Randomized trial of the short-term effects of dieting compared with dieting plus aerobic exercise on lactation performance[1–3]

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ABSTRACT

Background: Limiting postpartum weight retention is important for preventing adult obesity, but the effect of weight loss on lactation has not been studied adequately.

Objective: We evaluated whether weight loss by dieting, with or without aerobic exercise, adversely affects lactation performance.

Design: At 12 ± 4 wk postpartum, exclusively breast-feeding women were randomly assigned for 11 d to a diet group (35% energy deficit; n = 22), a diet plus exercise group (35% net energy deficit; n = 22), or a control group (n = 23). Milk volume, composition, and energy output; maternal weight, body composition, and plasma prolactin concentration; and infant weight were measured before and after the intervention.

Results: Weight loss averaged 1.9, 1.6, and 0.2 kg in the diet, diet + exercise, and control groups, respectively ($P < 0.0001$) and was composed of 67% fat in the diet group and nearly 100% fat in the diet + exercise group. Change in milk volume, composition, and energy output and infant weight did not differ significantly among groups. However, there was a significant interaction between group and baseline percentage body fat: in the diet group only, milk energy output increased in fatter women and decreased in leaner women. The plasma prolactin concentration was higher in the diet and diet + exercise groups than in the control group.

Conclusions: Short-term weight loss (≈1 kg/wk) through a combination of dieting and aerobic exercise appears safe for breast-feeding mothers and is preferable to weight loss achieved primarily by dieting because the latter reduces maternal lean body mass. Longer-term studies are needed to confirm these findings. *Am J Clin Nutr* 1999;69:959–67.

KEY WORDS Adipose tissue mobilization, body composition, breast milk, energy expenditure, energy intake, aerobic exercise, lactation, prolactin, obesity, weight loss, women

INTRODUCTION

Evidence that excessive postpartum weight retention contributes to the development of obesity (1, 2) underscores the need for guidelines for weight loss during lactation. The Institute of Medicine has stated that for overweight lactating women, weight loss of up to 0.5 kg/wk appears safe (3), but the effects of more rapid weight loss and how it is best achieved have not been evaluated.

In the general population, weight loss by aerobic exercise in combination with dietary energy restriction promotes fat utilization and prevents the loss of lean tissue and the decrease in metabolic rate that normally accompany dietary restriction alone (4–7). We reported previously that aerobic exercise (without dietary restriction) has no effect on breast-milk volume or composition in exclusively breast-feeding women (8–10). A 12-wk exercise program did not adversely affect lactation, but also did not enhance weight loss beyond that of the nonexercising control group (9, 10).

There are few experimental studies on weight loss during lactation. Consistent with established guidelines, Dusdieker et al (11) reported no effect of dietary restriction on breast-milk volume or composition among women who lost an average of 0.48 kg/wk for 10 wk. However, the study lacked a control group and 11 of the 33 women who originally enrolled withdrew. Strode et al (12) compared the lactation performance of women who voluntarily reduced their energy intake for 1 wk (n = 14) with that of a control group who did not change their intake (n = 8). Those who consumed ≥6.28 MJ/d (1500 kcal/d) had no change in milk volume, but those who consumed <6.28 MJ/d experienced a decrease in milk volume. Although methodologic limitations in both of these studies make it difficult to draw definitive conclusions (13), their results imply that a moderate energy deficit has no effect, but a more severe energy deficit may impair milk production. Experimental data in lactating baboons support this hypothesis (14): milk volume declined significantly when energy intake was reduced by 40% during a 10-wk period, but not when the reduction was only 20%.

Our objectives were to *1*) determine the effects of a relatively large energy deficit (35%) on the lactation performance of healthy, well-nourished women, and *2*) compare the effects of dietary restriction alone with dietary restriction combined with aerobic exercise.

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SUBJECTS AND METHODS

Study design

Exclusively breast-feeding women between 8 and 16 wk postpartum were recruited through local physicians' offices, childbirth classes, and letters to new parents. Subjects were eligible if they had no chronic illnesses; were not taking medication regularly; were nonsmokers; had delivered a single, healthy, term infant; and were willing to exercise 3 d/wk for ≥1 mo before the intervention (to prepare physically in case they were assigned to the group with intensive exercise).

During a 10–12-d baseline period, dietary intake, resting metabolic rate (RMR), energy expenditure, maximal oxygen consumption, body composition, milk volume and composition, and plasma prolactin concentrations were measured. Subjects were then randomly assigned to 1 of 3 groups for 11 d: *1*) a diet group (35% energy deficit), *2*) a diet plus exercise group (diet + exercise: 35% net energy deficit, 60% by dietary restriction and 40% by additional exercise), and *3*) a control group. The duration of the intervention was chosen to be longer than our previous 7-d study (12), but not so long as to compromise feasibility (ie, maintaining compliance was a concern) or pose a serious risk to the infants if there were any adverse effects on lactation. Random assignment of individuals was computer based and used the Moses-Oakford algorithm (15) with variable block size. During the intervention, body composition, milk volume and composition, and plasma prolactin measurements were repeated. Because of the study's measurement and exercise requirements, part-time child care of ≤28 h for the control and diet groups and 46 h for the diet + exercise group was offered. Subjects were encouraged to continue breast-feeding on demand throughout the study. If subjects in the diet or diet + exercise group experienced a decrease in milk volume and wished to withdraw from the intervention, they were free to do so, but measurements were continued whenever possible. To detect a group difference of ≥251 kJ/d (60 kcal/d, ≈10%) for the change in milk energy output during the intervention ($\alpha = 0.05$, $\beta = 0.20$), the necessary sample size was 23 per group on the basis of an SD of ±264 kJ/d for the change in milk energy output in our previous study (9). The protocol was approved by the Human Subjects Review Committee at the University of California, Davis.

Dietary intake

For 4 d during baseline, subjects weighed and recorded all food items consumed to the nearest 2 g (Lume-o-gram Lo-Pro; OHAUS, Florham Park, NJ). Nutrient intake was calculated by using the FOOD PROCESSOR II computer program (ESHA Research, Salem, OR), food-composition tables, and data supplied by food manufacturers.

Energy expenditure, maximal heart rate, and oxygen consumption

On 2 mornings during baseline, RMR was determined with a portable metabolic cart (CPX/Max/D; MedGraphics, Minneapolis) by using standard procedures described previously (9). Oxygen consumption and carbon dioxide production during steady state (usually the last 10 min of the 30-min measurement period) were converted to RMR by using Weir's formula (16) and normalized to a 24-h period by multiplying by 1440 min/d. The 2-d mean (±SD) RMR was used to calculate daily energy expenditure (CV: 3.8 ± 3.5%).

During the baseline period, subjects kept detailed activity records for 4 d by talking into a tape recorder every 15–30 min, and energy expenditure during sleep and daily activity was determined by factorial procedures as described previously (8, 17). Except for exercise (*see* below), tables compiled by Ainsworth et al (18) were used to estimate the energy cost of activities; energy expenditure during sleep was assumed to be equal to the RMR (19, 20).

Energy expended in exercise was determined by monitoring the heart rate as described elsewhere (21). First, the relation between subjects' heart rates and oxygen consumption was determined by linear regression of data collected while the subjects walked on a treadmill at 4 speeds and grades: 3.2 km/h (2 mph), 0% grade; 4.8 km/h (3 mph), 0% grade; 4.8 km/h, 3% grade; and 4.8 km/h, 6% grade. These measurements were carried out on the last morning of the baseline period and were repeated on the morning after the intervention period (day 12). Subjects in all 3 groups wore a portable heart rate monitor (Vantage XL; Polar-CIC, Port Washington, NY) during all exercise sessions in the baseline and intervention periods. Energy expenditure during exercise in the baseline period was estimated from each individual's baseline regression equation, whereas that during the intervention was estimated from each individual's average of the baseline and intervention equations (21). Subjects performed graded treadmill exercise to volitional fatigue according to standard procedures described previously (8).

Anthropometry and body composition

Height and weight were measured to the nearest 0.5 cm and 1 g, respectively. Body volume was measured by hydrostatic weighing in the first 23 subjects and by air-displacement plethysmography (BOD POD; Life Measurement Instruments, Concord, CA) (22) in the remaining subjects. In a previous validation study, body composition determined by these 2 methods did not differ significantly (22). For one subject who was not able to be tested by either of these methods, body density was estimated from anthropometric measurements by using an equation for women developed by Pollock et al (23):

$$\begin{aligned}\text{Body density} = 1.1023 &- (0.0005 \\ &\times \text{suprailiac skinfold, mm}) - (0.0003 \\ &\times \text{thigh skinfold thickness, mm}) \\ &- (0.0005 \times \text{waist circumference, cm}) \\ &- (0.0033 \times \text{bra cup size}) \qquad (1)\end{aligned}$$

where bra cup size A = 1, B = 2, C = 3, etc).

Skinfold thicknesses and waist circumference were measured by using Harpenden calipers (British Indicators Ltd, London) and a spring-ended anthropometric tape measure, respectively, according to standard techniques. Body fat mass and fat-free mass were estimated from body density by using Siri's formula (24). Infant weight was measured to the nearest 1 g on an electronic balance.

Plasma prolactin concentrations

Plasma prolactin concentrations, basal and in response to infant suckling, were measured as described previously (9) by immunoradiometric assay (Coat-A-Count IRMA; Diagnostic Products Corporation, Los Angeles) during baseline and on day 7 or 8 of the intervention period.

Breast-milk volume and composition

Twenty-four–hour milk volume, feeding frequency, and total time spent breast-feeding were assessed in the home on 4 consecutive

days during baseline and on days 5, 6, 9, and 10 of the intervention; milk volume was measured by standard test-weighing procedures (25) with an electronic scale (Sartorius 3826; Brinkmann Instruments, Westbury, NY). Differences in weight before and after each feeding were summed over each 24-h period and corrected for estimated insensible water loss as described previously (25) by using the following equation: $0.05\ g \cdot kg^{-1} \cdot min^{-1} \times$ infant weight (kg) $\times$ total time spent breast-feeding (min).

Milk samples were collected at home by 24-h alternate expression with an electric pump by using methods described elsewhere (26). On average, subjects expressed 44% of their total milk volume. Subjects were allowed to feed their infants the portion of expressed milk that was not required for analysis. Aliquots of milk proportional to the volume pumped at each feeding were pooled and stored at −20°C until analyzed further. Lipid was measured gravimetrically after a modified Folch extraction (27). Total nitrogen (TN) and nonprotein nitrogen (NPN) were analyzed by micro-Kjeldahl analysis (28) and protein concentration was calculated as $6.25 \times (TN - NPN)$. The mean (±SD) CVs for duplicate samples were 1.9 ± 1.7% (lipid), 2.1 ± 4.2% (TN), and 9.5 ± 7.8% (NPN). Milk energy output was calculated as milk energy density multiplied by the average 24-h milk volume. Gross energy density was predicted from the milk lipid concentration (g/dL) by using the following equation, which was developed from a previous study (29):

$$\text{Milk energy density (kJ/g)} = 1.464 + (0.397 \times \text{milk lipid}) \quad (2)$$

where $R^2 = 0.98$, $P < 0.001$.

Determination of intervention dietary intake and exercise prescription

The total energy requirement (TER) at baseline was determined individually by averaging energy expenditure (including breast-milk output and exercise) and intake. For the diet group, the amount of energy to be provided during the intervention was calculated as $0.65 \times$ TER and no additional exercise was prescribed. For the diet + exercise group, additional energy expenditure prescribed in exercise during the intervention was calculated as $0.40 \times 0.35 \times$ TER. Energy provided during the intervention for the diet + exercise group was calculated as $0.65 \times$ (TER + additional energy expenditure prescribed in exercise) so that a net 35% energy deficit would be achieved. The control group was asked to maintain their weight during the intervention by maintaining their usual diet and activity patterns.

For the diet and diet + exercise groups, diets were individually tailored and food was provided in preweighed amounts. Meals and snacks were prepared from fresh, prepackaged, and frozen commercial foods. Subjects were encouraged to drink plenty of water and other non-energy-containing beverages and a daily multivitamin and mineral supplement was provided. Diets were designed to keep macronutrient proportions identical to those reported at baseline, provided that the recommended dietary allowance for protein during lactation was met (15 g/d above that for nonlactating women; 17). If this was not the case, the protein intake was increased to meet this requirement and the carbohydrate intake was decreased to compensate; this was necessary for 10 subjects in the diet group and for 4 subjects in the diet + exercise group. Subjects were instructed to weigh leftovers and any additional foods consumed that had not been provided.

For the control and diet groups, exercise frequency and intensity were held constant between the baseline and intervention periods. During the intervention period, exercise for the diet + exercise group was prescribed in terms of a target heart rate range (50–70% of maximal heart rate) and total time, divided into 9 of the 11 d. Exercise sessions were self-supervised; the subjects exercised at their own convenience in one or more sessions per day. They were allowed to perform any aerobic exercise activity or combination thereof, including walking, jogging, low-impact aerobics, step aerobics, bicycling, swimming, and use of exercise machines such as stair steppers and stationary cycles. For all 3 groups, energy expended in exercise (based on heart rate monitoring) was checked every 1–3 d during the intervention period, and the prescription was adjusted as necessary to meet the total exercise expenditure goal for the diet + exercise group, or to maintain the baseline level for the diet and control groups.

Statistical analysis

Data were analyzed by using SAS-PC (30). Group characteristics were compared with one-way analysis of variance (ANOVA) or chi-square analysis. Changes over time were evaluated with repeated-measures ANOVA; differences among groups in changes over time were evaluated with analysis of covariance by using change variables as the outcomes (baseline − intervention) and with baseline values controlled for. Multiple pairwise group comparisons were made with Tukey's honestly significant difference test. Pearson correlations were calculated to determine associations among variables. Before analysis of plasma prolactin data, 3 variables were created: basal (prefeeding) concentration, peak concentration (the highest measured concentration), and the area under the curve (AUC) for prolactin response. The basal concentration and AUC for prolactin response required log transformation because they were not normally distributed. All statistical tests were two-tailed; a P value ≤0.05 was accepted as significant.

RESULTS

One hundred thirty-five women eligible for the study inquired about participation. Of these, 67 elected not to participate: 52 because of time constraints and 15 because of general disinterest. Of the 68 subjects enrolled, 1 withdrew after assignment to the diet + exercise group, but before the intervention began because she had difficulty completing the baseline measurements. There were no significant group differences in the characteristics of the remaining 67 subjects (**Table 1**). One subject in the diet + exercise group did not continue with the intervention after day 8 because of a previously unreported exercise-induced asthma condition; data for this subject were included in the analysis up to the time that she stopped participating in the intervention. At baseline, subjects were 12 ± 4 wk postpartum, aged 32 ± 5 y, and had a body mass index (in kg/m^2) of 25.2 ± 4.2 ($\bar{x}$ ± SD).

Information on dietary intake, energy expenditure, and energy deficit is provided in **Table 2**. Mean TER at baseline ranged from 11.4 to 11.9 MJ/d and did not differ significantly among groups. Dietary energy intake and the percentages of energy from fat, carbohydrate, and protein did not differ significantly among the 3 groups at baseline and did not change significantly between the baseline and intervention periods for the 2 experimental groups. On average, a 34% energy deficit was achieved in both the diet and the diet + exercise groups; to achieve this deficit, the diet + exercise group exercised an average of 86

TABLE 1
Characteristics of subjects in the 3 groups[1]

	Control (n = 23)	Diet (n = 22)	Diet + exercise (n = 22)
Age (y)	31.3 ± 5.7[2]	31.7 ± 5.2	31.6 ± 5.1
Education (y)	16.4 ± 2.6	16.6 ± 2.4	16.0 ± 1.9
Race or ethnic group (n)			
Non-Hispanic white	18	17	18
Hispanic	2	3	2
Black	3	0	0
Asian	0	2	2
Parity			
Primiparous	10	9	6
Multiparous	13	13	16
Height (cm)	166.5 ± 7.1	165.0 ± 10.01	164.6 ± 7.7
Prepregnancy weight (kg)	66.4 ± 8.7	67.3 ± 14.0	64.9 ±14.2
Pregnancy weight gain (kg)	16.4 ± 6.0	15.2 ± 5.0	16.1 ± 4.8
BMI at baseline (kg/m^2)	24.9 ± 3.8	25.3 ± 4.8	25.4 ± 4.1
Maximal O_2 uptake at baseline (mL·kg^{-1}·min^{-1})	33.4 ± 6.3	34.7 ± 5.4	34.2 ± 5.6
Sex of infant			
Female	8	9	12
Male	15	13	10
Birth weight (kg)	3.52 ± 0.41	3.58 ± 0.53	3.54 ± 0.49
Weight of infant at baseline (kg)	6.34 ± 0.37	6.39 ± 0.75	6.36 ± 0.75
Age of infant at baseline (wk)	12.6 ± 3.7	12.2 ± 3.5	12.0 ± 3.7

[1] There were no significant differences between groups.
[2] $\bar{x}$ ± SD.

min/session on 9 of the 11 d.

Body weight and body-composition changes are shown in **Table 3**. Weight loss did not differ significantly between the diet and diet + exercise groups (1.9 and 1.6 kg, respectively) and was minimal in the control group (0.2 kg); weight loss in the control group was significantly different from that in the diet and diet + exercise groups ($P < 0.0001$). The decrease in fat mass also did not differ significantly between the diet and diet + exercise groups; however, fat-free mass decreased by 0.7 kg in the diet group, but increased by 0.1 and 0.2 kg in the diet + exercise and control groups, respectively ($P = 0.003$). The change in percentage body fat also differed among groups, with the diet + exercise group having the biggest decrease and the control group having the smallest decrease.

Data on infant feeding and breast-milk volume and composition are shown in **Table 4**. Feeding frequency and total time spent breast-feeding did not differ significantly among groups. Milk volume during baseline was slightly, but not significantly, higher in the diet + exercise group than in the control and diet groups. Change in milk volume did not differ significantly among groups. Milk lipid concentration and thus energy density also did not change significantly as a result of the intervention in any of the groups. Milk protein concentration decreased significantly and to a similar extent in all 3 groups ($P = 0.004$), but no significant changes occurred in milk NPN. Milk energy output was significantly higher in the diet + exercise group than in the other 2 groups at baseline ($P = 0.04$). The group difference in change in milk energy output was marginally significant ($P = 0.10$), but not significant when adjusted for baseline values ($P = 0.58$). Infant weight gain did not differ significantly among groups: 163 ± 99, 194 ± 139, and 229 ± 122 g for the control, diet, and diet + exercise groups, respectively. None of the infants was below the 5th percentile of the National Center for Health Statistics weight-for-age reference at either the baseline or intervention time points.

Individual changes in milk energy output varied widely, ranging from −458 to 765 kJ/d in the control group, −656 to 481 kJ/d in the diet group, and −565 to 647 MJ/d in the diet + exercise group. Three women had decreases >418 kJ/d (100 kcal/d) (1 in each group) and 5 had increases >418 kJ/d (2 in the diet group, 1 in the diet + exercise group, and 2 in the control group). Further examination of these individual differences revealed a significant interaction ($P = 0.05$) between treatment group and baseline body composition, as shown in **Figure 1**. In the diet group, there was a significant positive association between percentage body fat at baseline and change in milk energy output. Such an association did not exist for the diet + exercise or control groups. These results persisted even after differences in milk energy output at baseline were controlled for, either within or among groups. The same interaction between baseline percentage body fat and treatment group existed for milk NPN.

Two of the 67 subjects perceived a decrease in milk volume. One subject in the diet group reported on day 5 of the intervention that her infant wanted to breast-feed more frequently and her breasts felt less full than usual. However, her milk volume on day 5 was similar to her baseline average; nonetheless, by the end of the intervention her average milk volume and energy output had decreased by 102 g/d and 377 kJ/d, respectively. The subject in the diet + exercise group who discontinued the intervention after day 8 also perceived that her milk volume was decreasing; however, by the end of day 8 her average milk volume and energy output had increased above baseline by 12 g/d and 109 kJ/d, respectively. When data for all subjects were combined, the change in milk energy density was inversely related to the change in milk volume ($r = -0.24$, $P = 0.05$).

Mean (±SD) plasma prolactin concentrations are shown in **Figure 2**. Basal (prefeeding) prolactin concentrations differed by parity [base-

TABLE 2
Dietary intake, energy expenditure, and energy deficit in the 3 groups of subjects[1]

	Control (n = 23)	Diet (n = 22)	Diet + exercise (n = 22)
Baseline			
Energy intake (MJ/d)	10.40 ± 2.04 (7.15–15.86)	11.13 ± 1.85 (6.91–14.23)	10.53 ± 1.92 (7.92–14.54)
Fat (% of energy)	28.2 ± 1.1 (15.1–38.2)	30.1 ± 1.3 (15.0–40.7)	28.8 ± 1.0 (18.9–41.0)
Carbohydrate (% of energy)	57.4 ± 1.3 (43.8–57.4)	55.9 ± 1.6 (40.4–70.5)	57.1 ± 1.2 (44.8–66.6)
Protein (% of energy)	15.5 ± 0.5 (11.2–19.4)	15.0 ± 0.4 (11.2–19.9)	15.6 ± 0.4 (10.8–19.0)
Exercise			
(d/wk)	3.0 ± 0.9 (2.0–6.0)	3.1 ± 1.0 (2.0–7.0)	3.3 ± 0.9 (2.0–6.0)
(min/wk)	122 ± 57 (40–279)	134 ± 80 (48–434)	130 ± 44 (70–210)
Energy expenditure (MJ/d)	10.31 ± 1.44 (8.65–14.47)	10.47 ± 1.55 (7.64–13.03)	10.35 ± 1.77 (7.84–14.21)
Milk energy output (MJ/d)	2.19 ± 0.36 (1.65–2.79)	2.29 ± 0.41 (1.59–3.25)	2.49 ± 0.45 (1.83–3.77)
Total energy requirement (MJ/d)	11.44 ± 1.19 (9.61–14.94)	11.94 ± 1.48 (8.54–14.30)	11.69 ± 1.74 (9.37–14.92)
Intervention			
Energy intake (MJ/d)	NA	7.84 ± 0.97 (5.56–9.28)	8.68 ± 1.26 (6.99–11.46)
Fat (% of energy)	NA	29.5 ± 1.4 (15.0–42.0)	28.1 ± 0.9 (20.0–40.0)
Carbohydrate (% of energy)	NA	54.8 ± 1.5 (40.0–70.0)	56.0 ± 1.0 (45.0–64.0)
Protein (% of energy)	NA	15.7 ± 0.3 (13.0–20.0)	15.6 ± 0.4 (12.0–19.0)
Exercise			
(d/wk)	2.8 ± 0.8 (1.3–3.8)	2.8 ± 0.8 (1.9–5.1)	5.8 ± 0.4[2] (5.1–6.4)
(min/wk)	135 ± 126 (44–657)	126 ± 86 (43–472)	499 ± 87[2] (300–685)
Net change in energy expended in exercise (MJ/d)[3]	0.00 ± 0.22 (−0.40–0.64)	−0.07 ± 0.13 (−0.29–0.14)	1.55 ± 0.38[2] (0.59–2.12)
Energy deficit			
(MJ/d)	NA	−4.10 ± 0.54 (−5.02 to −2.97)	−4.56 ± 0.94 (−6.43 to −2.80)
(% of total energy requirement)	NA	34 ± 1 (30–35)	34 ± 3 (27–43)

[1] $\bar{x}$ ± SD; range in parentheses. NA, not applicable.
[2] Significantly different from control and diet groups, $P < 0.0001$.
[3] Defined as energy expended in exercise per day during the intervention minus energy expended in exercise per day during baseline.

TABLE 3
Body-composition changes in the 3 groups of subjects

	Control (n = 23)	Diet (n = 22)	Diet + exercise (n = 22)[1]
Weight (kg)			
Baseline	68.5 ± 8.5[2]	68.3 ± 10.2	69.0 ± 12.8
Intervention	68.3 ± 8.6[a]	66.4 ± 9.8[b]	67.8 ± 12.7[b]
Change[3,4]	−0.2 ± 0.6[a]	−1.9 ± 0.7[b]	−1.6 ± 0.5[b]
	(−0.5, 0.1)[5]	(−2.2, −1.6)	(−1.9, −1.4)
Fat-free mass (kg)			
Baseline	46.2 ± 4.2	45.7 ± 4.9	45.7 ± 6.0
Intervention	46.4 ± 4.1[a]	45.1 ± 4.8[b]	46.0 ± 5.9[a]
Change[4]	0.2 ± 1.0[a]	−0.7 ± 0.6[b]	0.0 ± 0.9[a]
	(−0.2, 0.6)	(−0.9, −0.4)	(−0.43, 0.38)
Fat mass (kg)			
Baseline	22.3 ± 7.0	22.6 ± 7.1	23.3 ± 8.2
Intervention	21.9 ± 7.0[a]	21.3 ± 6.8[b]	21.9 ± 8.1[b]
Change[3,4]	−0.4 ± 1.1[a]	−1.3 ± 0.9[b]	−1.6 ± 1.0[b]
	(−0.9, 0.1)	(−1.6, −0.9)	(−2.1, −1.1)
Body fat (% of body wt)			
Baseline	32.0 ± 7.0	32.5 ± 6.2	32.9 ± 6.5
Intervention	31.5 ± 6.9	31.6 ± 6.2	31.4 ± 6.7
Change[3,4]	−0.5 ± 1.6[a]	−0.9 ± 0.9[b]	−1.6 ± 1.5[c]
	(−1.2, 0.2)	(−1.3, −0.5)	(−2.3, −0.9)

[1] n = 21 for intervention and change values. Values within a row with different superscript letters are significantly different, $P < 0.05$.
[2] $\bar{x}$ ± SD.
[3] Significant main effect of time, $P < 0.05$.
[4] Significant time-by-group interaction, $P < 0.05$.
[5] 95% CI.

line: primiparous, 48.3 ± 34.0 μg/L; multiparous, 33.3 ± 22.0 μg/L (P = 0.19); intervention: primiparous, 47.2 ± 21.4 μg/L; multiparous, 28.8 ± 17.8 μg/L ($P < 0.0001$)]. The peak prolactin response to infant suckling was positively correlated with feed duration during the intervention (Pearson's r = 0.40, P = 0.001), but not at baseline (r = −0.04, P = 0.77). The change in basal prolactin differed significantly among groups after parity and basal prolactin at baseline were controlled for (P = 0.02): basal prolactin decreased significantly more in the control group than in the diet and diet + exercise groups. The change in peak prolactin response to infant suckling also differed among groups (P = 0.09); the difference between the control and diet groups was significant after change in feed duration and peak prolactin at baseline were controlled for (P = 0.03). There were no significant differences among groups in the change in AUC.

DISCUSSION

In this short-term intervention, an energy deficit of 35% (≈4.2 MJ/d, or 1000 kcal/d) resulted in an average weight loss in the diet and diet + exercise groups of 1.8 kg in 11 d, a rate of 1.2 kg/wk. The slight weight loss in the control group (0.1 kg/wk, or 0.4 kg/mo) was similar to the average rate of weight loss typically observed during lactation (31). Although there was no significant difference in the amount of weight lost between the diet and diet + exercise groups, fat-free mass was conserved and fat loss was enhanced in the diet + exercise group. The body-composition changes observed in this study are comparable with those reported in other short- and long-term studies of nonlactating subjects in which the effects of dieting were compared with those of dieting plus exercise (4–7).

We observed no main effect of energy deficit on milk volume

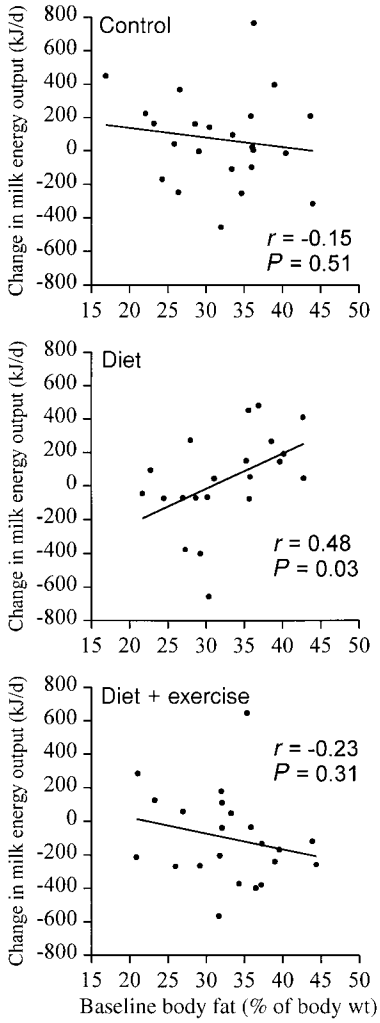

FIGURE 1. Interaction between baseline percentage body fat and the change in milk energy output in the control (n = 23), diet (n = 21, 1 missing value), and diet + exercise (n = 22) groups. There was a significant positive association in the diet group, but not in the control or diet + exercise group.

or energy output. In previous studies in humans and baboons, milk volume did not decrease in response to moderate energy restriction, but did when energy intake was restricted severely (11, 12, 14). However, those studies did not report total milk energy output, which may be less likely to decrease than milk volume because the infant presumably extracts a higher proportion of the high-fat hindmilk at each feeding as milk volume declines (as suggested by the inverse correlation between change in milk volume and change in milk energy density in this study). Although the 35% energy deficit in this study was similar to the 40% energy deficit that reduced milk volume in baboons (14), the duration of our intervention was much shorter (11 d compared with 10 wk). Whether an energy deficit will affect milk energy output may depend not only on the duration of the deficit but also on maternal energy reserves. In this study there was a significant interaction between baseline percentage body fat and treatment group: in the diet group (but not in the diet + exercise and control groups), fatter women tended to exhibit an increase

TABLE 4
Infant feeding, breast-milk volume, and breast-milk composition in the 3 groups of subjects[1]

	Control (n = 23)	Diet (n = 22)	Diet + exercise (n = 22)
Feeding frequency (times/d)			
Baseline	9.0 ± 2.1	8.0 ± 1.9	8.8 ± 2.1
Intervention	8.8 ± 2.3	7.6 ± 1.4	8.5 ± 2.0
Change	−0.2 ± 1.1	−0.4 ± 1.1	−0.2 ± 1.1
Total time breast-feeding[2] (min/d)			
Baseline	144 ± 44	139 ± 37	156 ± 55
Intervention	137 ± 38	143 ± 41	157 ± 54
Change	−7 ± 37	−4 ± 29	2 ± 21
Milk volume (g/d)			
Baseline	801 ± 115	830 ± 168	878 ± 165
Intervention	818 ± 140	829 ± 122	863 ± 156
Change	17 ± 81	−1 ± 76	−16 ± 84
Lipid concentration (g/L)			
Baseline	34.1 ± 6.0	35.4 ± 7.4	37.5 ± 9.3
Intervention	35.3 ± 8.3	36.2 ± 8.0	35.6 ± 6.1
Change	1.3 ± 7.8	0.8 ± 5.1	−2.0 ± 8.3
Protein concentration (g/L)			
Baseline	9.10 ± 1.36	8.96 ± 1.03	8.62 ± 1.66
Intervention	8.62 ± 1.58	8.39 ± 1.23	8.35 ± 1.55
Change[3]	−0.48 ± 1.01	−0.56 ± 0.99	−0.27 ± 1.46
Nonprotein nitrogen concentration (g/L)			
Baseline	0.44 ± 0.07	0.40 ± 0.09	0.46 ± 0.15
Intervention	0.46 ± 0.10	0.46 ± 0.11	0.49 ± 0.14
Change	0.02 ± 0.10	0.03 ± 0.11	0.03 ± 0.16
Energy density (MJ/L)			
Baseline	2.79 ± 0.24	2.87 ± 0.29	2.96 ± 0.37
Intervention	2.87 ± 0.33	2.90 ± 0.31	2.88 ± 0.24
Change	0.05 ± 0.31	0.03 ± 0.20	−0.08 ± 0.33
Energy output (MJ/d)			
Baseline	2.19 ± 0.36	2.29 ± 0.41	2.49 ± 0.45[4]
Intervention	2.26 ± 0.38	2.33 ± 0.31	2.39 ± 0.40
Change	0.07 ± 0.28	0.04 ± 0.28	−0.10 ± 0.27

[1] $\bar{x}$ ± SD; n = 21 for intervention and change values for lipid, protein, and nonprotein nitrogen concentrations, energy density, and energy output in the diet group.
[2] Defined as the average daily sum of the intervals between infant weight measurements before and after each feeding.
[3] Significant main effect of time, $P < 0.01$.
[4] Significantly different from diet and control groups, $P < 0.05$.

and leaner women a decrease in milk energy output (although only 3 subjects actually had a decrease >207 kJ/d, or 50 kcal/d). These results are consistent with the model proposed by Brown and Dewey (32), which predicts that only women with inadequate energy reserves will exhibit a decrease in milk energy output in response to a moderate-to-severe energy deficit.

The effect of an energy deficit on milk energy output may also depend on the capacity to mobilize and utilize adipose tissue to support lactation. In the present study, we did not observe a relation between initial fat reserves and the change in milk energy output in the diet + exercise group; thus, exercise may have exerted a protective effect in lean women. Exercise training has been shown to increase insulin sensitivity (33) and enhance fat utilization (4); these changes may serve to stabilize blood glucose concentrations and protect lactation when there is an energy deficit. Although the diet and control groups also exercised during the intervention, they did so less frequently and for a shorter duration (average 3 d/wk, 44 min/session) than did the diet + exercise group. Apparently, the moderate amount of exercise in the diet group did not have the same protective effect as did the high frequency and duration of exercise in the diet + exercise group.

In the diet group, the relative increase in plasma prolactin concentrations may explain the lack of a main effect on milk energy output. These results are consistent with the observation that prolactin concentrations were higher in unsupplemented, undernourished Gambian lactating women than in those given an energy supplement (34). Elevated prolactin concentrations are postulated to ensure the maintenance of milk synthesis by preferentially channeling nutrients to the mammary glands under conditions of energetic stress. The effects of prolactin are thought to occur (at least in part) via lipoprotein lipase activity, which increases at the mammary gland and decreases at other sites during lactation (35); in animal models this change in site-specific lipoprotein lipase activity was shown to be mediated by prolactin (36–38).

In conclusion, short-term weight loss ($\bar{x}$ ± SD: 1.1 ± 0.4 kg/wk) resulting from a combination of dieting and aerobic exercise appears safe for breast-feeding mothers and is preferable to weight loss achieved primarily by dieting because the latter reduces maternal lean body mass. Note that this conclusion may not apply

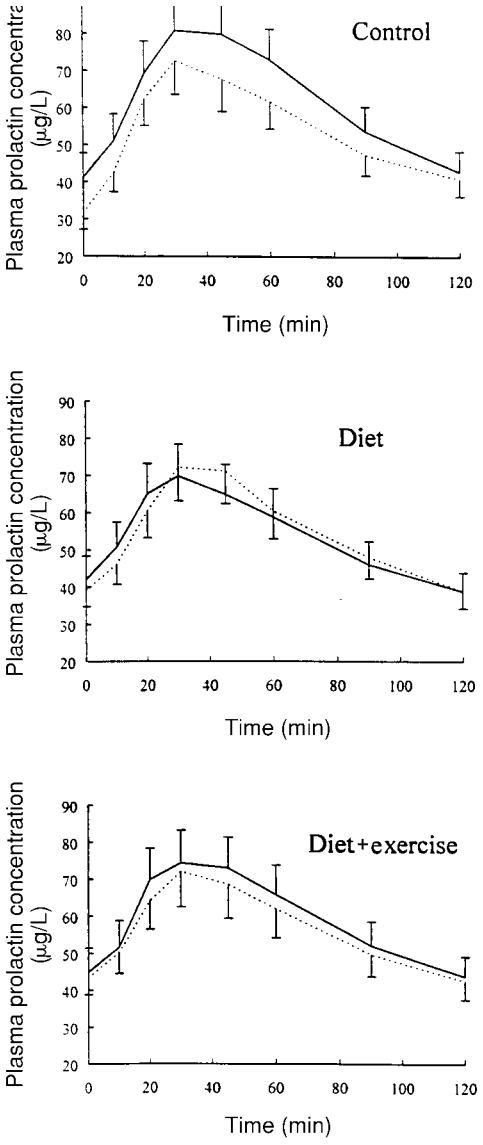

FIGURE 2. Mean (±SEM) basal (time 0) prolactin concentrations and concentrations in response to infant suckling, adjusted for parity group (primiparous compared with multiparous) and duration of feeding at baseline (—) and during the intervention (····). Basal prolactin concentrations decreased more in the control group than in the diet or diet + exercise group ($P = 0.02$, adjusted for parity group and basal prolactin at baseline; $n = 22$, 21, and 20 in the control, diet, and diet + exercise groups, respectively). Change in peak prolactin concentrations also differed among groups ($P = 0.09$); the difference between the control and diet groups was significant after the change in duration of feeding and peak prolactin at baseline were controlled for ($P = 0.03$; $n = 20$, 20, and 19 in the control, diet, and diet + exercise groups, respectively). Sample sizes vary because of missing data on duration of feeding for 4 subjects.

under other circumstances, such as during periods of weight loss >11 d or among women with lower initial body fatness than the women in the present study had. Moreover, a less severe energy deficit than the 35% imposed in this study may be more desirable and easier for overweight women to achieve in the long term. Further research should focus on the safety of long-term, moderate weight loss during lactation, particularly in lean women.

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