2000

The effects of an educational intervention on calcium intake and bone mineral content in young women with low calcium intake.

B. A. Peterson
R. C. Klesges
E. M. Kaufman
Theodore V. Cooper, *University of Texas at El Paso*
C. M. Vukadonovich

Nutrition

The Effects of an Educational Intervention on Calcium Intake and Bone Mineral Content in Young Women with Low Calcium Intake

Britt Ann Peterson, Robert C. Klesges, Elizabeth M. Kaufman, Theodore V. Cooper, Christopher M. Vukadinovich

Abstract

Purpose. *This study evaluated whether a combined behavioral and dietary intervention would affect young women's calcium intake and bone mineral content (BMC).*

Design. *The design was a two by three mixed design with one between-subjects factor (treatment vs. control) and one within-subjects factor (time—baseline, 3-month, and 6-month).*

Setting. *The study was conducted in a university setting in Memphis, Tennessee.*

Participants. *A total of 80 premenopausal women (ages 18 to 30) with low baseline calcium intake (< 700 mg/d) were included in the analyses. There were 40 women in the treatment group and 40 women in the control group.*

Measures. *Hertzler and Frary's rapid assessment questionnaire was employed to evaluate calcium intake, and dual-energy x-ray absorptiometry (DEXA) was employed to assess BMC.*

Results. *Repeated measures analysis of variance (RM ANOVA) was employed to analyze results. Results indicated that women in the treatment group made greater increases in total calcium intake and supplemental calcium than women in the control group and that all women made significant increases in dietary calcium intake. Additionally, analyses of BMC revealed that women in the treatment group did not experience significant changes in total BMC, and women in the control group experienced significant losses in total BMC.*

Conclusions. *In the current study, women were losing BMC, and the women who made the largest increases in calcium intake were able to retard this bone loss. There is increasing evidence that dietary calcium intake in young people is extremely low, and the results of the current study highlight the need for much more intensive evaluations investigating the factors that are positively associated with premenopausal bone mineral change. (Am J Health Promot 2000;14[3]:149–156.)*

Key Words: Bone Mineral Content, Osteoporosis, Calcium Intake

Britt Ann Peterson, Robert C. Klesges, Elizabeth M. Kaufman, Theodore V. Cooper, and Christopher M. Vukadinovich are at the University of Memphis Prevention Center, Memphis, Tennessee.

Send reprint requests to Robert C. Klesges, PhD, The University of Memphis Prevention Center, 3350 Poplar Avenue, Suite 675, Memphis, TN 38119.

This manuscript was submitted June 16, 1998; revisions were requested January 11, 1999; the manuscript was accepted for publication July 30, 1999.

Am J Health Promot 2000;14(3):149–156.

0890-1171/00/$5.00 + 0

PURPOSE

Osteoporosis is one of the more serious diseases affecting women. One-third of women over the age of 65 suffer from vertebral fractures, and one-third of women over the age of 80 experience hip fractures.[1] Among those individuals with hip fractures, the mortality rate increases by 20%, and as many as 50% of the survivors are unable to walk without assistance.[2,3] It has been estimated that the nationwide annual cost of osteoporosis is $10 billion and that in the absence of active prevention efforts, this figure will increase to as much as $200 billion by the year 2040.[2]

Recently, researchers interested in the prevention of osteoporosis have recognized the need for clarifying the relationship between calcium intake and premenopausal bone mass. Epidemiological data indicate that calcium intake is very low in a significant percentage of young people.[4] Although it is believed that the attainment of peak bone mass during the early premenopausal years may be the strongest prophylactic against the development of osteoporosis,[5–7] the extent to which calcium intake affects premenopausal bone remains poorly understood.

To date, studies that have attempted to delineate the relationship between calcium intake and premenopausal bone have produced conflicting findings. Though many cross-sectional studies have reported positive correlations between calcium intake

and premenopausal bone.[8–16] the majority of longitudinal investigations have failed to find significant results.[13–17] Intervention studies have also failed to clarify the relationship between calcium intake and premenopausal bone. Three intervention studies found that changes in calcium intake were associated with changes in premenopausal bone mass[18–20]; two other intervention studies did not replicate this pattern of findings.[21,22]

There are several possible explanations for the mixed findings regarding the role of calcium intake as a predictor of premenopausal bone mass. First, previous treatment studies of bone mass and calcium supplementation suffered from sample sizes that were extremely low, from 8 to 36 participants per cell. With varying baseline calcium intake and probable varying levels of adherence, small sample sizes leave the relationship between calcium intake and premenopausal bone unclear. Second, many of the previous studies included well-nourished populations with high baseline calcium intakes. Calcium intake appears to exhibit a threshold effect, namely, increasing the intake in those with inadequate intake appears to increase BMC, but increasing the calcium intake in those with adequate intake does not appear to accrue further incremental effects.[23,24] To accurately assess the relationship between calcium intake and premenopausal bone, intervention studies are needed that focus on individuals who have low levels of calcium intake. A third problem relates to the age of the participants selected. Although the studies to date have limited their target groups to premenopausal women, the age of these women has varied greatly, and participants are typically past ages when peak bone density is believed to occur. A fourth and final weakness of previous research relates to the fact that many studies have not controlled for lifestyle factors (e.g., sedentary lifestyle, tobacco smoking, oral contraceptive use, and family history of osteoporosis), which may be associated with bone mass.[14,25]

The purpose of the current investigation was twofold. First, we investigated whether a brief dietary intervention would have an impact on the calcium intake of young women consuming significantly below recommended dietary levels. Second, we explored whether increases in young women's calcium intake would lead to subsequent increases in bone content.

METHODS

Design

The present study was a controlled evaluation of the impact of an intervention designed to increase calcium intake on the calcium consumption and BMC of young, premenopausal women. The dependent variables in this study were change in calcium consumption, as measured by the calcium intake questionnaire, and change in BMC, as measured by DEXA. The design was a two by three mixed design with one between-subjects factor (treatment vs. control) and one within-subjects factor (time—baseline, 3-month, and 6-month).

Sample

A total of 255 women were recruited for the study through television and newspaper advertisements and through fliers distributed at local Memphis businesses and in The University of Memphis Psychology Department. Recruitment information clearly indicated that all women would be offered valuable health information and body composition scans for free. Additionally, participants recruited from the Psychology Department were offered course credit in return for participation.

Of the 255 women responding to the recruitment for the study, 122 qualified for participation. Sixty-two of these individuals were randomly assigned to the treatment condition, and 60 were randomly assigned to the control condition. To qualify for the study, an individual had to be aged 18 to 30 years and had to consume no more than 700 mg of calcium per day. By restricting the sample to individuals below the age of 30 and consuming less than 700 mg of calcium per day, we targeted the population most likely to benefit from this program and most likely to show bone content changes.[26] Additionally, respondents were screened to exclude pregnant or lactating women, women who were less than 1-year postpartum, women with skeletal disorders, women with menstrual dysfunction (women who did not menstruate every 21 to 35 days), and women with kidney disease or related disorders. Although most of the women who did not qualify for the study were excluded because they consumed more than 700 mg of calcium per day, six women were excluded because they were pregnant, planning to become pregnant, or less than 1-year postpartum, and 16 women were excluded because they had irregular menstrual cycles. Of the 111 women who were not included because they consumed more than 700 mg of calcium per day, 96 were excluded during initial telephone screening, and 15 were excluded when evaluation of their baseline data revealed calcium intake exceeding 700 mg per day. Only women who did not qualify for the study were informed of inclusion/exclusion criteria. Nutrient reference data from the third National Health and Nutrition Examination Survey indicate that 50% of American women between the ages of 20 and 29 consume less than 640 mg of calcium per day.[27] In the current study, 56% of the sample recruited were consuming less than 700 mg of calcium per day, and thus, the recruited sample appears to be representative of the population. Institutional Review Board approval was obtained for this study, and consent forms were completed by all participants during the initial laboratory meeting. Written consent for DEXA testing was also obtained at each assessment.

Participant Retention. Given the all-volunteer nature of the subject population, several efforts were made to minimize the rate of attrition. In addition to indicating that health information and body composition assessments would be provided for free and that students from the Psychology Department would be offered credit toward improving their course grades, we also informed participants

that when they completed their 6-month lab visit, they would be paid $10.00 for having completed the study.

As well as employing an incentive program, we also implemented a telephone protocol aimed at decreasing the rate of attrition. Throughout the study, all participants were given a telephone reminder call the evening before their appointment. If a participant failed to attend her scheduled lab, we immediately attempted to contact her to reschedule as quickly as possible. If a participant indicated a willingness to complete the study, she was rescheduled a minimum of three times per follow-up before being dropped from the study.

Measures

Demographics. Participants filled out standard background questions including age, education, marital status, and income. Additionally, because there are several potential predictors of BMC, it was necessary to monitor other variables that may affect bone composition, to ensure equal weighting of these factors as a result of random assignment. These variables include family history of osteoporosis, smoking status, oral contraceptive use, and physical activity. Physical activity was assessed by asking standard recommended levels of activity from the American College of Sports Medicine (ACSM), namely, how many times per week they engaged in activity that increased their heart rate for a minimum of 20 minutes. Although there was no formal published measure of physical activity administered, the operational definition of physically active individuals is well disseminated by the ACSM. All demographic variables were monitored at each assessment point so that any changes in status or failures of randomization could be assessed.

Bone Mineral Content. BMC was assessed by DEXA. DEXA provides highly accurate and precise measures of bone composition and density. The accuracy of the bone measurements does not depend on hydration status, and the DEXA procedure requires little participant cooperation.[28] The DEXA equipment used for all assessments was a Hologic-2000 (Hologic, Inc., Waltham, Massachusetts). This equipment provides a negligible radiation dose, ranging from 1 to 3 mrads per scan. In comparison, background environmental radiation exposure averages 3.5 mrads per week, and chest X-rays provide 8 to 10 mrads.[28] Levels of reliability and validity of DEXA are extremely high and are routinely used to diagnose not only low bone density but osteoporosis as well.[28]

The bone sites measured in this study included the femur, the forearm, and the total body. To date, criteria for selecting optimal skeletal measurement sites have not been well defined. We chose the femur, forearm, and total body because these sites provide further exploration of past studies that have been inconclusive and because these sites require minimal radiation exposure. For each participant, one scan was made on each assessment day, at each assessment site. A total of nine measurements were completed across sites and across time.

Calcium Intake Questionnaire. Hertzler and Frary's[29] rapid assessment questionnaire was employed to evaluate levels of dietary calcium intake. This questionnaire assesses the number of milligrams of calcium consumed on a typical day during the previous week. The rapid assessment questionnaire lists the major food sources of calcium, and participants are asked to record the number of daily servings consumed of each of the foods listed. These servings are then converted to milligrams of calcium to estimate the individual's level of calcium intake from food. Hertzler and Frary's scale has good stability with test–retest reliabilities over 3 weeks of .80 and validity with a correlation of .68 with 3-day diet records.[29] In addition to assessing calcium intake from food, we also assessed calcium supplement consumption.

Procedure. Recruitment was conducted through public media announcements and the psychology department's subject pool. Potential participants who responded to these announcements were contacted by telephone. During this initial telephone interview, all individuals were informed that we were evaluating a brief treatment designed to promote bone content that might lead to fewer stress fractures and decreased risk of osteoporosis in later life. Additionally, participants were informed that they would be randomly assigned to either a treatment or a control group. They were told that the treatment group would require attendance at three group meetings and that the focus of these meetings would be on lifestyle factors conducive to bone health. They were told that the control group would not receive intervention but would be given valuable feedback about their fat and lean tissue mass. Anyone not assigned to the treatment groups was able to attend intervention meetings after their final 6-month DEXA assessment.

If an individual expressed interest and met all of the preliminary criteria, then a baseline laboratory visit was scheduled at The University of Memphis. During the baseline laboratory visit, participants were again given an overview of the study; were asked to complete consent forms, baseline demographics, and calcium intake questionnaires; and were asked to have their first DEXA assessment. Because no safe radiation exposure has been established for a fetus, DEXA scans were rescheduled for six people reporting unprotected sex since their previous period, and two participants were dropped from the study because they became pregnant. All DEXA tests were conducted by a licensed x-ray technologist who was blind to treatment condition.

After participants completed all baseline measures, they were randomly assigned to the treatment or control condition. Participants assigned to the control condition did not return to the laboratory until the 3- and 6-month follow-up visits. At this time, all baseline measures were again completed. At the end of the 6-month lab visit, control participants were given complete DEXA feedback and were informed of the potential

benefits of increasing their calcium intake.

Participants assigned to receive treatment returned for 3- and 6-month lab visits and were also scheduled for three weekly calcium intervention sessions, which were conducted in small groups of 9 to 11 women. In the first session, the counselor focused on providing an explanation of osteoporosis and the impact it has on people's lives. Participants reviewed their DEXA evaluations and, because all participants had low calcium intakes, discussed their relative risk for osteoporosis. The second group session focused on providing education regarding dietary interventions for increasing dietary calcium, including low-fat sources of calcium, calcium-fortified foods, and calcium supplements. During this session, all treatment participants were also given supplements containing 1000 mg (333 mg × tid) of calcium as citrate and 400 IU (133 IU × tid) of vitamin D. The third group session focused on assessing the changes that participants had made in their calcium intake. Participants were asked to again complete the same calcium intake questionnaire. Additionally, participants were asked to turn in their calcium bottles and record the number of supplements consumed during the previous week. Each subject's current calcium consumption was compared with baseline calcium consumption. Time was spent with each participant, problem solving difficulties with supplement adherence and with dietary calcium consumption. At the end of the third group session, participants were provided with a 3-month supply of calcium supplements. They were provided with a phone number and were told to call this number if they experienced any side effects or concerns regarding their calcium intake. Participants in the intervention condition received another 3-month supply of calcium/vitamin D during their 3-month follow-up visits.

Analysis

Preliminary Analyses. Initial analyses compared the treatment and control groups on demographic variables and variables that have been associated with BMC changes. These variables included such characteristics as family history of osteoporosis, smoking status, oral contraceptive use, baseline calcium intake, physical activity, age, and race. The results of the preliminary analyses are presented in Table 1.

Table 1

Preliminary Analyses

Variables	Entire Sample		Treatment (n = 62)		Control (n = 60)		
	Mean or %	SD	Mean or %	SD	Mean or %	SD	*p*
Age (year)	21.3	3.4	21.6	3.8	21.1	2.9	0.39
Minority (%)	32.8		27.5		38.2		0.59
Full-time students (%)	86.9		81.2		92.6		0.05
Married (%)	9.5		13.0		5.9		0.15
Family history of osteoporosis (%)	10.2		8.7		11.8		0.55
Activity level (# of times/wk)	2.1	1.3	2.1	1.4	2.1	1.3	0.91
Oral contraceptive (OC) use (%)	46.0		42.0		50.0		0.36
Years of OC use	1.5	2.7	1.4	2.8	1.7	2.5	0.46
Smokers (%)	16.2		17.4		19.1		0.79
Number years smoked	0.8	2.1	0.7	2.2	1.0	2.0	0.40
Supplemental calcium intake (mg/d)	11.1	88.9	19.6	123.5	2.5	20.8	0.26
Dietary calcium intake (mg/d)	460.1	190.4	441.7	190.8	478.6	189.5	0.26
Total calcium intake (mg/d)	471.2	217.4	461.3	241.5	481.1	191.1	0.60

As indicated in Table 1, the two groups were very similar across virtually all variables. The only baseline variable for which there was a significant difference was student status. This variable was significant at the .05 level, indicating there were significantly more full-time students in the control group than in the treatment group. To rule out differences in the dependent variables due to baseline differences in student status, student status was entered into analyses as a covariate. These analyses did not change patterns of significance, and thus, the results reported were not based on analysis of covariance.

Attrition. The initial sample consisted of a total of 122 participants—62 treatment participants and 60 control participants. By the 6-month follow-up, 42 participants had dropped out of the study, leaving 40 participants in the treatment condition and 40 participants in the control condition. Although a small number of participants dropped out of the study due to pregnancy (n = 2), moving away from Memphis (n = 2), illness and broken bones (n = 3), and starting depo provera (n = 2), the majority of individuals who dropped out of the study failed to attend scheduled labs. A series of analyses were run comparing the characteristics of participants who dropped out of the study with the characteristics of participants who completed the 6-month lab. These analyses revealed only one significant between-group difference. The baseline calcium intake of participants dropping out of the study was significantly lower than the baseline calcium intake of participants who completed the study ($t = 2.51$, $p < .013$).

Approach to Analysis. To defend against inflated Type I error rates occurring when the assumption of sphericity is violated with RM factors, we corrected the degrees of freedom for the main effect and their interaction with Box's ϵ, unless it exceeded .70, in which case it was replaced by the Huynh-Feldt ϵ.[30] To follow up on all significant main effects of time, we employed one-way independent ANOVA to test the simple effects of time and one-way independent ANOVA to test the simple effects of condition (treatment vs. control). All simple effects tests employed error terms specific to the cells being com-

Table 2

Calcium Intake (mg/d) by Condition Across Time

Variable	Baseline M	Baseline SD	3-Month M	3-Month SD	6-Month M	6-Month SD	Significant Simple Effects*
Total calcium intake†							
Treatment (n = 37)	453.02$_a$	245.15	1526.94$_b$	329.33	1336.33$_c$	593.01	b > a p = 0.00 c > a p = 0.00
Control (n = 38)	470.63$_d$	163.78	652.08$_e$	309.32	696.44$_f$	316.37	e > d p = 0.00 f > d p = 0.00 b > e p = 0.00 c > f p = 0.00
Dietary calcium intake‡							
Treatment (n = 38)	418.17$_a$	136.94	725.82$_b$	334.28	755.28$_c$	305.25	$\frac{(b+e)}{2} > \frac{(a+d)}{2}$ p = 0.00
Control (n = 38)	470.15$_d$	155.81	634.16$_e$	302.44	676.96$_f$	315.00	$\frac{(c+f)}{2} > \frac{(a+d)}{2}$ p = 0.00
Supplemental calcium intake†							
Treatment (n = 37)	32.22$_a$	166.54	803.32$_b$	321.78	575.07$_c$	458.55	b > a p = 0.00 c > a p = 0.00
Control (n = 38)	0.49$_d$	37.76	17.92$_e$	113.68	19.49$_f$	67.81	b > e p = 0.00 c > f p = 0.00

* Letters in the simple effects column represent the mean that has the same letter as a subscript.
† Denotes a significant interaction between condition (treatment vs. control) and time.
‡ Denotes a significant main effect for time.

pared, and *post hoc* testing following significant RM simple effects tests was done with Bonferroni-adjusted paired *t*-tests. For all calcium and BMC measures, we performed two (condition—treatment vs. control) by three (time—baseline, 3-month, and 6-month) ANOVA with RM on the time factor only. Given the small n and mild nonsphericity, these ANOVAs were performed by the univariate method to maximize power. The clarity of the statistical findings in the following paragraphs will be aided by referring to the means in Tables 2 and 3.

RESULTS

Calcium Intake

Total Calcium Intake. The *F*-test for the condition by time interaction proved significant, $F(2, 146) = 35.44$, $p < .001$.

The analysis of the simple effects of condition by time found that *all* participants significantly increased their calcium intake from baseline to 3-month and from baseline to 6-month but that treated participants had made significantly greater increases in their calcium intake than control participants at the 3- and 6-month time points. Overall, total calcium intake increased in both groups, but the calcium intake in the treated group increased more. This was largely due to increases in supplemental calcium.

Dietary Calcium Intake. There was a significant main effect for time, $F(2, 148) = 30.34$, $p < .001$. There were no significant findings for condition, $F(1, 74) = .97$, $p = .329$, or for condition by time, $F(2, 148) = 2.28$, $p = .108$.

Follow-up tests revealed that participants made significant increases in their dietary calcium intake from baseline to 3-month and from baseline to 6-month.

Supplemental Calcium Intake. The *F*-test for the condition by time interaction was significant, $F(2, 146) = 52.36$, $p < .001$.

The analysis of the simple effects of condition by time found that participants assigned to the treatment group made significant increases in their supplemental calcium intake across time. In contrast, participants assigned to the control group did not make significant changes in their supplemental calcium intake.

Bone Mineral Content

Total. The condition by time interaction was significant, $F(2, 156) = 3.27$, $p < .041$.

The analysis of the simple effects of condition by time revealed that participants who did not receive treatment *lost* a significant amount of BMC from baseline to the 6-month follow-up period ($X = -22.24$) and from the 3-month to the 6-month follow-up period ($X = -15.75$). No significant decreases were observed, in contrast, in the treatment group.

The analyses for the femur and forearm revealed no significant effects for condition or time or for the condition by time interaction.

DISCUSSION

The current study evaluated whether or not a behavioral/nutrition intervention would affect young, at-risk women's calcium intake and

Table 3

Bone Mineral Content (gm/cm) by Condition Across Time

Variable	Baseline		3-Month		6-Month		Significant Simple Effects*
	M	SD	M	SD	M	SD	
Total†							
Treatment (n = 40)	1625.26	268.67	1622.69	264.73	1621.04	269.30	
Control (n = 40)	1696.13_a	308.11	1689.64_c	305.68	1673.89_c	290.01	a > c p = 0.00 b > c p = 0.01
Femur							
Treatment (n = 40)	27.98	4.60	28.00	4.63	28.18	4.66	
Control (n = 40)	28.95	5.06	28.95	5.34	29.60	5.08	
Forearm							
Treatment (n = 40)	12.26	1.77	12.49	1.83	12.10	2.62	
Control (n = 40)	12.36	1.54	12.58	1.62	12.54	1.65	

* Letters in the simple effects column represent the mean that has the same letter as a subscript.
† Denotes a significant interaction between condition (treatment vs. control) and time.

BMC. Results showed that women in the treatment group made greater increases in total calcium intake and supplemental calcium intake than women in the control group and that all women made significant increases in dietary calcium intake. Additionally, women in the treatment group did not experience significant changes in total BMC, and women in the control group experienced significant losses in total BMC.

The current investigation was the first calcium intervention to focus on young premenopausal women whose baseline calcium intake was low. It is interesting that all participants in this study, regardless of treatment condition, increased their dietary calcium intake. This suggests that the acts of monitoring calcium intake and participating in repeated DEXA scans may provide as much impetus for making dietary changes as participation in a behavioral/educational intervention. Unfortunately, however, evaluation of the strength of the dietary changes reveals the difficulty individuals have consuming sufficient levels of calcium through their diet alone. Only the treatment participants who received calcium supplements consumed calcium at levels that approached the recommended daily allowance. Further, the treatment participants, who made these significantly larger increases in their calcium intake, experienced greater benefits to their bone. The results of this study are consistent with previous intervention trials that have shown that increases in calcium intake retard the loss of premenopausal bone in older women.[18,29]

Perhaps the most remarkable finding was that the intervention may have prevented *losses* in BMC at a time when women should be gaining peak bone mass. The current intervention may have prevented losses in BMC at a time when other studies have shown increases for several reasons. First, the current intervention targeted women who were consuming far below the recommended dietary allowance of calcium, and these women may be at particularly high risk for early premenopausal bone loss. Some studies have shown a statistically significant relationship between adolescent calcium intake and premenopausal BMC levels.[31] Further, there have also been data showing greater bone loss in premenopausal women with smaller baseline bone mineral levels.[17] If the low calcium intake in the current study represents longstanding habits, the mean BMC of the sample studied may be significantly lower than the mean BMC of a matched sample of individuals with a history of adequate calcium intake. Further, these lower baseline values may put the sample studied at greater risk for experiencing BMC loss.

A second explanation for the inconsistent pattern of premenopausal bone changes may relate to the fact that a minimum amount of calcium may be necessary for premenopausal bone to increase. In the current intervention, treatment participants increased their calcium intake by an average of 883.3 mg between baseline and the 6-month follow-up. However, even with these significant increases in calcium intake, women were still having difficulty meeting the recommended daily allowance for calcium. It is not known how much calcium is needed to exert bone changes, and it may be that increasingly greater amounts of calcium must be consumed both to prevent bone loss and to promote bone gain. In the current study, we may have prevented losses in BMC at a time when other studies have shown increases, because the women in the current intervention may not have been consuming the minimum amount of calcium needed to promote bone gain.

A third explanation for the inconsistent pattern of findings relates to the need for larger scale investigations aimed at delineating the age at which peak bone mass is attained and the impact of calcium interventions on various age groups of premenopausal women. The current study included women ranging in age from 18 to 30. To date, the age at which peak bone mass is attained

remains unclear. Though some studies indicate that women stop accruing bone mass by their mid-20s, other studies indicate that bone mass may continue to accrue into the third decade of life.[3,32] In the current study, it may have been possible that calcium intake was facilitating bone growth in the youngest women but that these increases in bone went undetected due to losses in bone in the older women. In this study, age was not a significant predictor of change in BMC; however, the study was limited in its power to detect age effects, and the efficacy of the intervention may differ depending on a woman's age.

The patterns of BMC loss in the current investigation may reflect the need for interventions that not only further investigate how the effects of calcium vary by age but that also further explore the impact of lifestyle factors such as physical activity, cigarette smoking, and oral contraceptive use.[14,25] The risk factor receiving the most attention recently is physical activity. A number of cross-sectional studies have demonstrated greater BMC in individuals who engage in increased levels of physical activity.[33–35] Further, a recent meta-analysis indicated that physical activity increases BMC but only when calcium intake is adequate.[36] This meta-analysis suggests that physical activity and calcium play an interactive role. It may be that calcium and physical activity will have greatest impact when both are increased to a sufficient level. It would be beneficial to conduct more aggressive interventions, aimed at increasing both physical activity and calcium intake, to determine if these interventions not only retard the loss of bone but also promote bone gain.

Although there were a number of positive features in this study, such as focusing on a largely unstudied age and risk group of women, there were also limitations. Despite the provision of incentives and multiple attempts to reschedule participants for labs, 34% of the participants did not complete the study. Interestingly, those participants who dropped out of the study had significantly lower baseline calcium intake than participants who remained, and it is unknown whether the intervention would have had the same impact had there been a smaller attrition rate.

Additionally, the present study does not clearly indicate whether the educational component of the intervention was effective. Participants in the control group were not completely blind to the study objectives, and awareness of the study objectives may have influenced both treatment and control individuals' behaviors. As stated previously, both treatment and control participants increased their dietary calcium intake, and it is unclear whether the acts of monitoring calcium intake and body composition were enough to change dietary patterns. Further, it is also unclear whether participant behavior was influenced by the provision of incentives and whether patterns of calcium consumption would have been significantly different if supplements had not been provided for free. Future studies need to further explore the factors that will produce the behavior changes necessary to change bone content. Health scientists should explore the influence of educational interventions on osteoporosis-related knowledge and beliefs and should explore the impact of these interventions under conditions that can be applied to a broader population. Future studies should evaluate the efficacy of interventions when participants are not provided with monetary incentives, course credit, and free calcium supplements. Further, these studies should be conducted among larger, more diverse populations.

Finally, it must be recognized that the clinical significance of the present intervention is not completely evident due to the fact that follow-ups did not extend beyond a 6-month time period. Previous studies have shown that short-term changes in bone mineralization can be difficult to interpret due to seasonal variations in bone mineralization[37] and that the greatest impact of calcium supplementation may not appear until 2 and 3 years after supplementation.[18] In the present study, we controlled for the impact that seasonal variability could have on between-group differences by randomly assigning participants to groups and by running treatment and control groups at the same time. However, due to the 6-month nature of the design, it is not completely clear whether seasonal variations in bone mineralization could have affected within-group differences. Furthermore, it is not completely clear whether the between-group differences would have been maintained or would have become stronger as additional time allowed for additional bone remodeling. Although the women in the control group experienced statistically significant losses in total BMC, the clinical significance of these findings remains uncertain. Clearly, future interventions are needed with larger sample sizes and longer follow-up periods.

In summary, the current investigation was the first calcium intervention to focus on young premenopausal women whose baseline calcium intake was low. The results of this study are consistent with previous studies that have shown a positive relationship between calcium intake and premenopausal bone. Further, the results suggest the need for much more intensive interventions aimed at young women, as there is increasing evidence that dietary calcium in young people is extremely low.[4] In the current study, women were losing BMC, and the women who made the largest increases in calcium intake were able to retard this bone loss. If young women are to strengthen their bodies against the development of osteoporosis, it is critical that they make the behavioral changes necessary not only to prevent BMC loss but also to promote BMC gain. Future studies are needed that more aggressively investigate the factors that are positively associated with premenopausal bone mineral change.

SO WHAT? Implications for Health Promotion Practitioners and Researchers

This study seems to indicate that young women with low calcium intake are at risk for losses in BMC at a time when peak bone mass should be occurring and that

calcium interventions may protect against this loss. The results of this study are consistent with previous studies that have demonstrated a positive relationship between calcium intake and premenopausal bone. If these assertions hold true, then health promotion practitioners need to more aggressively target young women, with interventions aimed at increasing their calcium consumption, and health promotion researchers need to further examine the factors that will not only prevent bone loss but will also promote bone gain.

Acknowledgment

This project was supported in a grant from the National Institute of Health, AR42729 awarded to The University of Memphis, E.C.K.

References

1. Nelson ME, Fisher EC, Dilmanian FA, et al. A 1-y walking program and increased dietary calcium in postmenopausal women: effects on bone. Am J Clin Nutr 1991;53:1304–11.
2. McBean LD, Forpac T, Calvert SF. Osteoporosis: visions for care and prevention—a conference report. J Am Diet Assoc 1994;94:668–71.
3. Wardlaw GM. Putting osteoporosis in perspective. J Am Diet Assoc 1993;93:1000–6.
4. Eck LH, Hacken-Renner C. Calcium intake in youth: sex, age, and racial differences in NHANES II. Prev Med 1992;21:473–82.
5. Matkovic V. Calcium and peak bone mass. J Intern Med 1992;231:151–60.
6. Peck WA, Riggs LB, Bell NH. Physician's Resource Manual on Osteoporosis. Washington, DC: National Osteoporosis Foundation, 1987.
7. Sowers MR, Galuski DA. Epidemiology of bone mass in premenopausal women. Epidemiol Rev 1993;15:87–98.
8. Cumming RG. Calcium intake and bone mass: a quantitative review of the evidence. Calcif Tissue Int 1990;47:194–201.
9. Hansen MA. Assessment of age and risk factors on bone density and bone turnover in healthy premenopausal women. Osteoporos Int 1994;4:123–8.
10. Ho SC, Leung PC, Swaminathan R, et al. Determinants of bone mass in Chinese women aged 21–40 y. II. Pattern of dietary calcium intake and association with bone mineral density. Osteoporos Int 1994;4:167–75.
11. Ramsdale SJ, Bassey EJ, Pye DJ. Dietary calcium intake relates to bone mineral density in premenopausal women. Br J Nutr 1994;71:77–84.
12. Salamone LM, Glynn NW, Black DM, et al. Determinants of premenopausal bone mineral density: the interplay of genetic and lifestyle factors. J Bone Miner Res 1996;11:1557–65.
13. Houtkooper LB, Ritenbaugh C, Aickin M, et al. Nutrients, body composition, and exercise are related to change in bone mineral density in premenopausal women. J Nutr 1995;125:1229–37.
14. Mazess RB, Barden HS. Bone density in premenopausal women: effects of age, dietary intake, physical activity, smoking, and birth control pills. Am J Clin Nutr 1991;53:132–42.
15. Prior JC, Vigna YM, Barr SI, et al. Ovulatory premenopausal women lose cancellous spinal bone: a five year prospective study. Bone 1996;18:261–7.
16. Riggs BL, Wahner HW, Melton LJ, et al. Dietary calcium intake and rates of bone loss in women. J Clin Invest 1987;80:979–82.
17. Sowers MR, Clark K, Hollis B, et al. Radial bone mineral density in pre- and perimenopausal women: a prospective study for rates and risk factors for loss. J Bone Miner Res 1992;7:647–57.
18. Baran D, Sorensen A, Grimes J, et al. Dietary modification with dairy products for preventing vertebral bone loss in premenopausal women: a three year prospective study. J Clin Endocrinol Metab 1990;70:264–70.
19. Rico H, Revilla M, Villa LF, et al. Longitudinal study of the effect of calcium pidolate on bone mass in eugonadal women. Calcif Tissue Int 1994;54:477–80.
20. Smith EL, Gilligan C, Smith PE, et al. Calcium supplementation and bone loss in middle-aged women. J Clin Nutr 1989;50:833–42.
21. Freudenheim JL, Johnson NE, Smith EL. Relationships between usual nutrient intake and bone-mineral content of women 35–65 years of age: longitudinal and cross-sectional analysis. Am J Clin Nutr 1986;44:863–76.
22. Friedlander AL, Genant HK, Sadowsky S, et al. A two-year program of aerobics and weight training enhances bone mineral density of young women. J Bone Miner Res 1995;10:574–85.
23. Matkovic V, Heaney RP. Calcium balance during human growth: evidence for threshold behavior. Am J Clin Nutr 1992;55:992–6.
24. National Institutes of Health. Consensus Development Conference Statement: Optimal Calcium Intake. Bethesda, Maryland: National Institutes of Health, 1994.
25. Toss G. Effect of calcium intake vs. other lifestyle factors on bone mass. J Intern Med 1992;231:181–6.
26. Dawson-Hughes B, Jacques P, Shipp C. Dietary calcium intake and bone loss from the spine in healthy postmenopausal women. Am J Clin Nutr 1987;46:685–7.
27. Alaimo K, McDowell MA, Briefe RR, et al. Dietary intake of vitamins, minerals, and fiber of persons ages 2 months and over in the United States: Third National Health and Nutrition Examination Survey, Phase I, 1988–91. Adv Data 1994;258:1–28.
28. Kellie SE. Measurement of bone density with dual-energy x-ray absorptiometry (DEXA). JAMA 1992;267:286–94.
29. Hertzler AA, Frary RB. A dietary calcium rapid assessment method: RAM. Top Clin Nutr 1994;9:76–85.
30. Huynh H, Feldt LS. Estimation of the box correction for degrees of freedom from sample data in randomized block and split-plot designs. J Educ Stat 1976;1:69–82.
31. Halioua L, Anderson JJ. Lifetime calcium intake and physical habits: independent and combined effects on the radial bone of healthy premenopausal Caucasian women. Am J Clin Nutr 1989;49:534–41.
32. Recker RR, Davies M, Hinders SM, et al. Bone gain in young adult women. JAMA 1992;268:2403–8.
33. Buchanan J, Myers C, Lloyd T, et al. Determinants of peak trabecular bone density in women: the role of androgens, estrogen, and exercise. J Bone Miner Res 1988;3:673–80.
34. Davee A, Rosen C, Adler R. Exercise patterns and trabecular bone density in college women. J Bone Miner Res 1990;5:245–50.
35. Politzer W, Anderson J. Ethnic and genetic differences in bone mass: a review with a hereditary vs. environmental perspective. Am J Clin Nutr 1989;50:1244–59.
36. Specker B. Evidence for an interaction between calcium intake and physical activity on changes in bone mineral density. J Bone Miner Res 1996;11:1539–44.
37. Rico H, Revilla M, Cardenas JL, et al. Influence of weight and seasonal changes in radiogrammetry and bone densitometry. Calcif Tissue Int 1994;54:385–8.