# Metabolic assessment of female chronic dieters with either normal or low resting energy expenditures[1–4]

*Jacqui R Gingras, Vicki Harber, Catherine J Field, and Linda J McCargar*

**ABSTRACT**

**Background:** Chronic dieting syndrome can have negative physiologic and psychological consequences. Metabolic differences between female chronic dieters with normal and with low resting energy expenditures (REEs) have not been fully examined.

**Objective:** To determine whether differences existed between 2 groups ($n$ = 15/group) of female chronic dieters aged 21–49 y with either normal (≥100% of predicted) and with low (≤85% of predicted) REEs based on the equation of Mifflin et al.

**Design:** The sample was a nonrandomized convenience sample and the 2 groups were compared in an observational study design. Body composition, aerobic fitness, physical activity, glucose and insulin responses, leptin and thyroid hormone status, dietary intake, and dietary restraint were measured.

**Results:** Both groups were similar with respect to age, height, weight, and body mass index. The normal-REE group had a higher lean body mass and insulin response to a test meal, higher thyroxine and reverse triiodothyronine concentrations, and lower dietary restraint. Within both groups, leptin decreased significantly from baseline to 2 h after an oral-glucose-tolerance test. The groups did not differ significantly with respect to dietary intake, aerobic fitness, or physical activity.

**Conclusions:** Differences in insulin response were associated with higher ratios of abdominal to gluteal body fat in the normal-REE group. Leptin response appears to be due to normal diurnal variations in leptin production rather than a direct response to food consumption. It appears that a normal REE does not necessarily predict positive metabolic health among chronic dieters.

*Am J Clin Nutr* 2000;71:1413–20.

**KEY WORDS** Resting energy expenditure, chronic dieting syndrome, insulin, leptin, dietary restraint, metabolism, women

## INTRODUCTION

The chronic dieter is defined as an individual who consistently restricts energy intake to maintain an average or below-average body weight (1, 2). Chronic dieting syndrome describes individuals who *1*) have a persistent overconcern with body shape and weight, *2*) restrict their food choices for ≥2 y, and *3*) continually diet to achieve weight loss without success or with success but with weight regain (2). The physiologic consequences of chronic dieting are varied and may influence metabolism in the long-term (3).

Dieting continues to be a common practice among North American women. Dieting has become the norm—it is more common for a woman to be "on a diet" than not (4). It has also been suggested that deliberate, long-term energy restriction may lead to a decreased resting energy expenditure (REE) as an adaptive mechanism by the body to conserve energy (5–9). This theory has not been unanimously supported in the scientific literature (10–12). However, McCargar and McBurney (13) assessed the REEs of female chronic dieters ($n$ = 172) and found that a subset of women ($n$ = 30; 17.4%) had lower than normal REEs [≤85% of REE as predicted by the equation of Mifflin et al (14)]. Although researchers have continued to explore the effects of dieting on energy metabolism, questions still remain regarding the factors responsible for high or low REEs in female chronic dieters.

It appears that chronic dieting may have effects on body composition, aerobic fitness, biochemical indexes, dietary intake, and dietary restraint. Whether these effects vary when resting metabolism is different among chronic dieters is not known. In this study, women with either normal or low REEs were compared to further assess the relations between energy metabolism and associated metabolic variables.

## SUBJECTS AND METHODS

### Subjects

Participants from a previous study (13) who were chronic dieters and who had measured REEs of either ≤85% ($n$ = 30) or ≥100% ($n$ = 31) of predicted values were invited to participate in the present study. Of the 61 eligible participants, 30 women volunteered: $n$ = 15 in the normal-REE group (NREE) and $n$ = 15 in the

[1] From the Department of Agricultural, Food, and Nutritional Science and the Faculty of Physical Education and Recreation, University of Alberta, Edmonton, Canada.

[2] Preliminary data published in abstract form (FASEB J 1998:A204) and in a review article (J Clin Biochem Nutr 1999;26:77–84).

[3] Supported in part by The Canadian Fitness and Lifestyle Research Institute.

[4] Reprints not available. Address correspondence to LJ McCargar, 4-10 Agriculture/Forestry Centre, Department of Agricultural, Food, and Nutritional Science, University of Alberta, Edmonton, AB, Canada T6G 2 P5. E-mail: lmccargar@afns.ualberta.ca.

Received January 20, 1999.

Accepted for publication November 22, 1999.

low-REE group (LREE). Comparisons were made by using a cross-sectional, observational study design. Height, weight, REE, body composition, aerobic fitness, and biochemical indexes were measured and diet and activity questionnaires were completed. All measurements for an individual subject were completed in 4 visits to the study center over ≤4 wk.

All participants in the present study ($n$ = 30) confirmed that they met the criteria for chronic dieting syndrome (2) by answering "yes" to the following questions about dieting behavior: *1*) Are you persistently overconcerned with your body shape and weight?, *2*) Have you restricted your food choices for ≥2 y?, and *3*) Are you continually dieting to lose weight without success or with success but with weight regain?

Participation was also based on eligibility criteria, which included nonsmoking women aged 25–49 y with no chronic illness and no long-term use of medications known to affect carbohydrate metabolism or metabolic rate. Participants were screened for use of oral contraceptive pills (OCPs); no significant difference in OCP use was found between the NREE (20% OCP users) and LREE (27% OCP users) groups. The study received ethical approval from The University of Alberta, Faculty of Agriculture, Forestry, and Home Economics Human Ethics Review Committee. All participants provided informed consent before participating in the study.

### Anthropometry

Height was measured in subjects without shoes and was determined to the nearest 0.1 cm with a stadiometer. Weight was measured with participants in light clothing with a medical balance-beam scale (Healthometer; Continental Scale Corporation, Bridgeview, IL) and recorded to the nearest 0.1 kg. To determine the waist-to-hip ratio (WHR), the waist circumference was measured at the level of the umbilicus and the hip measurement was made at the level of maximal protrusion of the gluteal muscles.

### Resting energy expenditure

REE was measured by indirect calorimetry with a metabolic cart (VMax 29N; SensorMedics, Yorba Linda, CA). All measurements were obtained at the Department of Agricultural, Food, and Nutritional Science, Metabolic Testing Laboratory. Participants had their REEs measured during the follicular phase (days 1–7) of their menstrual cycles to confirm their placement in either the NREE or LREE group (13).

After a 12-h fast, the participants' REEs were assessed between 0800 and 1000. Participants rested in a supine position in a thermoneutral environment for ≥30 min before the start of the test. Use of stimulants—including caffeine, tobacco, and medication—and intense physical activity were prohibited 24 h before the REE measurement. Participants were instructed to remain awake, but relaxed, and to refrain from voluntary skeletal muscle activity for the duration of the test.

To calculate REE, a minimum of 15 min of steady state measurements were averaged. This typically required a total test period of 20–40 min. A respiratory quotient ≤0.85 was considered to reflect a fasted subject. Measured REE was calculated by using the Weir equation (15), which was adapted for use without the collection of urinary nitrogen.

To determine whether each participant had an NREE or LREE, actual REE was compared with predicted REE. The predicted REEs for these participants were calculated by using the equation of Mifflin et al (14). If the actual REE was equal to or greater than the predicted REE, the participant was placed in the NREE group. If the actual value was ≥15% less than the predicted REE, the participant was placed in the LREE group.

### Body composition

Body-composition measurements were performed by a trained technician using dual-energy X-ray absorptiometry (DXA) (Hologic QDR 4500A; Hologic, Inc, Waltham, MA). With the participant lying supine, wearing a hospital gown, and with all metal objects removed, a series of transverse scans were made from head to toe at a standardized transverse scan speed of 5 cm/s, with a total scan time of ≈3 min.

DXA technology assesses body composition by using a 3-compartment model [lean body mass (LBM), fat mass, and bone mass]; an X-ray tube and a K-edge filter generates and directs 2 energy levels of photon through tissue. On the basis of software calibrations, the ratio of soft tissue attenuation at the 2 energy levels is used to partition soft tissue into fat and lean compartments.

To quantify fat distribution, 2 additional regions were added to the DXA analysis. The central abdominal region ($R_1$) was defined by lateral borders aligned with the outer edge of the rib cage (excluding ≈30% of the abdominal subcutaneous fat), a superior border just above the L2 vertebrae, and an inferior border just below the L4 vertebrae, so the L2-L4 region was encompassed (16). This was an area shown by magnetic resonance imaging to contain a relatively high visceral and low subcutaneous fat content (17). The gluteal region ($R_2$) was defined by lateral borders along each side of the body, a superior border aligned with the highest points of the inner pelvis, and an inferior border through the midpoint of both femurs (18). $R_1$ and $R_2$ were determined after the participant's image was scanned into the Hologic software program. These regions were analyzed in terms of fat content and ratio of regional fat to total body fat. These data were compared with WHR, an additional measure of fat distribution.

### Aerobic fitness

All aerobic fitness testing was completed in the Exercise Physiology Laboratory (Faculty of Physical Education, University of Alberta, Edmonton) by a trained exercise physiologist using standardized procedures. Fitness tests were performed on a bicycle ergometer (model 818E; Monark, Turku, Sweden). The test began at a power output of ≈60 W, which was increased every 2 min by ≈30 W until volitional exhaustion. Gas exchange was monitored with a metabolic measurement cart (SensorMedics 2900). The peak or plateau oxygen consumption ($\dot{V}O_2$; <100 mL/min) during exercise at increasing power output was used to determine maximal oxygen consumption ($\dot{V}O_2$max). The test was stopped if one or more of the following criteria were met: the respiratory quotient was >1.1, the age-predicted or known maximal heart rate was reached, or there was volitional fatigue. Heart rate and revolutions were monitored and recorded every minute. $\dot{V}O_2$ data were recorded by computer every 30 s. Aerobic power was recorded as L $\dot{V}O_2$/min and as mL $O_2 \cdot min^{-1} kg^{-1}$. Measured fitness levels were subsequently compared with reported values for Canadian women (19).

Usual physical activity patterns were estimated by using the Baecke Questionnaire (20). Participants were asked questions pertaining to work, sport, and leisure activities. Responses were indicated on a Likert scale ranging from least to most (possible scores of 0–5 for each, for a total of 15). The total activity index was a sum of the 3 subscales (work, sport, and

**TABLE 1**
Characteristics of female chronic dieters in the normal- and low-REE groups[1]

| Variable | Normal REE ($n$ = 15) | Low REE ($n$ = 15) |
|---|---|---|
| Age (y) | 39.3 ± 5.5 | 39.5 ± 7.9 |
| Height (cm) | 162.0 ± 5.2 | 162.3 ± 6.0 |
| Weight (kg) | 84.9 ± 17.3 | 82.9 ± 26.5 |
| BMI (kg/m$^2$) | 32.0 ± 5.8 | 31.1 ± 8.9 |
| REE | | |
| Measured (kJ/d) | 6736 ± 816 | 5138 ± 1105[2] |
| Predicted (kJ/d)[3] | 6305 ± 858 | 6226 ± 1322 |
| Percentage of predicted (%)[4] | 107.1 ± 5.9 | 82.5 ± 2.8[6] |
| (kJ·kg body wt$^{-1}$·d$^{-1}$)[5] | 80.8 ± 8.4 | 63.6 ± 7.1[6] |
| (kJ·kg LBM$^{-1}$·d$^{-1}$) | 150.2 ± 9.6 | 128.5 ± 11.7[6] |
| RQ[7] | 0.79 ± 0.04 | 0.81 ± 0.04 |

[1] $\bar{x}$ ± SD. REE, resting energy expenditure; RQ, respiratory quotient.
[2,6] Significantly different from the normal REE group: [2]$P < 0.0001$, [6]$P < 0.00001$.
[3] Determined with the Mifflin et al equation (14).
[4] (Measured REE/predicted REE) × 100.
[5] Measured REE was used.
[7] Carbon dioxide production/oxygen consumption.

leisure); scores closer to 15 indicated that participants were very active, whereas scores closer to 0 indicated that participants were not very active.

### Biochemical analysis

Several biochemical indexes were measured, including glucose, insulin, leptin, thyroxine ($T_4$), triiodothyronine ($T_3$), and reverse $T_3$ ($rT_3$). Blood was collected during a 2-h oral-glucose-tolerance test (OGTT) at a public collection site (Dynacare-Kasper Medical Laboratory, Edmonton, Canada) by a trained technician.

Participants arrived at the collection site in the morning after an overnight fast (nothing to eat or drink except water for 12 h). After the collection of a fasting blood sample, participants were provided with 150 g white bread (75 g carbohydrate, 12 g protein, and 0.5 g fat) (21). The bread was cut into bite-size pieces and the weight was standardized before consumption. At the start of bread consumption, time 0 was indicated as defined by the National Diabetes Data Group (22). Participants were encouraged to consume all of the bread within 10–15 min.

Serum was analyzed for glucose and insulin at each time point (5 samples; at baseline and 30, 60, 90, and 120 min), leptin was determined at baseline and 120 min, and thyroid hormones ($T_4$, $T_3$, and $rT_3$) were determined at baseline only. The total amount of serum collected per participant was 35 mL.

Glucose concentrations were determined with a quantitative, enzymatic diagnostic assay (Sigma Diagnostics, St Louis). Insulin concentrations were determined by using a double-antibody enzyme linked immunosorbent assay (Boehringer Mannheim Immunodiagnostics, Amsterdam). Leptin concentrations were determined by using a human radioimmunoassay kit (Linco Research, St Charles, MO). $T_4$ and $T_3$ (Diagnostic Products Corporation, Los Angeles) and $rT_3$ (Biodata Diagnostics, Rome) concentrations were determined by solid-phase radioimmunoassay. Total glucose and insulin areas under the curve (AUCs) were determined by using the incremental model (23), which quantifies cumulative measures of these compounds in the blood over a given period.

### Dietary intake and restraint

Energy, carbohydrate, fat, and protein intakes were obtained by using a food-frequency questionnaire (FFQ) validated previously for determining individual dietary intakes (24). Portion sizes (small, medium, or large) were assigned to each item and a specific amount (ie, 125, 250, or 375 mL, or 0.5, 1.0, or 1.5 cups) was identified for each item. The FFQs were analyzed by using a computerized nutrition software program (version 6.0, THE FOOD PROCESSOR; ESHA Research, Salem, OR). The 10-item restrained-eating subscale (25) was used to determine deliberate weight control.

### Statistics

Statistical analyses were performed by using the software program SPSS (version 7.5; SPSS Inc, Chicago). All results are presented as means ± SDs. Independent $t$ tests were used to compare the metabolic data of each group. A repeated-measures two-way (group × time) analysis of variance (ANOVA) was conducted on data collected at more than one time point (glucose, insulin, and leptin). Pearson correlation coefficients were determined between relevant variables. The level of significance was set at $P < 0.05$.

## RESULTS

Descriptive characteristics of the 2 groups of female chronic dieters are presented in **Table 1**. Measured REE was significantly higher in the NREE group than in the LREE group. Group differences remained when REE was corrected for body weight and LBM.

The NREE group had a significantly greater LBM (44.8 ± 4.8 kg) than did the LREE group (39.8 ± 5.9 kg), as shown in **Table 2**. Total fat mass and bone mass did not differ significantly between groups. In addition to the standard measures of body composition, 2 regions ($R_1$ and $R_2$) were compared between participants. This comparison showed that the NREE group had higher ratios of abdominal to gluteal fat and WHR than did the LREE group.

Aerobic fitness and Baecke physical activity scores were not significantly different between the 2 groups. Both groups were

**TABLE 2**
Body composition and fat distribution in female dieters in the normal- and low-REE groups[1]

| Variable | Normal REE ($n$ = 15) | Low REE ($n$ = 15) |
|---|---|---|
| LBM (kg) | 44.8 ± 4.8 | 39.8 ± 5.9[2] |
| Fat mass (kg) | 34.7 ± 12.2 | 37.8 ± 18.4 |
| Bone mass (kg) | 2.2 ± 0.3 | 2.1 ± 0.3 |
| $R_1$ fat mass (kg) | 3.0 ± 1.3 | 2.8 ± 1.4 |
| $R_1$/total body fat | 0.09 ± 0.02 | 0.07 ± 0.01 |
| $R_2$ fat mass (kg) | 8.3 ± 2.9 | 9.8 ± 4.6 |
| $R_2$/total body fat | 0.24 ± 0.03 | 0.26 ± 0.62[3] |
| $R_1$ fat mass/$R_2$ fat mass | 0.37 ± 1.00 | 0.29 ± 0.07[4] |
| WHR | 0.81 ± 0.04[5] | 0.76 ± 0.06[6] |

[1] $\bar{x}$ ± SD. REE, resting energy expenditure; LBM, lean body mass; $R_1$, abdominal region as referenced from Carey et al (16); $R_2$, gluteal region as referenced from Ley et al (18); WHR, waist-to-hip ratio.
[2,4,6] Significantly different from the normal-REE group: [2]$P = 0.017$, [4]$P = 0.019$, [6]$P = 0.021$.
[3] Nearly significantly different from the normal-REE group, $P = 0.052$.
[5] $n = 12$.

**TABLE 3**
Physical fitness and activity of female chronic dieters in the normal and low-REE groups[1]

| Variable | Normal REE ($n$ = 15) | Low REE ($n$ = 14)[2] |
|---|---|---|
| Absolute $\dot{V}O_2$max (L $O_2$/min) | 2.34 ± 0.43 | 2.11 ± 0.46 |
| Relative $\dot{V}O_2$max (mL $O_2 \cdot min^{-1} \cdot kg$ body $wt^{-1}$)[3] | 28.13 ± 7.13 | 26.66 ± 5.42 |
| Relative $\dot{V}O_2$max (mL $O_2 \cdot min^{-1} \cdot kg$ $LBM^{-1}$) | 52.39 ± 8.70 | 53.70 ± 6.53 |
| Physical activity score[4] | 7.27 ± 1.22 | 7.02 ± 1.26 |
| Work index[5] | 2.52 ± 0.70 | 2.69 ± 1.00 |
| Sports index[5] | 2.13 ± 0.92 | 1.61 ± 0.63 |
| Leisure time index[5] | 2.62 ± 0.50 | 2.72 ± 0.42 |

[1] $\bar{x}$ ± SD. There were no significant differences between groups. REE, resting energy expenditure; $\dot{V}O_2$max, maximum oxygen consumption.
[2] One subject missing.
[3] Average value for women aged 30–39 y = 32.0 mL $O_2 \cdot min^{-1} \cdot kg$ body $wt^{-1}$ (19).
[4] From the Baecke questionnaire (20), scored on a 15-point Likert scale (score of 15 indicates high physical activity).
[5] From the Baecke questionnaire: the sum of these 3 activity indexes equals the total physical activity score, scored on a 5-point Likert scale (score of 5 for each index indicates high physical activity).

less physically fit than was the average Canadian woman aged 30–39 y, who had an average $\dot{V}O_2$max of 32 mL $O_2 \cdot min^{-1} \cdot kg$ body $wt^{-1}$ (19) (**Table 3**); $\dot{V}O_2$max values ranged from 26.7 to 28.1 mL $O_2 \cdot min^{-1} \cdot kg$ body $wt^{-1}$ for the 2 study groups.

A summary of the AUCs for total glucose and insulin are shown in **Figures 1** and **2**, respectively. Insulin responses differed between the 2 groups; the NREE group had almost twice the amount of insulin for an equivalent glucose load. There were no significant differences between the groups in terms of the average time required to consume the OGTT meal (NREE group: 16.7 ± 6.3 min; LREE group: 20.5 ± 6.6 min). Two women from the NREE group met the criteria for impaired glucose tolerance as determined from the classification guidelines of the National Diabetes Data Group (22). Removal of these data did not alter the significance of the results, so the data were included.

There were no significant differences in fasting and 120-min leptin values between the 2 groups; however, there were within-group differences (**Figure 3**). In both groups, leptin decreased significantly from fasting to 120 min ($P < 0.001$). Furthermore, the correlation between fasting insulin and fasting leptin was positive and significant ($r = 0.395$, $P < 0.05$).

The NREE group had significantly higher $T_4$ (115.28 ± 27.53 compared with 93.89 ± 14.20 nmol/L, respectively; $P = 0.014$) and $rT_3$ (0.22 ± 0.05 compared with 0.18 ± 0.01 nmol/L, respectively; $P = 0.019$) values than did the LREE group. There were no significant differences in $T_3$ values between groups (NREE group: 2.3 ± 0.5 nmol/L; LREE group: 2.2 ± 0.6 nmol/L). All thyroid values were within normal physiologic ranges. Of the 3 thyroid hormones tested, only $T_4$ correlated positively and significantly with REE ($r = 0.472$, $P < 0.05$)

There were no significant differences in dietary intakes (total energy, fat, carbohydrate, and protein) between the groups (**Table 4**). Average energy intakes were 9678 ± 3176 kJ (NREE group) and 8477 ± 2448 kJ (LREE group). The NREE group (2.81 ± 0.52) had significantly lower restraint scores ($P = 0.011$) than did the LREE group (3.52 ± 0.84).

## DISCUSSION

On the basis of these findings, female chronic dieters with normal REEs had higher LBMs, insulin responses, ratios of abdominal to gluteal body fat, and $T_4$ and $rT_3$ concentrations, and lower dietary restraint scores than did those with low REEs. These findings establish that these 2 types of chronic dieters have distinct physiologic and behavioral characteristics. Participants were considerably heavier than what is considered desirable on the basis of body mass index (BMI; in $kg/m^2$) (26). The relevance of BMI in determining health risk has been a subject of recent debate. Physical fitness, not BMI, has been suggested to be a better predictor of mortality or morbidity, even among overweight individuals (27).

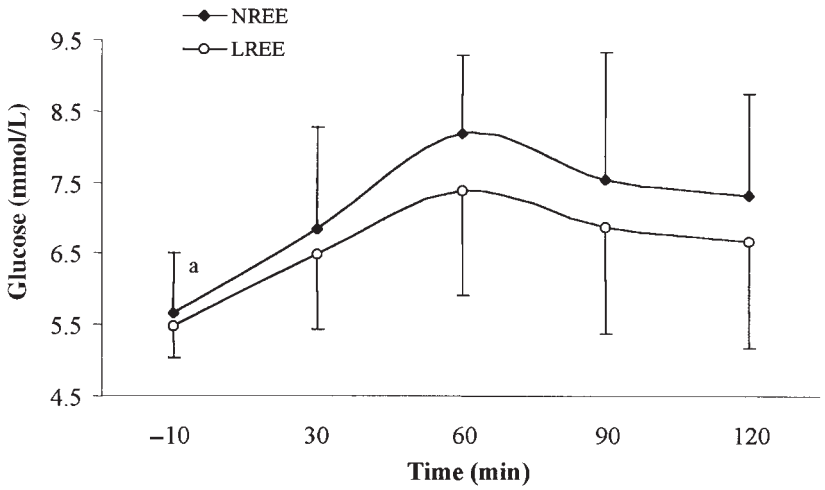


**FIGURE 1.** Mean (±SD) glucose concentrations in female chronic dieters with either normal (NREE) or low (LREE) resting energy expenditures before and after consumption of 75 g carbohydrate ("a" indicates the time of consumption). The area under the curve for glucose was 193.48 ± 71.24 mmol·min/L in the NREE group and 146.94 ± 112.60 mmol·min/L in the LREE group. There were no significant differences between groups.

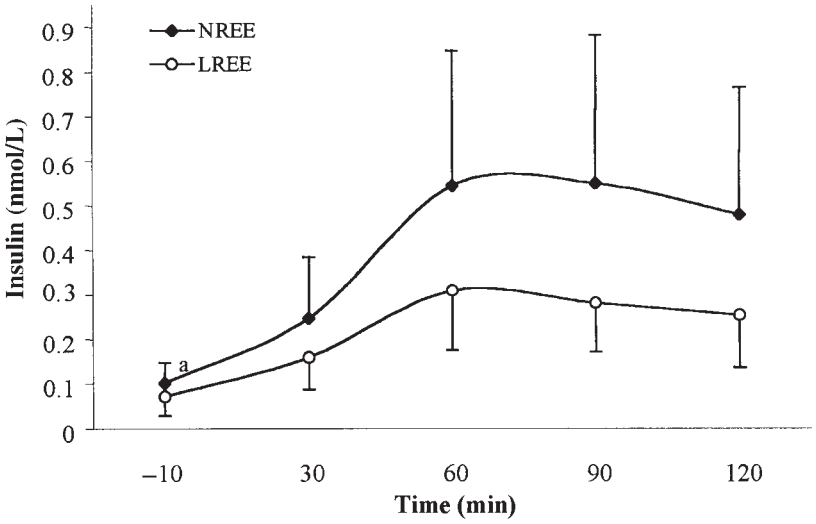


**FIGURE 2.** Mean (±SD) insulin concentrations in female chronic dieters with either normal (NREE) or low (LREE) resting energy expenditures before and after consumption of 75 g carbohydrate ("a" indicates the time of consumption). The area under the curve for insulin was 36.63 ± 20.16 nmol·min/L in the NREE group and 18.80 ± 7.86 nmol·min/L in the LREE group. The AUC of the NREE group was significantly greater than that of the LREE group, $P < 0.01$. There was no group × time interaction.

Assessment of body composition showed a significant difference in LBM between the NREE and LREE groups. LBM represents the most metabolically active tissue and accounts for 50–80% of the variation in REE (28–33). Other influences on REE include age, sex, and genetic factors (34). Genetic differences could explain the higher LBM of female chronic dieters with higher REEs (32). Although LBM differed significantly between the groups, there were no significant differences in fat mass, bone mass, or total body weight between the 2 groups when measured by DXA. The only body compartment that DXA does not assess directly is total body water; DXA assumes a total body water content of 73.2%. It is possible that there were differences in total body water between groups; however, such differences were not measured.

The higher WHR values in the NREE group supported regional DXA findings. Although differences in the ratio of abdominal to gluteal fat distribution were not anticipated, the higher ratio in the NREE group may have been the result of genetic predisposition, increased androgen hormonal activity, increased dietary fat intake, decreased physical activity, or altered rates of lipolysis (35). The present research could only rule out dietary fat intake and physical activity from the above variables.

Abdominal fatness is associated with an increased prevalence of glucose intolerance, insulin resistance, elevated blood pressure, and elevated blood lipids in both men and women (36). It has been suggested that the abdominal or android fat pattern may represent an increase in the size or number of more metabolically active intraabdominal fat cells, or both (37). These fat cells release fatty acids directly into the portal circulation, which may interfere with insulin clearance in the liver, thus affecting various metabolic processes (16, 35, 37). Therefore, we postulated that the higher ratio of abdominal to gluteal fat in the NREE group may have contributed to the observed insulin resistance in this group.

Previously, Manore et al (6) reported that dieters were significantly heavier and fatter than nondieting control subjects, but that fat-free mass was similar between groups. It is suggested that repeated bouts of dieting may alter body composition by increasing the efficiency of food utilization (38). However, it has not been determined whether women diet because they are heavier or whether they become heavier because they diet. As such, generalizations about the body compositions of chronic dieters being influenced by dieting behaviors cannot be inferred from the results presented here. Although the study participants as a group were considerably less fit than was the average Canadian woman aged 30–39 y (19), aerobic fitness and physical activity did not correlate with REE. These results are similar to those reported in the literature (39, 40).

Although serum insulin concentrations were significantly higher in the NREE than in the LREE group, these results were not considered clinically significant because all insulin values were within normal physiologic ranges. Circulating concentrations of insulin after a 2-h OGTT in 7 normal-weight subjects (3 women and 4 men with a mean BMI of 23.8 ± 1.0) were similar to those in the NREE group (31.0 ± 14.3 compared with 36.6 ± 20.2 pmol/L, respectively) (21). Catecholamines (epinephrine and norepinephrine) have been correlated with both a normal REE and insulin resistance. Further investigation of the relations of sympathetic nervous system activity, energy metabolism, and insulin resistance among chronic dieters is warranted.

The average 23.9% decrease in serum leptin values 120 min postprandially in all participants resulted from diurnal variations in leptin rather than from a direct response to feeding. Sinha et al (41) found that leptin concentrations decreased from ≈0300 to 0930 in lean, obese and obese, type 2 diabetic participants even when breakfast was consumed at 0800. In the present study, all leptin samples were drawn early in the morning. It appears that the decrease in serum leptin concentrations after breakfast was a continuation of the natural decline from nighttime to morning (41–43).

The relation between leptin and insulin metabolism has yet to be completely understood with respect to effects on abdominal

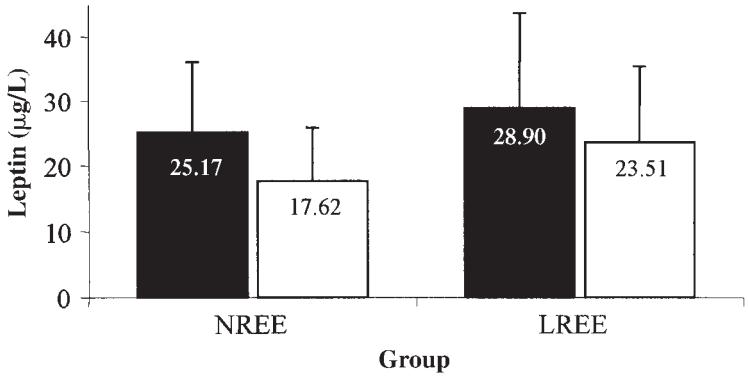


**FIGURE 3.** Mean (±SD) leptin concentrations in female chronic dieters with either normal (NREE) or low (LREE) resting energy expenditures before and after consumption of 75 g carbohydrate. There was a significant main effect of time ($P < 0.001$) but no group × time interaction.

fat distribution. Leptin is believed to regulate overall body fat content, which can then be directed by predominant sex hormones (free testosterone) to an upper body (abdominal) distribution (44, 45). Kennedy et al (45) determined that women experience hyperleptinemia (20% increase over baseline values) during the final 60 min of a 180-min induced hyperinsulinemic state (euglycemic hyperinsulinemic clamp). Kolaczynski et al (46) showed that women experience hyperleptinemia after 48 h of a prolonged (64–72 h) euglycemic hyperinsulinemic clamp protocol; however, the sample was small ($n = 8$). Relative insulin resistance as a function of abdominal body fat distribution would thus explain the association between insulin and high leptin concentrations. As such, insulin resistance would not be the direct consequence of hyperleptinemia but, rather, the byproduct of accumulated abdominal fat that occurs with progressive centralized obesity as observed in the NREE group in the present study.

Significant between-group differences were detected in serum $T_4$ and $rT_3$ but not in serum $T_3$. Although all group means were within normal physiologic ranges, the physiologic consequences of differences within the normal range are not known. The finding that $T_3$ concentrations were not significantly different between groups nor correlated with REE (absolute or relative) was unexpected. Serum $T_3$ concentrations often reflect states of energy deficiency (47) or suppressed resting metabolic rate (48); however, it appears that variations in energy expenditure as measured by indirect calorimetry are not related to $T_3$ in female chronic dieters.

As reported in the literature, changes in thyroid status are controlled by dietary intake (49, 50). Conversion of $T_4$ to $rT_3$ (5′-monodeiodination of $T_4$) increases with energy restriction, carbohydrate restriction, and prolonged exercise (51). Differences in thyroid status observed between the 2 groups of chronic dieters were evidence of increased amounts of $T_4$ being converted to $rT_3$, as occurs during thyroid hormone economy (52, 53). The greater concentrations of $T_4$ and $rT_3$ in the NREE group suggest that this group probably consumed less energy and exercised more than did the LREE group; however, this theory was not supported by the data in the present study.

Preoccupation with food and its nutritional composition are common characteristics of chronic dieters (54). However, several participants remarked while completing the FFQ that they did not realize how much of a certain food they ate until they had to record the amount on the FFQ. This realization may not have been possible with a diet record or a 24-h dietary recall.

Participants with normal REEs had lower dietary restraint scores than did those with low REEs. This finding was substantiated by Platte et al (5), who observed lower REEs in restrained eaters even after LBM was controlled for. The significantly lower REE in restrained eaters could be a cause or a consequence of their eating behavior. If lower REEs are the result of genetic predisposition, the restrained eating style may be a behavioral adaptation used to prevent weight gain. The LREE group, which appeared to have more restrictive eating behaviors, had dietary restraint scores similar to those of failed dieters (55) and individuals attending Weight Watchers (56).

The use of *t* tests for the statistical analysis may be considered a limitation of the study; however, we used the test for 2 reasons. First, our 2 comparison groups had significantly different REEs ($P < 0.0001$), which was our key interest and which we expected would influence all of the other variables. Additionally, the 2 groups were very similar in age, height, and weight and were of the same sex, all of which are factors that normally influence REE. However, in the present study these factors did not seem to have a direct effect. Thus, we were convinced that the 2 groups had clearly different resting metabolisms. Second, each variable that we measured, on the basis of the literature, was expected to be different because of the different metabolisms of the 2 groups. Thus, a Bonferroni correction was done. The sample size was calculated on the basis of a $P$ value of 0.05 and a β value of 0.10 and the requirement of 15 participants per group was achieved.

The results of the present study indicated differences in metabolic variables between the 2 groups of female chronic dieters; body-composition variables, particularly LBM, best predicted REE. The results also indicated that a normal REE was associated

**TABLE 4**
Energy and macronutrient intakes of female chronic dieters in the normal- and low-REE groups[1]

| Variable | Normal REE ($n = 15$) | Low REE ($n = 15$) |
|---|---|---|
| Energy (kJ/d) | 9678 ± 3176 | 8477 ± 2448 |
| Carbohydrate (% of energy) | 46.8 ± 7.4 | 51.8 ± 9.1 |
| Protein (% of energy) | 17.9 ± 3.2 | 18.0 ± 4.1 |
| Fat (% of energy) | 37.2 ± 7.7 | 32.0 ± 8.7 |

[1] $\bar{x}$ ± SD. There were no significant differences between groups. REE, resting energy expenditure.

with insulin resistance. Insulin resistance has been shown to correlate positively with a high ratio of abdominal to gluteal fat mass, which was shown in women in the NREE group. Differences in leptin were likely due to normal diurnal variations rather than to the influence of food consumption or REE. The higher $T_4$ and $rT_3$ values in the NREE group support the positive association between thyroid production and metabolic rate; however, this difference has been shown mainly under conditions of energy restriction, which was not the case for the NREE group. More research is required to elucidate the complex interactions of insulin, leptin, and thyroid hormones in energy metabolism. The LREE group had higher dietary restraint than did the NREE group. This may partly explain the lower REE of the LREE group because dietary restraint has been shown to the reduce metabolic rate.

The female chronic dieters in the LREE group in the present study had a low LBM, low metabolic hormone concentrations, and high dietary restraint. Health promotion programs that emphasize regular physical activity and normalized eating behaviors (ie, a healthy diet) may be beneficial for this group.

We gratefully acknowledge the support of the following individuals for their integral role in assisting with this research: Nigel Gann, Becky McBurney, Irene Holland, Susan Goruk, and Shirley Shostak.

## REFERENCES


1. Beals KA, Manore MM. The prevalence and consequences of subclinical eating disorders in female athletes. Int J Sport Nutr 1994; 4:175–95.
2. Grodner M. Forever dieting: chronic dieting syndrome. J Nutr Educ 1992;24:207–10.
3. Manore MM. Chronic dieting in active women: what are the health consequences? Women's Health Issues 1996;6:332–41.
4. Polivy J, Herman CP. Breaking the diet habit. New York: Basic Books, 1983.
5. Platte P, Wurmser H, Wade SE, Mercheril A, Pirke KM. Resting metabolic rate and diet-induced thermogenesis in restrained and unrestrained eaters. Int J Eat Disord 1996;20:33–41.
6. Manore MM, Berry TE, Skinner JS, Carroll SS. Energy expenditure at rest and during exercise in nonobese female cyclical dieters and in nondieting control subjects. Am J Clin Nutr 1991;54:41–6.
7. Blackburn GL, Wilson GT, Kanders BS, et al. Weight cycling: the experience of human dieters. Am J Clin Nutr 1989;49(suppl):1105–9.
8. Elliot DL, Goldberg L, Kuehl KS, Bennett WM. Sustained depression of the resting metabolic rate after massive weight loss. Am J Clin Nutr 1989;49:93–6.
9. Steen SN, Oppliger RA, Brownell KD. Metabolic effects of repeated weight loss and regain in adolescent wrestlers. JAMA 1993; 260:47–50.
10. Wing RR, Matthews KA, Kuller LH, et al. Environmental and familial contributions to insulin levels and change in insulin levels in middle-aged women. JAMA 1992;268:1890–5.
11. National Task Force on the Prevention and Treatment of Obesity. Weight cycling. JAMA 1994;272:1196–202.
12. Wadden TA, Bartlett S, Letizia KA, Foster GD, Stunkard AJ, Conill A. Relationship of dieting history to resting metabolic rate, body composition, eating behavior, and subsequent weight loss. Am J Clin Nutr 1991;56(suppl):203S–8S.
13. McCargar LJ, McBurney RF. The chronic dieting syndrome: metabolic and behavioral characteristics. Can J Diet Pract Res 1999; 60:227–30.
14. Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr 1990;51:241–7.
15. Weir JBV. New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol 1949;109:1–9.
16. Carey DG, Jenkins AB, Campbell LV, Freund J, Chisholm DJ. Abdominal fat and insulin resistance in normal and overweight women: direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. Diabetes 1996;45:633–8.
17. Ross R, Shaw KD, Martel Y, de Guise J, Avruch L. Adipose tissue distribution measured by magnetic resonance imaging in obese women. Am J Clin Nutr 1993;57:470–5.
18. Ley CJ, Lees B, Stevenson JC. Sex- and menopause-associated changes in body-fat distribution. Am J Clin Nutr 1992;55:950–4.
19. Fitness and Amateur Sport Canada. Canadian standardized test of fitness. 3rd ed. Ottawa: Fitness and Amateur Sport Canada, 1986.
20. Pols MA, Peeters PHM, Bueno-de-Mesquita HB, et al. Validity and repeatability of a modified Baecke questionnaire on physical activity. Int J Epidemiol 1995;24:381–8.
21. Wolever TMS, Bolognesi C. Source and amount of carbohydrate affect postprandial glucose and insulin in normal subjects. J Nutr 1996;126:2798–806.
22. National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes 1979;28:1039–57.
23. Wolever TMS, Jenkins DJA. The use of the glycemic index in predicting the blood glucose response to mixed meals. Am J Clin Nutr 1986;43:167–72.
24. Bright-See E, Catlin G, Godin G. Assessment of the relative validity of the Ontario Health Survey Food Frequency Questionnaire. J Can Diet Assoc 1994;55:33–8.
25. van Strien T, Frijters JER, Bergers GPA, Defares PB. The Dutch eating behaviour questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behaviour. Int J Eat Disord 1986; 5:295–315.
26. Pi-Sunyer FX, Becker DM, Bouchard C, et al. Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. JAMA 1998;98:1178–91.
27. Blair SN, Shaten J, Brownell KD, Collins G, Lissner L. Body weight change, all-cause mortality, and cause-specific mortality in the Multiple Risk Factor Intervention Trial. Ann Intern Med 1993; 119:749–57.
28. Ravussin E, Burnand B, Schutz Y, Jequier E. Twenty-four-hour energy expenditure and resting metabolic rate in obese, moderately obese, and control subjects. Am J Clin Nutr 1982;35:566–73.
29. Bogardus C, Lillioja S, Ravussin E, et al. Familial dependence of the resting metabolic rate. N Engl J Med 1986;315:96–100.
30. Sims EAH, Danforth E. Expenditure and storage of energy in man. J Clin Invest 1987;79:1019–25.
31. Ravussin E, Bogardus C. Relationship of genetics, age, and physical fitness to daily energy expenditure and fuel utilization. Am J Clin Nutr 1989;49:968–75.
32. Bouchard C, Tremblay A, Nadeau A, et al. Genetic effects in resting and exercise metabolic rates. Metabolism 1989;38:364–70.
33. Astrup A, Buemann B, Christensen NJ, et al. The contribution of body composition, substrates, and hormones to the variability in energy expenditure and substrate utilization in premenopausal women. J Clin Endocrinol Metab 1992;74:279–86.
34. Rice T, Tremblay A, Deriaz O, Perusse L, Rao DC, Bouchard C. A major gene for resting metabolic rate unassociated with body composition: results from the Quebec Family Study. Obes Res 1996;4:441–9.
35. Krotkiewski M. Can body fat patterning be changed? Acta Med Scand Suppl 1988;723:213–23.
36. Pouliot MC, Despres JP, Lemieux S, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol 1994;73:460–8.
37. Kissebah AH, Vydelingum N, Murray R, et al. Relation of body fat distribution to metabolic complications of obesity. J Clin Endocrinol Metab 1982;54:254–8.
38. Brownell KD, Steen SN, Wilmore JH. Weight regulation practices in athletes; analysis of metabolic and health effects. Med Sci Sports Exerc 1987;19:546–56.

39. Albanes D, Conway JM, Taylor PR, Moe PW, Judd J. Validation and comparison of eight physical activity questionnaires. Epidemiology 1990;1:65–71.
40. Cauley JA, LaPorte RE, Sandler RB, Schramm MM, Kriska AM. Comparison of methods to measure physical activity in postmenopausal women. Am J Clin Nutr 1987;45:14–22.
41. Sinha MK, Ohannesian JP, Heiman ML, et al. Nocturnal rise of leptin in lean, obese, and non-insulin dependent diabetes mellitus subjects. J Clin Invest 1996;97:1344–7.
42. Laughlin GA, Yen SSC. Hypoleptinemia in women athletes: absence of a diurnal rhythm with amenorrhea. J Clin Endocrinol Metab 1997;82:318–21.
43. Weigle DS, Duell PB, Connor WE, et al. Effect of fasting, refeeding, and dietary fat restriction on plasma leptin levels. J Clin Endocrinol Metab 1997;82:561–5.
44. Evans DJ, Hoffmann RG, Kalkhoff RG, Kissebah AH. Relationship of androgenic activity to body fat topography, fat cell morphology, and metabolic aberrations in premenopausal women. J Clin Endocrinol Metab 1983;57:304–10.
45. Kennedy A, Gettys TW, Watson P, et al. The metabolic significance of leptin in humans: gender-based differences in relationship to adiposity, insulin sensitivity, and energy expenditure. J Clin Endocrinol Metab 1997;82:1293–300.
46. Kolaczynski JW, Nyce MR, Considine RV, et al. Acute and chronic effect of insulin on leptin production in humans: studies in vivo and in vitro. Diabetes 1996;45:699–701.
47. Loucks AB, Callister R. Induction and prevention of low-T3 syndrome in exercising women. Am J Physiol 1993;264:R924–30.
48. Myerson M, Gutin B, Warren MP, et al. Resting metabolic rate and energy balance in amenorrheic and eumenorrheic runners. Med Sci Sports Exerc 1991;23:15–22.
49. Mathieson RA, Walberg JL, Gwazdauskas FC, Hinkle DE, Gregg JM. The effect of varying carbohydrate content of a very-low-caloric diet on resting metabolic rate and thyroid hormones. Metabolism 1986;35:394–8.
50. O'Brian JT, Bybee DE, Burman KD, et al. Thyroid hormone homeostasis in states of relative caloric deprivation. Metabolism 1980; 29:721–7.
51. O'Connell M, Robbins DC, Horton ES, Sims EAH, Danforth E. Changes in serum concentrations of triiodothyronine and reverse triiodothyronine during prolonged moderate exercise. J Clin Endocrinol Metab 1978;49:242–5.
52. Loucks AB, Laughlin GA, Mortola JF, Girton L, Nelson JC, Yen SSC. Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. J Clin Endocrinol Metab 1992;65:514–8.
53. van der Heyden JTM, Docter R, van Toor H, Wilson JHP, Hennemann G, Krenning EP. Effects of caloric deprivation on thyroid hormone tissue uptake and generation of low-$T_3$ syndrome. Am J Physiol 1986;251:E156–63.
54. Johansson L, Solvoll K, Bjorneboe GEA, Drevon CA. Under- and overreporting of energy intake related to weight status and lifestyle in a nationwide sample. Am J Clin Nutr 1998;68:266–74.
55. Ogden J. The measurement of restraint: confounding success and failure? Int J Eat Disord 1993;13:69–76.
56. Wardle J. Eating style: a validation study of the Dutch Eating Behaviour Questionnaire in normal subjects and women with eating disorders. J Psychosom Res 1987;31:161–9.


The American Journal of Clinical Nutrition