Aerobic exercise maintains regional bone mineral density during weight loss in postmenopausal women

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Ryan, Alice S., Barbara J. Nicklas, and Karen E. Dennis. Aerobic exercise maintains regional bone mineral density during weight loss in postmenopausal women. J. Appl. Physiol. 84(4): 1305–1310, 1998.—This study examines the effects of weight loss by caloric restriction (WL) and aerobic exercise plus weight loss (AEx+W) on total and regional bone mineral density (BMD) in older women. Healthy, postmenopausal women [age 63 ± 1 (SE) yr] not on hormone-replacement therapy underwent 6 mo of WL (n = 15) consisting of dietary counseling one time per week with a caloric deficit (250–350 kcal/day) or AEx+W (n = 15) consisting of treadmill exercise three times per week in addition to the weight loss. Maximal aerobic capacity increased only in the AEx+W group (P < 0.001). Body weight, percent fat, and fat mass decreased similarly in both groups (P < 0.005), with no changes in fat-free mass. Total body BMD (by dual-energy X-ray absorptiometry) decreased in both groups (P < 0.05). Femoral neck, Ward's triangle, and greater trochanter BMD decreased in the WL group (P = 0.05) but were not significantly different after AEx+W. L2–L4 BMD did not significantly change in either group. Thus WL and AEx+W both result in losses of total body BMD; however, AEx+W appears to prevent the loss in regional BMD seen with WL alone in healthy, older women. This suggests that the addition of exercise to weight-loss programs may reduce the risk for bone loss.

AGING IS ASSOCIATED with a loss of bone mineral density (BMD) (21) and fat-free mass (FFM) (31) and an increase in body fat (9). The decline in BMD may lead to osteoporosis while the increased body fat and the associated obesity contribute to an increased risk for cardiovascular disease (CVD). Yet, obesity appears to be protective for fracture risk in postmenopausal women (13, 17), with a higher body weight associated with greater BMD (18). Caloric restriction is the treatment of choice for the reduction of body weight in obese women (10). However, dietary-induced weight loss can result in a significant loss of total body bone mass in women (7, 27). A low-calorie diet also resulted in a significant decrease in total body, arm, and leg bone mineral, with no differences between pre- and postmenopausal women (14). There are few studies examining the effect of weight loss on vertebral, femoral, and total BMD in postmenopausal women, although one study showed that a high-fiber diet significantly increased the annual bone loss from the lumbar spine in postmenopausal women (2).

Increased physical activity is considered a beneficial therapy for osteoporosis (30). However, studies (5, 6, 8, 16, 26) examining the effects of aerobic exercise (AEx) alone on BMD in older women have utilized different modes, frequency, intensity, and duration of exercise with disparate results. Despite this, exercise is generally recognized as beneficial to bone. However, the addition of dietary-induced weight loss (WL) to an exercise training program resulted in a loss of lumbar spine BMD in postmenopausal women (33). Thus loss of body mass may negatively affect the benefits of exercise on BMD.

Because of the potential adverse health effects of decreased bone density and increased body fat with advancing age, we examined whether dietary-induced weight loss with and without aerobic exercise would maintain or result in a loss of total and regional BMD in healthy, obese older women not on estrogen therapy.

MATERIALS AND METHODS

Subjects. Forty-one women between the ages of 52 and 72 yr who were at least 2 yr postmenopausal and not on any medication, including hormone-replacement therapy, volunteered to participate in the study. Only women who were weight stable and who had not participated in a regular exercise program for a minimum of 6 mo before the study were recruited. Subjects were screened by medical history questionnaire, physical examination, fasting blood profile, and a graded exercise treadmill test to exclude those with CVD. All subjects were nonsmokers, free of diabetes and cancer, had not had a recent skeletal fracture (&gt;2 yr), and were not on any medications that would alter calcium or bone metabolism. After the dietary-stabilization period during which five women dropped out of the study, the remaining women were enrolled in either the WL (n = 18) or the AEx+W (n = 18) program on the basis of subject preference. The groups were similar in age and body mass index (BMI).

All methods and procedures for the study were approved by the Institutional Review Board of the University of Maryland. Each participant provided written informed consent.

Dietary analysis and WL program. To minimize any affect that dietary composition might have on the measured metabolic variables, 6 wk before the initiation of the study all subjects were instructed on the American Heart Association (AHA) Step I (1) diet by a registered dietitian. The composition of this diet was 50–55% carbohydrate, 15–20% protein, ≤30% fat, and ≤300 mg of cholesterol per day. The women were asked to maintain this diet composition throughout the study's duration (6 mo). Compliance was monitored by review of 7-day food records taken every 4 wk. The total calories and percentage of calories from carbohydrate, protein, and fat were calculated by using Nutritionist III (25). All subjects were weight stable 2 wk before each testing period. After the 6 wk of AHA stabilization diet and initial testing period, the women in both groups attended weekly WL classes led by a registered dietitian. Women were instructed to restrict their caloric intake by 250–350 kcal/day to induce ~0.25–0.5 kg weight loss per week during the study period. At the end of
the 6-mo program, both groups maintained a constant weight on the AHA diet for 2–4 wk before final testing. Those subjects in the AEx+WL intervention were asked to continue exercise training during the final testing period.

Maximal oxygen uptake ($V_O^{max}$). $V_O^{max}$ was measured before and after WL or AEx+WL to classify the fitness status of the subjects. A continuous treadmill test protocol was used as previously described (29). Briefly, speed was kept constant while the grade was increased from 0 to 4% at 2 min and then was increased 2% every minute after the third minute until the subject was unable to continue. Validation for attainment of $V_O^{max}$ included meeting two of the following three criteria: 1) a plateau in oxygen uptake with an increased workload as evidenced by a difference in oxygen uptake of $<2$ ml kg$^{-1}$ min$^{-1}$; 2) a respiratory exchange ratio $>1.10$; and 3) a maximal heart rate within 10 beats/min of the age-predicted maximal value.

AEx program. In addition to participation in the WL classes, subjects in the AEx+WL intervention walked or jogged on treadmills three times a week for 6 mo. Each exercise session included a 10-min stretching and warm-up phase and a 10-min cool-down phase on cycle ergometers. Exercise sessions began at relatively low levels of aerobic capacity on the basis of the subjects’ $V_O^{max}$ values ($50–60\%$ heart rate reserve) and gradually progressed in duration and intensity until the subjects were exercising at $>70\%$ $V_O^{max}$ for 35 min by the fourth month of training. The average caloric expenditure of the exercise program progressed from $\sim 200$ to $\sim 500$ kcal/wk. Attendance was taken at each exercise session to monitor compliance with the program. Subjects were contacted if an exercise session was missed. All sessions were monitored by an exercise physiologist and at least one exercise leader, both of whom were certified in cardiopulmonary resuscitation. Body weight was recorded every week throughout the study at the training sessions.

Body composition and BMD. All body composition measurements were performed after a 12-h overnight fast. Height (cm) and weight (kg) were measured to calculate BMI as weight (kg)/height (m$^2$). Waist circumference, measured at the narrowest point superior to the hip, was divided by the circumference of the hip, measured at its greatest gluteal protuberance, to obtain the waist-to-hip ratio (WHR), an index of body fat distribution. A total body scan was performed with dual-energy X-ray absorptiometry (DEXA; model DPX-L, LUNAR Radiation, Madison, WI). All scans were analyzed by using the LUNAR version 1.32 DPX-L extended-analysis program for body composition. Fat mass, lean tissue mass, and bone mineral content (BMC) were determined. FFM was calculated as lean tissue plus BMC. Reliability was assessed from five repeat scans of an older healthy volunteer who was repositioned after each scan in one visit. The coefficients of variation (CVs) for total body percent fat, fat mass, lean tissue mass, and BMC are 1.4, 1.4, 0.7, and 0.4%, respectively.

The same DEXA scanner and software were used to scan the total body, anteroposterior lumbar spine (L2–L4), and femur (femoral neck, Ward’s triangle, greater trochanter) to obtain total and regional BMD. Five repeat scans of the lumbar spine and femoral regions were obtained in two healthy volunteers who were repositioned for each scan. The CVs for these regions are 0.5, 0.8, 1.3, 4.2, and 1.3%, respectively. All scans were analyzed by the same investigator, who was blinded to the treatment groups, and all bone density tests took place after an overnight fast during one visit both before and after the interventions.

Statistical analyses. Differences between and within WL and AEx+WL groups for the dependent variables were calculated by using a one-way or repeated-measures analysis of variance, as appropriate. Pearson product-moment correlation coefficients were calculated to test for associations among body composition, aerobic capacity, and BMD. Statistical significance was set at $P < 0.05$ for all tests. All data were analyzed by SPSS statistical software (SPSS, Chicago, IL). All values are expressed as means ± SD unless otherwise noted.

RESULTS

Thirty of the 41 women completed either the WL (n = 15) or AEx+WL (n = 15) protocols. Five women dropped out of the study during the AHA dietary-stabilization period, with the remaining six women (3 from each group) leaving the study during the interventions because of personal reasons. In those women who completed the interventions, there was a $>90\%$ compliance for attendance at the exercise sessions and diet classes. Analyses were performed only on those women who completed either intervention. There were no differences in the baseline measurements between women who completed vs. those who withdrew from the study. Data were missing for the femur scan for one woman in each group.

The subject characteristics of each group before and after WL or AEx+WL are presented in Table 1. The WL and AEx+WL groups were not significantly different at baseline for initial age, body weight, BMI, $V_O^{max}$, percent fat, fat mass, or FFM. Body weight, BMI, fat mass, and percent fat decreased similarly after both WL and AEx+WL (all $P < 0.005$). Both waist and hip circumference decreased after the interventions in both groups ($P < 0.005$), although there was no change in the WHR. There was a significant time effect for FFM in the analysis of variance ($P = 0.01$), which was not significant in either group alone. $V_O^{max}$ (l/min) did not change significantly in either group alone. For all dependent variables, there was a significant group effect ($P = 0.005$) induced by the intervention. There was no significant group by time interaction ($P = 0.15$).

### Table 1. Characteristics of postmenopausal women before and after weight loss or aerobic exercise plus weight loss

<table>
<thead>
<tr>
<th></th>
<th>Weight Loss</th>
<th>Aerobic Exercise + Weight Loss</th>
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<tbody>
<tr>
<td><strong>Age, yr</strong></td>
<td>63 ± 6</td>
<td>62 ± 6</td>
</tr>
<tr>
<td><strong>Weight, kg</strong></td>
<td>83.1 ± 11.3</td>
<td>79.3 ± 8.0</td>
</tr>
<tr>
<td><strong>BMI, kg/m²</strong></td>
<td>30.9 ± 3.0</td>
<td>30.5 ± 2.8</td>
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<tr>
<td><strong>%Fat</strong></td>
<td>48.5 ± 4.7</td>
<td>46.5 ± 3.5</td>
</tr>
<tr>
<td><strong>Fat mass, kg</strong></td>
<td>40.1 ± 9.2</td>
<td>36.5 ± 5.9</td>
</tr>
<tr>
<td><strong>Fat-free mass, kg</strong></td>
<td>41.7 ± 3.4</td>
<td>41.7 ± 3.9</td>
</tr>
<tr>
<td><strong>Waist circumference, cm</strong></td>
<td>91.9 ± 5.4</td>
<td>89.1 ± 7.5</td>
</tr>
<tr>
<td><strong>Hip circumference, cm</strong></td>
<td>112.5 ± 6.7</td>
<td>111.9 ± 5.6</td>
</tr>
<tr>
<td><strong>WHR</strong></td>
<td>0.82 ± 0.07</td>
<td>0.04 ± 0.08</td>
</tr>
<tr>
<td><strong>$V_O^{max}$, l/min</strong></td>
<td>1.49 ± 0.28</td>
<td>0.19 ± 0.18</td>
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</tbody>
</table>

Values are means ± SD. BMI, body mass index; WHR, waist-to-hip ratio; $V_O^{max}$, maximal oxygen consumption. Significantly different from before the intervention. * $P < 0.05$; † $P < 0.001$. ‡ Significantly different between groups, $P < 0.05$.  


change after WL but increased significantly in the AEEx+WL group (P < 0.001). After the interventions, aerobic capacity was significantly higher in the AEEx+WL group than in the WL group (P < 0.05).

Food records were taken with the subjects in the weight-stable state after the AHA diet-stabilization period and after each of the interventions. Analysis of food records revealed no changes before vs. after WL or AEEx+WL in the percentage of calories from carbohydrate (WL: 57 ± 3% vs. 57 ± 5% and AEEx+WL: 55 ± 6% vs. 58 ± 9%), fat (WL: 20 ± 3% vs. 22 ± 4% and AEEx+WL: 26 ± 5% vs. 23 ± 8%), and protein (WL: 20 ± 2% vs. 19 ± 4% and AEEx+WL: 18 ± 3% vs. 18 ± 2%). Caloric intake was not significantly different between these two time periods in either group (WL: 1,406 ± 284 vs. 1,461 ± 253 kcal and AEEx+WL: 1,577 ± 220 vs. 1,488 ± 268 kcal). In addition, there were no significant changes in calcium or phosphorus levels in the WL group (768 ± 6 vs. 1,158 ± 306 vs. 1,043 ± 288 mg) or AEEx+WL group (798 ± 274 vs. 778 ± 329 mg and 1,196 ± 267 vs. 1,078 ± 319 mg).

The mean T scores (± SD measurement comparison to a young adult reference population) for BMD of the WL and AEEx+WL groups before each of the interventions are, respectively, −0.12 ± 1.04 and 0.18 ± 1.08 for the total body, −0.56 ± 1.28 and −0.61 ± 1.1 for L2–L4 spine, −0.77 ± 0.70 and −0.54 ± 1.14 for femoral neck, −1.29 ± 0.90 and −1.01 ± 1.48 for Ward’s triangle, and −0.01 ± 0.77 and −0.08 ± 1.17 for greater trochanter BMD. The mean T scores indicate that these women were within the normal range for young adults. There were four women (27%) in the AEEx+WL group who had T scores between −1 and −2.5 for the lumbar spine and seven (50%) women in this group with T scores between −1 and −2.5 for the femoral neck, representing low bone mass or osteopenia according to the World Health Organization (15). There were no T scores greater than −2.5 for these regions in the AEEx+WL group. In the WL group, three women (20%) had L2–L4 spine T scores between −1 and −2.5 or osteopenia and two women (13%) had L2–L4 spine T scores greater than −2.5 or osteoporosis (15). Five women (36%) in the WL group had femoral neck T scores between −1 and −2.5 or osteopenia.

Total body BMD significantly decreased in both the WL and AEEx+WL groups (Table 2, Fig. 1; both P < 0.05). Total body BMC significantly decreased in the WL group (2.391 ± 0.282 vs. 2.314 ± 0.279 kg; P < 0.05) but not in the AEEx+WL group (2.389 ± 0.315 vs. 2.360 ± 0.317 kg; not significant). Lumbar (L2–L4) spine BMD did not change significantly within groups after either WL or AEEx+WL (Table 2); however, the percent change between groups approached statistical significance (−1.0 vs. 1.4%; P = 0.07; Fig. 1). BMD of the femoral neck and Ward’s triangle significantly decreased in the WL group (P < 0.05), and the loss of BMD in the greater trochanter approached statistical significance in this group (P = 0.05). Femoral neck, Ward’s triangle, and greater trochanter BMD did not significantly change in the AEEx+WL group. The percent change in femoral neck BMD was significantly different (P = 0.01) between the WL and AEEx+WL groups (−2.6 vs. 1.2%, respectively; Fig. 1).

Body BMD, L2–L4 BMD, femoral neck, Ward’s triangle, or greater trochanter BMD did not significantly correlate with initial body weight in the women who completed the interventions (n = 30). BMD of the total body and L2–L4 spine correlated significantly with FFM (r = 0.44 and r = 0.40, respectively; both P < 0.05). All regions of the femur also correlated with FFM (femoral neck: r = 0.46, P < 0.01; Ward’s triangle: r = 0.37, P = 0.05; and greater trochanter: r = 0.50, P < 0.01). After adjustment for body weight, these relationships remained significant with the exception of Ward’s triangle. The relationships between total body, femoral neck, and greater trochanter BMD also correlate with lean tissue mass (all P < 0.05), whereas the lumbar spine and Ward’s triangle BMD approach statistical significance (P = 0.06 and 0.07, respectively). Total or regional BMD did not significantly correlate with fat mass in all women. BMDs of either the total body or regions of interest were not associated with aerobic capacity (L/min) in either group.

Changes in total body BMD did not correlate with absolute changes in body weight in the intervention groups. However, changes in body weight did correlate with changes in L2–L4 spine and greater trochanter BMD (both r = 0.38, P < 0.05) and tended to correlate with changes in the femoral neck and Ward’s triangle BMD (both r = 0.34, P = 0.08).

**DISCUSSION**

Because mechanical loading contributes to subsequent bone mass (20, 28), weight-bearing exercise is suggested as a therapy to increase BMD. In addition, the association between body mass and bone mass suggests that the stress to bones is greater with a

<table>
<thead>
<tr>
<th>Region</th>
<th>Before</th>
<th>After</th>
<th>Before</th>
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<tbody>
<tr>
<td>Lumbar spine (L2–L4)</td>
<td>1.133 ± 0.154</td>
<td>1.123 ± 0.166</td>
<td>1.127 ± 0.132</td>
<td>1.143 ± 0.133</td>
</tr>
<tr>
<td>Femoral neck</td>
<td>0.888 ± 0.084</td>
<td>0.865 ± 0.083*</td>
<td>0.908 ± 0.136</td>
<td>0.917 ± 0.127</td>
</tr>
<tr>
<td>Ward’s triangle</td>
<td>0.743 ± 0.117</td>
<td>0.711 ± 0.124*</td>
<td>0.769 ± 0.190</td>
<td>0.757 ± 0.185</td>
</tr>
<tr>
<td>Greater trochanter</td>
<td>0.789 ± 0.085</td>
<td>0.763 ± 0.102*</td>
<td>0.776 ± 0.126</td>
<td>0.775 ± 0.134</td>
</tr>
<tr>
<td>Total body</td>
<td>1.116 ± 0.084</td>
<td>1.097 ± 0.087*</td>
<td>1.140 ± 0.086</td>
<td>1.124 ± 0.087*</td>
</tr>
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Values are means ± SD in g/cm². Significantly different from before the intervention: *P < 0.05; †P = 0.05.
higher body weight, with BMD higher in obese than in lean premenopausal women (18). The present study demonstrates that a 6-mo WL program with or without AEx training results in a loss of total body bone mineral and that the addition of aerobic exercise to weight loss maintains regional bone density in obese postmenopausal women not on estrogen therapy.

Aerobic exercise training maintains or slows the loss of BMD in older women (5, 23), with both high- and low-impact exercise effective in maintaining BMD in early postmenopausal women (12). Whereas some studies demonstrate that bone loss in exercising postmenopausal women is similar to that in controls (3, 6, 26), others demonstrate that aerobic exercise training can increase BMD in postmenopausal women (8, 16). The combination of exercise and hormone-replacement therapy also increases BMD (16, 26). In the group of women who aerobically trained and lost weight in our study, BMD of the lumbar spine and femoral neck tended to increase and BMD in the other femoral regions was maintained, compared with women who lost a similar amount of weight but did not exercise. In contrast, all regional BMD measurements decreased in the women participating in WL alone, with significant losses detected in the femoral neck and Ward's triangle. Thus, although BMD did not significantly increase in the AEx+WL group, the maintenance of BMD when aerobic exercise is added to dietary-induced weight loss is clinically important to prevent loss of BMD in women with advancing age. Perhaps a longer training period or a larger sample size would permit detection of significant increases in BMD after AEx+WL. The addition of aerobic exercise to weight-loss regimens is important for cardiovascular benefits in obese postmenopausal women and at the same time may reduce the potential loss of regional bone density or more specifically that at the spine and hip, which are important clinical sites for fracture.

To our knowledge, only one other study combined a weight-loss program with aerobic exercise to examine changes in BMD (33). This 12-wk study demonstrated a decrease in lumbar spine BMD in both the diet and diet-plus-exercise groups (significant only in the latter group) in comparison to a control group of postmenopausal women. Total body BMD had a tendency to decrease, albeit not significantly, in both intervention groups. In contrast, we observed a significant decline in total body BMD in both groups (~1.7% with WL and ~1.4% with AEx+WL, Fig. 1) with no significant changes in lumbar spine BMD. These discrepancies could be attributable to the duration and amount of weight loss. Support for this rationale is provided by studies that examine the effects of weight loss alone on BMD (7, 14, 27). First, Ramsdale and Bassey (27) found no changes in BMD after 3 mo of caloric restriction that resulted in a modest weight loss in young women but a 0.7% loss in the total body BMD and a 0.5% loss in the lumbar spine BMD after 6 mo of caloric restriction (~3.3 kg). Second, severe dietary restriction with a weight loss of ~16 kg in 10 wk (7) and a 12-kg weight loss in 15 wk (14) resulted in significant 2.5 and 5.9% losses, respectively, in total body bone mineral. Thus the duration as well as the amount of weight loss appear to influence the loss of total body BMD.

An important issue when assessing bone changes with weight loss is the possible effect of tissue thickness loss on calculated bone loss. However, Jensen et al. (14) determined that the bone loss observed after weight loss by using a Hologic DEXA was not explained by an error effect by including a control group measured with varying thicknesses of lard. Moreover, recent reports have found no differences in total body BMD or total and regional BMC when packets of lard are placed on the ventral side or on the thighs and abdomen of human subjects using Lunar DPX and DPX-L systems with similar software and analyses programs (22, 32). This would in effect mimic changes in tissue thickness with a weight-loss program in obese humans and illustrate confidence in the Lunar system in the examination of BMD with tissue thickness and fat changes.

Another potential confounding factor is a change in calcium intake because a reduced absolute calcium intake was related to the change in total BMD after weight loss (27). In our study, calcium intake was similar between groups and there were no changes in calcium intake after either intervention. Thus this cannot explain the greater bone loss observed in the WL group. However, both groups were below the 1,500 mg/day recommended intake for postmenopausal women not on hormone-replacement therapy (24). We do not know whether an adequate calcium intake would have prevented the bone loss in the WL-only group. There were also no changes in phosphorus levels...
or dietary composition. This lack of change in dietary composition indicates that the dietary intake of nutrients important for normal bone metabolism most likely did not influence our results. The influence of estrogen in stimulating bone accretion was also not a factor because none of the women in the present study was on hormone-replacement therapy.

The lack of significant associations between body weight and bone mass in the women who completed the interventions in this study may be a result of the small sample size. Others have reported associations between body weight and bone density (4, 19). Total body bone mineral is significantly correlated with body weight as well as total body fat mass (19). Spinal bone density is also correlated with body weight and fat mass (4). When the baseline data were analyzed with 41 vs. 30 women (including all women who initially entered the study), significant positive associations between body weight and regional BMD were observed, suggesting that a larger sample size may be necessary to detect these relationships. It also is likely that homogeneity of obesity (percent fat) in these women masks the associations between body weight and bone mass. In a large cohort of older women (11), only weight exerted independent effects on bone density at weight-bearing sites, whereas adiposity was primarily related to BMD through its contribution to weight. We demonstrated significant associations between FFM and the total body, lumbar spine, and all regions of the femur in these obese postmenopausal women, which confirm previous reports of the positive associations between FFM and total body and spinal bone density in pre-and postmenopausal women (4, 19).

In conclusion, these results suggest that weight loss with or without exercise is accompanied by a loss of total body bone density in obese postmenopausal women. The observations that WL alone but not AEx+WL tends to result in a loss of BMD of the lumbar spine and significantly decreases BMD in the femoral regions suggests that the addition of weight-bearing aerobic exercise in WL programs may be beneficial with respect to maintaining regional bone density and thus may reduce risk for bone loss in dieting older women not on hormone-replacement therapy.

Our appreciation is extended to those women who participated in this study. We are grateful to Kelly Fitzpatrick, who assisted in the training of the women; to Linda Bunyard for dietary assistance; to Dr. Marc Hochberg for insightful comments; and to Dr. Andrew P. Goldberg for support.

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