancy with all its detrimental effects on both maternal and infant nutrition.

Frisch (1977) postulated that the attainment of a critical fatness altered the sensitivity of hypothalamus to gonodotrophins with consequent attainment of menarche. This hypothesis however has not been confirmed either by clinical observations or by studies in experimental animals. Pharmacokinetic studies both in experimental animals and in women have shown that following administration of exogenous steroids plasma levels were lower and urinary excretion was faster in undernutrition (Prasad, Narasinga Rao *et al.* 1979; Prasad, Sivakumar *et al.* 1979). It is possible that a similar mechanism might operate with respect to endogenous steroids as well. The circulating endogenous steroid levels are known to affect the pituitary–ovarian axis and the effect might be expected to be maximal at the time of establishment (as in menarche) or re-establishment (as in return of menstruation in lactating women) of normal pituitary–ovarian axis. It is possible that women with a higher body-weight have a more sustained ovarian steroid concentration for the same amount of endogenously-produced oestrogens, resulting in accelerated establishment of a normal pituitary–ovarian axis and target-organ changes resulting in faster return of menstruation. It is however difficult to design studies to verify whether the hypothesis is in fact correct.

Undernutrition, which is common in women belonging to low-income groups in developing countries, becomes aggravated during lactation. It is essential, therefore, that efforts be made to improve maternal nutritional status. Improvement in nutritional status may however lead to a faster return of menstruation and fertility in lactating women which in turn may shorten the interpregnancy interval. The early advent of the next pregnancy is likely to negate much of the benefit of nutritional supplements. It is therefore essential that efforts to improve health and nutritional status of lactating women should be integrated with contraceptive care.

The authors are grateful to Dr S. G. Srikantia, Director, National Institute of Nutrition, Hyderabad for his keen interest in this study.

REFERENCES

Belavady, B. (1979). *Proc. Swedish Nutr. Fdn Symp.* no. 14.

Chavez, C., Martinez, C., Bourges, H., Coronado, M., Lopez, M. & Basta, S. (1972). *Proc. 9th int. congr. Nutr. Mexico*, p. 90.

Chen, L. C., Ahmed, S., Gesche, M. & Mosley, W. H. (1974). *Pop. Stud.* **28**, 277.
Delgado, H., Lechtig, A., Yarbrough, C., Martorell, R., Klein, R. E. & Irwin, M. (1977). In *Nutritional Impacts on Women*, p. 133 [K. S. Moghissi and T. N. Evans editors]. London: Harper and Row.
Delvoye, P., Desmaegad, M., Uwayitu Nyampela & Robyn, C. (1978). *Am. J. Obstet. Gynecol.* **130**, 635.
Frisch, R. E. (1977). In *Nutrition and Human Reproduction*, p. 91 [W. H. Mosley, editor]. New York: Plenum Press.
Jain, A., Hsu, T. C., Freeman, R. & Chang, M. (1976). *Demography* **7**, 255.
Jelliffe, D. B. & Jelliffe, E. F. P. (1978). *Am. J. clin. Nutr.* **31**, 492.
Lunn, P. G., Prentice, A. M., Austin, S. & Whitehood, R. G. (1980). *Lancet*. i, 623.
Population Reports (1975). *Pop. Rep. Ser. J.* no. 4.
Prasad, K. V. S., Narasinga Rao, B. S., Sivakumar, B. & Prema, K. (1979). *Contraception* **20**, 77.
Prasad, K. V. S., Sivakumar, B. & Narasinga Rao, B. S. (1979). *Contraception* **20**, 619.
Prema, K., Naidu, A. N. & Neela Kumari, S. (1979). *Am. J. clin. Nutr.* **32**, 1298.
Tyson, J. E., Carter, J. N., Andreasson, B., Huth, J. & Smith, B. (1978). *Fertil. Steril.* **30**, 154.

Printed in Great Britain