Proceedings of the Nutrition Society (1995), **54**, 379–389

PROCEEDINGS OF THE NUTRITION SOCIETY

The Summer Meeting of the Nutrition Society was held at the University of Southampton on 2–5 August 1994

Symposium on 'Maternal–offspring interactions'

Maternal nutrition and the regulation of milk synthesis

BY PETER HARTMANN[1], JILLIAN SHERRIFF[2] AND JACQUELINE KENT[1]

[1]*Department of Biochemistry, The University of Western Australia, Nedlands, WA 6009, Australia and* [2]*School of Public Health, Curtin University of Technology, Bentley, WA 6102, Australia*

Recent research has demonstrated the unsurpassed value of breast-feeding in promoting the optimal nutrition of the human neonate. Breast milk has been shown to have a uniquely appropriate composition for the neonate at a time when growth and development are occurring at a near-maximal rate, yet when many of the body's systems (digestive, hepatic and renal) are relatively immature. The N required for growth is both qualitatively and quantitatively appropriate for the immaturity of these systems, and the bioavailability of many vitamins and minerals to the neonate is enhanced in breast milk. Trophic factors in human milk (peptides, growth factors, hormones, amino acids, glycoproteins, polyamines and nucleotides) promote the development of the immature gastrointestinal tract, and breast milk contains a number of enzymes (e.g. bile salt-stimulated lipase (*EC* 3.1.1.3)) with the potential to augment the infant's gastrointestinal digestive capacity (Harzer & Haschke, 1989). These diverse observations illustrate the advantages of human milk and provide the rationale for the recommendation that breast milk should be the infant's only source of nutriment for the first 4–6 months of life (National Health and Medical Research Council, 1992).

In contrast to the nutrition of the breast-fed infant, less definitive information is available on the relationship between maternal nutrition and the well-being of the lactating woman. Mothers who breast-feed their infants have prolonged postpartum amenorrhoea (Short *et al.* 1991) and this may assist in the maintenance of maternal Fe status. Breast-feeding may assist maternal weight loss, or on the other hand it may increase the risk of obesity (due to the failure to lose fat stored during pregnancy). An increased risk of osteoporosis is also a possible consequence.

Since the energy output in milk represents a significant proportion of the energy intake in the maternal diet of most mammals (approximately 25% for women exclusively breast-feeding their infants), it is reasonable to expect that the level of food intake would regulate milk yield. This expectation has received experimental support from research in dairy animals; however, a comprehensive review by Prentice *et al.* (1986) questioned its validity for lactating women. Prentice *et al.* (1986) demonstrated that milk yield from poorly nourished mothers in developing countries was similar to that from well-nourished mothers in developed countries and that improving maternal nutrition did not increase milk yield of women in developing countries. They concluded, therefore, that the most important determinant of milk yield in women from both groups of countries

was the optimization of the breast-feeding relationship between the infant and its mother. A generalization implicit from this conclusion is that the importance of maternal nutrition in the regulation of milk yield may be fundamentally different in traditional dairy animals (completely milked twice daily) compared with mammals which suckle their young according to natural behaviour patterns.

NUTRITION AND MATERNAL PHYSIOLOGY

Dietary energy

The increasing adiposity in people in Western societies is of considerable concern (Egger, 1992). For Australia, the 1989 Risk Factor Prevalence Study classified 36·9% of females as overweight (a body mass index (BMI) greater than 25 kg/m^2; National Heart Foundation of Australia, 1990). It has been suggested that pregnancy is a risk factor for the development of obesity (Sheldon, 1949; Ohlin & Rossner, 1990), as weight loss postpartum is not inevitable. Furthermore, it is possible that changes in energy metabolism associated with pregnancy and lactation remain after weaning, compounding the difficulty of weight loss. In view of the health risk and the image problems associated with excess body fat, and the high failure rates which accompany attempts to lose weight in the long term, it is important to assess the contribution of lactation to the development of this problem.

Some lactating mammals such as dairy cows and laboratory rats depend heavily on an increased food intake, and milk yield suffers if this is not met; while others such as some seals and whales rely entirely on body fat and protein reserves to sustain their lactations. Traditionally, weight gain in women during pregnancy has been viewed as including fat deposited for use during lactation (Goldberg *et al.* 1991; Hytten, 1991). It is further asserted that the altered hormonal environment required to support lactation will result in the mobilization of this additional fat (Greenwood *et al.* 1987) and, hence, the attainment of the prepregnancy body weight. However, human studies have not always been able to show a clear advantage of lactation with respect to weight loss, despite the additional energy cost of milk synthesis. Thus, women appear to be intermediate between the extremes of the dairy and aquatic mammals.

In a study of Dutch women, Rookus *et al.* (1987) compared the effect of pregnancy on subsequent BMI (n 49) with the change in BMI during the same period in non-pregnant women (n 400). They concluded that at 9 months postpartum, the women who did not breast-feed and those who breast-fed for less than 2 months did not undergo any greater increase in BMI than was to be expected from ageing. However, women who breast-fed for more than 2 months had a BMI 0·6 kg/m^2 greater than that experienced by the non-pregnant women. Conversely, Dugdale & Eaton-Evans (1989) reported that the duration of breast-feeding did not influence postpartum weight change in 151 Australian women, and Brewer *et al.* (1989), in a study of fifty women from Louisiana, USA, observed that there was no significant difference in the total weight loss in the 6-month postpartum period regardless of feeding mode. Ohlin & Rossner (1990) monitored 1423 women until 1 year postpartum and reported a significant but very weak influence of lactation (r −0·09) on the weight change since the prepregnant state.

It has become clear that weight loss during breast-feeding is not consistent at all stages of lactation but appears to be greatest between 3 and 6 months (Brewer *et al.* 1989; Ohlin & Rossner, 1990; Dewey *et al.* 1993). Thus, studies which have included women who

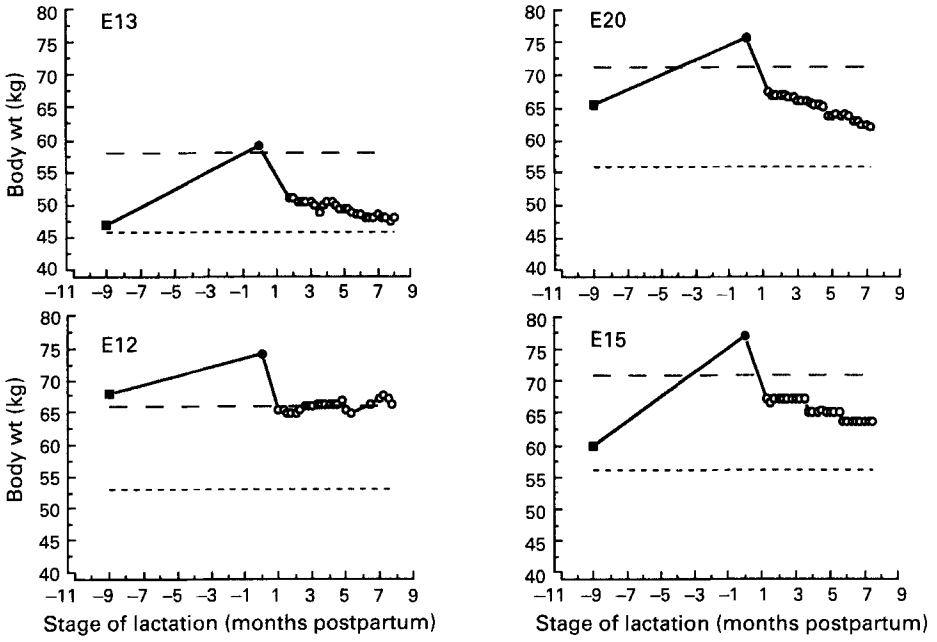

Fig. 1. The weekly change in body weight (—○—) for four subjects between the first and seventh months of lactation. Each subject's self-reported prepregnancy (■) (−9 months postpartum) and prepartum (●) weight (prepregnancy weight plus weight gain over pregnancy, 0 months postpartum) are included for comparison, along with the ideal body weight (IBW) range for a woman of her height (- - - -, minimum IBW; – – – –, maximum IBW).

have breast-fed for less than 6 months will confound the interpretation of the data. In addition, it has been postulated that women who do not breast-feed are more likely to diet in the postpartum period than those who are breast-feeding and, thus, studies may be flawed by large differences in energy intake between breast-feeding and non-breast-feeding mothers. In this connection, Dewey *et al.* (1993) were careful to exclude any subjects who were consciously restricting their energy intake, and reported that women who breast-fed during the first year postpartum lost an average of 2 kg more than those women who formula-fed between 1 and 12 months postpartum.

A further explanation for the apparent lack of a strong influence of lactation on weight loss postpartum is that the response of individuals' body weights to pregnancy and lactation is highly variable and that comparison of mean values distorts the true situation. Indeed, Ohlin & Rossner (1990) reported that 30% of their subjects lost weight, 56% had gained 0 to <5 kg and 14% had gained ≥ 5 kg at 1 year postpartum. In a study of twenty mothers at 1, 3, 5, and 7 months of lactation (four withdrew after 3 months), we observed that while the body weight at 5 months postpartum was significantly higher than the prepregnancy body weight, by 7 months the body weight was no longer significantly different from prepregnancy body weight (Sherriff & Hartmann, 1994). While most mothers lost weight over the lactation period (e.g. mothers E13 and E20; Fig. 1), three mothers gained more than 1·0 kg during lactation (e.g. mother E12; Fig. 1) and three mothers had not returned to their prepregnancy weight by 7 months postpartum (e.g.

https://doi.org/10.1079/PNS19950007 Published online by Cambridge University Press

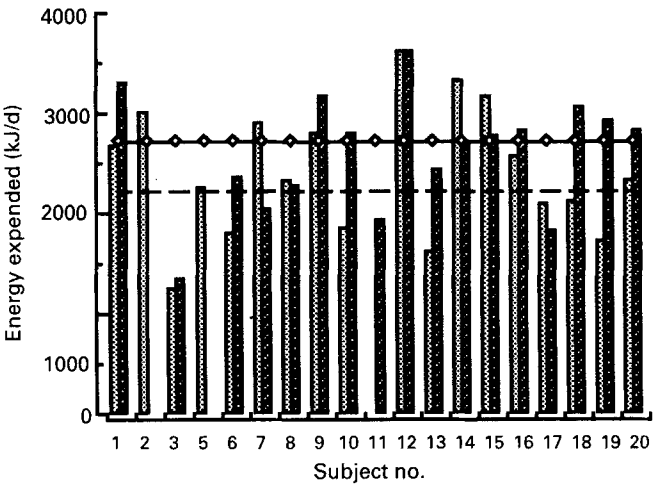

Fig. 2. A comparison of the calculated energy expended in milk synthesis by subjects at 1 (▩) and 3 (▨) months postpartum and (a) the additional energy consumption that is recommended in Australia during lactation (2200 kJ; – – –), and (b) the energy that is theoretically available from the additional energy intake and the mobilization of 2 kg adipose tissue in the first 6 months of lactation (2700 kJ; ◇–◇).

mother E15; Fig. 1). Clearly, either weight gain or failure to return to prepregnancy weight by 7 months postpartum was a problem for more than one-third of lactating mothers.

Anthropometric measurements in these women demonstrated that there was a significant decrease in the mean subscapular and suprailiac skinfolds as well as a decrease in thigh circumference from 1 to 7 months of lactation. On the other hand, eleven subjects underwent an increase in the thickness of the triceps skinfold between 1 and 3 months, whereas the remaining nine subjects either sustained a decrease or did not change. In addition, the mobilization of fat from both the subscapular and suprailiac sites did not translate into weight loss for more than one-third of the women, therefore, this fat must have been deposited elsewhere (presumably in the subcutaneous fat of the upper arm).

There was a large variation (coefficient of variation 25%) in the 24 h output of energy in milk, determined by the summation of the daily outputs of fat, lactose and protein, for infants who were exclusively breast-fed at 1 and 3 months of lactation. Furthermore, the current Australian recommendation for additional energy intake during lactation (2000–2400 kJ/d, assuming that an additional 500 kJ is made available from mobilized fat) exceeded the energy cost of milk synthesis in most women in our study (Fig. 2). However, the calculated cost of milk synthesis for three of the eighteen women at 1 month and four women at 3 months exceeded the upper level of the recommendation (2900 kJ/d) by at least 100 kJ/d.

Recommendations for energy intake during lactation need to address the issue of current weight status, and would serve individuals better if they were based on desired outcome rather than energy intake. From the point of view of the mother's weight loss, it must be stressed that breast-feeding should continue at least until prepregnancy weight is

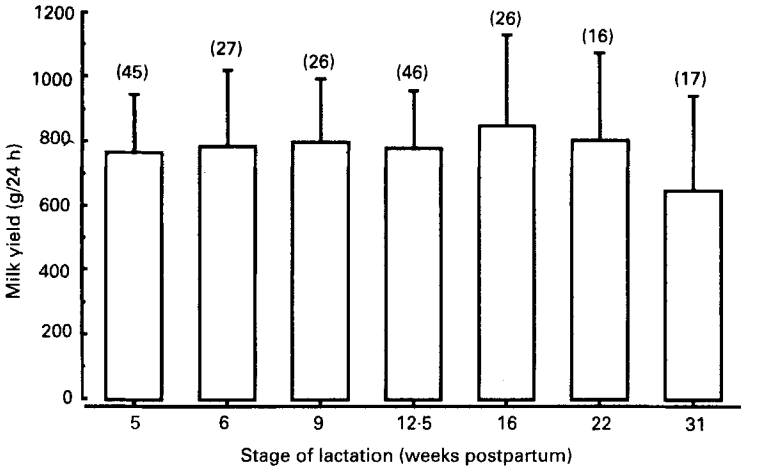

Fig. 3. Values for the 24 h milk yield of sixteen to forty-six women at various stages of lactation (weeks postpartum). Values are means and standard deviations represented by vertical bars for the no. of subjects shown in parentheses.

attained (up to 6–9 months). While it is appropriate to introduce other food sources to the infant from 4 to 6 months of age, the use of alternative milk sources (complementary feeding) will result in reduced energy output in milk and, hence, reduced maternal weight loss. Dewey *et al.* (1993) defined a limit of no more than 120 ml/d. Given the variability associated with both the demand for milk imposed by individual infants (Fig. 3) and the maternal fat stores that are available for use during lactation, universal prescriptive dietary recommendations are not appropriate. The recommendation for breast-feeding of longer duration than is currently practised by the majority of Western women is likely to be met with more success if presented in terms of weight control, as the desire to lose weight is acknowledged as a socio-behavioural factor affecting lactation (Allen *et al.* 1986). This strategy will in turn reduce future health risks to the mother associated with being overweight, particularly if the additional weight has resulted in an increased waist:hip ratio.

Minerals

The combined effects of pregnancy and lactation represent a considerable drain on Ca stores in women, the fetal skeleton requiring approximately 200 mg/d (5 mmol/d), and a similar amount being secreted postpartum in the milk (approximately 800 ml milk/d with a total Ca content of approximately 6·25 mmol). This demand for Ca could be met from increased Ca in the diet, increased efficiency of Ca intestinal absorption, conservation of renal Ca, and/or increased bone Ca mobilization with a net loss of bone. Increased fractional absorption of Ca has been measured using a dual non-radioactive Ca isotope technique in women in the last trimester of pregnancy (Kent *et al.* 1991). At the same time, increased renal losses of Ca have been demonstrated to occur (King *et al.* 1992), but the Ca balance remains positive (King *et al.* 1992); perhaps due to high oestrogen

levels which protect bone mass (Lissner, 1991). During lactation there is an increased capacity to reabsorb Ca in the renal tubule (Kent *et al.* 1991), but fractional absorption of Ca measured at 20 weeks of lactation is no longer elevated (Kent *et al.* 1990). During lactation the concentration of oestrogen in blood is low compared with pregnancy, thus the bone mass is not protected to the same extent as that during pregnancy. Indeed, while biochemical markers indicate a normal rate of bone resorption in late pregnancy (36 weeks; Kent *et al.* 1991), lactation is associated with a net drain of Ca from the body as a result of a selective reduction in trabecular bone (Kent *et al.* 1990; Lissner *et al.* 1991; Sowers *et al.* 1993). This reduction is independent of parathyroid hormone and 1,25-dihydroxycholecalciferol since the serum concentrations of these hormones are similar in lactating and non-pregnant, non-lactating, premenopausal women (Kent *et al.* 1990).

Thus, at first glance lactation would appear to represent a risk for the future development of osteoporosis. The largest completed investigation of factors associated with fractures (Bauer *et al.* 1993) found, however, that breast-feeding did not meet even minimal criteria for independent risk of low appendicular bone mass. This interpretation is limited, however, by the fact that the three sites used for the determination of bone mass (distal radius, the mid radius and the calcaneus) and, thus, the risk of fractures, are unlikely to have included significant trabecular bone. On the other hand, Kent *et al.* (1990) demonstrated that during weaning there is a period of imbalance between a normal rate of bone resorption and an elevated rate of bone formation resulting in recovery of bone mass. These authors concluded that by 4–6 months after weaning, the fore-arm bone mineral density was the same as that in the control women. This study clearly has implications with respect to birth spacing. Further studies are required to address the questions of recovery both after prolonged breast-feeding and in older women (for whom the age of peak bone mass attainment has passed). With the current trend toward delayed child-bearing, it might be expected that the effect of lactation on bone mass may not be able to be completely reversed before menopause. On the other hand, Western women now have a lower number of children than their predecessors, so any cumulative insult due to multiple, close pregnancies and lactations is likely to be reduced.

The relationship of maternal Ca intake to the previously described biochemical adaptations to the demand for Ca is not well studied. Vegetarian lactating mothers with an intake of Ca of less than half that of control mothers had similar indices of Ca metabolism, apart from increased 1,25-dihydroxycholecalciferol concentrations (Specker *et al.* 1987). Based on results of an oral Ca tolerance test, Kent *et al.* (1991) have suggested that an oral Ca supplement may benefit breast-feeding women, by reducing lactation-related elevated rate of bone resorption and consequent loss of trabecular bone.

NUTRITION AND THE REGULATION OF MILK SYNTHESIS

Synthesis and production

The quantification of lactational performance is of fundamental importance to the study of the role of maternal nutrition in the regulation of the synthesis and secretion of milk in women. Theoretically, lactational performance can be governed by the mother's capacity to produce milk (milk synthesis) and/or the infant's ability to remove milk from the

breast (milk production). We have defined milk synthesis as the accumulation of milk within the mammary gland and milk production as the volume of milk removed from the mammary gland (Daly & Hartmann, 1995*b*). In this context the term 'milk yield' is used to specify milk production measured over a period of 24 h. The measurement of milk production and milk synthesis in women has been reviewed recently by Daly & Hartmann (1995*a*,*b*).

We have measured milk yield by test-weighing the mother with correction for evaporative water loss (Arthur *et al.* 1987) from 1 month to 7 months postpartum (Fig. 3). It is of interest to note that the mean values obtained are somewhat less than our earlier reports (Rattigan *et al.* 1981; Saint *et al.* 1984) due to the correction of the weight changes for evaporative water loss and the use of selection criteria which did not include a previous successful lactation. The range for milk yields obtained at 1 and 3 months postpartum (Fig. 3) represented more than a twofold difference, despite the fact that all the subjects were apparently well-nourished, were exclusively breast-feeding their infants and were assessed using the same method to measure yield. A twofold range also was observed by Dewey *et al.* (1991) who measured the 24 h milk intakes of infants over a period of 4 d. Clearly these differences in milk yields will contribute to variation in the energy balances for lactating women at similar stages of lactation.

It is apparent from the milk yield of mothers reported in a variety of studies (for review, see Daly & Hartmann, 1995*a*) involving breast-feeding twins, tandem breast-feeding, expression of milk for milk banks and the post breast-feeding expression of as much extra milk as possible, that the capacity of the breasts to synthesize milk can often exceed the usual requirements of the infant. Furthermore, the demand-fed infant consumes irregular quantities of milk from the breasts at irregular times throughout the day (Fig. 4). Although these feeding patterns are consistent with most recent studies which indicate that it is the baby's appetite which determines milk yield, the question as to how the breast regulated milk synthesis to meet this unpredictable demand remained a puzzle until recently. Clearly, the normal practice of regularly emptying the breasts in order to determine the energy output in milk would overestimate the energy cost of lactation. Thus, in determining the energy output in milk, it is critical to distinguish between the volume of milk that the breast is capable of synthesizing (milk synthesis) and the volume removed by the infant (milk production).

Our laboratory has developed methods to measure the short-term (between breast-feeds) rates of milk synthesis in women by determining the change in breast volume over time (Arthur *et al.* 1989). Since the decrease in breast volume during a breast-feed was highly correlated with the amount of milk consumed by the infant, it was assumed that the increase in breast volume between breast-feeds represented the volume of newly synthesized milk (for review, see Daly & Hartmann, 1995*b*). In studies using computerized breast measurement (CBM) we (Daly *et al.* 1992, 1993) have observed that the short-term rates of milk synthesis were highly variable both between breasts and between interfeed intervals. Also, that infants did not usually drain the breasts of available milk at a breast-feed and that there was a positive relationship between the degree of breast emptying and the short-term rates of milk synthesis. All these observations are consistent with the local control of milk synthesis within individual breasts (for review, see also Hartmann *et al.* 1995).

Experimentation with frequent milking in goats and cows resulted in the demonstration of a unilateral level of feedback control of milk synthesis which is independent of

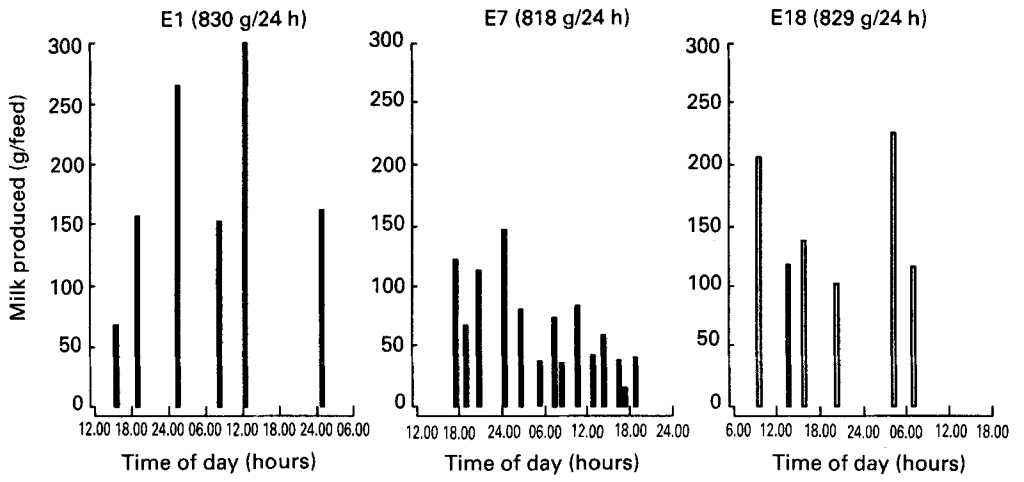

Fig. 4. Amount of milk consumed and feeding frequency of three infants over a 24 h period. The total amount of milk consumed during the 24 h period is shown in parentheses. This is the corrected 24 h yield of milk, since measurements were not made over exactly 24 h, calculated as

$$D = \frac{C24\,(B-1)}{AB},$$

where D is 24 h milk intake, A is the time (h) between first and last feeds, B is number of feeds and C is sum of individual milk intakes. Infants of E1 and E7 consumed milk from both breasts at each breast-feed, while infant of E18 consumed milk from only one breast at each feed.

the galactopoietic hormones (Wilde & Peaker, 1990), and is consistent with an autocrine control mechanism. Both *in vitro* and *in vivo* approaches have led to the hypothesis that an inhibitor is secreted and accumulates in milk over time. The mechanism of this feedback has not been elucidated in women; however, Prentice *et al.* (1989) have provided evidence for the existence of a protein (factor inhibiting lactation) in the whey fraction of human milk which can inhibit the secretion of lactose and casein in cell culture. These important findings not only provide a biochemical mechanism for the observed ability of women to adjust their milk synthesis to the amount of milk withdrawn by their infants, but also explain why substrate supply (i.e. maternal dietary intake) has little influence on their milk yield.

Milk fatty acids

While it is apparent that maternal nutrition has little influence on milk yield in lactating women, the composition of the mother's diet can influence the secretion of some of the important minor components of breast milk, particularly those that pass from blood to milk with little modification by the synthetic processes of the lactocyte.

The lipid fraction, in addition to its crucial contribution to the energy content of human milk, serves as a carrier of fat-soluble vitamins and certain fat-soluble hormones. Human milk triacylglycerols also provide the essential fatty acids, linoleic and α-linolenic acid, and the long-chain polyunsaturated fatty acids. The latter have a variety of specific

functions and, in particular, the proportions of arachidonic acid and docosahexaenoic acid are high in the membrane phospholipids of the grey matter of the brain and of the photoreceptor cells of the retina (Connor *et al.* 1992). Premature infants who were tube-fed mother's milk rather than formula had an IQ advantage of 8·3 points at 8 years of age (Lucas *et al.* 1992). Moreover, work from South Australia on the development of visual function in term infants (Gibson *et al.* 1993) highlights the importance of the long-chain polyunsaturated fatty acids in breast milk. There is considerable variation in the content of these fatty aids in human milk and it is likely that this is related to the variations in these fatty acids in the maternal diet (Van Beusekom *et al.* 1990). In this connection, the apparent redistribution of subcutaneous fat may serve to supply long-chain polyunsaturated fatty acids from maternal fat stores. Milk from vegans contains higher concentrations of linoleic and α-linolenic acid, and lower concentrations of docosahexaenoic acid than milk from omnivores (Sanders & Reddy, 1992), reflecting fatty acid composition of dietary triacylglycerols.

CONCLUSIONS

In reviewing maternal nutrition in pregnancy, James & Ralph (1991) concluded that, 'The new data emphasize the previous complete misunderstanding of energy balance in pregnancy which led to the suggestion that a woman should eat more than normal, i.e. "eat for two".' Similarly, during lactation many women do not need as much additional dietary energy as currently recommended. There is considerable variation between individual women both in the energy output in milk, even for exclusively breast-fed infants of a similar age, and in the energy mobilized from maternal stores for milk synthesis. Thus, recommendations for energy intake during lactation need to address the current weight status of the mother, and would serve individual women better if they were based on desired outcome for the mother's body weight after weaning rather than her current presumed energy requirements for milk production. It also may be appropriate to recommend a low-fat diet to encourage weight reduction (Rolls & Shide, 1992) and to maximize the *de novo* synthesis of fatty acids for milk triacylglycerols, but such a diet should ensure an adequate intake of long-chain polyunsaturated fatty acids and other nutrients such as protein, vitamins and Ca.

A perceived inability to produce milk is one of the most common reasons given by mothers for terminating breast-feeding. However, clinical experience suggests that it is either the infant's lack of access to the breast (e.g. by scheduled breast-feeding) or inappropriate suckling behaviour (poor positioning and attachment to the breast) that commonly lead to lactation failure. Therefore, it is crucial for mothers to be adequately instructed on the art of breast-feeding.

The promotion of the benefits of breast-feeding (National Health and Medical Research Council, 1992), together with an informed approach to the management of the art of breast-feeding (Phillips, 1991), has resulted in most mothers in Australia showing a greater commitment to breast-feeding now than was the case a generation ago. Unfortunately, a few mothers with both a strong desire to breast-feed and appropriate breast-feeding skills have low milk production and their babies fail to thrive (Gorman, 1994). Since in these mothers the 'low-milk syndrome' only can be attributed to the inability of the mother's breasts to synthesize sufficient milk, the metabolic cause(s) of this defect requires urgent investigation.

Studies conducted by the authors were supported by the National Health and Medical Research Council and Family Health International. The studies were approved by the Committee for Human Rights of The University of Western Australia.

REFERENCES

Allen, L. H., Ferris, A. M. & Pelto, G. H. (1986). Maternal factors affecting lactation. In *Human Lactation 2: Maternal and Environmental Factors*, pp. 51–60 [M. Hamosh and A. J. Goldman, editors]. New York: Plenum Press.

Arthur, P. G., Hartmann, P. E. & Smith, M. (1987). Measurement of milk intake of breast-fed infants. *Journal of Pediatric Gastroenterology and Nutrition* **6,** 758–763.

Arthur, P. G., Jones, T. J., Spruce, J. & Hartmann, P. E. (1989). Measuring short-term rates of milk synthesis in breast feeding mothers. *The Quarterly Journal of Experimental Physiology* **74,** 419–428.

Bauer, D. C., Browner, W. S., Cauley, J. A., Orwell, E. S., Scott, J. C., Black, D. M., Tao, J. L. & Cummings, S. R. (1993). Factors associated with appendicular bone mass in older women. *Annals of Internal Medicine* **118,** 657–665.

Brewer, M. M., Bates, M. R. & Vannoy, L. P. (1989). Postpartum changes in maternal weight and body fat depots in lactating versus non-lactating women. *American Journal of Clinical Nutrition* **49,** 259–265.

Connor, W. E., Neuringer, M. & Reisbick, J. (1992). Essential fatty acids: The importance of *n*-3 fatty acids in the retina and brain. *Nutrition Reviews* **50,** 21–29.

Daly, S. E. J. & Hartmann, P. E. (1995*a*). Infant demand and milk supply, Part 1: Infant demand and milk production in lactating women. *Journal of Human Lactation* **11,** 21–26.

Daly, S. E. J. & Hartmann, P. E. (1995*b*). Infant demand and milk supply, Part 2: The short-term control of milk synthesis in lactating women. *Journal of Human Lactation* **11,** 27–37.

Daly, S. E. J., Kent, J. C., Huynh, D. Q., Owens, R. A., Alexander, B. F., Ng, K. C. & Hartmann, P. E. (1992). The determination of short-term breast volume changes and the rate of synthesis of human milk using computerized breast measurement. *Experimental Physiology* **77,** 79–87.

Daly, S. E. J., Owens, R. A. & Hartmann, P. E. (1993). The short-term synthesis and infant-regulated removal of milk in lactating women. *Experimental Physiology* **78,** 209–220.

Dewey, K. G., Heinig, M. J. & Nommsen, L. A. (1993). Maternal weight-loss patterns during prolonged lactation. *American Journal of Clinical Nutrition* **58,** 162–166.

Dewey, K. G., Heinig, M. J., Nommsen, L. A. & Lonnerdal, B. (1991). Maternal versus infant factors related to breast milk intake and residual milk intake. *Pediatrics* **87,** 829–837.

Dugdale, A. E. & Eaton-Evans, J. (1989). The effect of lactation and other factors on postpartum changes in body weight and triceps skinfold thickness. *British Journal of Nutrition* **61,** 149–153.

Egger, G. (1992). The case for using waist to hip ratio measurements in routine medical checks. *Medical Journal of Australia* **156,** 280–284.

Gibson, R., Makrides, M., Neumann, M. & Simmer, K. (1993). Fatty acid status and visual function in term infants. In *Abstracts XV International Congress of Nutrition, Adelaide: 2*, p. 760. Adelaide: International Union of Nutritional Sciences.

Goldberg, G. R., Prentice, A. M., Coward, W. A., Davies, H. L., Murgatroyd, P. R., Sawyer, M. B., Ashford, J. & Black, A. E. (1991). Longitudinal assessment of the components of energy balance in well-nourished lactating women. *American Journal of Clinical Nutrition* **54,** 788–798.

Gorman, C. (1994). When breast-feeding fails. *Time Australia* **34,** 67.

Greenwood, M. R. C., Savard, R., West, D. B. & Kava, R. (1987). Energy metabolism and nutrient 'gating' in pregnancy and lactation. In *Recent Advances in Obesity Research*, vol. 5, pp. 258–623 [E. Berry, S. H. Blonheim, H. E. Eliahon and E. Shafrir, editors]. London: John Libbey and Co.

Hartmann, P. E., Atwood, C. S., Cox, D. B. & Daly, S. E. J. (1995). Endocrine and autocrine strategies for the control of lactation in women and sows. *Hannah Research Institute Conference on Intercellular Signalling in the Mammary Gland* [C. Wilde, D. H. Knight and M. Peaker, editors]. New York: Plenum Publishing Co. (In the Press).

Harzer, G. & Haschke, F. (1989). Micronutrients in human milk. In *Micronutrients in Milk and Milk-based Food Products*, pp. 146–148 [E. Renner, editor]. London and New York: Elsevier Applied Science.

Hytten, F. (1991). Weight gain in pregnancy. In *Clinical Physiology in Obstetrics*, 2nd ed., pp. 173–203 [F. Hytten and G. Chamberlain, editors]. Oxford: Blackwell Scientific Publications.

James, W. P. T. & Ralph, A. (1991). Maternal nutrition and pregnancy. In *Scientific Foundations of Obstetrics and Gynaecology*, p. 405 [E. Philipp, M. Setchell and J. Ginsburg, editors]. Oxford: Butterworth Heinemann.

Kent, G. N., Price, R. I., Gutteridge, D. H., Rosman, K. J., Smith, M., Allen, J. R., Hickling, C. J. & Blakeman, S. L. (1991). The efficiency of intestinal calcium absorption is increased in late pregnancy but not in established lactation. *Calcified Tissue International* **48,** 293–295.

Kent, G. N., Price, R. I., Gutteridge, D. H., Smith, M., Allen, J. R., Bhagat, C. I., Barnes, M. P., Hickling, C. J., Retallack, R. W., Wilson, S. G., Devlin, R. D., Davies, C. & St John, A. (1990). Human lactation: Forearm trabecular bone loss, increased bone turnover, renal conservation of calcium and inorganic phosphate with recovery of bone mass following weaning. *Journal of Bone and Mineral Research* **5,** 361–369.

King, J. C., Halloran, B. P., Huq, N., Diamond, T. & Buckendahl, P. E. (1992). Calcium metabolism during pregnancy and lactation. In *Mechanisms Regulating Lactation and Infant Nutrient Utilization*, pp. 129–146 [M. F. Picciano and B. Lönnerdal, editors]. New York: Wiley-Liss.

Lissner, L., Bengtsson, C. & Hasson, T. (1991). Bone mineral content in relation to lactation history in pre- and postmenopausal women. *Calcified Tissue International* **48,** 319–325.

Lucas, A., Morley, R., Cole, T. J., Lister, G. & Leeson-Payne, C. (1992). Breast milk and subsequent intelligence quotient in children born preterm. *Lancet* **339,** 1477–1481.

National Health and Medical Research Council (1992). *Dietary Guidelines for Australians*, pp. 84–92. Canberra: Australian Government Publishing Service.

National Heart Foundation of Australia (1990). *Risk Prevalence Study* no. 3, 1989. Canberra: National Heart Foundation of Australia and The Australian Institute of Health.

Ohlin, A. & Rossner, S. (1990). Maternal body weight development after pregnancy. *International Journal of Obesity* **14,** 159–173.

Phillips, V. (1991). *Successful Lactation*, 6th ed. Melbourne: NMAA.

Prentice, A., Addey, C. V. P. & Wilde, C. J. (1989). Evidence for local feedback control of human milk secretion. *Biochemical Society Transactions* **17,** 122.

Prentice, A., Paul, A., Prentice, A., Black, A., Cole, T. & Whitehead, R. (1986). Cross-cultural differences in lactational performance. In *Human Lactation 2: Maternal and Environmental Factors*, pp. 13–44 [M. Hamosh and A. S. Goldman, editors]. New York: Plenum Press.

Rattigan, S., Ghisalberti, A. V. & Hartmann, P. E. (1981). Breast milk production in Australian women. *British Journal of Nutrition* **45,** 243–249.

Rolls, B. J. & Shide, D. J. (1992). The influence of dietary fat and food intake on body weight. *Nutrition Reviews* **50,** 283–290.

Rookus, M. A., Rokebrand, P., Burema, J. & Deurenburg, P. (1987). The effect of pregnancy on BMI nine months postpartum in 49 females. *International Journal of Obesity* **11,** 609–618.

Saint, L., Smith, M. & Hartmann, P. E. (1984). The yield and nutrient content of colostrum and milk of women from giving birth to 1 month post-partum. *British Journal of Nutrition* **52,** 87–95.

Sanders, T. A. & Reddy, S. (1992). The influence of a vegetarian diet on the fatty acid composition of human milk and the essential fatty acid status of the infant. *Journal of Pediatrics* **120,** S71–S77.

Sheldon, J. H. (1949). Maternal obesity. *Lancet* **12,** 869–873.

Sherriff, J. L. & Hartmann, P. E. (1994). Changes in maternal anthropometry during lactation. *2nd National Conference of the Australian Lactation Consultants' Association*, Adelaide W15. Adelaide: South Australian Women's Association.

Short, R. V., Lewis, P. R., Renfree, M. B. & Shaw, G. (1991). Contraceptive effects of extended lactational amenorrhoea: beyond the Bellagio Consensus. *Lancet* **337,** 715–717.

Sowers, M. F., Corton, G., Shapiro, B., Jannausch, M. L., Crutchfield, M., Smith, M. L., Randolph, J. F. & Hollos, B. (1993). Changes in bone density with lactation. *Journal of the American Medical Association* **269,** 3130–3135.

Specker, B. L., Tsang, R. C., Ho, M. & Miller, D. (1987). Effect of a vegetarian diet on serum 1,25-dihydroxyvitamin D concentrations during lactation. *Obstetrics and Gynaecology* **70,** 870–874.

Van Beusekom, C., Martini, I. A., Rutgers, H. M., Boersma, E. R. & Muskiet, F. A. (1990). A carbohydrate-rich diet not only leads to incorporation of medium-chain fatty acids (6:0–14:0) in milk triglycerides but also in each milk-phospholipid subclass. *American Journal of Clinical Nutrition* **52,** 326–334.

Wilde, C. J. & Peaker, M. (1990). Autocrine control in milk secretion. *Journal of Agricultural Science* **114,** 235–238.

Printed in Great Britain