Energy balance during an 8-wk energy-restricted diet with and without exercise in obese women1–3

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ABSTRACT The effects of severe energy restriction alone (2.0 MJ/d for 4 wk and subsequently 3.5 MJ/d for 4 wk) or energy restriction plus moderate exercise on energy balance were studied in 20 healthy obese women. Subjects aged 25–50 y were matched on the basis of body mass index and percentage body fat and randomly assigned to diet alone (D) or diet and exercise (DE) for 8 wk. DE resulted in a significantly increased loss of fat mass compared with D (7.8 ± 0.8 compared with 5.5 ± 0.8 kg; P < 0.05). The average daily metabolic rate measured with doubly labeled water decreased with both treatments, with no differences between the treatments. Energy balance data show that the DE treatment resulted in a significantly greater energy deficit than the D treatment. The relative contribution of fat to energy expenditure during exercise was significantly enhanced by DE treatment whereas it did not change after D. The energy expended on physical activity was not changed at the end of both treatments, with no differences between the two groups. The unchanged energy expended on physical activity indicates that DE might be accompanied by partial compensation of daily physical activities outside the training for the energy expended during the training. The energy deficit due to energy restriction alone was not compensated by a decrease in free-living daily physical activities. Addition of moderate exercise to an energy-restriction program in obese women has advantages with respect to changes in body composition, energy expenditure, and substrate utilization. Am J Clin Nutr 1995;62:722–9.

KEY WORDS Metabolic rate, exercise, obesity, energy expenditure, fat oxidation, doubly labeled water, physical activity

INTRODUCTION

Decreased energy expenditure concomitant with weight loss has been suggested as one of the causes slowing subsequent weight loss and facilitating weight regain. Loss of fat-free mass and changes in thermogenic hormones contribute to these decreased energy expenditure rates (1). In a recent study by Rising et al (2) it was suggested that obesity is associated with low physical activity. Because physical activity is one of the most variable components of energy expenditure, addition of exercise to a weight loss program in obese subjects might have favorable effects. By increasing exercise, higher rates of energy expenditure can be achieved, and increased energy expenditure might result in a greater weight loss, when energy intake does not change. Furthermore, exercise might have stimulating effects on fat oxidation, which has been shown to be decreased with increasing obesity (3).

It has been suggested that the potential benefit of adding exercise to an energy-restriction regimen for achieving weight loss in obesity is mainly attenuation of fat-free mass losses. A recent meta-analysis by Ballor and Poehlman (4) suggests that the majority of the studies on the effects of adding modest regular exercise to a dietary-restriction regimen resulted in preservation of fat-free mass, rather than in larger weight loss. Because the extent of adaptation to diet alone or combined with exercise treatment is related to the magnitude of the energy deficit, the lack of conclusive results may be explained partially by concomitant changes in other components determining energy balance. Altered food intake or changes in normal daily activities (5) have been suggested as compensatory adaptations for the extra energy deficit from the addition of exercise. However, no compensation in habitual daily activities was shown in obese boys on an exercise treatment (6) or in obese women following a diet treatment alone (2.9–3.5 MJ/d) or combined with exercise (7). However, in the latter study, the observations were limited to measures obtained by using heart-rate monitoring and actometer recordings.

To evaluate the effects of adding of exercise to an energy-restriction regimen in the treatment of obesity, information about the net changes in total energy expenditure and its various components is necessary. The doubly labeled water (DLW) technique provides an excellent way to measure total energy expenditure over 1–3 wk under free-living conditions (8). Energy expenditure, measured with DLW, has an accuracy of 1–3% and a precision of 2–8% when validated against respiratory (9). Therefore, the present study focuses on the effects of a very-low-energy diet alone or in combination with an exercise program on total free-living energy expenditure and its components in obese women.

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722

SUBJECTS AND METHODS

Subjects

Twenty female subjects aged 25–50 y volunteered for the present study. They were a subsample from a larger group of obese premenopausal women who were recruited for a weight-loss program. All subjects were apparently healthy according to the results of a medical examination. Before treatment all subjects gave their written informed consent to participate in the study, which was approved by the University’s ethical committee. The subjects were matched on the basis of their body mass index (BMI; in kg/m²) and percentage body fat and randomly assigned to either a diet alone (D) or a diet and exercise group (DE). Baseline characteristics of the subjects participating in the present study are listed in Table 1.

Experimental design

The study lasted 10 wk. The first 2 wk were considered baseline, during which average daily metabolic rate (ADMR), sleeping metabolic rate (SMR), exercise-induced energy expenditure, body composition, and maximal oxygen uptake (VO₂max) were determined. After this period, the subjects followed the D or DE program for 8 wk. All measurements were repeated at the end of this 8-wk period.

Diet

The energy-restriction program took place over an 8-wk period that was divided into two parts. The first part consisted of a low-energy formula diet (Modifast; Sandoz, Bern, Switzerland) providing 2.0 MJ/d for 4 wk. It provided 50 g carbohydrate, 52 g protein, 7 g fat, and a micronutrient content that meets the Dutch recommended daily intake (10). The second part consisted of a mixed diet providing 3.5 MJ/d from week 5 until week 9. This diet contained 1.4 MJ/d of the formula diet and was supplemented to 3.5 MJ/d by a free choice of foodstuffs. During the entire period subjects were instructed to record their food intake. During the energy-restriction period the subjects came to the laboratory once a week to have their weight measured and food records checked.

Exercise training

The DE group participated in a training program that took place at a professional sports center, three times per week during the entire period. The training session was supervised by a professional trainer twice per week. These sessions lasted 90 min and consisted of alternating sessions of aerobic dancing and fitness. The aerobic dance sessions were performed to music and consisted of routines that combined various dance steps with other whole-body movements including knee lifts, trunk twists, jumping jacks, kicks, hops, and jogging in place. The fitness sessions included cardiocircuit and strength training using free weights and mechanical gym equipment, regularly presented as circuit weight training. The session started with a 15-min warm-up of cycling, stepping, and/or rowing, followed by 60 min of aerobic or fitness activities as described, and ended in a 15-min cool-down with stretching. Additionally, at least one time per week, subjects trained individually according to a personal fitness schedule for 60 min. The attendance of the subjects was monitored. The exercise intensities were regularly determined by heart-rate recordings (Sporttester; Polar Electro, Kempele, Finland). The prescribed intensity of the exercise program was 50–60% of VO₂max.

Measurements

Maximal aerobic capacity

Before and at the end of the D or DE treatment, each subject’s VO₂max and peak mechanical power (Wmax) were determined by using a progressive-continuous cycling test on an ergometer (Lode, Groningen, Netherlands). After an initial 5-min period of cycling at 40 W, followed by 4 min at 80 W, the workload was increased by 20 W/min until the subjects were exhausted. Wmax was defined as the highest load a subject could maintain for 1 min. During the test, ventilatory and gas-exchange responses were measured continuously by using a computerized open-circuit indirect calorimetry system (Oxycon Beta; Minntech, Netherlands). Heart rate was recorded continuously by electrocardiogram. Criteria for maximal exercise were forced ventilation, leveling off of oxygen uptake, or a respiratory-exchange ratio (RER) > 1.1 at maximal exercise. The highest oxygen uptake achieved for ≥30 s was taken as VO₂max. Values of Wmax at weeks 0 and 8 were used to determine each individual intensity of a standardized activity test at weeks 0 and 8, respectively.

Energy expenditure

SMR was measured during an overnight stay (1900–0700 h) in a computerized open-circuit indirect calorimeter (14 m²) equipped with a bed, toilet, television set, and chair. The volume of air drawn through the chamber was measured by a dry gas meter (Schlumberger, Dordrecht, The Netherlands) and continuously analyzed by a paramagnetic oxygen analyzer (Servomex, Crowborough, UK) and an infrared carbon dioxide analyzer (Hartmann & Braun, Frankfurt, Germany). Air-flow rate and the oxygen and carbon dioxide concentrations of the ingoing and outgoing air were used to compute oxygen consumption and carbon dioxide production on-line through an automatic acquisition system interfaced with a Macintosh computer (Cupertino, CA). Basal RER and energy expenditure (SMR) were calculated from oxygen consumption and carbon dioxide production (11) during the sleeping period between 0300 and 0600 h and controlled for extra physical activity by a Doppler radar system.

On the morning after the overnight stay in the respiration chamber, exercise-induced energy expenditure was measured, still in the fasting state, to examine the energy costs of a

| TABLE 1 Baseline characteristics of subjects treated with 8 wk of diet alone (D) or diet and exercise (DE) |
|-------------|-------------|-------------|-------------|-------------|
| D (n = 10)  | DE (n = 10) |
| Age (y)     | 36.6 ± 2.3  | 39.3 ± 5.4  |
| Weight (kg) | 88.3 ± 1.8  | 90.4 ± 2.9  |
| Body mass index (kg/m²) | 31.7 ± 0.9  | 32.4 ± 1.3  |
| Body fat (%)| 41.8 ± 1.5  | 41.6 ± 1.5  |
| Fat mass (kg) | 37.0 ± 1.8  | 38.0 ± 2.9  |
| Fat-free mass (kg) | 51.4 ± 1.3  | 52.4 ± 1.8  |
| Waist-hip ratio | 0.84 ± 0.02 | 0.83 ± 0.02 |

\( ^{\text{7}}^{\text{x}} \) ± SEM.
specific standardized activity. The exercise protocol consisted of 45 min of exercise on a cycle ergometer at a workload of 45% of the measured Wmax. The exercise was preceded by a 30-min supine resting period (basal measurement) and followed by a 60-min recovery period with the person in supine rest. Respiratory gas exchange measurements were done continuously during the basal and recovery periods by means of a computerized open-circuit ventilated hood system. During exercise, respiratory gas-exchange responses were measured for a total 20 min, integrated over 5-min intervals, by using computerized indirect calorimetry (Oxycon Beta). Both systems were calibrated with standard gases before each measurement and during the measurement periods. Measured RER was used to calculate carbohydrate and fat oxidation rates, assuming that the RER reflected the nonprotein respiratory quotient (12).

During the first 2 wk of the study (baseline period) and the last 2 wk of the energy restriction with or without exercise treatment, ADMR was assessed by the DLW method (9). The dosage calculation was based on body mass and percentage fat to create 300 ppm excess 18O and 150 ppm excess 2H. The isotope drink was administered between 2200 and 2300 after subjects had emptied their bladders and baseline urine samples had been collected. Further urine samples were collected on days 1, 8, and 15 after the first voiding and between 2000 and 2300. Isotopes were measured in urine with an Aqua Sira mass spectrometer (VG-Isogas Ltd., Middlewich, Cheshire, UK). Carbon dioxide production was calculated from the isotope ratios in baseline, initial, and final samples with the equation of Schoeller et al. (13) adapted for the measured ratio of the dilution spaces for 18O and 2H of L041 (9). The total body water (TBW) in the equation was calculated from the deuterium dilution space at the start of the observation period. Carbon dioxide production was converted to ADMR by using an energy equivalent based on the individual macronutrient composition of the diet and the use of body fat reserves (14).

Energy expenditure of daily physical activity was assessed by expressing ADMR in multiples of SMR [physical activity index (PAI) = ADMR/SMR], or by expressing the energy expended on physical activity plus diet-induced thermogenesis (DIT) as ADMR minus SMR. Because of differences in DIT due to the lower energy intake, before and at the end of treatment, energy expended on physical activity (EEPA) was also calculated as ADMR - (SMR + DIT). DIT at the start of the treatments was assumed to be 10% of ADMR (15). Because of the diet prescribed, energy intake at the end of the treatments was < 10% of ADMR. Therefore, DIT values at the end of the interventions were calculated as 10% of the actual energy intake. Because it is known that obese subjects underreport their dietary intake (16, 17) and energy intake equals ADMR minus released energy from body tissue, the actual energy intake was calculated as the average ADMR of weeks 0 and 8, minus the energy equivalent of the average daily change in body tissue as a result of the 8-wk treatment.

Body composition

Before and at the end of the study body weight was measured on a digital balance accurate to 0.1 kg (model D-7476; Sauter, Echingen, Germany). Height was measured to the nearest 0.1 cm by using a wall-mounted stadiometer. BMI was calculated from weight and height. Waist circumference was measured as the smallest circumference between the rib cage and the iliac crest, with the subject in standing position and breathing quietly. The hip circumference was measured at the level of the widest circumference between the waist and the thighs. Waist-hip ratio (WHR) was calculated as waist circumference divided by hip circumference.

Before and after the D and DE periods body composition was measured with two different techniques: isotope dilution and underwater weighing with simultaneous lung-volume measurement (helium dilution). The percentage of body fat was calculated from body density and TBW by using the method proposed by Siri (18). Deuterium dilution was used to measure TBW. Before going to bed at night during the stay in the respiration chamber the subjects each drank a diluted sample of 2H2O after emptying their bladders (baseline urine sample). The dosage calculation was based on body mass to create an 2H excess of 100 ppm. A second urine sample was collected in the morning of the next day, from the second voiding between 0600 and 1000. Deuterium was measured in urine samples with an isotope-ratio mass spectrometer (VG Aqua Sira). TBW was calculated as the measured deuterium dilution space divided by 1.04 (9). The same morning, whole-body density was determined by hydrostatic weighing with the subject in the fasted state and before the measurement of exercise-induced energy expenditure. Underwater weight was measured to the nearest 0.1 kg (Sauter). Residual lung volume was measured with a spirometer (model 2000; Volutograph, Mijnhards, Netherlands) at the moment of underwater weighing. The measurements were done in triplicate and the average value was used in the statistical analysis.

Data analysis

Data in the text, tables, and figures are given as mean ± SEM. Exercise-induced energy expenditure was calculated as the total integrated change over baseline values (areas under the curve from t = 0 to t = 45 min exercise; AUCt-0,45). Diet and exercise interactions were tested by two-way repeated-measures analysis of variance (ANOVA) with group (exercise and no exercise) and time (before and after diet) as grouping variables. Post hoc testing was performed by paired (within group) or unpaired (between group) Student's t tests. Significance levels were adjusted for multiple comparisons according to Bonferroni inequalities. Regression analysis was used to assess associations between measured variables. Analysis of covariance using fat-free mass as the covariate was used to adjust for differences in body weight when comparing metabolic rate values. P < 0.05 was accepted as statistically significant. All analyses were performed by using STATVIEW 512+ (Brainpower Inc., Calabasas, CA).

RESULTS

As shown in Table 1, no significant differences in variables between the D and DE groups were observed at the start of the study. From the heart-rate recording measurements, it appeared that the mean intensity of the training sessions was 52% (range 45-60%) of measured VO2max. Changes in body weight and body composition for the D and DE groups are shown in Table 2. Both energy restriction alone and combined with exercise treatment resulted in significant decrements of weight, fat mass, and fat-free mass (P < 0.0001, ANOVA). However, the
DE treatment resulted in a significantly increased loss of fat mass compared with the D treatment ($P < 0.05$, ANOVA), whereas total weight loss and loss of fat-free mass were not different between the two groups. The percentage of weight lost as fat mass was 77.1 ± 4.0% for the D group and 88.6 ± 5.1% ($P = 0.10$) for the DE group.

Changes in VO$_{2\text{max}}$, expressed on the basis of fat-free mass, are shown in Figure 1. For the D group the absolute VO$_{2\text{max}}$ decreased 6.2% at the end of energy restriction ($P < 0.05$, ANOVA). The DE group showed a tendency toward a small increase of 5.4% after treatment ($P = 0.051$, ANOVA). Expressed per kilogram body weight, the VO$_{2\text{max}}$ group showed a significant increase in VO$_{2\text{max}}$ of 13.8% (26.0 ± 1.5 compared with 29.5 ± 1.3 mL·min$^{-1}$·kg$^{-1}$ for weeks 0 and 8, respectively; $P < 0.0001$, ANOVA), whereas the D group showed no change (25.5 ± 1.0 compared with 25.4 ± 1.2 mL·min$^{-1}$·kg$^{-1}$ for weeks 0 and 8, respectively; NS). For both groups, Wmax did not significantly change in response to treatment (D group: 175 ± 7 compared with 167 ± 10 W for weeks 0 and 8; NS; DE group: 187 ± 5 compared with 186 ± 7 W for weeks 0 and 8; NS).

Results of the SMR and ADMR measurements are shown in Table 3. Both the D and DE groups showed decreases of 10% in SMR at the end of the 8-wk treatment ($P < 0.0001$, ANOVA). There were no significant differences in response to treatment with no significant differences between the D and DE groups (D group: 0.81 ± 0.01 compared with 0.77 ± 0.01 for weeks 0 and 8; $P = 0.06$; DE group: 0.81 ± 0.01 compared with 0.77 ± 0.01 for weeks 0 and 8; $P < 0.05$).

![Figure 1](https://via.placeholder.com/150)

**Figure 1.** Maximal oxygen consumption (VO$_{2\text{max}}$) values of subjects treated with 8 wk of diet alone (D; $n = 10$) or diet and exercise (DE; $n = 10$). Error bars = SEM; FFM, fat-free mass.
FIGURE 2. Fat and carbohydrate oxidation during 45 min of exercise [at 45% maximal power (Wmax)] for the diet (D; n = 10) and diet and exercise (DE; n = 10) groups. *Significantly different from week 0, P < 0.05.

was 3.1 MJ for both groups, consisting of 89 g carbohydrate, 62 g protein, 15 g fat, and a micronutrient intake that meets the Dutch recommended daily intake. However, estimation of the actual food intake is very difficult and obese people in particular tend to underreport their actual food intake (16, 17). The actual energy intake equals approximately the mean ADMR of weeks 0 and 8 minus energy released from the average daily change of body tissue. This method assumes linear decreases in ADMR and body tissue during the 8-wk treatment. For body tissue this assumption appeared to be reasonably justified because 55% of the weight loss occurred during the first half of the treatment. Assuming that 1 kg fat provides 38.3 MJ and 1 kg fat-free mass provides 3.5 MJ, the energy equivalent of the weight loss as a result of the treatments can be calculated. It appears that the total energy equivalent of the weight lost during the 8-wk treatment for the D group was 216.3 MJ, whereas that of the DE group amounted to 303.5 MJ. Consequently, the D group obtained on average 3.9 MJ/d, whereas the DE group obtained 5.4 MJ/d by releasing stored body energy. From this, it can be concluded that in the present study food intake is considerably underestimated by approximately 4.5 MJ/d for the D group and 8.9 MJ/d for the DE group. During the last 2 wk of the diet the average daily energy intakes for the D and DE groups were 9.6 and 12.2 MJ, respectively. Table 4 shows the reported and adjusted energy intake, ADMR, and energy balance data during the last 2 wk of both treatments. From these results it appears that the DE group showed a greater negative energy balance compared with the D group.

Physical activity level was calculated as the PAI. Measured PAIs are given in Table 5. There were no significant differences in PAI between the D and DE groups, before or after treatment. Table 5 also shows the values of ADMR = SMR and energy expended on physical activity (EE_{act}) expressed as ADMR = (SMR + DIT). Assuming DIT values of 10% of ADMR (16) before treatment, and based on an adjusted energy intake of 0.76 and 0.62 MJ/d for the D and DE groups, respectively, during treatments, it appeared that there were no

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Energy intake (EI), expenditure, and balance during last 2 wk of an 8-wk diet alone (D) or diet and exercise (DE)</th>
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</thead>
<tbody>
<tr>
<td>D</td>
<td>(n = 10)</td>
</tr>
<tr>
<td>DE</td>
<td>(n = 10)</td>
</tr>
<tr>
<td>Reported EI</td>
<td>3.1 ± 0.4</td>
</tr>
<tr>
<td>Adjusted EI</td>
<td>7.6 ± 0.7</td>
</tr>
<tr>
<td>ADMR</td>
<td>10.8 ± 0.5</td>
</tr>
<tr>
<td>Energy balance</td>
<td>−3.2 ± 0.5</td>
</tr>
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<td>−4.9 ± 0.6</td>
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</tbody>
</table>

*SEM.

2 Adjusted for changes in body weight; calculated as the average of average daily metabolic rate (ADMR) of week 0 and week 8, minus the energy equivalent of the average daily change of body tissue as a result of the 8-wk treatment.

4 Daily EI minus ADMR.

Significantly different from D, P < 0.05 (two-way ANOVA for repeated measures).

Differences between the D and DE groups before and at the end of the treatments for EE_{act}.

DISCUSSION

In the present study, free-living total daily energy expenditure was assessed before and at the end of an 8-wk, very-low-energy-diet treatment alone or combined with an exercise program by using the DLW method. Simultaneously, SMR was measured to assess the energy expenditure of daily physical activity by expressing physical activity in multiples of SMR or by expressing the energy expended on physical activity as ADMR = (SMR + DIT). Also, the energy cost of a standardized activity was measured to evaluate the energy expended for a certain activity. By assessing these components of energy balance, it is possible to determine adaptations of the different
TABLE 5
Physical activity levels of obese subjects treated with 8 wk of diet alone (D) or diet and exercise (DE)\(^1\)

<table>
<thead>
<tr>
<th></th>
<th>D ((n = 10))</th>
<th>DE ((n = 10))</th>
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</thead>
<tbody>
<tr>
<td>PAI ((\times) SMR)(^2)</td>
<td></td>
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<tr>
<td>week 0</td>
<td>1.72 ± 0.06</td>
<td>1.72 ± 0.07</td>
</tr>
<tr>
<td>week 8</td>
<td>1.74 ± 0.06</td>
<td>1.75 ± 0.10</td>
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<tr>
<td>ADMR – SMR (MJ/d)</td>
<td></td>
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</tr>
<tr>
<td>week 0</td>
<td>5.24 ± 0.37</td>
<td>4.98 ± 0.45</td>
</tr>
<tr>
<td>week 8</td>
<td>4.60 ± 0.40</td>
<td>4.50 ± 0.57</td>
</tr>
<tr>
<td>EEact (MJ/d)(^3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>week 0</td>
<td>4.01 ± 0.34</td>
<td>3.77 ± 0.41</td>
</tr>
<tr>
<td>week 8</td>
<td>3.94 ± 0.32</td>
<td>4.02 ± 0.52</td>
</tr>
<tr>
<td>EEact (kJ·d(^{-1})·kg body wt(^{-1}))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>week 0</td>
<td>45.6 ± 4.0</td>
<td>42.6 ± 4.8</td>
</tr>
<tr>
<td>week 8</td>
<td>47.5 ± 3.7</td>
<td>50.5 ± 7.1</td>
</tr>
</tbody>
</table>

\(^1\) ± SEM.
\(^2\) Physical activity index calculated as average daily metabolic rate (ADMR) divided by sleeping metabolic rate (SMR).
\(^3\) Energy expended on physical activity calculated as ADMR minus (SMR plus diet-induced thermogenesis).

components of energy balance to D or DE treatment. There are many studies in the literature that investigated basal energy expenditure (7, 20–22), physical activity (7), or DIT, and several studies have determined total free-living energy expenditure in obese subjects (23), but to our knowledge there are no published studies that used the DLW method for measurement of total energy expenditure during weight reduction as a result of D or DE treatment in obese women.

The present study showed that addition of moderate exercise to an energy-restriction treatment did not accelerate weight loss, which confirms the majority of previous studies (4, 24). Addition of exercise to the diet treatment resulted in an extra fat loss in the present study, as was shown also in several other studies that used moderate or severe energy restriction (20, 22, 25, 26). The degree of dietary restriction (27, 28) and type, intensity, duration, and frequency of exercise (7, 21, 28) may be important factors causing the inconsistent results concerning the effects of diet alone compared with diet plus exercise.

In particular, regular moderate aerobic exercise, which was used in the present study, has been shown to result in adaptations favoring fat mobilization and oxidation during exercise. Although there tended to be differences in baseline contributions of carbohydrates and fat to exercise-induced energy expenditure between the groups (\(P = 0.059\)), the relative contribution of fat was significantly increased as a result of combined diet and exercise treatment in the present study, whereas no significant change occurred as a result of diet alone. Addition of exercise to dietary treatment, therefore, seems of particular importance for treatment of obesity because macronutrient balance, which has been shown to be disturbed in the obese state (3), is positively influenced. Increased fat oxidation from training seems to be of minor importance in the resting state, because during the night no differences in RER could be observed between the D and DE treatments.

A study by van Dale et al (7) showed that free-living total energy expenditure, measured with heart-rate recordings, significantly decreased as a result of both a very-low-energy-diet and diet and exercise treatment. However, this decline was more pronounced from diet treatment alone than from diet combined with exercise. In the present study, the ADMR, as measured with the DLW technique, declined significantly by 12.2% and 9.1% in the D and DE groups, respectively, with no differences between the groups and similar changes in fat-free mass. On the basis of these results there are no indications for different adaptations in total free-living energy expenditure to a very-low-energy diet alone or diet and exercise treatment.

The ADMR of a subject involves the BMR, which has been shown to be ~5% higher than the SMR (29), the DIT, and the energy costs of physical activity. The decrease in absolute SMR because of the diet alone or because of combined diet and exercise treatment were of similar magnitude in the present study. Also, when corrected appropriately for changes in fat-free mass, it appears that there were no differences between the groups. Similar decrements for diet and diet and exercise treatment are in line with the findings of several studies that used severe or moderate energy restriction (20–22, 30), whereas other studies did report differences in the decline of RMR or SMR between a severe or moderate energy-restrictive diet alone or diet and exercise treatment (7, 25, 31). Regarding these equivocal results, the short-term stimulating effect of exercise on energy expenditure has been suggested to play a role (32), although others reported that exercise must be of a high intensity and long duration before a significant elevation in metabolic rate is observed beyond several hours (33). Discrepancies in the research of postexercise effects on energy expenditure might also be attributed in part to lack of adequate corrections for changes in body composition and type of the added exercise (34). Furthermore, Bailor (27) suggested that the dietary intake might interact with the effects of addition of exercise on RMR.

The discrepancy between reported energy intake, total energy expenditure, and changes in body energy stores observed in the present study confirms previous studies, suggesting that obese subjects underreport habitual energy intake (16, 17). However, although subjects of both groups underreported their energy intake, it appears that compliance with the prescribed diet was slightly better in the exercising subjects. It is therefore indicated that addition of exercise to very-low-energy-diet treatment promotes better dietary adherence, which is an additional benefit of exercise that may have even greater importance during treatment of obesity over the long term.

PAI and energy expended on physical activity (EE\(_{\text{act}}\)) calculated as ADMR – (SMR + DIT), were not changed by either the D or DE treatment and no differences between the two groups were observed. Furthermore, if EE\(_{\text{act}}\) data are expressed per kilogram body weight, there were no changes due to the treatment or differences between the treatments. Therefore, it may be concluded that the level of physical activity is not increased by combined diet and exercise treatment in obese women compared with diet alone. The DE group trained for 4 h/wk. It can be calculated that the energy expended during the training amounted to 5.9 MJ/wk. Assuming a mean activity level for the D group based on the ADMR divided by 24 h (0.45 MJ/h) compared with the measured 52% VO\(_{2}\max\) for the DE group, an extra energy deficit of 0.6 MJ/d from the extra physical activity can be calculated for the DE group. Because a difference < 0.6 MJ/d was detected by the DLW method in the present study, it might be indicated that the energy expended during the training is partly compensated by
a decrease in daily physical activities outside the training sessions. Based on a one-sided significance level of 0.05, a power of 90% and assuming an SD of 0.4 MJ/d on the basis of a 4% precision of the DLW method (9), a sample size of eight subjects would enable us to identify a difference of 0.5 MJ/d. Therefore, with the sample size used in the present study it should have been possible to detect a difference between the groups of 0.5 MJ/d.

A finding of compensation for added exercise seems to be not in agreement with previous studies using DLW, showing no compensatory decrease in daily physical activities after a training program alone in obese boys (6) and after endurance training in lean men and women (35). However, because of different groups of subjects, these studies appear to be not adequately comparable with obese women in the present study. No compensation in daily physical activities for addition of exercise to very-low-energy-diet treatment was also shown in obese women (7). However, measurements of total energy expenditure and physical activity were performed by heart-rate monitoring and actiometer recordings in the studies of van Dale et al (7). With these methods, estimates of energy expenditure from physical activity are not as precise as those with the DLW method, particularly because of uncertainties about the relation between heart rate and energy expenditure during sedentary activities. The present study used the DLW method during weight reduction by D or DE treatment for estimating energy expended on physical activity, and the results might indicate partial compensation in physical activity for addition of training to dietary treatment during the nonexercise part of the day.

The results also suggest that there is no decline in daily absolute physical activities due to very-low-energy-diet treatment alone, in agreement with another study in obese women that observed no significant change in dynamic activities (EE > 30% of VO2max) as measured by actiometer recordings (7). A tendency toward increased thermogenic efficiency during a standardized activity, as demonstrated in the present study, combined with lower energy costs for weight-bearing activities due to the decreased body mass might even indicate a slightly increased physical activity level after energy-restriction treatment. On the other hand, it was suggested that compensation for a food-energy-deficit occurs at the level of leisure activities in healthy men (36). Whether leisure activities decrease in obese women on a very-low-energy diet is uncertain. In contrast, it is also possible that these activities increase because of improved mobility as a result of decreased body weight.

In summary, addition of exercise to a very-low-energy diet in obese women has advantages with respect to changes in body composition, substrate utilization, and energy expenditure. In particular, loss of fat mass was favored, the contribution of fat to oxidation during exercise was increased, and a larger energy deficit was created, compared with diet treatment alone.

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