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THE INFLUENCE OF MATERNAL NUTRITION ON LACTATION

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CONTENTS

INTRODUCTION

The first objective of this review is to examine the effects of general over- or undernutrition on lactational performance. There are many ways to evaluate lactational performance, and for this review the focus is on infant milk and nutrient intake and infant growth. In each section, data from studies in experimental species are used to develop hypotheses that are then explored in the data from studies of lactating women. The second objective of this review is to go beyond the recent book, *Nutrition During Lactation* (18), to examine the effect of maternal nutritional status on lactational performance within a

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framework for evaluating causality. A biologically based conceptual model is developed to guide this analysis.

CONCEPTUAL FRAMEWORK

Important factors in the relationship between maternal dietary intake and various measures of lactational performance are identified in Figure 1. To simplify the diagram, I have omitted any possible effects that a change in maternal dietary intake may have on either maternal physical activity or the thermic effect of food or maternal and infant illness. Also omitted are the clear effects of maternal dietary intake before and during pregnancy on maternal adipose tissue and nutrient stores at parturition, lactational capacity, and infant size at birth. This diagram assumes that infants are exclusively breast-fed because the effect of direct supplementation of the infant, which would increase infant growth and decrease milk intake, is omitted. The diagram is both more specific and inclusive than the causal sequence (maternal nutritional status → lactational performance → infant growth) that has traditionally guided research in this field.

It is important to distinguish between maternal dietary intake (what the mother actually consumes) and nutritional status (manifestations of that consumption, such as blood nutrient concentrations or measures of body composition). In general, interventions have selected lactating women based on

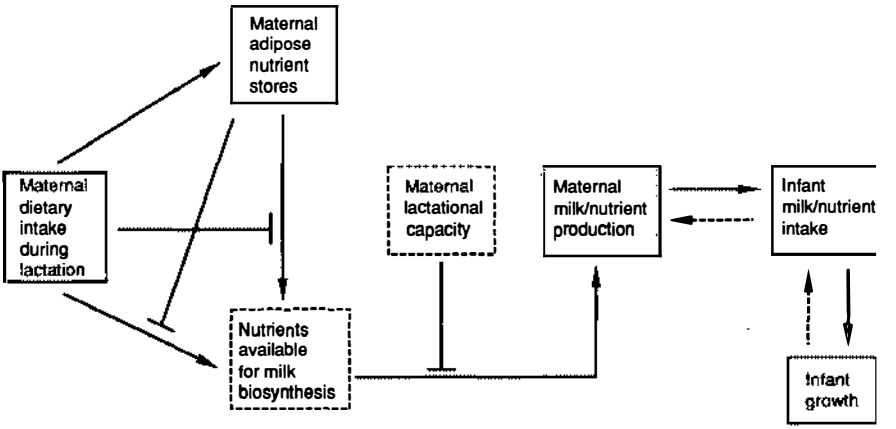

Figure 1 Relationships among maternal dietary intake, maternal nutritional status, milk production, and infant growth at any one time during lactation. Shown in *dotted boxes* are two important, but usually unmeasured, variables: nutrients available for milk biosynthesis and maternal lactational capacity (see text). *Solid lines with arrowheads* denote direct transfer of nutrients or energy-producing compounds. *Dotted line with arrowheads* denote the direct influence of one factor on another that does not occur via nutrient flux. *Solid lines ending with a bar* denote possible modification of effect on the *arrow* on which they abut.

some measure of nutritional status and then have sought to change dietary intake. The proportion of ingested nutrients partitioned for milk biosynthesis may depend on maternal nutrient stores. Nutrient stores also may be mobilized to contribute to the nutrients available for milk biosynthesis, and it is likely that the extent of nutrient mobilization is conditioned upon dietary intake. In experimental species, nutrients available for milk biosynthesis can be calculated from values for blood nutrient concentrations and from blood flow to the mammary glands.

The amount of milk a woman actually produces is limited by her lactational capacity, defined here as a woman's ability to produce milk at any given time. Lactational capacity is probably a function of genetic heritage, age (17), and parity (16) as well as breast enlargement during pregnancy (16) and nutritional history (42). It can respond to changing circumstances (improved dietary intake, increased infant demand) and, thus, might have a different value at some later time during lactation. Lactational capacity has usually been approximated by measuring milk production (infant milk intake plus residual milk). For well-nourished women, lactational capacity is likely to be greater than milk production, which, in turn, is usually greater than short-term infant milk intake [by 100 ml per day in one recent study (8)]. For poorly nourished women, lactational capacity, milk production, and infant milk intake are expected to (25) have more similar values. In the single study (25) that has examined this proposition, it was, in fact, not the case.

The amount of milk a woman actually produces also responds to infant milk intake, which itself responds to infant size and growth rate (infant vigor, suckling stimulus). In recent years, studies have emphasized that the infant's demand determines milk production via the prolactin response to suckling (8, 10, 23, 50). However, this concept is only valid when maternal lactational capacity is not limiting for milk production.

This model leads to the prediction that, given adequate infant demand, lactational capacity would not be limiting in obese women, who are likely to have sufficient dietary intake or nutrient stores to maintain lactation. This model also leads to the prediction that, among undernourished women, milk production might be limited by either lactational capacity or the availability of substrates for milk biosynthesis or both, even when infant demand is adequate.

EFFECT OF MATERNAL OVERNUTRITION ON LACTATIONAL PERFORMANCE

Several different experimental approaches have been used in animals and provide useful information about the effects of overnutrition on lactational performance (milk volume and composition, growth of the suckling young). Only very limited information is available from human subjects.

Studies in Experimental Animals

EFFECTS OF OVERFEEDING DURING LACTATION AMONG OBESE ANIMALS In rats, maternal obesity can have negative effects on the pups. Forty-four per cent of the obese rats studied by Rolls et al (40) were unable to maintain their litters after 6 days of life. These rat dams were made obese by consumption of the "cafeteria diet" (stock diet plus access to salami, crackers, and cookies) before conception and during pregnancy and lactation. The pups who survived were significantly smaller than those of the controls. Similar findings in rats have been reported by others (51) who used a protein-supplemented, high-fat diet to produce maternal obesity before conception.

These obese animals weighed at least 30% more at conception than ad libitum–fed controls (41). In these investigations, overweight is assumed to be overfat: in general, data on body composition were not obtained. Obese dams did not develop the hyperphagia characteristic of lactation and, as a result, consumed less energy than did control animals (39). Among those obese dams whose pups survived until weaning, the heavier the mothers were at delivery, the more weight they lost during lactation. Although the weight lost probably consisted mostly of fat (48), the amount of maternal weight lost during lactation was unrelated to the weight of the pups at weaning (39).

The immediate predictors of pup growth in this model have been investigated. Studies have shown that obese dams produce 25% less milk (35), but this milk had 26% more energy (37) because it contained nearly double the usual proportion of lipid. The milk of the obese dams also contained 21% lower protein and 10% lower lactose concentrations as well as a reduced proportion of medium-chain fatty acids and an increased proportion of long-chain fatty acids (36). These changes in milk composition resulted primarily from cafeteria feeding during lactation (27). The higher fat concentration also may have resulted from the increased rate of mobilization of adipose tissue stores during lactation that is characteristic of obese animals (38). That the pups grew less well with an apparently adequate energy intake suggests that the combination of the other changes in milk composition with the reduction in volume resulted in a total nutrient intake by the pups that was limiting for their growth.

The effect of obesity on lactational performance as measured by pup weight also has been studied in golden hamsters. They were overfed with a high-fat diet (stock diet plus access to sunflower seeds and a 1:3 mixture of peanut butter:vegetable shortening) before mating, during pregnancy, and during lactation (11). The overfed hamsters weighed about 30% more at delivery and lost weight at the same rate during lactation as controls and, thus, ended lactation with the same difference in body weight that had existed at delivery. In contrast to observations in rats, no effect of maternal body weight on litter growth or survival was observed.

The cafeteria diet has a low protein-energy ratio and, from the available data, diet composition cannot be excluded as the cause of poor lactational performance of the rats or the difference in results between rats and hamsters. Investigation of the effects on lactational performance of other experimental paradigms for producing obesity in laboratory species is warranted. Further exploration of this relationship is of public health interest because of the high proportion of American women of reproductive age who are overweight or obese (52) and national health goals to increase breast-feeding rates (7).

EFFECTS OF OVERFEEDING DURING LACTATION AMONG LEAN ANIMALS Conflicting results about lactational performance have been obtained from studies of the effects of supplementing lean (i.e. control or nonobese) rats during lactation. Investigators have employed cafeteria feeding (37, 41) or a purified diet supplemented with a homogenous mixture of eggs and oil (34). In the studies that employed cafeteria feeding, the supplemented rats increased their energy intake compared to controls fed a stock diet (40, 41), but their pups did not grow as well as those of the controls (41). These supplemented rats produced milk with a lower protein concentration and a higher fat concentration (resulting in a higher energy density) than the milk of control rats (37). Their milk contained a lower proportion of medium-chain and a higher proportion of long-chain fatty acids than did the milk of control rats. The milk production of lean rats fed the cafeteria diet only during lactation has not been reported.

In contrast, when lean rats were fed a purified diet supplemented with eggs and oil, they gained weight, reduced their fat mobilization during lactation, produced more milk, and had larger pups than control rats (34). Milk composition was not examined in this experiment. The authors attributed these positive findings to the higher protein-energy ratio of their supplement compared to the supplemental foods used in cafeteria feeding. These results (34) indicate that ad libitum–fed lactating animals are capable of increasing their food intake with beneficial effects on lactational performance. These results also suggest that, in the amounts usually consumed, the stock diet does not maximize either milk production or litter growth. Thus, a reexamination of the nutrient requirements of the rat and the possible formulation of a more appropriate stock diet for use during lactation merits attention.

EFFECTS OF UNDERFEEDING OBESE ANIMALS DURING LACTATION When obese rats were switched at parturition from cafeteria feeding to a stock diet, they consumed less energy and lost about twice as much weight as obese rats maintained on the cafeteria diet (41). Their milk production has not been reported. Milk protein concentrations were higher than those of obese rats continued on cafeteria feeding; fat concentration and energy density did not differ between these two groups (37). Their pups grew much less well than

those of the obese rats maintained on the cafeteria diet who, as described above, grew less well than lean controls fed the stock diet (41). Thus, although these animals had free access to a theoretically adequate diet during lactation as well as adequate adipose tissue reserves at parturition (which they mobilized during lactation), pup growth remained poor. This finding was not attributable to low milk protein concentration or energy density, but the role of milk production in causing the poor pup growth remains unknown.

This experimental approach represents an animal model for dieting during lactation, a subject of concern to women who were overweight before conception or who gained excessive amounts of weight during pregnancy. As such, these data tell a cautionary tale. However, the poor growth of the pups of the dieting rats may result from intrauterine factors attributable to the low protein-energy ratio of the cafeteria diet fed during pregnancy or may result from inadequate dietary intake during lactation. Again, other means of producing obesity might be informative and deserve exploration.

Studies in Women

In contrast to the interest in and concern about obesity and the outcome of pregnancy among women, little attention has been given to any possible relation between preexisting or pregnancy-related obesity and any aspect of lactation.

Among well-nourished women studied longitudinally between 1 and 4 months postpartum, dietary intake accounted for 13% of the variability in infant milk intake (4). Anthropometric indicators of nutritional status were not associated with lactational performance in these women. Among well-nourished women studied longitudinally from 3 to 12 months postpartum (26), some associations were found between maternal nutritional status and milk composition. These relationships were stronger at later stages of lactation.

Butte & Garza (3) commented that their failure to detect an association between maternal nutritional status and lactational performance might have resulted from (*a*) the relative homogeneity of their study population in body size, (*b*) the relatively greater importance of diet compared to tissue reserves for maintaining energy balance during lactation, (*c*) variability in energy needs for maintenance and activity, or (*d*) imprecision in the anthropometric measurements used to estimate body composition. These limitations also apply to the other longitudinal studies of well-nourished women that have been conducted.

The effect of short-term caloric restriction has been investigated in one group of well-nourished women 6–24 weeks after delivery (47). Women who chose to restrict their dietary intake for one week were somewhat heavier and had gained more weight during pregnancy than those who chose not to do so.

During the period of restricted intake, milk volume and composition were maintained, but in the week afterward, infant milk intake and weight gain were less than in the pre-dieting period. The negative effects of dieting were greater among the mothers who restricted their intake to < 1500 kcal/day.

In summary, the available data from human subjects have not included enough overweight or obese subjects to examine overnutrition as a separate predictor of lactational performance. Within the range of maternal body weight that has been examined, infant milk intake is minimally responsive to variations in dietary intake or anthropometric indicators of maternal nutritional status.

EFFECT OF MATERNAL UNDERNUTRITION ON LACTATIONAL PERFORMANCE

Much more extensive information exists about the effects of various forms of undernutrition on lactational performance from both experimental species and human subjects. In animal models, the experimental approaches used parallel those described above for studying the effects of overnutrition. In human subjects, data are available from both observational and experimental studies, and the latter have included both nonrandomized and randomized designs. Emphasis is placed on the last of these research designs.

Studies in Experimental Animals

It is well-known that various dietary regimens (including chronically or acutely restricted food intake, low protein or carbohydrate intake, and poor protein quality) compromise lactational performance (milk volume and composition, pup growth) in rats (15, 19, 22, 44–46, 50). Of particular interest are the conditions under which these negative effects occur and to what extent they can be reversed by improving dietary intake during lactation. In this section, data are selected to focus on these two issues because they are the key to understanding the data from human subjects. Emphasis is placed on animal models of chronic food restriction because they are more applicable to the nutritional stress encountered by lactating women living under poor circumstances than are models of particular nutrient deficits. In this context, chronic food restriction refers to animals fed limited amounts of a nutritious diet beginning before conception and continuing until peak lactation (day 14). Acute food restriction refers to animals fed limited amounts of a nutritious diet for shorter periods (usually 7–14 days during lactation).

The extent of compromise in lactational performance depends on the degree and duration of food restriction. Litters of rats subjected to acute (15, 21) or chronic (21, 43, 50, 53) food restriction ingest less milk. This occurs at chronic maternal dietary intakes of 70% or less of ad libitum intake or at acute

dietary intakes of 60% or less of ad libitum intake. Chronic dietary restriction to 70% (43) or 50% (21) of ad libitum intake affected milk composition: protein and fat concentrations rose and lactose concentration fell; caloric density increased. No reduction in pup nutrient intake (nitrogen or energy) was observed when dams were chronically restricted to 70% of ad libitum intake (43), but at 50% of ad libitum intake, pup caloric intake was only 32% of control values (21). Pup growth was reduced at chronic maternal dietary intakes of 75% or less of ad libitum intake (15, 43, 53).

The effects of acute food restriction have also been modelled in baboons (33). Infant milk intake was not significantly reduced in animals fed 80% of ad libitum intake between 2 and 10 weeks postpartum but was 37% lower in those fed 60% of ad libitum intake. Milk composition was not affected by either level of dietary restriction. Infants of baboons in both food-restricted groups grew less well than those of baboons in the control group. Baboons in the 60% group lost four times as much weight during the experimental period as the other groups. To protect milk production, animals compensated for their reduced intake by increasing the efficiency of energy utilization by 17–25%, principally by reducing energy expenditure.

The effect of giving food supplements to undernourished lactating women has been modelled in rats by permitting chronically food-restricted dams to eat ad libitum from delivery until peak lactation (21, 32, 50). Milk intake by the litters of these dams increased dramatically compared to that of litters of rats whose food intake remained restricted and equaled that of litters of ad libitum-fed animals (21). Milk composition values for the refed animals were intermediate between those of chronically food-restricted rats and ad libitum-fed controls. As a result, the total caloric intake of the pups of the refed dams was the highest of these dietary treatment groups. Although the growth rate of the pups of the refed dams initially lagged that of control pups, by peak lactation their growth rate was the same or higher. Thus, provision of adequate dietary intake during lactation reversed the negative effects of prior chronic undernutrition on lactational performance.

The effect of refeeding on maternal nutritional status was also dramatic, but less complete. Refed dams gained weight during lactation and increased their proportion of body fat compared to that of chronically food-restricted rats (20); however, the refed rats remained significantly smaller, lighter, and leaner than control animals. Food consumption by the refed dams was similar to that of ad libitum-fed controls (49). A dietary intake similar to that of the controls represents a large food supplement because the refed rats weighed only 56% as much as the ad libitum–fed rats at parturition.

Important to an overall interpretation of the studies from rats is an understanding of the condition of the pups at birth. Chronically food-restricted dams deliver fewer and smaller pups than ad libitum–fed controls (2, 20, 31).

In addition, food restriction during pregnancy reduces the weight and compromises the development of the mammary gland (42). Thus, at parturition both maternal lactational capacity and the suckling stimulus of the pups is lower in chronically undernourished rats than in controls.

These studies in animal models show that maternal nutritional status at delivery as well as dietary intake during lactation influence lactational performance separately and jointly. Furthermore, these data show that the effects of prior maternal food restriction on milk and nutrient intake by the nursing litter and on litter growth are reversible with ad libitum feeding. These results from animal models are likely to be more dramatic than those that could be obtained from human subjects because: (*a*) some of the animals have been subjected to more severe undernutrition than is common among lactating women and, thus, the animals have more potential to respond to improvements in their dietary intake, and (*b*) their response is not constrained by the confounding factors present in studies of lactating women (e.g. direct supplementation of the nursing infant, sharing of the supplement with other family members, or changes in labor patterns).

Studies in Women

Studies of the effects of general undernutrition on women's lactational performance were reviewed recently (18). Evidence from observational studies is mixed. Although women living under poor circumstances are reported to eat less than women with better living conditions, they don't necessarily produce less milk (29). However, among such women, seasonal food shortages are associated with decreases in infant milk intake (27).

Data are available from two types of food supplementation trials: those with or without a randomized design. For reasons related to the study design and methods employed, the data from the three nonrandomized interventions that have provided food supplements to free-living women in rural areas of Mexico (5), the Gambia (28, 30), and India (13) are difficult to interpret (18).

Lessons learned from these early experimental studies include the importance of: (*a*) studying women who, because of their own poor nutritional status or inadequate current dietary intake, are likely to benefit from the supplement by improving their own health or nutritional status or by improving the nutrient intake or growth of their infants; (*b*) making this assessment at the time of peak lactational stress; (*c*) excluding the effect of concurrent supplementation of the infant (which is likely to decrease the infant's demand for milk and, thereby, milk production); and (*d*) assessing both milk volume and composition because maternal supplementation may change either or both factors and is likely to act through their product, the infant's total nutrient intake, to improve infant growth.

More recently, two randomized studies of the effect of food supplementa-

tion on lactational performance have been completed, one in Burma (24) and the other in Guatemala (14). Both experiments have designs that are suitable for causal inference and report that milk intake increased among infants of the women who received the food supplements. However, some aspects of both studies remain unexplained.

The study in Burma (24) included 21 women, 1–4 months postpartum, who were selected because their weight-for-height was <80% of an international standard. The supplement, a curry of animal protein cooked in oil, was home-delivered to the experimental group twice daily for 14 days. Supplemented women increased their dietary intakes, which were not low initially (2425 kcal per day), by a net of 900 kcal and 39 g of protein.

Infant milk intake was unchanged among control women, who received no treatment at all, but increased by 102 ml per day among supplemented women. No effect of the supplement on the protein concentration of breast milk was noted. Despite their increase in milk intake, infants of the supplemented mothers did not gain significantly more weight during the experimental period than infants of control mothers, which the authors attributed to the short duration of the experiment. The supplement also increased maternal body fat content as assessed from the sum of skinfold thicknesses at four sites.

This study is important because it shows that both maternal nutritional status and infant milk intake can be improved simultaneously. However, various aspects of this brief report are puzzling. One is how such a large net increase in dietary intake was achieved by women whose home dietary intakes were as high as these. Also puzzling is why women with these reported home dietary intakes were nonetheless so underweight. The usual activity pattern of these women was not discussed by the authors.

The study in Guatemala has only been reported as an abstract (14). It included 111 women who were selected because they had a low calf-circumference value during the last trimester of pregnancy. The subjects received either a low-energy (140 kcal per day) or a high-energy (500 kcal per day) supplement in the form of cookies delivered to their homes on weekdays for 20 weeks (from week 5 to week 25 of lactation).

In this study, infant milk intake was 47 g per day higher in the high-energy than the low-energy group at week 20 of lactation. A higher proportion of infants in the high-energy group was exclusively breast-fed at weeks 10 and 20 of lactation. This study is the first to document a causal relationship between maternal food supplementation and breast-feeding pattern. However, a complete understanding of this experiment must await full publication of its results.

In interpreting this study, one must remember that women in the low-energy group received a supplement. Therefore, they could have improved

their lactational performance as well as their own nutritional status during the intervention period compared to unsupplemented women in the community. Because such women were not studied by González-Cossío and her colleagues (14), the absolute degree to which the low-energy group may have benefited will remain unknown. However, it is likely that the difference between the two treated groups underestimates the full impact of food supplementation in this population.

A crude comparison across studies of the effect of food supplementation on infant milk intake can be constructed from the available data. In Burma (24), daily milk intake increased by about 12 g per 100 additional kcal. In Guatemala (14), this increase was very similar: 13 g per 100 kcal from supplement in the high-energy groups compared to the low-energy group.

The data from the study in Burma (24) reveal benefits of supplementation for the mother: the increase in maternal weight did not reach statistical significance, but sum of skinfold thickness did. Results from the nonrandomized study conducted in The Gambia are congruent: supplemented women gained weight, were less likely to report being ill (28), and were said to perform more agricultural labor (6).

The positive effects of food supplementation on infant milk intake found in the two randomized intervention trials support a causal link of maternal dietary intake → infant milk intake (Figure 1). These positive effects contrast sharply with mixed effects found in the nonrandomized studies. This suggests that confounding factors may have compromised the ability of the nonrandomized studies to demonstrate an impact of food supplementation on infant milk intake. Finding effects is likely to be conditional on conducting impact evaluations in populations with the potential to benefit from supplementation. Finding effects also is likely to depend on selecting an amount and type of supplement that both addresses the underlying nutritional deficits in the lactating women studied and will be accepted by them.

To evaluate the link of infant milk/nutrient intake → infant growth in this causal sequence requires demonstrable improvements in the infant's total nutrient intake (Figure 1). There is every reason to expect that infants whose nutrient intake increases will grow more rapidly (12). The data now available show that any improvements in infant growth that result from maternal food supplementation during lactation are too small or too variable to be detected with the number of subjects studied. This may be because improvements in infant nutrient intake have been too small to produce discernibly better growth during the relatively short intervals in which growth has been evaluated.

It is necessary to establish association, time order, and direction for a full evaluation of a possible causal sequence. Direction is especially problematical for the link infant milk/nutrient intake → infant growth. It is likely that the effect of improving maternal dietary intake operates *first* in this direction.

However, because milk production responds to the suckling stimulus (10), once milk production begins to improve and the baby grows more rapidly, the infant's demand will likely cause *further* increases in milk production as long as maternal lactational capacity is not limiting for milk production. This direction of action cannot be excluded from the available data and deserves further study.

As illustrated in Figure 1, the availability of adequate nutrients for milk biosynthesis is a necessary but not sufficient condition for increasing milk production: milk production will increase only with adequate infant demand. Similarly, adequate infant demand is a necessary but not sufficient condition for increasing milk production: milk production will increase only with adequate lactational capacity. Thus, for a valid test of the effect of improving maternal dietary intake on infant milk intake and growth, factors constraining lactational capacity must be removed *and* the infant must be capable of growing better in response to increased milk intake. The maximum benefit to the infant of maternal food supplementation will be achieved when the infant's response to increased milk intake elicits further improvements in milk intake. Factors that constrain this improvement in milk intake (e.g. a premature infant with low suckling vigor or fewer daily breast-feedings because better-fed mothers are away from home for longer hours to perform more agricultural work) reduce the possibility that improving maternal dietary intake will improve infant growth directly via increased milk intake.

CONCLUSIONS

Although cafeteria feeding may represent an accurate animal model for producing obesity in human subjects, the negative effects of long- or short-term use of this diet on lactational performance may relate to its low protein-energy ratio. Thus, other animal models of obesity should be investigated. The proportion of women of childbearing age in the United States and other developed countries who are overweight or obese indicates that the effect of maternal overnutrition on lactational performance deserves more systematic study.

In experimental animals, chronic undernutrition has deleterious effects on milk and nutrient intake of the young as well as on their growth. Improving previously poor maternal dietary intake during lactation increases milk intake by the young and corrects their growth deficit; it also improves maternal nutritional status, but only incompletely.

The newer data from randomized intervention trials among undernourished women show that improving maternal dietary intake increases infant milk intake. These data are less persuasive in demonstrating a direct causal effect of maternal food supplementation on infant growth. The finding from one

study that food supplementation increased the proportion of exclusively breast-fed infants has important, positive implications for infant health and justifies further experimental studies on this subject.

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