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Effect of Calcium Supplementation on Weight and Fat Loss in Women

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Abstract

Data suggest that a diet deficient in calcium is associated with higher body weight and that augmenting calcium intake may reduce weight and fat gain or enhance loss. Our aim was to determine whether calcium supplementation during a weight loss intervention affects body fat or weight loss. Data were combined from three separate 25-wk randomized, double blind, placebo-controlled trials of 1000 mg/d calcium supplementation in 100 premenopausal and postmenopausal women. The primary outcome measures were change in body weight and fat mass adjusted for baseline values.

There were no significant differences in body weight or fat mass change between the placebo and the calcium-supplemented groups in the pooled analysis (adjusted mean ± SE; body weight, placebo –6.2 ± 0.7 *vs.* Ca –7.0 ± 0.7 kg; fat mass, placebo –4.5 ± 0.6 *vs.* Ca –5.5 ± 0.6 kg), and no significant interactions of calcium supplementation with menopausal/diet status. Analysis as separate trials also found no significant differences between the placebo and the calcium groups. Calcium supplementation did not significantly affect amount of weight or fat lost by women counseled to follow a moderately restricted diet for 25 wk. Nevertheless, the magnitude and direction of the differences for group means are consistent with a hypothesized small effect.

Calcium is generally considered a key element for maintaining bone mineral homeostasis. New evidence and review of earlier studies supports the view that calcium also plays a role in adipocyte lipid kinetics at the cellular level and in moderating fatness at the population level. Within adipocytes, intracellular calcium levels alter the balance between lipid synthesis and breakdown, favoring lipogenesis when cytosolic calcium levels are high (1). National Health and Nutrition Examination Surveys I and III both provide cross-sectional evidence for an inverse association between calcium intake and body mass index (BMI; kilograms per square meter) (2, 3). These population observations are supported by retrospective analysis of a number of data sets that included calcium intake information and, in some cases, longitudinal administration of calcium as part of an osteoporosis trial (4).

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Both the mechanism and magnitude of the calcium-body weight effect remain uncertain. One theory, proposed by Zemel *et al*. (3), is that low calcium intake stimulates dihydroxy vitamin D and PTH and that, in turn, these calcitropic circulating substances stimulate adipocyte calcium uptake. As noted, high intracellular calcium levels promote lipogenesis and inhibit lipolysis. The theory suggests that dietary calcium increases lipolysis and preserves thermogenesis, thereby accelerating weight loss. Intracellular Ca^{2+} has a key role in regulating adipocyte lipid metabolism and triglyceride storage, with increased intracellular Ca^{2+} resulting in stimulation of lipogenic gene expression and lipogenesis, suppression of lipolysis, and adiposity (5). It is also suggested that the increased calcitriol released in response to low-calcium diets may contribute to the Ca^{2+} influx in human adipocytes and adiposity (6). Compared with low-calcium diets, transgenic mice expressing the agouti gene on high-calcium diets show reduced lipogenesis, increased lipolysis and thermogenesis, and suppressed body fat and weight gain. The beneficial effect of a high-calcium diet on body fat in the aP2-agouti transgenic was also observed during caloric restriction (1). A second potential mechanism involves stimulation of increased fecal energy losses due to formation of nonabsorbed complexes of calcium and fat (7–9).

We present data gathered during earlier 25-wk weight loss trials that included double-blind randomization of subjects to diet management with or without added calcium as a means of limiting diet-related bone loss. Some of the findings on weight loss, bone turnover, and bone mass from these studies have been reported previously (10–11). An additional 17 subjects not included in the original studies were included in this analysis because they completed dual-energy x-ray absorptiometry (DXA) measurements, but they did not complete blood samples and/or urine collections or lose sufficient weight to be included in the previous publications. This data set afforded us the opportunity to test, using a randomized trial design, the purported effects of calcium ingestion on body weight and fat loss during dieting.

Subjects and Methods

Obese postmenopausal women (>3 yr beyond menopause) and premenopausal women with a BMI ranging from 28–42 kg/m^2 were recruited for a 6-month weight loss program. Women who were taking any medication (including hormone replacement therapy or oral contraceptives) or had a disease state known to influence bone metabolism (*i.e.* cancer or diabetes) were excluded. In addition, for premenopausal women, only those who had not been pregnant or lactating within the previous year and had a history of a regular menstrual cycle were included. Subjects had to be weight-stable (±5% of body weight) for the previous 3 months. The study was approved by the Institutional Review Boards of Rutgers University (New Brunswick, NJ) and St. Luke's-Roosevelt Hospital (New York, NY), and all subjects provided written informed consent.

Randomization and treatment

Women were randomly assigned in a double-blind manner to one of two weight loss groups: 1) 1000-mg elemental calcium in the form of calcium citrate malate or calcium citrate; or 2) placebo tablets (Proctor and Gamble, Cincinnati, OH; Mission Pharmaceuticals, San

Antonio, TX). A computerized randomization program was used for the random assignment. Subjects were asked to consume two divided doses taken at breakfast and evening during this 6-month study. Compliance was assessed by pill counts every 2 months. The degree of compliance for the postmenopausal women in the regular diet group and Slim Fast (Slim Fast Foods Co., West Palm Beach, FL) group was 90 and 85%, respectively. Compliance for the premenopausal women was 91%.

Procedure

At baseline, subjects' food frequency and 24-h diet recalls were assessed to determine usual calorie and nutrient intake. A reduced-calorie diet was individually created based on both their energy expenditure and usual dietary patterns using the American Diabetic Association Exchange List. For each subject, a registered dietitian estimated energy expenditure, as per the Harris-Benedict formula, added an individual activity factor, and subtracted 2100 kJ from this total. Subjects in all weight loss groups were asked to adhere to this caloric deficit of 2100 kJ/d. Study participants were asked to stop taking vitamin/mineral supplements 1 month before the study period.

A registered dietitian encouraged weight loss using behavior modification and nutrition education. For all women, there were weekly group meetings for the first 16 wk, and then every other week thereafter for a total of 25 wk. Each group consisted of approximately 6–14 women and began in either the fall or winter. There were seven dietitians carrying out these group weight loss sessions from 1995–1998. During the diet period, the women were encouraged to continue their regular exercise patterns. Each subject was required to keep a diary containing daily diet recalls and physical activity, and the premenopausal women also kept a record of menstrual cycles.

A subset of postmenopausal women were encouraged to lose weight by consuming an obligatory one third of their total kilocalories from dietary supplements (Slim Fast) for the first 8 wk. Additional energy consumption was permitted from conventional foods in the form of snacks and one well-balanced meal. Dietary supplements were consumed for 8 wk, providing an additional 2.8 mg of vitamin D and 700 mg of calcium per day. Thereafter, women were counseled to replace dietary supplements with their usual reduced-calorie foods containing adequate dietary calcium and vitamin D for the remaining 16 wk.

Diet analysis—Twenty-four-hour dietary recalls (one weekend and two weekdays) at baseline and during the diet period (average of wk 3 and 20) were used to assess energy and calcium intake (Nutritionist IV, Salem, OR). Analysis of these records indicates that mean baseline calcium intakes ranged from 600 to 1000 mg/d, and that during the diet period mean calcium intakes of supplemented groups exceeded the calcium intakes of placebo groups by about 1000 mg/d.

Body fat mass—Whole-body DXA (DPX, Lunar Corp., Madison, WI) measurements were taken at baseline and upon completion of the weight loss regimen to determine changes in total body fat mass.

Weight and height—Subjects were weighed on a medical grade balance scale (accuracy, ±0.5%) and measured for height. The same scale was used throughout the 6-month trial. Weights were taken weekly at approximately 0900 h. BMI was calculated as weight (kilograms)/height (meters^2).

Statistical analysis

Because these studies were originally designed with the objective of studying bone mineral change with calcium supplementation and weight loss (9, 10), no *a priori* power calculations for weight loss were carried out. *Post hoc* power is discussed in the *Results* section.

These data were originally collected in separate studies on premenopausal and postmenopausal women, but they were combined for analysis to provide greater power to detect an effect of supplementation. Data were analyzed as a 2 × 3 factorial design with calcium supplementation (1000 mg daily or placebo) and menopausal status (premenopausal, postmenopausal, postmenopausal special diet) as between-subject factors and change in weight and body fat after 25 wk as the outcome variables. In addition, the results of separate analyses of the three studies are presented. All subjects for whom weight and fat mass change measures were available are included in the analyses.

Except for age, equivalence of groups at randomization was tested with ANOVA on baseline measures. In the pooled analysis the dependent variables, change in weight and fat, were analyzed in a two-way analysis of covariance with initial value as a covariate. In the analyses as separate studies there were no significant covariates so unadjusted group means were compared. Assumptions of equal variance were confirmed by Levine's test. Homogeneity of regression coefficients for analysis of covariance was also confirmed. Analyses were conducted using SPSS version 10 (SPSS, Chicago, IL). Type 1 error was set at 0.05, two-tailed. Values in the text are presented as mean ± SE.

Results

A total of 176 women were recruited for these studies; 165 were randomized, and body composition data were available for 100 of those who completed the study (Fig. 1). Initial characteristics of the subjects who completed the studies are shown in Table 1. Calcium intakes at baseline and during the intervention as estimated by dietary recalls are in Table 2. Two-way ANOVA on initial weight and fat mass found a statistically significant difference in fat mass between calcium-supplemented and placebo groups at randomization, with a greater mean initial fat mass in the placebo group compared with the calcium-supplemented group (43.5 ± 1.1 *vs.* 39.9 ± 1.2 kg; $P = 0.04$). Differences in initial values were taken into account by using initial values as covariates in subsequent analyses. ANOVA on baseline calcium intake indicates that premenopausal women had more calcium in their diet at baseline than the postmenopausal group, with the postmenopausal special diet group intermediate ($P < 0.05$). The premenopausal groups were run in the later years of the study, during a period when osteoporosis and calcium were receiving much attention in the local media, and the higher intakes may reflect this influence.

Analysis of covariance on weight change in the pooled data showed no statistically significant differences between the calcium and placebo conditions (placebo –6.2 ± 0.7 *vs.* calcium –7.0 ± 0.7 kg; $F_{(1,93)} = 0.64$; $P = 0.43$). The menopause status factor was not significant ($F_{(2,93)} = 2.60$; $P = 0.08$), and there was no significant interaction effect, indicating that calcium supplementation did not affect weight loss differently as a function of menopausal status ($F_{(2,93)} = 0.75$; $P = 0.48$). Analysis of covariance on change in fat mass in the pooled data also showed no statistically significant differences between the calcium and placebo conditions (placebo –4.5 ± 0.6 *vs.* calcium –5.5 ± 0.6 kg; $F_{(1,93)} = 1.29$; $P = 0.23$), no significant effect of the menopausal status factor ($F_{(2,93)} = 2.71$; $P = 0.07$), and no significant interaction ($F_{(2,96)} = 0.58$; $P = 0.56$). Statistical analysis of the entire population showed that mean weight and fat change adjusted for initial body weight or fat in these groups were not significantly different (Table 3A). When the three studies were analyzed separately, covariate adjustments for initial weight and fat were not required, so unadjusted means were compared (Table 3B). The mean change in body weight and fat were not significantly different between calcium and placebo treatments in any of the studies (Table 3B).

Examination of the distribution of weight and fat changes suggested that several subjects (three cases from the weight loss data and four cases from the fat loss data) showed unusually large changes (>2.5 SD from mean of their distribution and may be considered outliers. Exclusion of these subjects from analyses reduced error variability by about one third but did not result in any changes in the conclusions regarding calcium supplementation effects. However, the differences in fat loss among the menopausal/diet groups reached statistical significance in this analysis ($P = 0.03$), with the premenopausal group losing less fat than the post-menopausal groups.

Using the observed differences in means and the variability in weight (SD, 4.5 kg) and body fat change (SD, 4.1 kg) after adjusting for covariates in the pooled data, a study would require about 500 subjects per group to attain 80% power to detect a 0.8-kg difference in amount of weight change between two groups, and about 265 per group to detect a 1.0-kg fat mass difference, holding type 1 two-tailed error at 0.05.

Discussion

The results did not support the hypothesis that calcium supplementation at 1000 mg/d in conjunction with moderate dietary restriction has a beneficial effect on fat mass or weight loss over a 25-wk intervention. An effect of a magnitude such as to be of use in a moderate weight loss program was not seen. The dose levels used here are comparable to those used in previous human work both in terms of initial values and amount of supplementation (3, 4).

In this study, the premenopausal women tended to experience less weight and fat loss than the postmenopausal women, and this effect reached statistical significance for fat loss once outliers were excluded. The reason for this difference is not clear, and it may be simply a chance effect of different diet counselors who were more or less effective in their skills to promote weight reduction.

There have been previous human studies (3, 12–18) that have examined the association between dietary calcium and body weight and fat. Zemel *et al.* (3) examined epidemiological National Health and Nutrition Examination Survey III data and found that after controlling for energy intake, the relative risk of being in the highest quartile of body fat was greatest for those consuming diets lowest in calcium. This inverse relationship between body fat and calcium intake was found for both men and women. Zemel *et al.* (3) also report that increasing calcium from 400-1000 mg/d for 1 yr resulted in a mean 4.9-kg reduction in body fat in obese African-Americans. In a retrospective analysis by Davies *et al.* (4), five clinical trials were reevaluated for associations between calcium intake and body weight. The original outcome variable of these studies was a skeletal endpoint, and four of the five trials were observational studies with calcium intake estimated from food, whereas one trial was a randomized control trial using calcium carbonate. These authors (4) noted a negative association between calcium intake and weight in young, middle-aged, and older women, and calculated that a 1-g calcium intake difference was associated with an 8-kg difference in body weight. In addition, calcium intake was found to explain approximately 3% of the variance in body weight. A subsequent analysis of these data (4) by Heaney (13) showed that at mid-life there is a 0.4-kg/yr weight gain that is absent in women who consume the recommended calcium intake. This is supported by a higher fat oxidation rate in young healthy adults (male and female) who consume higher total dietary calcium (14). Other preliminary data also suggest an inverse relationship between calcium intake and body weight (16–18). However, one recent study in more than 9000 adults showed a positive effect of calcium on body weight in men, but not in women (15).

Some studies have shown that people perceive dairy foods as fattening and will avoid dairy when adhering to a weight loss regimen (19–21). This often results in inadequate calcium and vitamin D intake during voluntary weight loss using a standard low-calorie diet (10, 19–21), but this effect can be minimized with appropriate counseling (22). It has been suggested that weight loss attempts are unsuccessful for many individuals because of the tendency to eliminate milk, resulting in a low calcium intake (23). However, our data at two levels of calcium intake fail to show that calcium influenced body weight or fat loss in obese women with serum vitamin D levels within the normal range (10, 11).

There are many important differences between this study and other published human data [*e.g.* Zemel *et al.* (3)]: that study was not double-blind; fat was measured by bioelectrical impedance analysis and not DXA; and the sample was obese African-American men rather than our primarily Caucasian sample of women. Also, in the Zemel *et al.* study (3), calcium was supplemented as two cups of yogurt per day rather than capsules of calcium citrate. Rodent studies suggest that the source of calcium (*i.e.* dairy *vs.* supplements) may influence the effects on body weight (3). Zemel (23) has suggested that there may be other bioactive compounds in diary products that may contribute to a greater effect of dairy calcium than calcium alone on body fat reduction. Consistent with these findings, the CARDIA study found that dairy consumption was inversely associated with body weight in a study of more than 3000 young adults (24). More recently, a rodent study showed that high calcium intake containing dairy protein impairs fat absorption, thereby reducing weight and fat gain (9). It is also possible that calcium supplementation has more pronounced effects if subjects have

very low baseline intake levels (*e.g.* <500 mg/d), as was the case in some studies (3). However, our results do not support a threshold effect of calcium because there was a higher baseline calcium intake in the pre- than postmenopausal women, yet there was a tendency for the premenopausal women to show a greater fat loss response to supplemental calcium. These findings may be influenced by age or estrogen status. Initial adiposity (13) and gender may also affect the response of body weight or fat to calcium intake. These factors, among others, may account for the different results among studies and should be considered in planning future research.

In the current study, we restricted caloric intake, whereas other studies have looked at calcium effects with unrestricted intakes (3, 12–18). It is possible that the dramatic effect of caloric restriction on weight loss masks to some degree the smaller effects of supplemental calcium. However, preliminary data from Zemel *et al.* (25) suggest that the effect is still present during caloric restriction. Our results also indicate that the nonsignificant differences in means observed between supplemented and unsupplemented groups after 6 months (0.8 kg more body weight lost, 1.0 kg more fat mass lost) are in the direction of greater loss in the supplemented groups, so there may be an effect, albeit one too small to reach statistical significance in this study. It is noteworthy also that the effect size seems to be larger for measures of fat change than weight change and that the proportion of weight lost as fat is greater in the supplemented than unsupplemented group (78% *vs.* 72%). Given that the proposed mechanism of action is on the adipocyte (3, 5, 6) and/or the metabolism of fat (7–9, 14), studies that use weight change rather than fat change as the outcome measure may be incorporating unnecessary variability into the data.

In summary, an antiobesity effect of calcium reported by others during weight-stable conditions led us to hypothesize that supplemental calcium during caloric restriction would enhance loss of body weight and fat. Although we did not find a statistically significant effect of supplemental calcium on fat or body weight in obese women undergoing moderate weight loss, the direction of change suggests that over a longer period of time and with a larger number of subjects a reliable effect might be observed.

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Abbreviations

BMI	Body mass index
DXA	dual-energy x-ray absorptiometry

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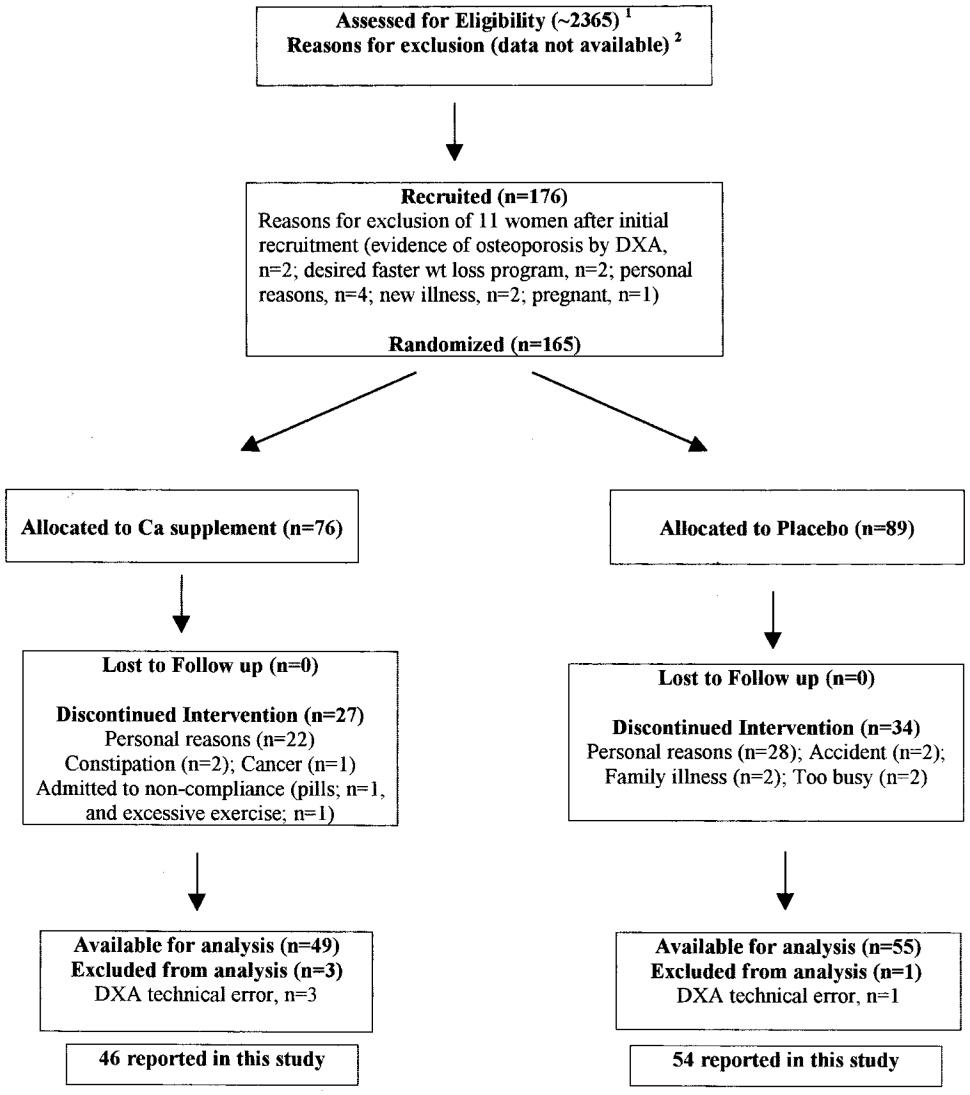

Fig. 1.

Initial characteristics of subjects who completed the studies. [1]Assessed for eligibility: this was tabulated for two of the eight recruitments. In these two recruitments, we have data to show that 7% of the subjects screened were eligible for recruitment into the study (40 eligible subjects from 584 individuals that were interviewed). For 162 women who were recruited, it can be estimated that we screened 2365 subjects during this 4.5-yr period. [2]Reasons for exclusion: due to noninvasive measures in this study and access to weekly weight loss sessions with a registered dietitian, there were almost no obese persons who refused to participate if they were eligible (weekly classes were available either weekdays or weekends to accommodate individual schedules). Those who were not eligible either had a disease state or medication known to influence calcium metabolism and were therefore excluded.

TABLE 1

Subject characteristics at randomization

	Age (yr)		BMI (kg/m^2)		Weight (kg)		Fat (kg)	
	Ca	**Pl**	**Ca**	**Pl**	**Ca**	**Pl**	**Ca**	**Pl**
Postmenopausal								
Mean	61.6	57.0	32.1	32.8	84.1	89.4	38.3	42.0
SD	8.6	8.2	3.5	4.2	9.4	10.3	6.9	7.5
N	17	19	17	19	17	19	17	19
Postmenopausal (special diet group)								
Mean	58.0	54.0	32.8	35.0	85.9	94.2	39.4	45.6
SD	5.5	5.5	4.1	4.1	9.2	15.7	6.3	9.6
N	11	11	11	11	11	11	11	11
Premenopausal								
Mean	40.4	41.5	33.9	34.7	93.7	93.5	42.1	42.8
SD	5.4	6.8	3.9	5.9	13.6	14.3	6.2	9.9
N	18	24	18	24	18	24	18	24

Ca, Calcium-supplemented group; Pl, placebo group.

TABLE 2

Daily calcium intakes (mg) at randomization and during weight loss intervention

	Calcium supplements		Placebo	
	Baseline	**Intervention**	**Baseline**	**Intervention**
Postmenopausal	705 ± 251	1514 ± 170	600 ± 210	511 ± 139
Postmenopausal special diet group	851 ± 432	1737 ± 175 (wk 3)	799 ± 392	733 ± 131 (wk 3)
		1525 ± 179 (wk 20)		557 ± 195 (wk 20)
Premenopausal	1019 ± 380	1782 ± 297	826 ± 389	695 ± 342

Because these subjects changed their diet from a meal replacement product to a diet of regular foods after 8 wk, the calcium values at 3 and 20 wk are given separately. Values are reported as X ± SD.

TABLE 3

Mean changes in body weight and fat mass (kg) adjusted for initial weight and fat, and analyzed as three separate studies (unadjusted means)

	Weight[a]		Fat[a]	
	Ca	**Pl**	**Ca**	**Pl**
Changes adjusted for initial weight and fat				
Postmenopausal	–7.5	–7.4	–6.4	–6.1
Postmenopausal special diet group	–7.2	–7.2	–4.9	–4.5
Premenopausal	–6.4	–4.1	–5.1	–2.9
Changes analyzed as 3 separate studies				
Postmenopausal	–7.0 ± 4.6	–7.3 ± 5.3	–6.1 ± 4.2	–6.1 ± 4.7
Postmenopausal special diet group	–6.7 ± 2.6	–7.6 ± 5.7	–4.8 ± 2.1	–4.8 ± 3.9
Premenopausal	–6.7 ± 5.5	–4.3 ± 3.5	–5.2 ± 5.0	–3.0 ± 3.5

Values are reported as X ± SD. Ca, Calcium-supplemented group; Pl, placebo group.

[a]Within group SD values based on mean square error from analysis of covariance are 4.5 for weight, 4.1 for fat.