EFFECTS OF ADDITION OF EXERCISE TO ENERGY RESTRICTION ON 24-HOUR ENERGY EXPENDITURE, SLEEPING METABOLIC RATE AND DAILY PHYSICAL ACTIVITY

D. van DALE, P. F. M. SCHOFFELEN, F. ten HOOR and W. H. M. SARIS

Department of Human Biology, University of Limburg, P. O. Box 616, 6200 MD Maastricht, The Netherlands

Body composition, sleeping metabolic rate (SMR), 24-h energy expenditure (24-EE) and daily physical activity were determined in 12 obese women during and after 12 weeks of exercise (4 h per week on 55 per cent of VO2 max) and/or energy restriction (2.9–3.5 MJ/d). Diet (D) and diet-exercise (DE) groups were formed by matching the subjects on their body mass index (BMI, kg/m²; mean 30.3).

After 12 weeks no significant differences were shown in loss of weight (D 12.2 and DE 13.2 kg) and loss of fat mass (D 9.4 and DE 10.9 kg). Both groups reduced their SMR (D 29.9 per cent and DE 21.7 per cent) and their metabolic rate during the entire night measured by indirect calorimetry (12-EE) (D 36.4 per cent and DE 28.6 per cent; P < 0.05). Energy expenditure over 24 h, estimated by means of heart-rate monitoring, was reduced by 22.1 per cent for D and by 19.6 per cent for DE (n.s.). Daily physical activity, which was determined during 5 d using an actometer, was increased after 12 weeks for DE (27 per cent; P < 0.05) and D (10 per cent; n.s.).

The suggestion that a reduction in normal activities of daily life in a diet-exercise group is the explanation for the absence of significant differences in weight and fat loss between a diet-exercise and a diet group is not confirmed in this study. Daily physical activity showed a significantly higher increase for the diet-exercise group than for the diet group, while the decline of SMR and 24-EE tended to be smaller.

The combination of diet and exercise has not resulted in consistently better results in weight loss and fat loss or in prevention of the dietary-induced fall of RMR, compared to energy restriction alone (Warwick & Garrow, 1981; Schultz et al., 1980; Lennon et al., 1985; Pavlou et al., 1985; Henson et al., 1987; Hill et al., 1987). Although methodological differences between studies may play a role in the inconsistency of results (Belko et al., 1987; van Dale et al., 1987), they provide no conclusive explanation for the lack of significant differences between a diet and a diet-exercise treatment.

Another factor which might contribute to the absence of significant differences between a diet and a diet-exercise group has been suggested by Epstein & Wing (1980). Since the extent of adaptation in energy expenditure is related to the magnitude of the energy deficit, subjects in the diet-exercise group might reduce the activities of their normal daily life or their non-exercise activities in order to compensate for the greater negative energy balance due to exercise. Such a possible reduction in daily life activities might compensate completely for the energy expenditure of the extra physical training, resulting in a similar energy expenditure and activity level for the diet-exercise and
the diet group. With similar values for energy expenditure and activity level no differences in weight and fat loss can be expected.

In a previous study comparing a diet and diet-exercise treatment (van Dale et al., 1987) we found no differences between the treatments (diet or diet-exercise) in changes in body composition and sleeping metabolic rate. Using the data of the aforementioned study, the aim of the present study was to compare the changes in 24-h EE, SMR and daily physical activity during 12 weeks of diet or diet-exercise treatment.

Subjects and methods

Subjects

Twelve healthy female subjects, aged 20-45 years, were matched on the basis of their BMI (mean 30.3) and admitted to a diet (D) or a diet-exercise group (DE). Descriptive data of the subjects are shown in Table 1. They were fully informed about the nature of the investigation and gave their informed consent.

Procedure

The study period lasted 12 weeks. Measurements of body composition, SMR, 24-EE and daily activities were made before the start of the study, after 4 weeks (in week 5) and after 12 weeks (in week 13). An outline of the study design is given in Fig. 1.

### Table 1. Descriptive data of the experimental subjects.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Diet group (D)</th>
<th>Diet-exercise group (DE)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean s.e.m.</td>
<td>Mean s.e.m.</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>87.3 ± 2.0</td>
<td>81.2 ± 4.0</td>
</tr>
<tr>
<td>Height, cm</td>
<td>170.0 ± 2.1</td>
<td>163.0 ± 2.4</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>30.3 ± 1.1</td>
<td>30.3 ± 0.8</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>37.4 ± 1.5</td>
<td>39.0 ± 0.6</td>
</tr>
<tr>
<td>Body fat, kg</td>
<td>32.9 ± 1.8</td>
<td>31.7 ± 1.7</td>
</tr>
</tbody>
</table>

D vs DE: *P < 0.05

Group: 12 females

<table>
<thead>
<tr>
<th>Study period (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-1 0 1 2 3 4 5 6 7 8 9 10 11 12 13</td>
</tr>
<tr>
<td>DIET (N=6)</td>
</tr>
<tr>
<td>2.9 MJ 3.5 MJ</td>
</tr>
<tr>
<td>DIET + EXERCISE (N=6)</td>
</tr>
<tr>
<td>Measurements</td>
</tr>
<tr>
<td>12-EEₘₑₛ,SMR</td>
</tr>
<tr>
<td>24-EEₘₑₛ</td>
</tr>
<tr>
<td>Densitometry</td>
</tr>
<tr>
<td>VO₂max</td>
</tr>
<tr>
<td>Daily physical activity</td>
</tr>
</tbody>
</table>

Fig. 1. Design of the study.
Table 2. Energy intakes of the subjects during the formula diet and the mixed diet.

<table>
<thead>
<tr>
<th>Subjects No.</th>
<th>Formula diet (MJ/d)</th>
<th>Mixed diet (MJ/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>3.0</td>
<td>3.5</td>
</tr>
<tr>
<td>7</td>
<td>3.5</td>
<td>4.2</td>
</tr>
<tr>
<td>9</td>
<td>2.8</td>
<td>3.3</td>
</tr>
<tr>
<td>12</td>
<td>2.6</td>
<td>2.6</td>
</tr>
<tr>
<td>13</td>
<td>2.5</td>
<td>3.3</td>
</tr>
<tr>
<td>14</td>
<td>2.6</td>
<td>2.8</td>
</tr>
<tr>
<td>mean</td>
<td>2.8</td>
<td>3.3</td>
</tr>
<tr>
<td>s.e.m.</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Diet-exercise group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>2.8</td>
<td>2.9</td>
</tr>
<tr>
<td>2</td>
<td>3.3</td>
<td>3.8</td>
</tr>
<tr>
<td>4</td>
<td>2.7</td>
<td>3.6</td>
</tr>
<tr>
<td>3</td>
<td>2.8</td>
<td>3.7</td>
</tr>
<tr>
<td>6</td>
<td>3.2</td>
<td>3.5</td>
</tr>
<tr>
<td>15</td>
<td>2.6</td>
<td>4.3</td>
</tr>
<tr>
<td>mean</td>
<td>2.9</td>
<td>3.6</td>
</tr>
<tr>
<td>s.e.m.</td>
<td>0.1</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Formula diet: Meritene, Wander, Switzerland. Mixed diet consisted of 1.7 MJ formula diet, which was supplemented with normal food.

underwater weighing the residual lung volume was measured using the helium dilution method. The average of six determinations was used as the true body weight under water. The percentage of body fat was calculated from body density using the equations of Siri (1956).

Sleeping metabolic rate (SMR)
For the measurement of SMR each subject stayed for 12 h (20.00–8.00) 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 chambers was measured by a dry gas meter (Dort, The Netherlands) and continuously analysed by a paramagnetic O₂-analysers (Servomex Taylor, England) and an infrared CO₂-analysers (Hartmann and Braun, West Germany). From the air flow rate, and the O₂- and CO₂-concentration of the in- and out-flowing air VO₂ and VCO₂ were computed on-line through an automatic acquisition system interfaced with an Apple 2E computer (Schofleien et al., 1985). SMR was calculated during the period of sleeping from 3.00 to 6.00 a.m.; the metabolic rate during the 12-h stay in the respiration chamber was also determined (12-EFrest). SMR has been reported to be 5 per cent lower than RMR (Goldberg et al., 1988).

Maximal aerobic power
Maximal aerobic power was measured using an incremental exercise test on an electrically braked bicycle ergometer (Lode, The Netherlands). After an initial period of 4 min of cycling of 0 W, 4 min at 40 W and 5 min at 80 W, the workload was increased by 20 W/min until the subjects were exhausted. Criteria for maximal exercise were: levelling off of oxygen uptake, forced ventilation, and a R value higher than 1.1. The highest VO₂ achieved on the test was taken as VO₂max. During the test, ventilatory and gas exchange responses were measured continuously by a computerized open system (Ergoacreer, Switzerland).

Exercise
The training was performed 4 d per week at a professional fitness centre and consisted of 2 h aerobic training and 2 h of fitness training per week. The fitness trainings also included 20 min of aerobic training. On the basis of heart-rate measurements the intensity of the training was calculated to be 55 per cent of the VO₂max. All trainings were under the guidance of a professional trainer and attendance of the subjects was monitored.

Body composition
Body composition was estimated by means of hydrostatic weighing. At the moment of
Energy expenditure over 24 h (24-EE)
For the estimation of 24-EE, the heart rate was recorded continuously for 24 h using a heart-rate memory system (24-EEHm) (Depex, Biltonaven). Heart rate (HR) was measured 1 d (24 h) before the study, after 4 weeks (in the beginning of week 5), and after 12 weeks (week 13), when the subjects were still exercising and/or dieting. The recordings took place on a day without physical training, because of practical problems like showering after exercise. The heart-rate memory (HRM) system was worn in a side pocket attached to a belt around the waist. Two electrodes were attached to the thorax, one at the manubrium sterni and the other at the iliacus. Heart rate was recorded and stored in the memory system. Using a read-out unit, the mean heart rate and actual time spent in the various heart-rate registers were read and analysed by computer. Detailed description of the HRM system apparatus is given by Saris (1982). Using heart rate as an indicator of physical activity, it is necessary to establish individual calibration curves because of interindividual differences between heart rate and oxygen uptake. Various standard activities were used for calibration: lying, sitting, and walking on a treadmill (3 km/h, 4.5 km/h and 4.5 km/h with a 10 per cent slope). Furthermore the bicycle ergometer was used with three workloads: 4 min with no workload, 4 min at 40 W and 4 min at 80 W. Measurements were carried out after 5–10 min rest. During the calibration measurements, respiratory gases were collected in Douglas bags. Gas volumes were measured with a dry gas meter (Dort, The Netherlands). Gas samples were analysed for O2 and CO2 content with a computerized open system (Ergoscreen, Switzerland). The ergoscreen was calibrated before each test using room air and calibration gas. Heart rate was continuously recorded during the standard activities by a conventional electrocardiogram apparatus. In addition to these calibration points, mean oxygen consumption and heart rate during the sleeping period from 3:00 to 6:00 a.m. were used.

Since the linear relationship between heart rate and exercise is less clear in the lower ranges than in the higher ranges, two regression equations were calculated: one for the activities lying, sitting and standing and one for the activities walking and cycling. It was necessary to determine a critical heart rate, above which the regression equation of dynamic activities such as walking and cycling was used to calculate VO2, and below which the regression equation for the resting activi-

![Graph of VO2 vs. Heart Rate](image)

Fig. 2. Calibration curves of one subject assessed at the start of the study, after 4 weeks and after 12 weeks of diet-exercise treatment. Two calibration curves were calculated: one during resting activities such as lying, sitting, and standing, and one during dynamic activities such as walking and cycling. • resting activities; • dynamic activities.
ties was used for that particular minute. The cut-off point was determined by calculating the average value between the highest heart rate at rest and the lowest heart rate during cycling or walking.

For each individual, heart rate and oxygen uptake during calibration activities were plotted, and two linear regression equations: \( \text{VO}_2 = a + b \text{HR} \) were calculated. An example is given in Fig. 2. It was then possible to substitute the mean heart rate over 24 h in the regression line and to calculate energy expenditure. The amount of energy expended per min was calculated from the formula proposed by Consolazio (1971), with a conversion factor of 4.92, as used by Passmore, Thomson & Warnock (1952). To calculate energy expenditure for the dynamic activities (walking and cycling) over 24 h, the 30 per cent level of \( \text{VO}_2 \text{max} \) was taken as a minimal level. The heart-rate recordings above this level were used in the regression equation to calculate the active 24-EE.

For the measurement of daily physical activity over a longer period a movement counter was used (actometer). The actometer is a self-winding wrist-watch from which the escape mechanism has been removed, so that the rotor is directly connected to the hands of the watch. The results can be read in days and hours (actometer units). The instrument records acceleration and deceleration with a component in the same plane as the face of the watch. In effect, it records not only the movement but also the intensity. The rotor turns more often as the intensity of the movement increases. A detailed description of this apparatus is given by Saris & Binkhorst (1977a). The actometer was worn on the left ankle for 5 consecutive days, including 2 days of training, and during a weekend.

Statistical results are expressed as means \( \pm \) s.e.m. Statistical differences between the diet and the diet-exercise groups were assessed using Student’s t-test for unpaired means. Within-group comparisons were calculated by Student’s t-test for paired means.

Results

As shown in Table 3, no significant differences in weight loss, fat loss or loss of fat-free mass between the diet and the diet-exercise group were observed. Similarly, exercise did not have a preventive effect on the decrease in SMR due to energy restriction, although the diet-exercise group tended to have a smaller decline in SMR after 12 weeks. An

<table>
<thead>
<tr>
<th>Week 0</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss (kg)</td>
<td>Fat loss (kg)</td>
<td>SMR (kJ/kgFFM/h)</td>
<td>( \text{VO}_2 \text{max} ) (ml/kgBW/min)</td>
</tr>
<tr>
<td>D</td>
<td>5.6 ± 0.2</td>
<td>28.3 ± 3.6</td>
<td></td>
</tr>
<tr>
<td>DE</td>
<td>5.6 ± 0.2</td>
<td>31.3 ± 1.9</td>
<td></td>
</tr>
<tr>
<td>After 4 weeks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>7.9 ± 0.9</td>
<td>6.0 ± 0.8</td>
<td>4.5 ± 0.4**</td>
</tr>
<tr>
<td>DE</td>
<td>8.2 ± 0.7</td>
<td>6.7 ± 0.6</td>
<td>4.9 ± 0.3</td>
</tr>
<tr>
<td>After 12 weeks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>12.2 ± 1.9</td>
<td>9.4 ± 1.4</td>
<td>4.1 ± 0.3**</td>
</tr>
<tr>
<td>DE</td>
<td>13.2 ± 0.6</td>
<td>10.9 ± 0.6</td>
<td>4.3 ± 2.0**</td>
</tr>
</tbody>
</table>

Significance of difference within D or DE at start: *\( P < 0.05 \); **\( P < 0.01 \); between D and DE \( P < 0.01 \).
Table 4. Energy expenditure over 24 h, determined by heart-rate monitoring (24-EE_{hr}), energy expenditure, determined during 12 h in the respiration chamber (12-EE_{resp}) and sleeping metabolic rate (SMR) at week 0, after 4 weeks and after 12 weeks for the diet group (D) and the diet-exercise group DE (mean ± s.e.m.).

<table>
<thead>
<tr>
<th></th>
<th>Week 0</th>
<th>4 weeks</th>
<th>12 weeks</th>
<th>% change 0-4 weeks</th>
<th>% change 0-12 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>24-EE_{hr} D</td>
<td>11.2 ± 0.5</td>
<td>9.0 ± 0.7</td>
<td>8.5 ± 1.0</td>
<td>19.2 ± 4.2**†</td>
<td>22.1 ± 6.3**</td>
</tr>
<tr>
<td>(MJ)</td>
<td>DE</td>
<td>10.8 ± 0.3</td>
<td>9.1 ± 0.7</td>
<td>9.0 ± 0.6</td>
<td>16.6 ± 6.6*</td>
</tr>
<tr>
<td>24-EE_{hr} D</td>
<td>205 ± 12.9</td>
<td>171 ± 9.5</td>
<td>164 ± 13.2</td>
<td>16.3 ± 4.2**</td>
<td>20.3 ± 5.3**</td>
</tr>
<tr>
<td>(kJ/kgFFM) DE</td>
<td>227 ± 8.3</td>
<td>191 ± 7.1</td>
<td>190 ± 4.7</td>
<td>13.3 ± 7.8*</td>
<td>17.3 ± 7.1**</td>
</tr>
<tr>
<td>12-EE_{resp} D</td>
<td>4.6 ± 0.2</td>
<td>3.2 ± 0.3</td>
<td>2.9 ± 0.3</td>
<td>28.6 ± 3.8**</td>
<td>36.4 ± 5.6**</td>
</tr>
<tr>
<td>(MJ)</td>
<td>DE</td>
<td>4.1 ± 0.3</td>
<td>3.4 ± 0.3</td>
<td>3.1 ± 0.2</td>
<td>15.1 ± 6.8*</td>
</tr>
<tr>
<td>12-EE_{resp} D</td>
<td>84.5 ± 2.5</td>
<td>61 ± 4.3</td>
<td>56.5 ± 5.3</td>
<td>28.2 ± 4.4**</td>
<td>33.1 ± 5.7**</td>
</tr>
<tr>
<td>(kJ/kgFFM) DE</td>
<td>83.5 ± 4.2</td>
<td>72 ± 3.9</td>
<td>65.5 ± 2.9</td>
<td>12.1 ± 6.2*</td>
<td>20.4 ± 5.6**</td>
</tr>
<tr>
<td>SMR (24 h) D</td>
<td>7.3 ± 0.3</td>
<td>5.5 ± 0.5</td>
<td>5.2 ± 0.6</td>
<td>24.1 ± 4.8*</td>
<td>29.9 ± 6.4**</td>
</tr>
<tr>
<td>(MJ)</td>
<td>DE</td>
<td>6.7 ± 0.4</td>
<td>5.4 ± 0.5</td>
<td>5.2 ± 0.3</td>
<td>13.9 ± 6.8*</td>
</tr>
</tbody>
</table>

*P < 0.05, **P < 0.01 paired observations with respect to week 0. †P < 0.05 D vs DE.

important and determining factor in the appearance of differences in weight and fat loss between diet and diet-exercise groups is the intensity or frequency of the exercise.

In order to evaluate the influence of frequency and intensity of the exercise on aerobic capacity, VO_{2}max was measured. After 4 weeks the diet-exercise group showed a significantly improved aerobic capacity (P < 0.05), whereas the diet group showed a tendency to decrease its aerobic capacity. After 12 weeks the diet group had adapted to energy restriction and returned to baseline levels, whereas the diet-exercise group remained on the improved aerobic capacity level attained in the fifth week.

The total daily energy expenditure, based on the 24-h heart-rate recordings (24-EE_{hr}), varied considerably between the subjects (range 7.0–14.6 MJ/d). At the start, 24-EE_{hr} was the same for the diet and the diet-exercise groups (11.2 and 10.8 MJ/d) (Table 4). Similarly, no significant differences were observed in the changes in 24-EE_{hr} after a period of exercise and/or dieting. Energy expenditure over 24 h in the diet group decreased by 19.2 and 22.1 per cent after 4 and 12 weeks respectively, while that in the diet-exercise group decreased by 16.6 and 19.6 per cent respectively. Standardized for FFM, 24-EE_{hr} was still reduced for both groups and there were no significant differences between the diet and the diet-exercise groups.

Energy expenditure during the entire night (12-EE_{resp}), measured during the 12-h stay (20.00–0.00) in the respiration chamber, showed a large decrease for the diet group (29 and 36 per cent after 4 and 12 weeks). For the diet-exercise group the decline of metabolic rate during the night was significantly smaller after 4 and 12 weeks (15 and 22 per cent; P < 0.05).

Similar decreases were found for SMR, which dropped by 24 and 30 per cent after 4 and 12 weeks for the diet group, whereas the diet-exercise group tended to show a smaller decline (14 and 22 per cent after 4 and 12 weeks respectively).

In Fig. 3 the energy expenditure above the 30 per cent VO_{2}max level is shown as a measure for the dynamic activities such as walking and cycling. Figure 3 shows that at the start of the study the groups were not significantly different in their dynamic activities. After 4 weeks on a restricted diet the diet group had not changed, whereas the diet-exercise group tended to increase their activities. The increase was not significant, probably because of the large variation between the individuals. At the end of the study a slight increase was also observed for the diet group.
Fig. 3. Changes in daily physical activity, defined as
the energy expenditure spent above the relative level of
30 per cent of the individual's maximal aerobic power
at week 0, after 4 weeks and 12 weeks (mean ±
s.e.m.). ■ diet group; ● diet-exercise group.

The actometer was used to measure
daily physical activities over a longer
period, including days of physical training.
Adding exercise made the results of the
actometer show a significant increase of 32
per cent for DE after 4 weeks (P < 0.05)
(Fig. 4). After 12 weeks the activities still
showed an increase or remained increased
with 27 per cent (P < 0.05). For D no
significant increase in activities was
observed after 4 and 12 weeks (increases of
7.5 and 10 per cent respectively).

Fig. 4. Changes in daily physical activity measured
with the actometer during 5 consecutive days, including
exercise days, and during a weekend, at week 0, after 4
and after 12 weeks (mean ± s.e.m.). ■ diet group;
● diet-exercise group.
* P < 0.05 vs Week 0.

Discussion
In the present study total daily energy
expenditure during an exercise and/or
weight reduction treatment was assessed
by a combination of the heart-rate method
and actometer measurements, together
with the determination of energy expendi-
ture by indirect calorimetry. Recently, the
combination of heart-rate monitoring and
actometer measurements has been shown
to yield a satisfactory estimation of energy
expenditure in free-living conditions
(Avons et al., 1988). Also the heart-rate
method, which had been validated against
indirect calorimetry, has been reported to
estimate closely total daily energy expendi-
ture (Spurr et al., 1988). However, both
the heart-rate method and the actometer
have their drawbacks (Christensen et al.,
1983; Geissler, Dzumbira & Noor, 1986).
Many psychological and physiological
factors may affect heart rate, especially
at low levels of activity, without appreciably
affecting EE. This may result in an
overestimation of 3 to 26 per cent (Daun-
cey & James, 1979). However, at higher
oxygen consumption levels the prediction
from heart-rate recordings is more accu-
rate and less affected by non-physical
constraints like emotional stress. There-
fore, in the present study the change in
physical activity was analysed separately
to adjust for possible psychological influ-
ces on heart rate. Of special interest
were activities like walking, running or
cycling. These so-called dynamic activities
have an expenditure level above 30 per
cent of the individual VO_{2max} (Astrand
& Rodahl, 1977). In the present study
physical activity was defined as the energy
expenditure above the 30 per cent level of
the individual VO_{2max}.

The actometer enables us to make
measurements for several days, which
reflect more accurately the actual activity
level than only one day of measurements.
The actometer recorded acceleration of the
movements of the leg. It has been
suggested (Saris & Binkhorst, 1977b) that
differences in physical activity are mainly
due to the differences in the levels of
activity in the lower extremities, i.e.,
walking and running. Small changes in the
activity level in the upper part of the body
are less accurately measured. Changes in
the way someone sits, from active to
passive, which has been suggested as a
possible adaptation to energy restriction (Kurzer, 1987), may not be measured precisely. Furthermore, a large intra-
instrumental variation is found (Saris & Binkhorst, 1977a, b). To adjust for this variation, each subject consistently wore the same actometer and only the changes in actometer units were used for analysis.

With the methods used in this study, no indications of a compensation for the extra physical training in normal daily life activities were detected. Energy expenditure over 24-h, measured with the heart-
rate method (24-EEHr), showed decreases after 4 and 12 weeks of exercise and/or diet. No significant changes were detected between the D and DE groups, although the DE group tended to maintain their level of EE after 4 weeks, whereas for the D group a further decrease was observed (n.s.). Other studies using similar energy intakes and showing similar weight losses demonstrated the same decreases in 24-h EE (Bessard, Schutz & Jéquier, 1983; Ravussin et al., 1983; de Boer et al., 1986), while smaller energy deficits, resulting in less weight loss did not have an effect on 24-EE (Warnold, Carlgren & Krotkiewski, 1978). Furthermore, the decline of 24-EEH was found to be smaller than the decline of 12-EEcomp. This can be explained by the determination of 12-EEcomp being for the most part based on measurements during the night, when the lowest values of EE(SMR) are observed (Goldberg et al., 1988). Moreover, as a result of their weight loss, subjects are probably more active, because of their improved mobility. Although 24-h energy expenditure decreased as a result of the decrease in SMR, the increase in activities could have contributed to a smaller decline in 24-EE than in 12-EE or SMR.

Like the total energy expenditure, SMR also decreased as a result of body weight loss and fat loss. After standardization for differences in fat-free mass both SMR and 24-EE still remained decreased compared to baseline values. This finding is in contrast with the results of Ravussin et al. (1985), who found no decrease in RMR after adjusting for differences in fat-free mass; the loss of fat-free mass completely accounted for the decline in RMR. Our results were in accordance with the results of de Boer et al. (1986), Hill et al. (1987) and de Groot (1988), who also observed a greater decrease in RMR and 24-EE than could be explained by the loss of fat-free mass. Adaptations in hormones and the sympathetic nervous system might play an important role in the decrease of RMR during dieting and exercise (Jung, Shetty & James, 1980).

Recently it has been demonstrated that Na-K-ATPase activity was decreased during dieting. Since this activity is controlled by the sympathetic nervous system, it can be concluded that during dieting sympathetic nervous system activity is also decreased (Pasquali et al., 1988). Adding exercise to dieting did not result in a prevention of the decrease in SMR for DE, although smaller decreases were observed for DE than for diet alone. Up to now only a few investigators (Domahs et al., 1984; Lennon et al., 1985) have confirmed the study of Schultz et al. (1980), which reported a significant increase in RMR after the addition of exercise to energy restriction. Other studies did not demonstrate preventive effects of exercise (Krotkiewski et al., 1981; Henson et al., 1987; Hill et al., 1987). The degree of energy restriction might play an important role, leading to a less favourable effect of exercise on RMR and SMR when food intake is severely restricted.

With respect to differences in 24-EE and weight loss, one important remark has to be made. In this study selection of the subjects for both groups was based on BMI. At the start of the study it turned out that, although the percentage of fat was the same for both groups, the diet group weighed 6.1 kg more. This higher body weight was reflected in a higher energy expenditure at the start. Since the same diet was provided for both groups during the study, the energy deficit, calculated from energy intake and 24-h energy expenditure, was larger in the diet group than in the diet-exercise group (total
energy deficit over 12 weeks for D: 509.6 MJ; for DE: 502.6 MJ). On the other hand, the diet-exercise group was involved in a training programme for 4 h per/week. Assuming that the activity level in the diet group was about 15 per cent VO2max compared to the measured 55 per cent VO2max in the diet-exercise group during the exercise session, an extra energy deficit of about 4 MJ per week, or 48 MJ due to the extra physical activity over the entire 12 weeks, could be calculated. In addition, the body composition changes in the two groups and an estimated energy value for adipose tissue and fat-free mass of 32 MJ and 4 MJ respectively, allows the calculation of an extra energy release of about 45 MJ in the DE group over the entire period. This is in accordance with the theoretical calculations.

When body weight was about 6 kg more for the diet-exercise group energy expenditure might have been higher (1 kg weight gain raises RMR 50 kJ/d (Ravussin et al., 1982), which is an increase of 300 kJ/d for 6 kg). In 12 weeks this 300 kJ/d extra could result in 21600 kJ extra energy expenditure for the diet-exercise, assuming that the adaptation to the diet is the same for both groups. This 21600 kJ is equivalent to about 0.73 kg adipose tissue. The difference between the groups could have been 0.73 kg in favour of the diet-exercise group. Moreover, the purpose of this study was to find out if there was compensation in activities for the diet-exercise group. The 6 kg lower body weight was not crucial for the possible compensation in activities. This compensation due to the extra energy expenditure during training could appear even though their body weight was 6 kg lower. A theoretical compensation in daily activities leading to a reduction of 5 per cent yields a surplus of energy of 42 MJ. In such a situation no difference in body weight loss is to be expected between the D and DE groups. Since there was an observed difference of about 40 MJ, based on the calculation of body composition, one can deduce from these theoretical considerations, together with the data obtained, that there is no compensation in daily activity for the diet-exercise group.

It may be objected that the measurements of daily physical activities on the basis of heart-rate recordings were done on a non-exercise day. This excluded the direct measurement of the increase in energy expenditure due to the increase in exercise and a possible compensation in the period directly after the training. It was for that reason that the actometer was used to measure decrease in activity immediately after exercise. Similar values in actometer units for the diet and diet-exercise group were expected, in case of a decrease in activities. At the start the values were indeed similar, but during a period of training the values for DE increased significantly, while those for D increased only slightly. Should compensation only occur in quiet activities like sitting, then our conclusion that there is no compensation becomes doubtful, because the actometer cannot measure such changes (eg, in the way of sitting). On the other hand, if that is the case such decreases will not lead to important changes in total EE and daily physical activity. After all, the decrease in sitting activities cannot counterbalance the increase in activity due to the added exercise. Changes in activities like walking and cycling between the training sessions are indeed measured by the actometer and if these activities are carried out at a lower rate after training, the increase as a result of training should be counterbalanced by the decrease in these activities. However, there was a considerable increase in the diet-exercise group, which contradicts this possibility.

In summary, the unexpectedly similar weight and fat losses in the diet and diet-exercise groups cannot be attributed to reduced levels of physical activity of DE outside the exercise sessions. Although there was a decrease in 24-EE, this decrease was found in both groups and was due to the decline in SMR and reduced weight-bearing activities. Exercise training increased daily physical activity for the diet-exercise group.
References


