Journal of Human Kinetics volume 25 2010, 41-47
Section II - Exercise Physiology & Sports Medicine DOI 10.2478/v10078-010-0030-5

Is Walking Exercise a Protective Factor for Osteoporosis in Young Obese Women?

by
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Twenty obese (BMI>30) women with an average age of 22.00 ± 1.50 years volunteered to participate in this study. They were randomly assigned to exercise (n=10) and control (n=10) groups. Before and after the training program, both groups were evaluated for anthropometric measurements and blood analysis. Bone mass density (BMD) was evaluated by using dual-energy X-ray absorptiometry (DXA) at the hip and spine (L_2 – L_4). Each walking session was 30 min walking at 50-75% of maximal heart rate, 3 days per week, for 2 months. After 2 months, the exercise group showed no significant effect on BMD at the spine (L_2– L_4) and hip ($p>0.05$). However, there was a slightly increase in BMD at both regions, while the control group did not. Also, no change was observed in bloods factors . Percent body fat, fat mass and lean mass changes in response to training were significant in the exercise group (all $p=0.000$). As a result, it may be suggested that walking programs of longer duration and higher intensity can influence BMD and related factors in obese girls, however to achieve this result, more studies are needed.

Key words: bone mass density, walking exercise, bone loss, obese women.

Introduction

Osteoporosis is a condition of decreased bone mass and density. This leads to fragile bones which are at an increased risk for fractures (Sambrook and Cooper, 2006). Although symptoms of osteoporosis do not generally occur until after menopause, recent evidence suggests that bone loss starts much earlier in life and it may be associated with an increasingly sedentary lifestyle (Hata et al., 2003). Over the past 10 years, osteoporosis has emerged as a major clinical challenge for physicians, health care professionals and patients, related to its prevalence, morbidity and morality of associated fractures. The incidence of osteoporotic fractures have been increasing, and half of the elderly female population in most Asian and western countries are affected. It was estimated that in the years to come, new cases would be added yearly to the existing ones (Cummings and Melton, 2002). In spite of development of increasing numbes of those diagnosed with osteoporosis, still the preventive measure of osteoporosis is neglected and the already staggering medical, social and economic costs can be expected to increase unless effective prophylactic and therapeutic regimens are developed (Abdy, 1998).

Epidemiological evidence suggests that obesity has an effect on bone density and it appears to be protective for fracture risk in women (Bener et al., 2005). On the other hand, body weight impacts bone density and is, therefore, an important risk factor for osteoporosis. Obesity has been identified as a risk factor for many illnesses. The consequence of excessive weight can have a profound negative effect on bones and joints (Cobayashi et al., 2005). An increased body mass index (BMI) has been associated with many orthopedic conditions, such as arthritis,

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Authors submitted their contribution of the article to the editorial board.
Accepted for pinting in Journal of Human Kinetics vol. 25/2010 on June 2010.

osteoporosis and joint immobility (Yanai et al., 1997). However, results from these types of investigations are not very conclusive. Haffner and Bauer (1992) reported that excess androgencity only partially explains the relationship between obesity and bone density in premenopausal women. Further, obesity can be considered as protective factor for osteoporosis, improving bone mass and maintaining higher levels of estrogen during menopause (Albala et al., 1996). Another study performed in Greece reported that in postmenopausal women, obesity is inversely associated with BMD and did not find a simultaneous increase in BMD and BMI which was shown to influence the rate of bone turnover in a negative manner (Ribot et al., 1987). Izmozhrova (2008) also showed that obese postmenopausal osteoporosis had significantly higher frequency of arterial hypertension, chronic heart failure, osteoarthritis and glucose metabolism disorders than those with normal body mass. Obese persons also had more severe menopausal symptoms than women with normal body mass(Izmozherova and Popov, 2008). The best treatment for osteoporosis is weight control (Bacon et al., 2004). This condition is more common among obese women. Because of all the negative physiologic and psychosocial consequences associated with an excessive amount of body fat, obese people frequently try to lose weight (Pennington 1953; Klem et al., 1997). Although reduction of overweight reduces the risk for several chronic diseases, it may lead to bone loss (Rosen and Bouxsin, 2006).

Research indicates that maintaining regular physical activity throughout life may slow bone loss, whereas bed rest or weightlessness increases bone loss (Alioa et al., 1987; Iwamoto et al., 2001). The level of mechanical loading is one of the many factors that contribute to the homeostasis of the skeletal system (Ehrlich and Lanyonl, 2002). Several studies examining the effects of aerobic exercise on BMD in older women have utilized different modes, frequency, intensity, and duration of exercise with disparate results. While various forms of weight–bearing activity may slow loss of BMD, or possibly increase BMD at various skeletal sites through mechanical loading of bone, walking as an exercise intervention may be accepted by a board range of patients(Palombaro, 2005). Evidence in the literature links physical inactivity to bone loss. Puntila et al. (2001) reported an annual loss of lumbar spine BMD to be 23% less in physically active peri-and postmenopausal women. This loss is 52% less for lumbar BMD in women reporting walking as their sole form of weight-bearing exercise. Thorsen et al. (1996), similarly, found that brisk outdoor walking for 90 minutes at 50% of VO_2max had significant positive effect on the markers of collagen metabolism. Consequently, there may be a therapeutic role for walking exercise in the prevention and management of osteoporosis. Furthermore, it is popular and feasible for the obese population. Although exercise is generally recognized as beneficial to bone and as a way to help protect against bone loss, it has not been adequately defined for obese women in particular. Given the apparent lack of conclusive data in the relationship between the change in body overweight and bone mass statues, the aim of this prospective study was to assess whether walking exercise maintain BMD or restored bone loss among obese sedentary young women.

Materials and methods

Subject and study design

Twenty untrained obese (BMI>30) women with an average age of 22.00 ± 1.50 years volunteered to participate in this study. Then they were randomly assigned to exercise and control groups (Experimental n=10, Control n=10). Written informed consent for all procedures was obtained from all participants prior to entering the study, and this study was approved by the local Committee of Ethics. The criteria for the invitation were the willingness to participate, clinically healthy (no cardiovascular, musculoskeletal, respiratory, or other chronic diseases that might limit training or testing), no menstrual irregularities, not using medication that affect bone mass density and no beta-blockers sedentary life style (no regular sports activities for at least 2 years), nondieting, nonsmoking, and no apparent occupational or leisure time responsibilities that impede their participation. The following measurements were made at baseline prior to the start of the exercise program and at after completion of the 2- month training program.

Dietary Intake

Caloric expenditure was calculated based on the weight of the subject. To minimize any affect that dietary composition might have on the measured metabolic variables in the experimental group, at the initiation of the study all subjects were instructed on

the American Health Association (AHA) diet by a registered dietitian. The composition of this diet was 50-55% carbohydrate, 15-20% protein, <30% fat (New et al., 1997). The subjects were asked to maintain this diet composition throughout the study's duration (2 months). Compliance was monitored by review of 7-day food records taken every week.

Anthropometric measurement

Body weight and height were recorded and body mass index (BMI) was calculated as weight (kg) divided by height (m) squared. Fat mass, percent body fat and lean mass were assessed with bioelectrical impedance equipment (BIA-106, RJL Systems, USA). In addition, all subjects were weighed every week so that no subject gained or lost > 2.2 kg body weight over the entire study period.

BMD assessment

The main endpoints of the study were the change in bone mass density of the hip and the lumbar spine ($L_2 - L_4$). BMD (g/cm^2) was measured with the dual X-ray absorptiometry scans (DXA) (Norland XR-26, WI, USA). All the scanning and analyses were done by the same operator. The in-vivo day-to-day (coefficient of variation) BMD measurements in our laboratory ranged from 0.7 to 1.7%. The scanner was calibrated daily, and its performance was followed with our quality assurance protocol. There was no significant machine drift during the study period.

Blood analysis

Blood samples were collected after an overnight fast (>12 h) in a sitting position and centrifuged at 1500 rpm for 30 minutes at 4^o C within 2 h. Serum samples from each participant were stored frozen at -20^o C until analyzed. Serum estrogen level was assessed by radioimmunoassay (Amersham Biosciences, Piscataway, NJ, USA) in follicular stage in each subject's menstrual cycle, and serum calcium and phosphorus levels were measured by standard automated laboratory techniques.

Exercise program

The program included warming-up phase for 5 minutes of stretching exercises, 30 minutes walking at 50-75% of maximum heart rat and cooling-down phase for 5 minutes of stretching, three times a week for 2 months. Stretching exercises were performed for the arms, legs, back and stomach. A target heart rate range between 50-75% of age adjusted maximum heart rate intensity was calculated by each walker from her age and waking supine resting heart rate (Karvaonen and Vuorimaa, 1988). Heart rate was measured with an electronic heart rate monitor (Sport Tester PE, Polar Electro, Finland). The exercise program was accompanied by music. All sessions were supervised by a professional exercise physiologist leader.

Statistical Analysis

The data were analyzed using the SPSS statistical package (SPSS 13 for Windows; SPSS, Chicago, USA). Mean and standard deviation (SD) was used as descriptive statistics. Student's t-test was used for normally distributed variables. Paired t-test was used to assess the change in BMI, body weight, serum calcium, phosphorus, and estrogen before and after exercise intervention. One-way analysis of variance (ANOVA) was used to detect significant change and differences in response over time between groups. The final level of significance was accepted as $p<0.05$ for all comparisons.

Results

Twenty subjects (100%) completed the training program. No major change in menstrual status was observed during the study. Table 1 shows the physi-

Table 1

Changes in Anthropometric variables in pre- and post-test exercise (X ±SD)

variable	**Obese** (Exe)		**Obese** (Con)		P value
	pre	**post**	**pre**	**post**	
Age (year)	22.22 ± 1.98	-	22.67±1.50	-	
Height (cm)	157.78±5.11	-	159.11±1.50	-	
Weight (kg)	74.98± 8.11	73.27±7.74	78.11±10.88	78.06±10.14	0.000*
BMI (kg/m^2)	30.20 ± 1.83	28.88±2.10	30.93±3.57	30.41±3.05	0.000*
Lean mass (kg)	43.27±5.25	44.38±6.21	43.86±6.03	43.25±6.67	0.000*
Fat mass (kg)	29.11±4.54	27.17±6.30	31.16±6.28	31.42±7.13	0.000*
% Body fat	38.80±3.97	36.35±6.84	39.97±3.51	39.00±5.16	0.000*

Table 2

Changes in BMD and blood variables in pre- and post-test exercise (X ±SD)

variable	**Obese** (Exe)		**Obese** (Con)		
	pre	**post**	**pre**	**post**	**P value**
Hip BMD (g/cm²)	0.967± 0.106	0.983± 0.095	0.958 ± 0.085	0.945 ±0.092	0.868
Spine (L_2– L_4) BMD (g/cm²)	1.113 ±0.167	1.147 ±0.155	1.137±0173	1.128 ±.17	0.562
Estrogen (pg/ml)	30.42 ±15.60	46.99 ±18.55	34.35 ±24.17	39.51±17.24	0.967
Calcium (ml/dl)	9.47 ±024	9.42 ±028	9.50 ± 0.46	9.38 ±0.306	0.410
Phosphorous (ml/dl)	3.80 ±039	3.65 ±063	3.84 ±0.46	3.53 ± 0.44	0.939

cal characteristics of the study subjects (pre, post study). Initially, there were no significant differences in mean age, height and BMI between the two groups. Changes in percent body fat (2.2%), fat mass (2%) and lean mass (1.1%) in response to training were significant in the exercise group. The lean mass in the exercise group was significantly increased, but the percent body fat and fat mass were significantly decreased (all $p = 0.000$). The mean body weight was 1.3% lower at the end of the study. Body mass index (BMI) in the exercise group (2.3%) significantly differed from pre-test intervention, compared with the control group ($p<0.05$).

All subjects showed normal ranges of serum calcium, phosphorus and estrogen levels at baseline, and analysis of data showed that post-test differences between the groups were not significant *(p>0.05).* This lack of change in serum calcium, phosphorus and estrogen indicates that they are not important factors for normal bone metabolism and did not influence our results. No significant change was observed in BMD of the spine (L_2– L_4) and hip in the exercise group *(p<0.05).* However, the exercise group experienced slightly increased BMD at both regions, while the control group did not. The changes in BMD and serum samples are given in Table 2.

Figures 1, 2 show the change in hip and spinal (L_2– L_4) BMD over the study period and the differences in both the experimental and control groups.

Discussion

Exercise was reported to play an important role in maintaining or increasing bone density. With exercise training, skeletal involution might be reversed without the untoward side effects seen in medical fitness and quality of life (Caplan et al., 1999; Zylstra et al., 1989). Some of related studies, especially those with cross-sectional design, have demonstrated a significant relationship between physical activity and bone mass. Physically active subjects have significantly higher BMD than their age matched sedentary counterparts (Berard et al., 1997). Epidemiological, clinical and experimental exercise studies have suggested that exercise enhances bone development and augments bone mineral density during adolescence and may prevent osteoporosis and fractures during old age (Elizabeth and Bess, 1994; Lord et al., 1996). However, exercise will only play a part in preventative strategy to decrease the incidence of osteoporosis if the amount and type of exercise needed to be effective is perceived as attainable by the

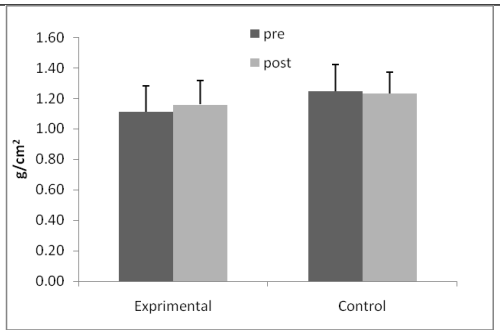

Figure 1

Change (mean ±SD) from baseline in spine (L_2– L_4) BMD (g/cm²) during the study period (pre, post study) between the experimental and control groups

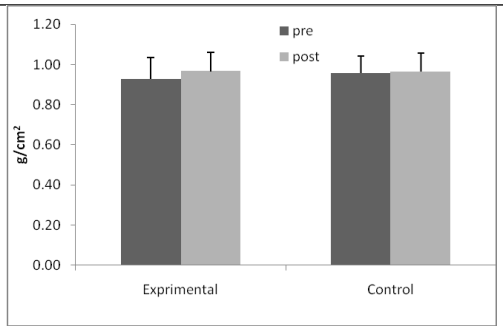

Figure 2

Change (mean ±SD) from baseline in Hip BMD (g/cm²) during the study period (pre, post study) between the experimental and control groups

majority of young-aged and older women. Though it may be desirable to design a regimen likely to be maximally effective at a number of different skeletal sites, such a program may be difficult to implement. Walking may not involve high loading but it is accessible to a majority of all women (Kato et al., 2005).

Comparisons among studies evaluating the effect of walking on bone density at various skeletal sites are limited by differences in methods to measure walking activity of the study population. A recent study by Mulhim et al. (2004) shown that 30 minutes of walking at 1.5 km/hour increased lumbar spinE and femoral BMD and decreased total body weight in sedentary woman. Among postmenopausal women, Nelson (1991) demonstrated the beneficial effects on BMD and body weight in walking at 75-80% of maximum heart rate for 50 minutes, 3-4 times per week, wearing a leaded belt. Chow et al. (1987) similarly reported an increase in bone mass as a result of treadmill exercise at 80% of the maximum heart rate during walking exercise. These finding confirm that bone maintenance effect of exercise during the premenopausal and postmenopausal period may be an essential factor, making a favorable difference compared with sedentary women.

The degree and extent of any exercise should be adapted to the age, the physical ability and the skeletal condition of the individual. Dalen and Olsen (1974) found no significant change in bone mineral content after a three-month walking program. Cavnaugh and Cann (1988) also reported that aerobic exercise, such as a walking program, did not prevent bone loss in postmenopausal women. However, Hotori et al. (1993) showed that walking for 30 minutes above the anaerobic threshold (AT) was effective in increasing BMD, whereas exercise below the AT was not. Martin and Notelovitz (1993) similarly observed that walking speeds of less than 6.4 km did not increase BMD.

In our patients, we shown that a simple 30 minute walking program at the range of 50-75% maximum heart rate was not enough to significantly change BMD in obese exercise women. The lack of significant improvement in bone density in the exercise group, compared with the control group, was most likely due to exercise being of insufficient intensity and/or duration of time or frequency. On the other hand, such results are likely due to bone being more compact and less quickly reactive to treatment. Several well-controlled studies similarly supported a positive effect of exercise on BMD, indicating either less reduction or more gain in BMD for the training group (>1%) compared with the control group (<1%) (Wolff et al., 1999). Some studies also showed that exercise of relatively short duration did not enhance BMD, but rather a decline in BMD was reported in non-exercise groups (Wavell et al., 2001). Result of the blood parameters also showed that neither estrogen nor calcium and phosphorous levels were significantly altered as a result of the two-month training regimen, suggesting that estrogen, calcium and phosphorous did not mediate the observed skeletal changes in the both groups.

However, the study finding revealed that 2 months walking exercise was of sufficient duration and intensity to result in significant improvements in all components of body composition in the obese exercise group. The decrease in body weight in the exercise group accounts for the responses of body weight to walking exercise, compared with the control group. Thompson et al. (2004) similarly indicated an inverse association between body composition and daily walking exercise in middle-aged women. Furthermore, there was a relation between weight change and an increase in BMD in both regions. In fact, the exercise program decreased fat mass in the exercise group without any losses at BMD. Ryan et al. (1998) also demonstrated that weight-bearing aerobic exercise, such as walking, may be beneficial with respect to maintaining regional bone density, in spite of reducing body weight, and thus may decrease risk for bone loss in older women.

In conclusion, this study demonstrated that walking exercise may reduce the risk of bone loss, even though it did not significantly increase bone density. As a result, it may be suggest that a walking program with longer duration and higher intensity, can be effective on BMD and related factors in obese girls, however, to demonstrate this result, more studies are needed.

Also, educating and raising awareness of osteoporosis among young women is very important and needed.

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Acknowlegdement

The work was supported by grants from the University of Guilan. The authors gratefully acknowledge all subjects who cooperated in this investigation.

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