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ORIGINAL COMMUNICATION

Low bone mass in premenopausal chronic dieting obese women

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Background: Obese premenopausal women are thought to be at low risk for osteoporosis due to increased body weight and effects of estrogen on weight-bearing bone.
Objective: To examine the effect of restrained eating on obese women, we examined bone mineral density (BMD) and content (BMC) of the spine and femur in obese women who were restrained eaters, with emphasis on the relationship between BMC and determinants of bone mass, and current eating behaviors, dietary intake, physical activity, and indices of calcium regulation, bone metabolism, stress and inflammation.
Design: A total of 78 obese, Caucasian, female, restrained eaters, ages 30–45 y, were enrolled in a weight lose program. Height, weight, bone turnover markers, serum parathyroid hormone (PTH), cortisol, c-reactive protein (CRP), dietary intake, eating behaviors, physical activity, and BMD and BMC were measured.
Setting: This study was conducted at the University of California, in Davis, CA, USA.
Results: In all, 31% of women had osteopenia or osteoporosis (OSTEO). In the OSTEO group, 87.5% of women had osteoporosis or osteopenia of the lumbar spine and 12.5% of the women had osteoporosis or osteopenia in femur. A significant positive correlation between BMC and energy expenditure ($r = 0.256$), and a significant negative correlation between BMC and number of times on a weight loss diet ($r = -0.250$) and cognitive restraint ($r = -0.239$) were observed. No significant differences were observed between OSTEO women and nonosteoporotic women for current eating behaviors, dietary intake, physical activity habits, bone turnover, calcium regulation, stress, or inflammation.
Conclusions: Obese restrained eaters are at risk for low bone mass. Prior dieting may be responsible. Chronic dieters should be encouraged to decrease their dietary restraint, develop healthy eating habits and increase physical activity.
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Keywords: bone mineral density; obesity; osteoporosis; restrained eating

Introduction

Osteoporosis is a major public health threat, affecting more than half of North American women, with Caucasians at particularly high risk (Christiansen *et al*, 1987). It is a debilitating disease, although generally preventable. Since there is no cure for osteoporosis and it can develop and progress undetected for decades, early diagnosis and treatment is critical in its prevention.

Measurement of bone mineral density (BMD) is the best tool available to assess osteoporotic fracture risk, and BMD measurements of the skeleton are the accepted clinical tool for diagnosis of osteopenia and osteoporosis (Kanis, 2002). Dual X-ray absorptiometry (DXA) is used to assess bone mineral content (BMC) of the entire skeleton and specific sites, and, when divided by the area measured, can be used to derive a value for BMD. Both the lumbar spine and femur are commonly accepted measurement sites, for the diagnosis of osteopenia and osteoporosis (Kleerekoper & Nelson, 1997).

Obese women are thought to be at decreased risk for osteoporosis due to the effects of increased body weight on BMD. Premenopausal women are similarly at reduced risk due to the effects of estrogen on bone. Therefore, obese premenopausal women are unlikely candidates for osteoporosis screening. In contrast, restrained eaters (chronic dieters) are at increased risk for osteoporosis as research

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indicates an association between high levels of restrained eating (conscious limitation of energy intake) and reduced bone mass in nonobese women (Barr *et al*, 1994; Van Loan & Keim, 2000; Van Loan *et al*, 2000). It is not known if obese restrained eaters are at increased risk. Additionally, moderate energy restriction may increase bone resorption in obese postmenopausal women (Ricci *et al*, 2001).

This investigation was a smaller component of a larger wellness and healthy living project conducted with premenopausal obese women. Our purpose was to examine the relationship between chronic dieting behavior and bone health, for example BMD and BMC, physical activity, dietary intake, bone metabolism, and markers of stress and inflammation.

Methods

Procedure

Subjects were recruited from Davis, California and the surrounding area. Participants were screened prior to enrollment in the protocol and met the following criteria: Caucasian; female; age 30–45 y; body mass index (BMI) 30–45 m/kg^2; nonsmoker; not pregnant or lactating; restraint scale (Polivy *et al*, 1988) score >15, indicating a history of chronic dieting; no recent myocardial infarction; no active neoplasms, no Type I diabetes or insulin-dependent Type II diabetes, nor history of cerebrovascular or renal disease. During the screening process five women were excluded due to perceived inability to participate effectively in a group. Accurate diagnoses could not be determined in the limited contact, but the five were excluded based on the following concerns: depression, borderline intellectual functioning, narcissistic personality disorder, and alcoholism. All other women who completed the screening process were enrolled. The study was approved in accordance with the guidelines of the Human Subject Review Board of the University of California, and written informed consent was obtained.

Anthropometry

Weight was measured to the nearest 0.1 kg on a calibrated electronic scale without shoes and wearing hospital scrubs. Height was measured, without shoes, to the nearest 0.1 cm using a wall-mounted stadiometer.

Bone parameters

BMC and BMD were assessed with a Lunar fan-beam Dual energy X-ray Absorptiometer (DXA) Prodigy Model (GE Medical Corp., Madison, WI, USA). Analysis was performed using software version 2.05. The Lunar DXA (Prodigy model) uses an array detector composed of cadmium–zinc–telluride (CZT) elements and directly converts X-rays into a digital signal. The direct conversion results in a higher efficiency and beam penetration at a lower dose to the subject. Standard calibration procedures were followed in accordance with the manufacturer's specifications and were conducted on a daily basis. Internal laboratory calibration of the instrument demonstrated a reliability of 0.08% coefficient of variation for BMD. Reliability for the estimate of fat mass, expressed as the coefficient of variation, was 1.0%. To maintain quality control all scans were analyzed by the same operator.

Dietary intake

The Block 98 Food Frequency Questionnaire provided information about the previous year's dietary intake including nutrients from food; nutrients from vitamin supplements; amount and sources of fiber; and the frequency of consumption of various food groups. Nutrients important to skeletal development and maintenance, such as calcium, magnesium, and vitamin D, were estimated from the food frequency.

Eating behaviors

The Three Factor Eating Inventory (TFEI) (Stunkard & Messick, 1988) assessed eating habits, including cognitive restraint of eating, disinhibition (the loss of control that follows self-imposed rules), and susceptibility to perceptions of hunger. All three subscales of the TFEI demonstrate high internal consistency (Cronbach's α for all ≥ 0.79) (Allison *et al*, 1995) as well as high test–retest reliability (0.91) (Laessle *et al*, 1989). The Eating Disorders Inventory-2 (Garner, 1991) assessed attitudes and behaviors towards weight, body shape, and eating, as well as more general psychological characteristics that are clinically relevant to eating disorders. Reliability (internal consistency), test–retest reliability, concurrent validity and discriminant validity have been determined for all subscales (Allison, 1995).

Physical activity

The Stanford Seven-Day Physical Activity Recall (Blair *et al*, 1985) was used to evaluate leisure time and occupational activities. Data were collected by interview and provided an estimate of energy expenditure. To eliminate interexaminer error and reduce variability in the data, all interviews were conducted by the same examiner.

Biochemical markers

Assessment of biochemical parameters included parathyroid hormone as a marker of calcium regulation, osteocalcin, and pyridinoline crosslinks as markers for bone turnover, cortisol as a marker of stress, and c-reactive protein as a marker of inflammation.

Parathyroid hormone was assessed in serum using an enzymatically amplified two-step sandwich-type immunoassay (ELISA, Diagnostic Systems Laboratories, Inc., Webster, TX, USA). In the assay, standards, controls, and unknowns

are incubated with biotinylated antiparathyroid hormone of defined specificity in microtitration wells precoated with an affinity-purified goat anti-human PTH antibody of defined and unique epitope specificity. Coefficient of variation (CV) on duplicate measurements was 5.14%.

Osteocalcin, a vitamin K-dependent, calcium-binding protein, is a major component of the noncollagenous bone matrix. It is synthesized by osteoblasts in bone and only small amounts are synthesized by dentin; synthesis does not occur in nonbone tissue. Therefore, serum osteocalcin originates exclusively from bone and measurements of serum osteocalcin provide a marker of osteoblast activity. Serum osteocalcin was measured via an enzymatically amplified two-step sandwich-type immunoassay (ELISA, Diagnostic Systems Laboratories, Inc. Webster, TX, USA). In the assay, standards, controls, and unknowns are incubated with antiosteocalcin polyclonal detection antibody labeled with the enzyme horseradish peroxidase microtitration wells coated with an affinity-purified antiosteocalcin mouse monoclonal antibody. CV on duplicate measurements was 6.79%.

Serum pyridinoline crosslinks (Pyd) were also assayed. Structural collagens such as type I and type II are present in bone and cartilage and are crosslinked within their α-chains and between adjacent molecules to provide rigidity and strength to the resulting collagen fibril. When bone and cartilage are degraded, pyridinoline is released into circulation and thereby serves as a marker for bone resorption. The serum Pyd assay is a competitive enzyme immunoassay in a microtiter plate format, (Metra Biosystems, Inc. Mountain View, CA 94043, USA). CV on duplicate measurements was 8.57%.

The cortisol assay was a solid-phase, chemiluminescent immunoassay run on an Immulite (Diagnostic Products Corporation, (DPC) Los Angeles, CA, USA). The assay uses a polyclonal rabbit antibody specific for cortisol. Two separate controls were used. Con6 (DPC, Inc.) Lot #018 is a tri-level control. The low-level control has an expected range of 3.3–5.1 μg/dl and the actual values were mean = 4.2 μg/dl, sd = 0.2 μg/dl, CV = 5.45%. The mid-level control expected values were 9.7–14.7 μg/dl and the actual values were mean = 12.7 μg/dl, sd = 1.4 μg/dl, CV = 10.97%. The high-level control expected range is 24.0–36.0 μg/dl and the actual values were mean = 31.3 μg/dl, sd = 3.4 μg/dl, CV = 10.8%. The second control was from BioRad (Richmond, CA, USA), lot #40000, and was also a tri-level control. The low-level control has an expected range of 1.8–4.2 μg/dl and the actual values were mean = 2.7 μg/dl, sd = 0.2 μg/dl, CV = 6.55%. The mid-level control expected values were 14.0–25.0 μg/dl and the actual values were mean = 19.9 μg/dl, sd = 1.3 μg/dl, CV = 6.59%. The high-level control expected range is 26.0–42.0 μg/dl and the actual values were mean = 40.0 μg/dl, sd = 4.8 μg/dl, CV = 12.0%.

C-reactive protein is a two-site chemiluminescent immunometric assay and uses a ligand-labeled monoclonal antibody and separation by antiligand-coated solid phase (Diagnostic Products Corporation, Los Angeles, CA, USA). The control was bi-level (CRP Control Module, DPC, Lot #0002). The low-level control has an expected range of 1.0–1.24 mg/dl. The actual values were mean = 1.09 mg/dl, sd = 0.05 mg/dl, CV = 4.90%. The high-level control expected range is 13.5–18.1 mg/dl. The actual values were mean = 15.6 mg/dl, sd = 1.4 mg/dl, CV = 8.96%.

Statistics

Student's *t*-test was used to compare characteristics between the two groups using Statistica Version 5.1 (Statsoft, Inc., Tulsa, Okl, USA). Values are presented as group means and standard deviations. For all statistics, a *P*-value of 0.05 was considered significant.

Results

Subjects

Physical characteristics can be found in Table 1. The average age was 39.3 ± 4.5 y (standard deviation), and ranged from 30–45 years. The average weight was 103.8 ± 25.6 kg and average BMI was 37.6 ± 3.8 kg/m^2.

Prevalence and location of osteopenia/osteoporosis

In all, 31% of the subjects had low bone mass based on the World Health Organization *z*-score values for population norms for BMD (WHO Study Group, 1994) (Figure 1). For the women with low bone mass, the osteopenia or osteoporosis was observed in the lumbar spine in 87.5% of the women, whereas only 12.5% of the women with low bone mass met the WHO criteria for osteopenia/osteoporosis based on measurements of the femur.

Group differences

There were no significant differences between 'normal' women and those with osteopenia or osteoporosis with

Table 1 Subject characteristics ($n = 78$)

	Average
Age (y)	39.3 ± 4.5
Weight (kg)	103.8 ± 25.6
BMI (kg/m^2)	37.6 ± 3.8
Fat-free mass (kg)	46.0 ± 4.6
Fat Mass (kg)	47.0 ± 7.2
Total body BMC (g)	2841.8 ± 386.2
Total body BMD (g/cm^2)	1.21 ± 07
Spine BMC (g)	69.72 ± 11.05
Spine BMD (g/cm^2)	1.27 ± 0.14
Right femur BMC (g)	35.70 ± 4.09
Right femur BMD (g/cm^2)	1.12 ± 0.11

BMI: body mass index; BMC: bone mineral content; BMD: bone mineral density. Values are mean ± s.d.

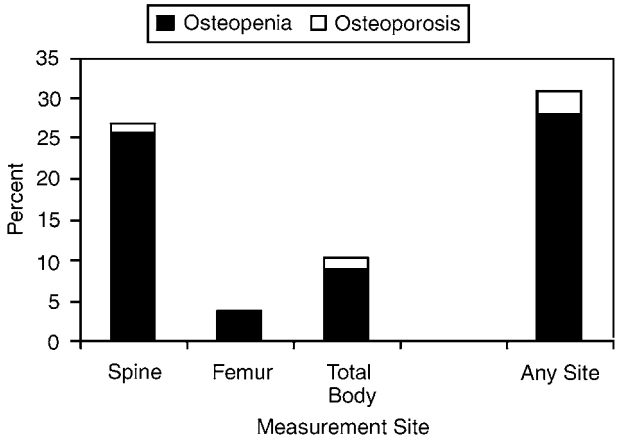

Figure 1 Prevalence of reduced bone density by severity and location.

Table 2 Anthropometric and bone parameters in obese normal and osteoporotic/osteopenia (OSTEO) women

	Normal (n = *54*)	*OSTEO* (n = *24*)	*% Difference*
Height (cm)	166.5 ± 5.7	165.2 ± 4.7	
Weight (kg)	103.6 ± 27.0	103.8 ± 23.1	
BMI (kg/m^2)	37.3 ± 3.7	36.9 ± 3.8	
Fat-free mass (kg)	45.9 ± 4.1	46.2 ± 5.8	
Fat mass (kg)	46.9 ± 7.1	47.2 ± 7.6	
Total body BMC (g)	2925.6 ± 378.7	2653.4 ± 339.7*	−9.3%
Total body BMD (g/cm^2)	1.24 ± .06	1.16 ± 0.06*	−6.5%
Spine BMC (g)	73.75 ± 9.26	60.66 ± 9.32*	−17.8%
Spine BMD (g/cm^2)	1.34 ± 0.10	1.12 ± 0.10*	−16.4%
Right femur BMC (g)	36.60 ± 3.67	33.79 ± 4.40*	−7.7%
Right femur BMD (g/cm^2)	1.15 ± 0.10	1.06 ± 0.11*	−7.8%

BMI: body mass index; BMC: bone mineral content; BMD: bone mineral density. Values are mean ± s.d.*Indicates a significant difference ($P < 0.05$).OSTEO group includes all women who are OSTEO at one or more sites, as per the WHO standard for population norms: osteopoenia is between −1 and −2.5 standard deviations below that of a 'young normal' adult and osteoporosis is more than −2.5 s.d. below that of a 'young normal' adult.

respect to height, weight, (BMI), fat mass, or fat-free mass. However, there were significant between-group differences in BMC and BMD as would be expected due to OSTEO grouping (Table 2). Total body BMC was 9.3% lower and total body BMD was 6.5% lower in the low bone mass group compared to the women with normal BMC and BMD values; spinal BMC was 17.8% lower and spinal BMD was 16.4% lower; femoral BMC was 7.7% lower and femoral BMD was 7.8% lower. When eating behaviors, physical activity habits, and reproductive health history were examined, no significant differences were observed between groups (Table 3). Similarly, when bone-related dietary intakes and biochemical indices were compared, no significant differences were observed between the women with low bone mass and those with normal bone mass (Table 4).

Table 3 Reproductive health history for obese normal and OSTEO women

	Normal (n = *54*)	*OSTEO* (n = *24*)
No of menses/y	10.8 ± 3.2	10.3 ± 3.9
Oral contraceptive use (%)	88.3	83.3
Age at first oral contraceptive use (%)	18.7 ± 2.5	20.7 ± 5.5
Average no. of pregnancies	2.5 ± 1.6	2.3 ± 1.5
No. of children/women	1–7 range	1–6 range
Ever pregnant (%)	78.3 (47/60)	88.9 (16/18)

Values are mean ± s.d. unless otherwise noted. No significant differences between groups ($P > 0.05$).

Table 4 Biochemical indices of bone turnover, stress, and inflammation obese normal and OSTEO women

	Normal (n = *54*)	*OSTEO* (n = *24*)
Osteocalcin (ng/ml)	7.39 ± 2.3	8.07 ± 3.0
Pyridinoline x-links (pg/ml)	1.56 ± 0.5	1.47 ± 0.5
PTH (pg/ml)	24.4 ± 12.8	22.2 ± 15.4
Cortisol (μg/dl)	11.4 ± 4.4	10.9 ± 2.3
C-reactive protein (mg/dl)	0.5 ± 0.0	0.7 ± 1.0

Values are mean ± s.d. PTH is parathyroid hormone. No significant differences $P > 0.05$.

Biochemical indices

Associations. A significant positive correlation was found between spine BMC, femur BMC, and several anthropometric variables, including weight, height, fat mass, and fat-free mass (Table 5). A significant positive correlation was also observed between spinal BMC and energy expenditure ($r = 0.256$) and femoral BMC and energy expenditure ($r = 0.316$). Energy expenditure was estimated from the Block Food Frequency Questionnaire 98. There were significant negative correlations between the number of times on a weight loss diet and femur BMC ($r = -0.250$) and between cognitive dietary restraint and femur BMC ($r = -0.239$), but no significant associations were observed for spine BMC and these parameters. No associations were observed between BMC and any of the bone-related dietary nutrients, or between BMC and any of the biochemical markers for calcium regulation, bone metabolism, stress (cortisol) or inflammation (c-reactive protein). Significant associations also were observed between spine or femur BMD and height, weight, and FFM (results not shown).

Discussion

It is generally believed that obese premenopausal women are at low risk for osteoporosis. However, this study of obese premenopausal women, with a history of chronic dieting behavior, illustrates a high occurrence of osteoporosis or osteopenia (30.8%). Two important associations were found:

Table 5 Significant correlations of spine and femur BMC with other variables

		Spine BMC		*Right femur BMC*	
Category	*Variable*	*Correlation*	P-*value*	*Correlation*	P-*value*
Anthropometry	Weight	0.305	0.007	0.324	0.006
	Height	0.512	0.000	0.441	0.000
	Fat mass	0.225	0.048	0.320	0.006
	Fat-free mass	0.355	0.001	0.506	0.000
	Total body BMC	0.553	0.000	0.362	0.002
	Spine BMC	1.0		0.507	0.000
	R. femur BMC	0.507	0.000	1.0	
Energy expenditure	(Physical activity record)	0.256	0.024	0.316	0.007
Eating behavior	No. of times on diet			−0.250	0.035
	Cognitive restraint			−0.239	0.044

Significance was set at the 0.05 level of probability.

(1) a negative association between the number of times the women dieted to lose weight and present BMC values and (2) a negative association between high cognitive dietary restraint and low BMC. The only other association found was the positive correlation between BMC and energy expenditure, which was expected as it is well supported in the literature (Madsen *et al*, 1998). No correlations were seen between BMC and any of the bone-related dietary nutrients, or between BMC and any of the biochemical markers for calcium regulation, bone turnover, stress or inflammation. These results suggest that the low BMC values observed in this study were not a recent occurrence and thus cannot be explained by present daily activities and behaviors, or by a current imbalance between bone formation and resorption. Instead, it suggests that previous activities and/or behaviors were responsible for these findings.

Given the age group of these women, 30–45 y, one might expect that the low BMC values were due to early perimenopausal bone loss. However, this is not a likely explanation since 61% of the women in the normal bone group were >40 y of age compared to only 29% of the women in the OSTEO group being 40 y of age and older.

Previous research with restrained eaters has shown a disruption in sex hormones (Barr *et al*, 1994), but Barr and colleagues were unable to demonstrate a relationship between cognitive dietary restraint, menstrual disturbances, and bone density. Since all women in the present study reported having a normal menstrual cycle, indicative of a normal hormone profile, it is not a likely explanation for the lower bone mass.

Although the women in the present study were obese, the finding of a high cognitive restraint score and lower BMC is supported by the work of Van Loan and Keim (2000). Further confirmation of the effects of dieting on bone health has been documented by Fogelholm *et al* (1997). Fogelholm and colleagues found that in obese people, after adjusting for weight and age of menarche, spine and radial BMD were significantly lower in individuals with a history of weight cycling compared to the women without a history of weight cycling. Weight cycling is a form of cognitive restraint, defined as control of food intake to maintain or reduce body weight, followed by a period of disinhibition, for example weight regain. Furthermore, Fogelholm *et al* (2001) observed a decline in total body, spine, trochanter, and distal radius BMD during weight reduction. BMC also declined with weight reduction. Following a period of 3–36 months of weight regain, they observed that although BMD and BMC values increased they were significantly lower than before weight reduction. The degree to which changes occurred in BMD and BMC was associated with the amount of weight lost. Additionally, Van Loan *et al* (2000) reported that during adolescence the drive for thinness and dieting to lose weight were significant negative predictors of adult bone mass. Our data and those of others suggest that these dieting and eating behaviors, especially when engaged in as an adolescent, can have a negative impact on adult bone mass. More recently Gallagher *et al* (2002) showed that women with a history of weight cycling did not have a lower total body bone mineral content (TBBMC); specifically, 99% of the Gallagher sample were within one sd of the age-matched normative values for femur BMD. The work by Gallagher and colleagues did not include a measurement of the lumbar spine, as we did, and therefore would have missed the low BMD that we observed for this site in our obese weight cycling. We also observed that the majority of our obese women (86%) had 'normal' BMD values when the femur was the measurement being examined. However, this was not the case when we examined the lumbar spine. It is our opinion that this oversight by Gallagher *et al*, reinforces our position that lumbar spine BMD measurements must be included when examining obese women to be sure that the appropriate interpretation of the relationship between dieting behavior and bone health is made. Thus, it appears that previous eating and dieting behaviors during adolescence and young adulthood may have played a critical role in the present day low bone mass observed in our obese women.

Finally, several studies have shown that DXA measurements are affected by tissue thickness (Jebb *et al*, 1995; Jebb,

1997). If technical difficulties with DXA, due to the large size of the women, were the cause of the observed osteopenia or osteoporosis, then all scans for all of the women would have shown similar results, for example, low BMD and BMC. To the contrary, 70% of the women had normal DXA values for spine and femur. Thus we, can only conclude that technical aspects of the DXA were not the cause of the observed osteopenia and osteoporosis in these obese women.

In conclusion, osteopenia or osteoporosis was observed in 31% of our sample of obese premenopausal women with a history of restrained eating. The observed low bone values were associated with a history of restrained eating, and were not explained by present dietary intake, levels of physical activity, or indices of stress, inflammation or bone turnover. These findings provide further evidence that chronic dieting negatively impacts bone health, even in obese women who heretofore have not been considered 'at risk' for osteoporosis.

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References

Allison DB (1995): *Handbook of Assessment Methods for Eating Behaviors and Weight-related Problems*. Thousand Oaks: Sage Publications.

Barr SI, Prior JC & Vigna YM (1994): Restrained eating and ovulatory disturbances: Possible implications for bone health. *Am. J. Clin. Nutr.* **59**, 92–97.

Blair SN, Haskell WL, Ho P, Paffenbarger RS, Vranizan KM, Farguhar JW & Wood PD (1985): Assessment of habitual physical activity by a seven-day recall in a community survey and controlled experiments. *Am. J. Epidem.* **122**, 794–804.

Christiansen C, Riis BJ & Ridbro P (1987): Prediction of rapid bone loss in postmenopausal women. *Lancet* **1**, 1105–1108.

Fogelholm M, Sievanen H, Heinonen A, Virtanen M, Uusi-Rasi K, Pasanen M & Vuori L (1997): Association between weight cycling history and bone mineral density in premenopausal women. *Osteoporos. Int.* **7**, 354–358.

Fogelholm GM, Sievanen HT, Kukkonen-Harjula K & Pasanen ME (2001): Bone mineral density during reduction, maintenance and regain of body weight in premenopausal, obese women. *Osteoporos. Int.* **12**, 199–206.

Gallagher KI, Jakici JM, Kiel DP, Page ML, Ferguson ES & Marcus BH (2002): Impact of weight-cycling history on bone density in obese women. *Obes. Res.* **10**, 896–902.

Garner DM (1991): *Eating Disorder Inventory-2 Manual*. Odessa, FL: Psychological Assessment Resources, Inc.

Jebb S (1997): Measurement of soft tissue composition by dual energy X-ray absorptiometry. *Brit. J. Nutr.* **77**, 151–163.

Jebb S, Goldberg G, Jennings G & Elia M (1995): Dual-energy X-ray absorptiometry measurements of body composition: Effects of depth and tissue thickness, including comparisons with direct analysis. *Clin. Sci.* **88**, 319–324.

Kanis J (2002): Diagnosis of osteoporosis and assessment of fracture risk. *Lancet* **359**, 1929–1936.

Kleerekoper M & Nelson DA (1997): Which bone density measurement? *J. Bone Miner. Res.* **12**, 712–714.

Laessle R, Tuschl RJ, Kotthaus BC & Pirke KM (1989): A comparison of the validity of three scales for the assessment of dietary restraint. *J. Abnorm. Psych.* **98**, 304–307.

Madsen KL, Adams W & Van Loan MD (1998): Effects of physical activity, body weight and composition, and muscular strength on bone density in young women. *Med. Sci. Sports. Exerc.* **30**, 114–120.

Polivy J, Herman CP & Howard KI (1988): Restraint Scale: Assessment of Dieting. In *Dictionary of Behavioral Assessment Techniques*. eds Hersen M & Bellack AS, New York: Pergamon. pp 377–380.

Ricci TA, Heymsfield SB, Pierson Jr RN, Chowdhury HA & Shapses SA (2001): Moderate energy restriction increases bone resorption in obese postmenopausal women. *Am. J. Clin. Nutr.* **73**, 347–352.

Stunkard AJ & Messick S (1988): *The Eating Inventory*. San Antonio, TX: Psychological Corporation.

Van Loan MD & Keim NL (2000): Influence of cognitive eating restraint on total-body measurements of bone mineral density and bone mineral content in premenopausal women 18-45 y: a cross-sectional study. *Am. J. Clin. Nutr.* **72**, 837–843.

Van Loan MD, Bachrach LK, Wang MC & Crawford PB (2000): Effect of drive for thinness during adolescence on adult bone mass. *J. Bone. Miner. Res.* **15**, S412.

WHO Study Group (1994): *Assessment of Fracture Risk and its Application to Screening for Postmenopausal Osteoporosis*. Geneva, Switzerland: World Health Organization.