# A prospective investigation of the relations among cognitive dietary restraint, subclinical ovulatory disturbances, physical activity, and bone mass in healthy young women[1–3]

*Esther J Waugh, Janet Polivy, Rowena Ridout, and Gillian A Hawker*

**ABSTRACT**

**Background:** Cognitive dietary restraint (CDR) may mediate subclinical ovulatory disturbances, which may result in loss of bone mineral density (BMD). CDR is associated with greater physical activity, which may modify the effect of CDR and ovulatory disturbances on bone mass.

**Objective:** We aimed to investigate the relations among CDR, ovulatory disturbances, and physical activity and their effect on BMD in healthy premenopausal women over a 2-y period.

**Design:** In this prospective cohort study, key explanatory factors, important covariates, and BMD were measured at baseline and at 12 and 24 mo; 225 women completed the baseline assessment, and 189 completed the study. CDR was measured with the Three-Factor Eating Questionnaire, and physical activity was measured with the Baecke scale. An average of 9.8 menstrual cycles in 2 y were monitored by using salivary progesterone measurements and urinary ovulation detection kits. Ovulatory disturbances included anovulatory cycles or short luteal phase lengths of $<10$ d. BMD at the lumbar spine, femoral neck, and total body was measured by using dual-energy X-ray absorptiometry. General linear mixed modeling was used to determine predictors of change in BMD over time.

**Results:** CDR was not associated with ovulatory disturbances or changes in BMD. The average annual rate of change in lumbar spine BMD was decreased by 0.01 g/cm$^2$ in women who had experienced $\geq$3 monitored cycles with ovulatory disturbances ($P = 0.02$).

**Conclusions:** CDR did not predict bone loss, and there was no relation between CDR and ovulatory disturbances. Ovulatory disturbances had a negative effect on the rate of change at the lumbar spine. The cause of these disturbances is unknown. *Am J Clin Nutr* 2007;86:1791–801.

**KEY WORDS** Dietary restraint, bone mineral density, BMD, ovulatory disturbances, premenopausal women, physical activity

## INTRODUCTION

Cognitive dietary restraint (CDR) is defined as the conscious attempt to limit and monitor food intake to achieve or maintain a desired weight. CDR is associated with eating behavior that is governed by cognitive processes rather than by physiologic mechanisms such as hunger and satiety (1). This CDR behavior may lead to impairment of the psychophysiologic regulation of food intake (2), which could result in alternating periods of restricted eating and overeating and in weight fluctuations (3). Other characteristics associated with CDR include heightened anxiety (4) and greater physical activity levels (5, 6).

CDR may have important biological consequences in healthy premenopausal women, particularly with respect to menstrual function and bone health. Three previous studies reported that CDR is associated with ovulatory disturbances, including a greater proportion of anovulatory cycles and short luteal phase lengths (7–9). These disturbances may have a negative effect on bone mass. Prior et al (10) found that anovulatory cycles and short luteal phase lengths were related to spinal bone loss over a 1-y period; 2 studies by others did not confirm these findings (11, 12). Four studies directly investigated the relation between CDR and bone mineral density (BMD) in healthy premenopausal women, and they provide modest evidence for a negative association (8, 13–15). One study found that total-body (TB) bone mineral content (BMC) but not BMD was lower in women with high restraint scores and body weight $<$ 71 kg (15). A second study reported that the restraint score was a negative predictor of TB BMD and BMC in normal-weight university students (14), and a third study found a negative correlation between femoral BMC and cognitive restraint in obese premenopausal women with a history of chronic dieting (13). The fourth study did not find an association between restraint and bone density, but it was limited by a small sample size (8).

Further investigation of the relation among CDR, ovulatory disturbances, and BMD in healthy young women is required. Four of the prior studies (7–9, 14) were conducted in normal-weight or underweight women, who may represent a unique subgroup of successful restrained eaters who may experience more pronounced physiologic and metabolic effects. Evidence of

[1] From the Osteoporosis Research Program, Women's College Hospital, Toronto, Canada (EJW and GAH), and the Departments of Health Policy, Management and Evaluation (EJW and GAH), Psychology (JP), and Medicine (RR and GAH), University of Toronto, Toronto, Canada.

[2] Supported by grant no. MT15645 from the Canadian Institutes of Health Research and by a doctoral fellowship from the Canadian Institutes of Health Research (to EJW). GAH was a Canadian Institutes of Health Research Scientist and is the FM Hill Chair in Academic Women's Medicine at the University of Toronto.

[3] Reprints not available. Address correspondence to EJ Waugh, Osteoporosis Research Program, Women's College Hospital, 76 Grenville Street, 8th Floor E, Room 812C, Toronto, ON M5S 1B2, Canada. E-mail: e.waugh@utoronto.ca.

Received January 15, 2007.

Accepted for publication August 9, 2007.

a negative relation between subclinical ovulatory disturbances and BMD remains inconsistent. Moreover, the effect of physical activity, which is known to have a protective effect on BMD (16), but which may be associated with both CDR (5, 6) and ovulatory disturbances (17, 18), has been addressed in only one study (14), which reported that the positive effect of exercise was moderated in women with high restraint. Finally, none of the studies concurrently evaluated the relations among CDR, ovulatory disturbances, physical activity, and BMD in one sample.

The purpose of the present study was to build on these prior findings by investigating these relations over a 2-y period in a large sample of healthy premenopausal women who represent a broad spectrum of body weights. Specifically, we addressed 4 questions. *1)* Is CDR associated with subclinical ovulatory disturbances? *2)* Does CDR or subclinical ovulatory disturbances (or both) predict loss of BMD? *3)* Is the relation between CDR and changes in BMD moderated by ovulatory disturbances, physical activity, or body weight? 4) What are the key independent predictors of change in BMD over 2 y?

## SUBJECTS AND METHODS

### Subjects

Participants were healthy, white, premenopausal women aged 21–40 y who were recruited from the community. Between June 1999 and August 2001, participants were recruited from 2 groups: from an existing cohort that was described previously (19) and from women who responded to advertisements in local newspapers or to posted flyers seeking volunteers for a longitudinal study on determinants of peak bone mass. Of the 842 women who initially agreed to participate, 510 were ineligible, and 107 completed the baseline assessment but subsequently refused to monitor their menstrual cycles; thus, the baseline sample was 225 women. Twelve participants did not return for BMD testing at year 1, and an additional 24 did not complete year 2. A total of 189 participants completed the study (**Figure 1**). Exclusion criteria for the study included prior diagnosis of osteoporosis or specific comorbid conditions known to be associated with bone loss (ie, Crohn disease, symptomatic hyperthyroidism, rheumatoid arthritis, hysterectomy or bilateral oophorectomy, or anorexia nervosa); medications known to affect bone metabolism (ie, oral contraceptives or other exogenous hormones in the previous 3 mo, systematic glucocorticoids, hydrochlorothiazide and bisphosphonates in the previous 6 mo, treatment for infertility or endometriosis, use at any time of anticonvulsants for $\geq$3 mo, or daily use of inhaled corticosteroids); $<$8 menstrual cycles in the previous year; or pregnancy or active breastfeeding at the time of the study or within the previous 12 mo.

Written informed consent was obtained from all subjects. Ethical approval was obtained from the ethics review committees of

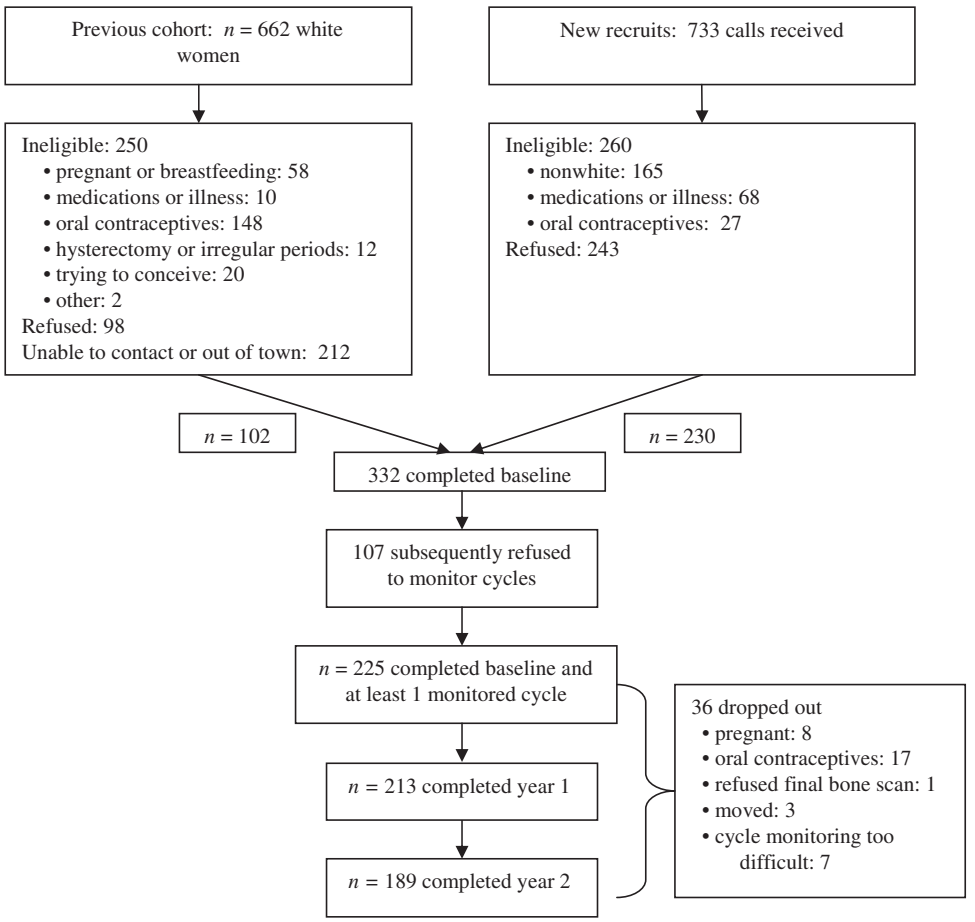


**FIGURE 1.** Flow chart depicting recruitment of participants from a previous cohort and new community sample to obtain a sample of 225 participants who completed baseline assessment, 213 who completed the year 1 assessment, and 189 who completed the study.

the Sunnybrook and Women's College Health Sciences Centre and the University of Toronto.

### Study design

This study was a 2-y prospective cohort study. Key explanatory factors, important covariates, and BMD were measured at baseline and at 12 and 24 mo. Menstrual function was measured over the 2-y study period.

### Physical measurements

Height (cm) was measured with a stadiometer and weight (kg) was measured with a balance-beam scale. These measurements were used to calculate body mass index (BMI; in $kg/m^2$). Body composition (lean mass and fat mass) was measured by using dual-energy X-ray absorptiometry (Lunar DPX-L bone densitometer; Lunar Corporation, Madison, WI) (20).

### Questionnaires

Participants completed a detailed osteoporosis risk factor questionnaire to elicit information regarding lifestyle factors (ie, cigarette smoking and alcohol and caffeine consumption), family history of osteoporosis, calcium and vitamin D supplementation, medication use, and prior surgeries and illnesses. CDR was measured by using the restraint subscale of the Three-Factor Eating Questionnaire (TFEQ-R) (21). The TFEQ-R is widely used to measure the construct of CDR (21). Higher scores on this measure are predictive of reduced caloric intake, avoidance of dietary fat, consumption of greater amounts of artificial sweeteners and diet products, and an intense concern about one's physical appearance in the absence of typical features of a psychiatric illness or eating disorder (22–24). The TFEQ-R consists of 21 items, each scored on a 2-point scale (0 or 1); a higher score is indicative of greater cognitive restraint. Participants also completed the State Trait Anxiety Inventory (STAI), a widely used measure of trait anxiety consisting of 20 items (25); a higher score indicates greater levels of general psychological stress. Physical activity level was determined by using the Baecke questionnaire, a valid and reliable self-administered questionnaire developed for the measurement of habitual physical activity in epidemiologic studies (26, 27). The Baecke questionnaire consists of work, leisure-time, sports, and total activity indexes. Finally, an interviewer-administered food-frequency questionnaire was completed to measure daily dietary intake of calcium. Food models and standardized portion sizes were used to aid participant recall and accuracy.

### Assessment of ovulatory disturbances and hormone analyses

Three consecutive menstrual cycles were evaluated twice during each of the 2 y of the study (total of 12 cycles) with the use of salivary progesterone measurements and urinary ovulation detection kits. Each block of 3 menstrual cycles was monitored during a different season so that all 4 seasons were included in the 2-y study. Salivary progesterone measurement is an established method by which to study luteal function in epidemiologic studies (28, 29). For each of the monitored cycles, participants collected saliva samples each morning before breakfast or tooth brushing. Samples were stored at −20 °C until they were analyzed. Salivary samples underwent radioimmunoassay (Diagnostic Systems Laboratories, Webster, TX) for progesterone with the use of a modified version of the method of Walker et al (30), described previously (31), in the laboratory of M Steiner (Women's Health Concerns Clinic, St Joseph's Healthcare, (Hamilton, Canada). The interassay and intraassay CVs were 15.7% and 13.9%, respectively.

Luteal phase samples were pooled to derive time-integrated mean luteal and mean midluteal progesterone values for each cycle. Specifically, mean luteal progesterone values were determined by pooling aliquots from the day when a urinary luteinizing hormone (LH) surge was detected until the day before the next menstrual cycle commenced. The days used to determine mean midluteal progesterone values were dependent on the day of the LH surge. For example, in a cycle in which the LH surge was detected 14 d before the start of the next menstrual cycle, mean midluteal progesterone was determined by pooling 5 samples from the span of time 9–5 d before commencement of the next cycle. To detect an LH surge, participants were provided with urinary ovulation detection kits (ClearPlan Easy; Unipath Ltd, Bedford, United Kingdom). These kits provide a valid and reliable method of identifying the LH surge that indicates the onset of ovulation, and thus they allow determination of the luteal phase length (32). ClearPlan Easy has been found to have an 82–88% positive predictive value for ovulation within 1 d (33). Participants followed the instructions provided with the kit, including testing at the same time of the day and not urinating for 4 h before testing.

On the basis of information on menstrual cycle patterns that was provided during the baseline visit, participants were asked to begin urinary testing 14 d before the anticipated end of the cycle and to continue testing for 5 consecutive days or until the test became positive. A positive test indicated that the LH surge had occurred. The duration of the luteal phase was calculated from the day after the LH surge until the day before the onset of menstrual flow. The follicular phase was the the period from first day of menstrual flow to the day before the onset of the luteal phase. The criterion for a short luteal phase length based on urinary LH peak data was $<10$ d (17, 34–36). When no urinary LH surge was detected, salivary progesterone concentrations were assayed consecutively from samples collected beginning 14 d before day 1 of the following cycle. If there was no rise in salivary progesterone to $\geq 2$ SDs above the mean progesterone concentrations of the previous days after 7 d of testing, and if these concentrations did not remain elevated for $\geq 2$ d, the cycle was considered to be anovulatory (31, 37–40). Participants kept a menstrual calendar for the duration of the study, in which they recorded testing days, the day on which the test became positive, and the onset of each menstrual period.

To measure serum concentrations of estradiol and free testosterone, we obtained serum samples once between 0800 and 1000 on days 3–5 of 1 of the 12 monitored cycles, chosen at random. All samples were frozen at −70 °C and analyzed by using commercial radioimmunoassays in the Endocrinology and Metabolism Laboratory at the University of Toronto (Toronto, Canada).

### Measurement of bone density

Lumbar spine (L1–L4) (LSP), hip (femoral neck) (FN), and TB BMD measurements were obtained with the use of dual-energy X-ray absorptiometry. Measurements were performed by a single, certified densitometry technician. The CVs, determined by test-retest with repositioning, were 1.18% at LSP, 1.56% at FN, and 0.72% at TB.

### Statistical analysis

All analyses were performed by using SAS statistical software (version 9.1; SAS Institute Inc, Cary, NC). The level of significance was established at $P \leq 0.05$ (2-tailed). BMD at the LSP was considered the primary outcome, because it consists primarily of trabecular bone. It was anticipated that the variables being investigated would have the most demonstrable effect on this site. To ensure completeness and to enable comparisons with other studies, BMD at the FN and TB was also included in the analysis as secondary outcomes.

All variables were assessed for normality. Paired *t* tests were used to evaluate changes in mean BMD values and in predictor variables between year 2 and baseline. Because there was minimal change in predictor variables over the 2-y study, evaluation of potential associations between and among variables was conducted on baseline values. Participants were categorized into 2 groups according to whether they had experienced <3 or ≥3 monitored menstrual cycles with subclinical ovulatory disturbances (anovulatory cycles or short luteal phases). Participants were also categorized into tertiles of CDR on the basis of their baseline TFEQ-R scores. Comparisons of physical and lifestyle characteristics and hormone values between ovulatory disturbance groups and CDR groups were done by using unpaired *t* tests or Wilcoxon rank-sum tests as appropriate for continuous variables and chi-square tests for categorical variables. The lowest and middle tertiles of CDR were compared with the highest tertile.

A general linear mixed method was used to model the effects of predictor variables on interindividual and intraindividual changes in BMD. The mixed model extends the general linear model by the addition of random effects, which allows each subject's initial BMD status (intercept) and rate of change (slope) to vary from the population average. This method also enables the estimation of individual-specific and population-specific fixed effects through the inclusion of time-varying and time-invariant variables, respectively (41–43). An unstructured covariance structure was specified, and maximum likelihood estimation method was used for parameter estimation (42). Random effects for both intercept and slope were included. The change variable was named "time" and was centered at time 0 to facilitate interpretation of the intercept (42). SAS PROC MIXED was used for this analysis.

Our initial models evaluated the effects of CDR and subclinical ovulatory disturbances on changes in BMD after control for BMI and physical activity (total Baecke score). Interactions between CDR and ovulatory disturbances, between CDR and BMI, and between CDR and physical activity were evaluated to determine whether the effect of CDR on BMD was moderated by these variables. Extended models to determine all key predictors of change in BMD were then generated. Time-varying predictors that were included in the complex model were BMI, percentage lean mass, CDR (TFEQ-R score), calcium intake (does not meet recommended daily amount or meets recommended daily amount), and smoking status (nonsmoker or current smoker); time-invariant predictors included were subclinical ovulatory disturbances (<3 or ≥3 cycles with disturbances), hormone values (ie, estradiol, testosterone, and mean luteal progesterone), age at menarche, Baecke total score, physically active as an adolescent (no or yes), alcohol consumption (no. of drinks/mo), age, and family history of osteoporosis (<3 or ≥3 relatives). Variables were removed one-by-one if $P \leq 0.05$, starting with the variable with the highest *P* value, to produce the final model (backward stepwise selection).

## RESULTS

Two hundred twenty-five women consented to the study and completed baseline assessments and ≥1 monitored cycle. Two hundred thirteen women completed year 1 assessments, and 189 women completed the 2-y study (16% were lost to follow-up). Of those who did not complete the study, 25 became ineligible because of pregnancy or commencing use of oral contraceptives, 7 found menstrual cycle monitoring too difficult, 3 moved, and 1 refused a third BMD test because of radiation concerns. Baseline demographic characteristics of the sample are summarized in **Table 1**. Physical and lifestyle characteristics and BMD values at each of the 3 time points are summarized in **Table 2**. There was a small but significant ($P < 0.0001$) increase of 1.4% in mean BMD at LSP over the 2 y in the 189 participants who completed the study. There were nonsignificant increases of 0.5% at the FN ($P = 0.09$) and 0.09% at the TB ($P = 0.45$). Of the predictor variables, only calcium intake changed significantly, increasing by 12.7% ($P = 0.001$).

### Subclinical ovulatory disturbances

A total of 2213 menstrual cycles were monitored over the 2-y study period. Sixty percent of participants monitored 12 cycles, and 14% monitored <6 cycles (average: 9.8 ± 3.4 cycles/participant). Blood samples were obtained from 205 women.

The mean menstrual cycle length was 28.9 ± 3.9 d (range: 23.6–55.2 d). Eight women had long mean cycle lengths (>36 d), and none had short mean cycle lengths (<21 d). The menstrual characteristics, hormone values, and subclinical ovulatory disturbances experienced by these women are summarized in **Table 3**. One hundred forty-one cycles (6.4%) showed ovulatory disturbances, as indicated by short luteal phase lengths (72 cycles) or anovulation (69 cycles). Forty-one women had ≥1 short luteal phase (<10 d), and 45 women had ≥1 anovulatory cycle. In total, 75 women (33.3%) experienced ≥1 subclinical ovulatory disturbance, and 16 women experienced ≥3 cycles with disturbances. Eight women experienced ovulatory disturbances

**TABLE 1**
Demographic characteristics of the sample of 225 white premenopausal women

| Characteristic | Value |
|---|---|
| Age (y) | 32.4 ± 4.6[1] |
| Education (%) | |
| Elementary school | 0.9 |
| High school | 12.0 |
| Postsecondary school | 87.1 |
| Marital status (%) | |
| Single | 57.3 |
| Married or common-law spouse | 39.1 |
| Separated or divorced | 3.6 |
| Reproductive status (%) | |
| Nulliparous | 74.2 |
| 1 birth | 5.3 |
| >1 birth | 20.5 |
| History of ≥3 relatives with osteoporosis (%) | 8.5 |

[1] $\bar{x}$ ± SD.

**TABLE 2**
Summary of physical and lifestyle characteristics and bone mineral density values at baseline and at 12 and 24 mo[1]

| | Baseline ($n$ = 225) | Year 1 (12 mo) ($n$ = 213) | Year 2 (24 mo) ($n$ = 189) |
|---|---|---|---|
| BMI (kg/m$^2$) | 24.3 ± 4.6[2] | 24.6 ± 4.7 | 24.7 ± 4.9 |
| Lean mass (%) | 66.1 ± 9.5 | 65.6 ± 9.6 | 65.6 ± 9.7 |
| Baecke total score (/15) | 8.3 ± 1.3 | 8.3 ± 1.3 | 8.4 ± 1.4 |
| Active as an adolescent (%) | 82.2 | 81.7 | 81 |
| TFEQ-R (/21) | 7.6 ± 4.0 | 7.3 ± 3.9 | 7.5 ± 4.2 |
| STAI (/80) | 37.0 ± 9.0 | 36.8 ± 8.7 | 36.1 ± 8.6 |
| Calcium intake | | | |
| (mg/d) | 876.5 ± 440.7 | 811.5 ± 440.4[3] | 965.2 ± 447.2[4] |
| Met RDA of 1000 mg/d (%) | 32.4 | 27.2 | 42.8 |
| Alcohol consumption (drinks/mo) | 10.4 ± 12.8 | 11.2 ± 12.6 | 12.5 ± 14.7 |
| Smoking status (%) | | | |
| Current smoker | 6.2 | 7.5 | 7.4 |
| Past smoker | 16.9 | 16.0 | 14.8 |
| Bone mineral density (g/cm$^2$) | | | |
| Lumbar spine (L1–L4) | 1.200 ± 0.138 | 1.218 ± 0.143[3] | 1.223 ± 0.148[3] |
| Femoral neck | 1.013 ± 0.139 | 1.016 ± 0.144 | 1.023 ± 0.144[4] |
| Total body | 1.181 ± 0.140 | 1.183 ± 0.081 | 1.183 ± 0.080 |

[1] TFEQ-R, Three Factor Eating Questionnaire–Restraint subscale; STAI, State Trait Anxiety Inventory; RDA, recommended daily amount.
[2] $\bar{x}$ ± SD (all such values).
[3] Significantly different from baseline in the 189 participants who completed the study. $P \leq 0.05$ (paired $t$ test).
[4] Significantly different from Year 1 in the 189 participants who completed the study, $P \leq 0.05$ (paired $t$ test).

in >40% of their monitored cycles. Baseline characteristics of women who experienced ovulatory disturbances over the 2-y study are shown in **Table 4**. There were no significant differences in physical and lifestyle characteristics or hormone values between women with <3 cycles with ovulatory disturbances and women with ≥3 such cycles. Having a greater percentage of cycles with ovulatory disturbances was associated with significantly lower mean luteal and mean midluteal progesterone ($P$ = 0.0009 and $P < 0.001$, respectively) but not with significantly lower estradiol ($P = 0.77$) or testosterone ($P = 0.11$).

### Cognitive dietary restraint and subclinical ovulatory disturbances

TFEQ-R scores ranged from 0 to 18 out of a possible score of 21 with a weighted mean over the 2 y of 7.5 ± 4.0. Analysis of baseline scores showed that TFEQ-R scores were correlated with BMI ($r = 0.264$, $P < 0.0001$), percentage lean mass ($r = -0.167$, $P = 0.01$), Baecke physical activity score ($r = 0.163$, $P = 0.01$), Baecke sport index score ($r = 0.22$, $P = 0.0005$), and STAI score ($r = 0.193$, $P = 0.007$). The physical characteristics, menstrual characteristics, and hormone values for women in each tertile of CDR at baseline are shown in **Table 5**. Women who were in the highest tertile had significantly higher BMIs ($P = 0.0002$), lower percentage lean mass ($P = 0.008$), and higher Baecke sport index scores ($P = 0.006$) but not significantly higher total Baecke scores ($P = 0.08$) than did those who were in the lowest tertile. Women in the highest tertile also had significantly ($P = 0.03$) higher BMIs than did those in the middle tertile. The mean menstrual cycle length and mean luteal phase length did not differ significantly, and the percentage of women with ≥3 cycles with ovulatory disturbances did not differ significantly across the restraint groups. There were no significant differences in estradiol, testosterone, or mean luteal progesterone values between the lowest, middle, and highest tertiles.

### Cognitive dietary restraint, subclinical ovulatory disturbances, and 2-y change in BMD

The results of the mixed-model analysis to evaluate the effect of CDR and subclinical ovulatory disturbances on changes in BMD, after adjustment for BMI and Baecke activity scores, are shown in **Table 6**. CDR had no effect on the average initial values or change in BMD at the LSP. Women who had ≥3 cycles with ovulatory disturbances had an average annual rate of change in LSP BMD (−0.01 g/cm$^2$) significantly ($P = 0.03$) different from that in women with <3 such cycles. There was no difference between these 2 groups in initial BMD values. There were no significant interactions between CDR and ovulatory disturbances, BMIs, or Baecke score at the LSP. Neither CDR nor ovulatory disturbances had significant effects at the FN or TB, and there were no significant interactions between CDR and the other 3 variables.

### Expanded predictive models of 2-y change in bone mineral density

Final mixed models are summarized in **Table 7**. The results for the LSP show that BMI was positively associated with BMD and a family history of osteoporosis and not being physically active as an adolescent had a negative affect on average initial BMD values, whereas each additional alcoholic drink/mo had a positive effect on initial BMD values. Having ≥3 cycles with ovulatory disturbances had a negative effect on rate of change. The model indicates that average initial LSP values (when BMI is zero) in women who had no family history of osteoporosis, who were physically active as an adolescent, and who consumed no alcohol was 1.0780 g/cm$^2$. Women with a family history of osteoporosis had a difference of −0.0784 g/cm$^2$ in average initial BMD ($P = 0.01$), women who did not exercise as an adolescent had a difference of −0.0569 g/cm$^2$ in average initial BMD ($P$ = 0.01), and each additional alcoholic drink/mo was associated

**TABLE 3**
Summary of menstrual characteristics, hormone values, and subclinical ovulatory disturbances based on assessment of 9.8 ± 3.4 menstrual cycles over 2 y in a sample of 225 premenopausal women[1]

| | Value |
|---|---|
| Menstrual characteristics | |
| Age at menarche (y) | 12.7 ± 1.5[1] |
| Gynecologic age (y) | 19.7 ± 4.9 |
| Mean cycle length (d) | 28.9 ± 3.9 |
| Mean luteal phase length (d) | 13.6 ± 1.7 |
| Hormone values[2] | |
| Estradiol (pg/mL) | 37.9 ± 36.2 |
| Free testosterone (pg/mL) | 1.3 ± 0.6 |
| Mean luteal progesterone (pmol/L) | 373.4 ± 155.6 |
| Mean midluteal progesterone (pmol/L) | 492.7 ± 222.5 |
| Subclinical disturbances | |
| Ever experienced short luteal phase length [*n* (%)] | 41 (18.2) |
| 1 short luteal phase (*n*) | 23 |
| 2 short luteal phases (*n*) | 8 |
| 3 short luteal phases (*n*) | 8 |
| 4 short luteal phases (*n*) | 1 |
| 5 short luteal phases (*n*) | 1 |
| Ever experienced anovulatory cycle [n (%)] | 45 (20.0) |
| 1 anovulatory cycle (*n*) | 32 |
| 2 anovulatory cycles (*n*) | 7 |
| 3 anovulatory cycles (*n*) | 3 |
| 4 anovulatory cycles (*n*) | 2 |
| 6 anovulatory cycles (*n*) | 1 |
| Ever experienced ≥1 short luteal phase length or anovulatory cycle [*n* (%)] | 75 (33.3) |
| 1 cycle with ovulatory disturbance (*n*) | 39 |
| 2 cycles with ovulatory disturbance (*n*) | 20 |
| 3 cycles with ovulatory disturbance (*n*) | 9 |
| 4 cycles with ovulatory disturbance (*n*) | 2 |
| 5 cycles with ovulatory disturbance (*n*) | 3 |
| 6 cycles with ovulatory disturbance (*n*) | 2 |
| Monitored cycles with ovulatory disturbances per participant [*n* (%)] | |
| 0% | 150 (66.7) |
| 1–10% | 30 (13.3) |
| 11–20% | 26 (11.6) |
| 21–30% | 9 (4.0) |
| 31–40% | 2 (0.9) |
| 41–50% | 7 (3.1) |
| 51–60% | 1 (0.4) |
| >60% | 0 |

[1] $\bar{x}$ ± SD (all such values).
[2] Estradiol and testosterone were collected once in 205 subjects.

with an increase of 0.0016 g/cm$^2$ in average initial BMD ($P$ = 0.02) Each 1-point increase in BMI at each time point was associated with a BMD increase of 0.0052 g/cm$^2$ ($P < 0.0001$). These variables did not affect the average annual rate of change, which was 0.0096 g/cm$^2$ ($P < 0.0001$), after control for BMI. This rate of change was decreased by 0.0109 g/cm$^2$ in women with ≥3 cycles with ovulatory disturbances, so that they experienced an annual loss of 0.0013 g/cm$^2$ ($P$ = 0.02) (**Figure 2**). Having ovulatory disturbances did not affect initial BMD values.

The FN model indicates that there was no significant annual change in BMD ($P$ = 0.08). BMI was positively associated with BMD, having a family history of osteoporosis had a negative effect on initial values and each 1-point increase in the Baecke total score had a positive effect on initial values. The model indicates that initial FN BMD (when BMI is zero) was 0.7972 g/cm$^2$ for women who did not have a family history of osteoporosis and who were not physically active (Baecke score of zero). Women with a family history of osteoporosis had a difference of −0.0732 g/cm$^2$ in average initial BMD ($P$ = 0.02), and every 1-point increase in the Baecke total score was associated with an increase of 0.0157 g/cm$^2$ in average initial BMD ($P$ = 0.02). Each 1-point increase in BMI at each time point was associated with a BMD increase of 0.0035 g/cm$^2$ ($P$ = 0.0005).

The TB model indicates that there was no significant annual change in BMD ($P$ = 0.77). BMI and percentage lean mass were positively associated with BMD, and not being physically active as an adolescent had a negative effect on initial values. The model indicates that average initial TB BMD (when BMI and percentage lean mass are zero) was 1.0212 g/cm$^2$ in women who were physically active as adolescents. Women who did not exercise as adolescents had a difference of −0.0319 g/cm$^2$ in average initial BMI ($P$ = 0.008). Each 1-point increase in BMI at each time point was associated with a BMD increase of 0.0048 g/cm$^2$ ($P < 0.0001$), and each 1% increase in lean mass at each time point was associated with a BMD increase of 0.00074 g/cm$^2$ ($P$ = 0.02).

## DISCUSSION

The primary purpose of this study was to evaluate the relations among CDR, subclinical ovulatory disturbances, and physical activity and their effect on change in BMD over a 2-y period in healthy, premenopausal women. In this large community cohort of young women, we did not confirm prior reports of either a

**TABLE 4**
Baseline characteristics of women with subclinical ovulatory disturbances categorized as having <3 or ≥3 cycles with short luteal phase lengths or anovulation[1]

| | Had < 3 cycles with ovulatory disturbances ($n$ = 209) | Had ≥ 3 cycles with ovulatory disturbances ($n$ = 16) |
|---|---|---|
| Physical and lifestyle characteristics | | |
| Age (y) | 31.5[2] | 33 |
| Gynecologic age (y) | 18.5 | 20 |
| Age at menarche (y) | 12.6 ± 1.0[3] | 12.7 ± 1.5 |
| BMI (kg/m$^2$) | 24.7 | 23.5 |
| Lean mass (%) | 64.6 ± 9.6 | 66.2 ± 9.5 |
| Baecke total score (/15) | 7.9 ± 1.2 | 8.3 ± 1.3 |
| TFEQ-R score (/21) | 8.4 ± 4.2 | 7.6 ± 4.0 |
| STAI score (/80) | 35.7 ± 8.9 | 37.2 ± 9.0 |
| Menstrual cycle length (d) | 27.5 | 28.0 |
| Hormone values | | |
| Estradiol (pg/mL) | 30.2 | 30.7 |
| Free testosterone (pg/mL) | 1.1 | 1.2 |
| Mean luteal progesterone (pmol/L) | 320.3 | 353.6 |
| Mean midluteal progesterone (pmol/L) | 412.1 | 468.3 |

[1] TFEQ-R, Three Factor Eating Questionnaire–Restraint subscale; STAI, State Trait Anxiety Inventory. Characteristics were compared between the 2 groups by $t$ test for normally distributed variables and by Wilcoxon rank-sum test for skewed variables; level of significance was $P \leq 0.05$.
[2] Median (all such values).
[3] $\bar{x}$ ± SD (all such values).

**TABLE 5**
Baseline characteristics of women categorized by tertile of baseline dietary restraint scores[1]

| | Lowest tertile of restraint (score < 5) ($n$ = 76) | Middle tertile of restraint ($n$ = 70) | Highest tertile of restraint (score > 9.4) ($n$ = 79) |
|---|---|---|---|
| Physical or lifestyle characteristics | | | |
| Age (y) | 34[2] | 33 | 34 |
| Age at menarche (y) | 12.9 ± 1.5[3] | 12.8 ± 1.5 | 12.4 ± 1.4 |
| BMI (kg/m$^2$) | 21.2[4] | 23.5[5] | 25.2 |
| Lean mass (%) | 67.9 ± 9.6[6] | 66.4 ± 1.5 | 64.1 ± 8.3 |
| Baecke total score | | | |
| (/15) | 8.0 ± 1.5 | 8.4 ± 1.3 | 8.4 ± 1.2 |
| Sport score (/5) | 2.7 ± 0.8[6] | 2.9 ± 0.7 | 3.1 ± 0.8 |
| TFEQ-R score (/21) | 3.5 ± 1.2[6] | 7.1 ± 1.3[7] | 12.1 ± 2.4 |
| STAI score (/80) | 35.2 ± 8.7 | 38.0 ± 9.4 | 38.0 ± 8.7 |
| Alcohol intake (drinks/mo) | 5 | 6.3 | 7 |
| Calcium intake (mg/d) | 827.2 | 844.5 | 806.9 |
| Current smoker (%) | 6.6 | 5.7 | 6.3 |
| Menstrual characteristics | | | |
| Mean cycle length (d) | 28.0 | 28.0 | 27.6 |
| Mean luteal phase length (d) | 13.8 | 13.9 | 13.7 |
| Had ≥3 cycles with ovulatory disturbances (%) | 5.3 | 10 | 6.3 |
| Hormone values | | | |
| Estradiol (pg/mL) | 30.2 | 29.2 | 32.1 |
| Free testosterone (pg/mL) | 1.3 | 1.1 | 1.2 |
| Mean luteal progesterone (pmol/L) | 345.9 | 342.0 | 364.2 |

[1] TFEQ-R, Three Factor Eating Questionnaire–Restraint subscale; STAI, State Trait Anxiety Inventory. Characteristics of the lowest and middle tertiles are each compared with those of the highest tertile by $t$ test for normally distributed variables, Wilcoxon rank-sum test for skewed variables, and chi-square for categorical variables.
[2] Median (all such values).
[3] $\bar{x}$ ± SD (all such values).
[4] Significantly different from highest tertile, $P \leq 0.05$ (Wilcoxon rank-sum test).
[5] Significantly different from highest tertile, $P \leq 0.05$ (Wilcoxon rank-sum test).
[6] Significantly different from highest tertile, $P \leq 0.05$ ($t$ test).
[7] Significantly different from highest tertile, $P \leq 0.05$ ($t$ test).

negative relation between CDR and BMD or an association of CDR with subclinical ovulatory disturbances. Although unrelated to CDR or physical activity, having subclinical ovulatory disturbances negatively affected the rate of change in LSP BMD over 2 y, and it resulted in slight bone loss, whereas women with <3 cycles with disturbances had a slight increase in bone density (Figure 2).

The inconsistency of our findings with those of previous studies examining the effects of CDR on ovulatory function and BMD may be the result of important differences in the populations studied—namely, the range in BMI values and the mean age of the participants in our cohort—and of our method of determining ovulatory disturbances. First, in contrast to prior studies that evaluated CDR in normal-weight persons (BMI 18–25) (7–9, 14), our sample was composed of women with a broad range of BMI values (16.2–39.3), and it included obese, normal-weight, and low-weight persons. In our study, higher CDR scores were associated with higher BMI, lower mean percentage lean mass, and higher Baecke sport scores, which suggests that these scores were indicative of overweight women who were attempting to diet and exercise to lose weight. We hypothesized that normal-weight or underweight persons who have high CDR scores may be a unique subgroup who are "successful" dieters and are consistently restricting caloric intake. This caloric restriction, if severe enough, may be contributing to the previously documented ovulatory disturbances. Our participants may represent "typical" restrained eaters who are "unsuccessful" dieters (3, 44). Our results suggest that, in this population, CDR does not induce alterations in the reproductive axis. We examined the interaction between CDR and BMI to determine whether the effect of CDR on BMD was different in women with lower weight, but we did not confirm this hypothesis. Further investigation is required to clarify the relation found in prior studies between CDR and ovulatory function in normal-weight and underweight women.

A second important difference between the population of the present study and the populations of previous studies was the age of our participants: the subjects in the present study had a greater mean age than did subjects in prior studies that found significant associations between CDR and BMD (14) or between CDR and ovulatory disturbances (7, 8). Our sample represents women who have reached gynecologic maturity, but who are not yet in the perimenopausal period. Gynecologic maturity has been found to be associated with reduced variability in cycle length (45, 46). In addition, durability of the reproductive axis in response to a moderate endurance training program was shown in a group of gynecologically mature, eumenorrheic women (gynecologic age: 17.8 ± 0.9 y) (47). This would suggest that the women in our study may have more robust reproductive systems and therefore

**TABLE 6**
Summary of results of general linear mixed-model analysis to evaluate the effect of cognitive dietary restraint (CDR) and subclinical ovulatory disturbances on 2-y changes in bone mineral density (BMD) after adjustment for BMI and physical activity (Baecke total score)[1]

| | Parameter estimate | *P* |
|---|---|---|
| Spinal BMD (L1–L4) | | |
| Intercept (initial status) | 0.9899 | <0.0001 |
| Time (annual rate of change) | 0.0123 | <0.0001 |
| CDR | | |
| Lowest vs highest tertile | 0.0047 | 0.36 |
| Middle vs highest tertile | 0.0068 | 0.15 |
| Subclinical ovulatory disturbances | −0.0292 | 0.39 |
| BMI (kg/m$^2$) | 0.0053 | <0.0001 |
| Baecke total score (/15) | 0.0099 | 0.13 |
| CDR × time | | |
| Lowest vs highest tertile | −0.0046 | 0.15 |
| Middle vs highest tertile | −0.0042 | 0.22 |
| Subclinical ovulatory disturbances × time | −0.0103 | 0.03 |
| Femoral neck BMD | | |
| Intercept (initial status) | 0.7952 | <0.0001 |
| Time (annual rate of change) | 0.0048 | 0.10 |
| CDR | | |
| Lowest vs highest tertile | −0.0058 | 0.36 |
| Middle vs highest tertile | −0.0035 | 0.54 |
| Subclinical ovulatory disturbances | −0.0037 | 0.91 |
| BMI (kg/m$^2$) | 0.0040 | 0.0001 |
| Baecke total score (/15) | 0.0146 | 0.03 |
| CDR × time | | |
| Lowest vs highest tertile | −0.0033 | 0.40 |
| Middle vs highest tertile | −0.0025 | 0.56 |
| Subclinical ovulatory disturbances × time | 0.0021 | 0.74 |
| Total-body BMD | | |
| Intercept (initial status) | 1.0324 | <0.0001 |
| Time (annual rate of change) | −0.0001 | 0.96 |
| CDR | | |
| Lowest vs highest tertile | −0.0016 | 0.58 |
| Middle vs highest tertile | 0.0017 | 0.51 |
| Subclinical ovulatory disturbances | −0.0105 | 0.56 |
| BMI (kg/m$^2$) | 0.0034 | <0.0001 |
| Baecke total score (/15) | 0.0077 | 0.03 |
| CDR × time | | |
| Lowest vs highest tertile | 0.0001 | 0.95 |
| Middle vs highest tertile | −0.0011 | 0.57 |
| Subclinical ovulatory disturbances × time | 0.0002 | 0.95 |

[1] Interactions between CDR and each of BMI, ovulatory disturbances, and Baecke score were analyzed; all were not statistically significant ($P \geq 0.05$); only main effects are included in this table for simplicity. CDR was categorized by tertile on the basis of scores on the Three Factor Eating Questionnaire–Restraint subscale. Subclinical ovulatory disturbances were defined as having ≥ 3 cycles with anovulation or short luteal phase lengths (< 10 d). Time was set at 0, 1, and 2, which represented baseline and 12- and 24-mo BMD assessments, respectively.

may be less likely to experience ovulatory disturbances in response to minor physiologic stressors, such as CDR or moderate exercise, than would women in the earlier studies who were of a younger age.

Our method of determining ovulatory function differed from that used in prior studies documenting a relation between ovulatory disturbances and CDR (7, 8) and ovulatory disturbances and BMD (10), and that difference may have contributed to our different results. Those other studies used the basal body temperature method, which has been shown to be an unreliable method of determining ovulation and which may have led to misclassification (48–50). Indeed, those investigators reported a much higher prevalence of ovulatory disturbances (67–80% of participants had disturbances) (7, 10) than we and others (12, 51) who used progesterone measurements have reported. Both salivary and urinary progesterone are highly correlated with serum progesterone, and measuring them is a reliable method of assessing ovarian function (28, 30, 52). We also used urinary ovulation detection kits as our primary method of determining ovulation; these kits have been shown to have very good psychometric properties (33).

Despite the different method of ovulatory assessment, and although we did not find a relation between CDR and ovulatory disturbances, we confirmed the results of Prior et al (10, 53) that ovulatory disturbances are predictive of spinal bone loss. No significant change in BMD at FN or TB was observed over the 2-y study, and that lack of such an observation was likely due to slower bone turnover rates at these sites. Ovulatory disturbances also may have a negative effect on these sites if observed over a longer period. Debate continues over the underlying cause of reduced bone mass due to ovulatory disturbances. Prior et al (10) proposed that the observed bone loss was due to reduced luteal progesterone production in the presence of normal concentrations of estrogen, but this possibility has not been supported by other investigators. Sowers et al (51) found lower concentrations of both luteal phase estrogen and progesterone urinary metabolites in women with BMD in the bottom 10th percentile than in women with BMD in the 50–75th percentile. DeSouza et al (11) observed lower luteal phase progesterone in women with short luteal phases (but found no difference in BMD) than in women with normal luteal phases. DeSouza et al concluded that, if estradiol status is maintained, BMD is not affected by a disturbance of progesterone production associated with luteal phase abnormalities. In the present study, although decreased luteal progesterone was associated with ovulatory disturbances, we found no relation between progesterone and bone loss.

The extended models of all key predictors of BMD indicated that BMI was positively associated with BMD at each skeletal site. The strong association between body weight and bone density has been documented in many prior studies of premenopausal women (54). Physical activity also was consistently predictive of higher BMD. Because activity scores remained constant throughout the study, the relation between activity and change in BMD could not be evaluated to determine the extent of the benefit of activity or the amount of activity required to increase BMD in premenopausal women. Although current activity was the best predictor at the FN, being physically active as an adolescent had a stronger effect at the LSP and TB. This finding provides support for the findings of others of the existence of a critical window of opportunity during puberty, when the skeleton is particularly responsive to mechanical loading that results in the optimization of peak bone mass (55). Additional significant predictors included a family history of osteoporosis (negative effect) and the subject's alcohol consumption (positive effect), which were previously reported (19, 56–58). Finally, lean mass was found to have a positive effect on BMD at TB after control for BMI and physical activity. Although questions remain about the relative benefit of lean mass and fat mass for bone density, recent studies have shown that lean mass has a greater effect than fat

**TABLE 7**
Key predictors of 2-y change in bone mineral density (BMD) at the lumbar spine (L1–L4), femoral neck, and total body determined by general linear mixed-model analysis[1]

| | Parameter estimate | 95% CI | *P* |
|---|---|---|---|
| Spinal BMD (L1–L4) | | | |
| Intercept (initial status) | 1.0779 | 1.0286, 1.1272 | <0.0001 |
| Time (annual rate of change) | 0.0096 | 0.0071, 0.0121 | <0.0001 |
| BMI (kg/m$^2$) | 0.0052 | 0.0034, 0.0070 | <0.0001 |
| Family history of osteoporosis (≥3 relatives) | −0.0755 | −0.1350, −0.0161 | 0.01 |
| Not physically active as an adolescent | −0.0524 | −0.0962, −0.0085 | 0.02 |
| Alcohol consumption (drinks/mo) | 0.0016 | 0.0003, 0.0030 | 0.02 |
| Subclinical ovulatory disturbances | −0.0351 | −0.0995, 0.0292 | 0.28 |
| Subclinical ovulatory disturbances × time | −0.0109 | −0.0200, −0.0018 | 0.02 |
| Femoral neck BMD | | | |
| Intercept (initial status) | 0.7972 | 0.6735, 0.9209 | <0.0001 |
| Time (annual rate of change) | 0.0028 | −0.0004, 0.0060 | 0.08 |
| BMI (kg/m$^2$) | 0.0037 | 0.0016, 0.0057 | 0.0005 |
| Family history of osteoporosis (≥3 relatives) | −0.0732 | −0.1353, −0.0112 | 0.02 |
| Baecke total score (/15) | 0.0157 | 0.0028, 0.0287 | 0.02 |
| Total-body BMD | | | |
| Intercept (initial status) | 1.0212 | 0.9493, 1.0932 | <0.0001 |
| Time (annual rate of change) | −0.0002 | −0.0016, 0.0012 | 0.77 |
| BMI (kg/m$^2$) | 0.0048 | 0.0033, 0.0063 | <0.0001 |
| Lean mass (%) | 0.0007 | 0.0001, 0.0013 | 0.02 |
| Not physically active as an adolescent | −0.0319 | −0.0555, −0.0082 | 0.008 |

[1] Final reduced model was determined through backward stepwise selection from complex model that included the following variables: time (varying)—BMI, percentage lean mass, cognitive dietary restraint (Three Factor Eating Questionnaire–Restraint scale score), smoking status (nonsmoker or current smoker), calcium intake (does not meet or meets recommended daily amount); time (invariant)—subclinical ovulatory disturbances (<3 or ≥3 cycles with disturbances), hormone values (ie, estradiol, testosterone, and mean luteal progesterone), age at menarche, Baecke total score, physically active as an adolescent (no or yes), alcohol consumption (no. of drinks/mo), age, and family history of osteoporosis (<3 or ≥3 relatives). Time was set at 0, 1, and 2, which represented baseline and 12- and 24-mo BMD assessments, respectively. Subclinical ovulatory disturbances were defined as having ≥3 cycles with anovulation or short luteal phase lengths (<10 d).

mass in younger women (59–61). This finding suggests that the dynamic load generated by muscle contraction stimulates a greater osteogenic response than that generated by the static load associated with body weight (62). This relation may not hold true in postmenopausal women (59, 63), and further research is needed to clarify the relative effects of the components of body composition across different age groups.

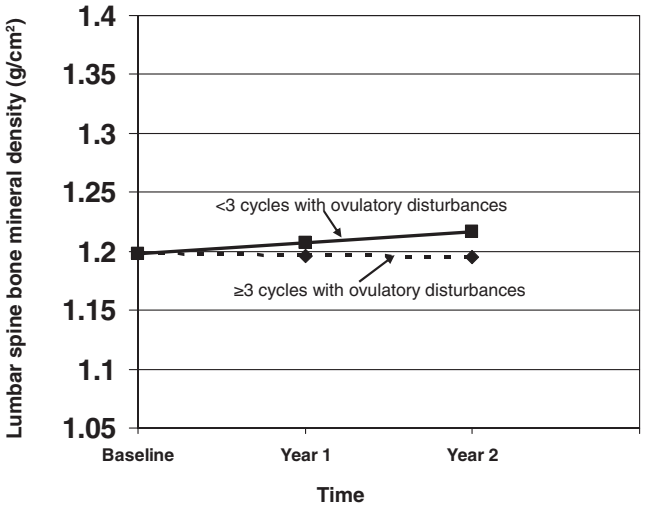


**FIGURE 2.** Line graph illustrating the effect of subclinical ovulatory disturbances on changes in lumbar spine bone mineral density over a 2-y period in an average participant with a stable BMI of 23 and no family history of osteoporosis, who was physically active as an adolescent, and who does not consume alcohol. The graph is based on the mixed model summarized in Table 7.

The strengths of the present study include its large sample size; the prospective design; the large number of menstrual cycles monitored over a 2-y period; the inclusion of women across a broad spectrum of body weight, BMD, and CDR values; and the measurement of important covariates. The limitations of the study include the single measurement of estradiol and testosterone and the nondiverse demographic characteristics of our volunteer sample (ie, well-educated white women), which may hinder generalizability of the results.

In summary, in a large sample of healthy premenopausal women, CDR was not associated with ovulatory disturbances or with bone loss over a 2-y period. Subclinical ovulatory disturbances, which were not uncommon, had a negative effect on the rate of change in BMD at the LSP. The underlying cause of these ovulatory disturbances in otherwise healthy young women warrants further investigation so that this risk factor may potentially be modified.

We acknowledge the work of Melissa French, who coordinated the study and collected the data. We also thank the dedicated group of women who participated in this study.

The authors' responsibilities were as follows: GAH, JP, and RR: study design; EJW: data analysis and writing of the manuscript; and GAH, JP, and RR: assistance with data interpretation and manuscript preparation. None of the authors had a personal or financial conflict of interest.

## REFERENCES


1. Herman CP, Mack D. Restrained and unrestrained eating. J Pers 1975; 43:647–60.
2. Tuschl RJ, Platte P, Laessle RG, Stichler W, Pirke K-M. Energy expenditure and everyday eating behavior in healthy young women. Am J Clin Nutr 1990;52:81–6.
3. Heatherton TF, Polivy J, Herman CP. Restraint, weight loss and variability of body weight. J Abnorm Psychol 1991;100:78–83.
4. Herman CP, Polivy J. Anxiety, restraint and eating behaviour. J Abnorm Psychol 1975;84:666–72.
5. McLean JA, Barr SI. Cognitive dietary restraint is associated with eating behaviors, lifestyle practices, personality characteristics and menstrual irregularity in college women. Appetite 2003;40:185–92.
6. Tepper BJ, Trail AC, Shaffer SE. Diet and physical activity in restrained eaters. Appetite 1996;27:51–64.
7. Barr SI, Janelle KC, Prior JC. Vegetarian vs nonvegetarian diets, dietary restraint, and subclinical ovulatory disturbances: prospective 6-mo study. Am J Clin Nutr 1994;60:887–94.
8. Barr SI, Prior JC, Vigna YM. Restrained eating and ovulatory disturbances: possible implications for bone health. Am J Clin Nutr 1994;59: 92–7.
9. Schweiger U, Tuschl RJ, Platte P, Broocks A, Laessle RG, Pirke K-M. Everyday eating behaviour and menstrual function in young women. Fertil Steril 1992;57:771–5.
10. Prior JC, Vigna YM, Schechter MT, Burgess AE. Spinal bone loss and ovulatory disturbances. N Engl J Med 1990;323:1221–7.
11. De Souza MJ, Miller BE, Sequenzia LC, et al. Bone health is not affected by luteal phase abnormalities and decreased ovarian progesterone production in female runners. J Clin Endocrinol Metab 1997;82:2867–76.
12. Waller K, Reim J, Fenster L, et al. Bone mass and subtle abnormalities in ovulatory function in healthy women. J Clin Endocrinol Metab 1996; 81:663–8.
13. Bacon L, Stern JS, Keim NL, Van Loan MD. Low bone mass in premenopausal chronic dieting obese women. Eur J Clin Nutr 2004;58: 966–71.
14. McLean JA, Barr SI, Prior JC. Dietary restraint, exercise and bone density in young women: are they related? Med Sci Sports Exerc 2000; 33:1292–6.
15. Van Loan MD, Klein NL. Influence of cognitive eating restraint on total-body measurements of bone mineral density and bone mineral content in premenopausal women aged 18–45 y: a cross-sectional study. Am J Clin Nutr 2000;72:837–43.
16. Wolff I, van Croonenborg JJ, Kemper HCG, Kostense PJ, Twisk J. The effect of exercise training programs on bone mass: a meta-analysis of published controlled trials in pre- and postmenopausal women. Osteoporos Int 1999;9:1–12.
17. De Souza MJ, Miller BE, Loucks AB, et al. High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. J Clin Endocrinol Metab 1998;83:4220–32.
18. de Souza MJ, Van Heest J, Demers LM, Lasley BL. Luteal phase deficiency in recreational runners: evidence for a hypometabolic state. J Clin Endocrinol Metab 2003;88:337–46.
19. Rubin LA, Hawker GA, Peltekova VD, Fielding LJ, Ridout R, Cole DE. Determinants of peak bone mass: clinical and genetic analyses in a young female Canadian cohort. J Bone Miner Res 1999;14:633–43.
20. Van Loan MD, Mayclin PL. Body composition assessment: dual-energy X-ray absorptiometry (DEXA) compared to reference methods. Eur J Clin Nutr 1992;46:125–30.
21. Stunkard AJ, Messick S. The Three-Factor Eating Questionnaire to measure dietary restraint, disinhibition and hunger. J Psychosom Res 1985;29:71–83.
22. deLauzon B, Romon M, Deschamps V, et al. The Three-Factor Eating Questionnaire-R18 is able to distinguish among different eating patterns in the general population. J Nutr 2004;134:2372–80.
23. Laessle RG, Tuschl RJ, Waadt S, Pirke K-M. The specific psychopathology of bulimia nervosa: a comparison with restrained and unrestrained (normal) eaters. J Consult Clin Psychol 1989;57:772–5.
24. Rideout CA, McLean JA, Barr SI. Women with high scores for cognitive dietary restraint choose foods lower in fat and energy. J Am Dietet Assn 2004;104:1154–7.
25. Speilberger C, Gorsuch R, Lushene R, Vagg PR, Jacobs GA. Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychological Press, 1983.
26. Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. Am J Clin Nutr 1982;36:936–42.
27. Philippaerts R, Westerterp KR, Lefevre J. Doubly labelled water validation of three physical activity questionnaires. Int J Sports Med 1999; 20:284–9.
28. Riad-Fahmy D, Read GF, Walker RF. Salivary steroid assays for assessing variation in endocrine activity. J Steroid Biochem 1983;19:265–72.
29. Walker SM, Walker RF, Riad-Fahmy D. Longitudinal studies of luteal function by salivary progesterone determinations. Horm Res 1984;20: 231–40.
30. Walker RF, Read GF, Riad-Fahmy D. Radioimmunoassay of progesterone in saliva: application to the assessment of ovarian function. Clin Chem 1979;25:2030–3.
31. Ellison PT, Lager C. Moderate recreational running is associated with lowered salivary progesterone profiles in women. Am J Obstet Gynecol 1986;154:1000–3.
32. Miller PB, Soules MR. The usefulness of a urinary LH kit for ovulation detection during menstrual cycles of normal women. Obstet Gynecol 1996;87:13–7.
33. Gudgeon K, Leader L, Howard B. Evaluation of the accuracy of the home ovulation detection kit, Clearplan, at predicting ovulation. Med J Aust 1990;152:344–9.
34. De Souza MJ. Menstrual disturbances in athletes: a focus on luteal phase defects. Med Sci Sports Exerc 2003;25:1553–63.
35. Sherman BM, Korenman SG. Measurement of plasma LH, FSH, estradiol and progesterone in disorders of the human menstrual cycle: the short luteal phase. J Clin Endocrinol Metab 1974;38:89–93.
36. Strott CA, Cargille CM, Ross GT, Lipsett MB. The short luteal phase. J Clin Endocrinol Metab 1970;30:246–51.
37. Ellison PT. Measurements of salivary progesterone. Ann N Y Acad Sci 1993;694:161–76.
38. Ellison PT, Lager C, Calfee BA. Low profiles of salivary progesterone among college undergraduate women. J Adolesc Health Care 1987;8: 204–7.
39. Vuorento T, Hovatta O, Kurunmaki H, Ratsula K, Huhtaniemi I. Measurements of salivary progesterone throughout the menstrual cycle in women suffering from unexplained infertility reveal high frequency of luteal phase defects. Fertil Steril 1990;54:211–16.
40. Vuorento T, Huhtaniemi I. Daily levels of salivary progesterone during menstrual cycle in adolescent girls. Fertil Steril 1992;58:685–90.
41. Hedeker D, Gibbons RD. Longitudinal data analysis. Hoboken, NJ: Wiley & Sons, Inc, 2006.
42. Singer JD, Willett JB. Applied longitudinal data analysis: modeling change and event occurrence. New York, NY: Oxford University Press, Inc, 2003.
43. Twisk JWR. Applied longitudinal data analysis for epidemiology: a practical guide. Cambridge, United Kingdom: Cambridge University Press, 2003.
44. Polivy J, Herman CP. Dieting and binging: a causal analysis. Am Psychol 1985;40:193–201.
45. Burleson MH, Gregory WL, Trevathan WR. Heterosexual activity and cycle length variability: effect of gynecological maturity. Physiol Behav 1991;50:863–6.
46. Treloar AE, Boynton RE, Behn BG, Brown BW. Variation in the human menstrual cycle through reproductive life. Int J Fertil 1967;12:77–126.
47. Rogol AD, Weltman A, Weltman JY, et al. Durability of the reproductive axis in eumenorrheic women during 1 yr of endurance training. J Appl Physiol 1992;72:1571–80.
48. Bauman JE. Basal body temperature: unreliable method of ovulation detection. Fertil Steril 1981;36:729–33.
49. McCarthy JJ Jr, Rockette HE. Prediction of ovulation with basal body temperature. J Reprod Med 1986;31:742–7.
50. Quagliarello J, Arny M. Inaccuracy of basal body temperature charts in predicting urinary luteinizing hormone surges. Fertil Steril 1986;45: 334–7.
51. Sowers M, Randolph JFJ, Crutchfield M, et al. Urinary ovarian and gonadotropin hormone levels in premenopausal women with low bone mass. J Bone Miner Res 1998;13:1191–202.
52. Metcalfe MG, Evans JJ, Mackenzie JA. Indices of ovulation: comparison of plasma and salivary levels of progesterone with urinary pregnanediol. J Endocrinol 1984;100:75–80.

53. Prior JC, Vigna YM, Barr SI, Kennedy S, Schulzer M, Li DKB. Ovulatory premenopausal women lose cancellous spinal bone: a five year prospective study. Bone 1996;18:261–7.
54. Tudor-Locke C, McColl RS. Factors related to variation in premenopausal bone mineral status: a health promotion approach. Osteoporos Int 2000;11:1–24.
55. MacKelvie KJ, Khan KM, McKay HA. Is there a critical period for bone response to weight-bearing exercise in children and adolescents? A systematic review. Br J Sports Med 2002;36:250–7.
56. Cobb KL, Kelsey JL, Sidney S, Ettinger B, Lewis CE. Oral contraceptives and bone mineral density in white and black women in CARDIA. Osteoporos Int 2002;13:893–900.
57. Kroger H, Tuppurainen M, Honkanen R, Alhava E, Saarikoski S. Bone mineral density and risk factors for osteoporosis—a population-based study of 1600 perimenopausal women. Calcif Tissue Int 1994;55:1–7.
58. New SA, Bolton-Smith C, Grubb DA, Reid DM. Nutritional influences on bone mineral density: a cross-sectional study in premenopausal women. Am J Clin Nutr 1997;65:1831–9.
59. Cui L-H, Shin M-H, Kweon S-S, et al. Relative contribution of body composition to bone mineral density at different sites in men and women in South Korea. J Bone Miner Metab 2007;25:165–71.
60. Janicka A, Wren TAL, Sanchez MM, et al. Fat mass is not beneficial to bone in adolescents and young adults. J Clin Endocrinol Metab 2007; 92:143–7.
61. Wang MC, Bachrach LK, Van Loan MD, Hudes M, Flegal KM, Crawford PB. The relative contributions of lean tissue mass and fat mass to bone density in young women. Bone 2005;37:474–81.
62. Petit MA, Beck TJ, Shults J, Zemel BS, Foster BJ, Leonard MB. Proximal femur bone geometry is appropriately adpated to lean mass in overweight children and adolescents. Bone 2005;36:568–76.
63. Reid IR, Ames R, Evans MC, et al. Determinants of total body and regional bone mineral density in normal postmenopausal women-key role for fat mass. J Clin Endocrinol Metab 1992;75:45–51.