

Effect of an 18-wk weight-training program on energy expenditure and physical activity

LUDO M. L. A. VAN ETTEN, KLAAS R. WESTERTERP,
FRANS T. J. VERSTAPPEN, BART J. B. BOON, AND WIM H. M. SARIS
*Department of Human Biology and Department of Movement Sciences,
University of Limburg, 6200 MD Maastricht, The Netherlands*

Van Etten, Ludo M. L. A., Klaas R. Westerterp, Frans T. J. Verstappen, Bart J. B. Boon, and Wim H. M. Saris. Effect of an 18-wk weight-training program on energy expenditure and physical activity. *J. Appl. Physiol.* 82(1): 298–304, 1997.—The purpose of this study was to examine the effect of an 18-wk weight-training program on average daily metabolic rate (ADMR). Before the intervention and in weeks 8 and 18 (T_0 , T_8 , and T_{18} , respectively) data on body composition, sleeping metabolic rate (SMR), food intake, energy cost of the weight-training program (EE_{ex}), and nontraining physical activity (accelerometer) were collected in the exercise group (EXER, $n = 18$ males). ADMR was determined in a subgroup (EX12, $n = 12$) by using doubly labeled water. At T_0 and T_{18} , data (except ADMR) were also collected in a control group (Con, $n = 8$). Body mass did not change in EXER or Con. Fat-free mass increased only in EXER with 2.1 ± 1.2 kg, whereas fat mass decreased in EXER as well as Con (2.0 ± 1.8 and 1.4 ± 1.0 kg, respectively). Initial ADMR (12.4 ± 1.2 MJ/day) increased at T_8 (13.5 ± 1.3 MJ/day, $P < 0.001$) with no further increase at T_{18} (13.5 ± 1.9 MJ/day). SMR did not change in EXER (4.8 ± 0.5 , 4.9 ± 0.5 , 4.8 ± 0.5 kJ/min) or Con (4.7 ± 0.4 , 4.8 ± 0.4 kJ/min). Energy intake did not change in EXER (10.1 ± 1.8 , 9.7 ± 1.8 , 9.2 ± 1.9 MJ/day) or Con (10.2 ± 2.6 , 9.4 ± 1.8 , 10.1 ± 1.5 MJ/day) and was systematically underreported in EX12 (-21 ± 14 , -28 ± 18 , $-34 \pm 14\%$, $P < 0.001$). EE_{ex} (0.47 ± 0.20 , 0.50 ± 0.18 MJ/day) could only explain 40% of the increase in ADMR. Nontraining physical activity did not change in both groups. In conclusion, although of modest energy cost, weight-training induces a significant increase in ADMR.

doubly labeled water; accelerometer; sleeping metabolic rate; food intake; physical exercise

EVIDENCE FOR AN IMPORTANT ROLE of increased physical activity in the quality of life and the primary prevention of coronary heart disease and cancer has grown in recent years (1a, 2, 7). For the majority of the people having a sedentary job, the recommended raise in general physical activity can be achieved by exercise, sport, recreation, and life-style activities. Depending on the field of interest and available time, a choice can be made from an extensive selection of different sports activities. Weight training is an example of an individual sport that is not restricted by time of the day or weather conditions, and most forms of weight training are reported to be safe, even for hypertensive and cardiac patients and the elderly (15, 16). With the introduction of the easy-to-use weight stack machines and electronic ergometers applied during warming-up and cooling-down exercises (treadmills, bicycles, and rower and step machines), the popularity of weight training has increased. Research on the effect of weight

training on health and fitness determinants revealed that weight training, like other types of exercise, positively affects physical performance and body composition and a number of health parameters (21, 23, 27, 30). Almost every study revealed an increase in muscular strength, whereas the effect on aerobic power is inconsistent and dependent on the type of weight training [e.g., circuit vs. heavy resistance training (28), high vs. low volume weight training (27)]. Compared with running and cycling, the weight-training-induced changes in body composition consist of a larger increase in fat-free mass, whereas the decrease in fat mass seems to be somewhat smaller. The modest effect on fat mass might be attributed to the lower energy costs of a single weight-training workout (17). The latter finding seems to make this kind of exercise less effective in programs of weight control and weight reduction (17). However, previous studies suggested that the effect of physical exercise on average daily metabolic rate (ADMR) exceeds the energy cost of the training work itself. This finding initiated research on the effect of exercise on other components of ADMR like the thermic effect of feeding (22) and sleeping/resting metabolic rate (33) or on excess postexercise energy expenditure (20). Studies that measured ADMR confirmed that the energy demand of the added physical exercise explains only partly the increase in ADMR (5, 11, 35). A validated method to measure ADMR in free living subjects is the doubly labeled water method ($^2H_2^{18}O$). Due to the high cost, only a few studies used this technique. Until now, no study investigated the effect of weight training on ADMR.

The purpose of this study was to investigate the effect of an 18-wk weight-training program on ADMR and the components sleeping metabolic rate and the energy cost of physical activity (nontraining and exercise activity) to quantify their contribution to an ensuing change in ADMR. A 3-day food record was used to estimate energy intake and food composition. A triaxial accelerometer was used to register nontraining physical activity.

METHODS

The overall design of the study is presented in Fig. 1.

Subjects. Twenty-six healthy sedentary men were selected to participate in the study. None of the subjects had engaged in a regular exercise program for at least 2 yr before the study. Detailed information concerning the purpose and all methods used in the study was provided, and written consent was obtained. Eighteen subjects participated in the exercise group (EXER), and eight subjects served as inactive controls (Con).

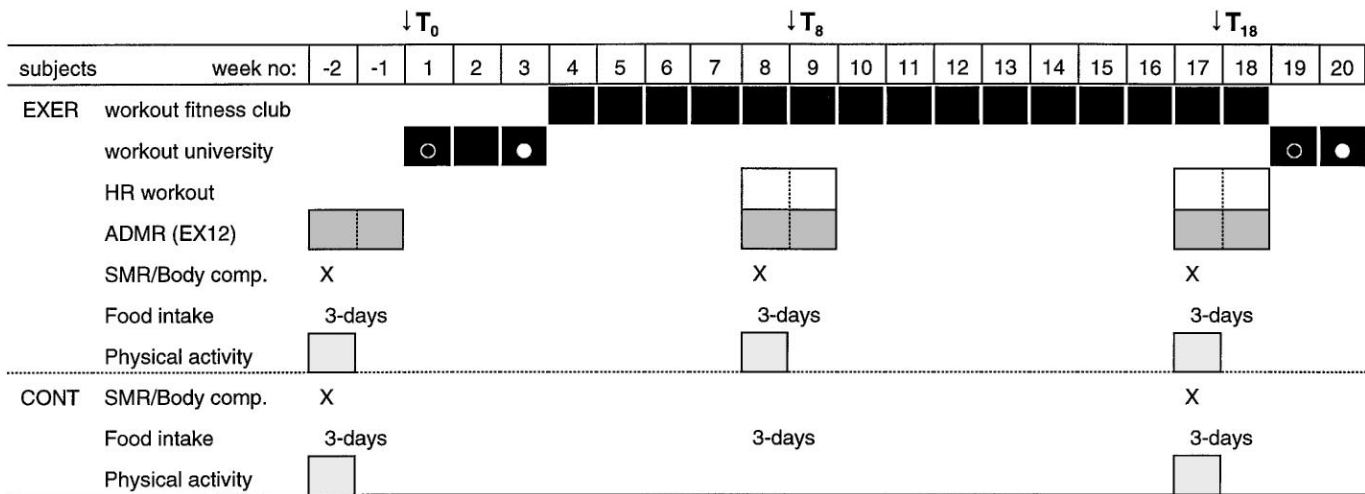


Fig. 1. Overall design. Actual training program consisted of 18 wk of weight training (■). The first 3 wk were performed at the university to determine the relationship between energy expenditure and heart rate (HR) in week 3 (white solid circle). Weeks 19 and 20 were not considered as part of the training program but were used to check whether the energy expenditure-HR relationship was changed. During weeks 1 and 19, an incremental cycling test was performed (white open circle). ADMR, average daily metabolic rate; EX 12, subgroup of 12 subjects; comp, composition; SMR, sleeping metabolic rate; EXER, exercise group; CONT, control group.

Average daily metabolic rate (ADMR). ADMR was measured over a period of 2 wk, using the doubly labeled water ($^2\text{H}_2^{18}\text{O}$) method according to the Maastricht protocol (37). Due to the high cost of this technique, ADMR was only measured in 12 subjects (EX12) randomly selected from the exercise group. The ADMR measurement was blinded by giving all subjects an isotope drink; the selected 12 subjects received $^2\text{H}_2^{18}\text{O}$, and the remaining subjects received $^2\text{H}_2\text{O}$ for body composition measurements only (see *Physical Characteristics*). The sampling medium for measuring the isotope enrichment and disappearance was urine. After a background urine sample was collected, the isotope dose was administered as the last consumption before the night (~2230). Urine samples from the second (0800–1000) and last (2100–2300) voiding of the day were collected at days 1, 8, and 14 (in all, 1 background + 6 enriched samples). Samples were analyzed with isotope ratio mass spectrometry (Aqua Sira, VG).

Sleeping metabolic rate (SMR). SMR was measured during an overnight stay in a respiration chamber (1800–0800). Dinner was eaten before subjects entered the chamber, and subjects were not allowed to eat again until body composition was determined the next morning. To minimize the residual effects of training on energy expenditure, subjects were asked to refrain from any exercise or strenuous activity on the day before the measurement. Hence, SMR measurement was always >8 h after the last meal and >30 h after the last training session. More technical information on the respiration chamber is described elsewhere (32). SMR was defined as the lowest mean energy expenditure over a shiftable 3-h interval between 0000 and 0600.

Physical characteristics. Physical characteristics are shown in Table 1. Anthropometric measurements were taken the morning after the controlled stay at the laboratory. After subjects had voided, body mass was measured to an accuracy of 5 g on an electronic scale (Mettler, E1200). Body volume was determined by using the hydrostatic weighing technique. Residual lung volume was simultaneously measured by using the helium dilution technique (Volugraph 200, Mijnhart). Total body water (TBW) was measured using deuterium ($^2\text{H}_2\text{O}$) dilution. Body composition was assessed by using the three-component model of Siri that combines body density with TBW (13).

Nontraining physical activity. Physical activity over a 7-day period was registered by using a triaxial accelerometer consisting of three uniaxial piezoresistive accelerometers (9). The accelerometer was attached to the low back with the use of an elastic belt. The accelerometer was connected to a portable data unit for on-line processing and storage of acceleration data. The data logger was programmed to calculate the sum of the integrated acceleration curves from all three directions. The time period for integration was set at 1 min, and physical activity was expressed as counts per minute. Subjects were instructed to wear the accelerometer during waking hours, except during bathing and workouts at the fitness club. Furthermore, subjects were asked to record the periods they traveled by vehicles for periods >15 min. These periods were excluded from analysis. Furthermore, all data with a value >8,000 counts/min were excluded because they were assumed not to be produced by human movement (8).

Food intake. To estimate food intake and food composition, a 3-day food record (including 1 weekend day) was used. After completion, the food record was checked with the subject to clarify the records and eliminate inconsistencies.

Training program. The subjects trained two times a week on nonconsecutive days for 18 wk. The first 3 wk, the training sessions were performed at the university (see *Energy cost of weight training*). The workouts at the fitness club consisted of 10 exercises using weight stack machines (Sportesse) and free weights. Three sets of 15 repetitions were performed on

Table 1. Characteristics for exercise group and control group

	Exercise		Control	
Age, yr	33 ± 6	23–41	35 ± 6	24–41
Height, m	1.82 ± 0.07	1.71–1.97	1.82 ± 0.05	1.75–1.88
Body mass, kg	78.8 ± 9.7	64.4–103.1	78.0 ± 10.8	56.1–88.8
Fat mass, kg	19.1 ± 4.7	13.7–31.2	17.7 ± 6.1	6.4–26.1
Fat-free mass, kg	59.7 ± 6.6	49.2–71.9	60.3 ± 7.2	49.7–69.9
Fat, %	24 ± 4	17–30	22 ± 6	11–29

Values are means ± SD or ranges; exercise group, $n = 18$; control group, $n = 8$. Analysis revealed no differences between groups.

the following exercises: bench press, flies, squat, leg curl, leg extension, seated rowing, lat pull down, dumbbell curl, triceps push down, and sit ups. Additionally, each workout included a 10-min warming up and cooling down of cycling at a self-selected intensity. All workouts were supervised by a fitness instructor.

Energy cost of weight training. Energy expenditure during the workouts at the fitness center (EE_{ex}) was predicted from exercise heart rate (HR). Because the relationship between energy expenditure and heart rate (EE/HR relationship) is different for weight-training exercises and leg cycling, two separate equations were used.

To determine the EE/HR relationship during weight training, the first 3 wk of the weight-training program were performed at the university (total of 6 sessions). The computer-paced sessions consisted of the following exercises: bench press, lunges, dumbbell pull, squat, and overhead lats pulley (3×15 repetitions). This sequence of five exercises was completed twice to increase the volume of the session without increasing the number of different exercises. Duration was 42 min, whereas exercise time was equally split between upper body and leg exercise. Work-to-rest ratio was 1:1. Besides these 10 exercises, the session consisted of a 10-min warming up and cooling down, being cycling at 100 or 150 W, depending on the subjects W_{max} ($<70\% W_{max}$). The first 2 wk (i.e., first 4 sessions) were used to practice the techniques and to get accommodated to the laboratory setting and computerized pacing. During the two sessions in week 3, gas exchange (Oxycon Beta, Mijnhart) and HR (Polar Sport Tester model PE3000) were continuously measured. Gas exchange was converted to energy expenditure with the use of the Weir formula (12). Data of both sessions were pooled before calculation of the linear regression equation of EE over HR.

To determine the EE/HR relationship during cycling, a stepwise incremental cycling test was performed in week 1. The test started with 4 min of cycling at 50 W. Subsequently, load was increased every 3 min with 50 W if HR was <160 beats/min and 25 W if HR ≥ 160 beats/min, until exhaustion.

To check whether the training program induced a change in the EE/HR relationship (preintervention equation), the calibration protocol was repeated after the 18 wk of training (postintervention equation). However, three instead of four sessions were used for accommodation.

Statistical analysis. The analysis of variance (ANOVA) for repeated measures was used to compare means, including the two groups as a between-subject factor. Between-group initial values were compared by using a simple factorial ANOVA. Statistical significance was set at $P < 0.05$.

RESULTS

Where appropriate, the results are presented for the entire exercise group (EXER) as well as the subgroup (EX12) in which ADMR was measured.

Changes in physical characteristics. As shown in Table 2, after 18 wk both the EXER and Con had lost a comparable amount of fat mass (2.0 ± 1.8 kg; $P < 0.001$ and 1.4 ± 1.0 kg; $P < 0.05$, respectively) whereas fat-free mass increased only in EXER [2.1 ± 1.2 kg, $P < 0.001$, and 0.4 ± 1.8 kg, not significant (NS), respectively]. The combined changes in fat mass and fat-free mass resulted in an unaltered body mass [0.1 ± 1.5 kg (NS) and -1.0 ± 1.2 kg (NS), respectively] but a similar decrease in percentage fat of $-2.6 \pm 2.0\%$; $P < 0.001$ and $-1.6 \pm 1.5\%$; $P < 0.05$, respectively. In EX12, body

Table 2. Changes in body composition in exercise and control groups

	Exercise, T ₈	Exercise, T ₁₈	Control, T ₁₈
Body mass, kg	0.4 ± 2.2	0.1 ± 1.5	-1.0 ± 1.2
Fat mass, kg	-0.8 ± 1.8	$-2.0 \pm 1.8\ddagger$	$-1.4 \pm 1.0^*$
Fat-free mass, kg	$1.3 \pm 1.3\ddagger$	$2.1 \pm 1.2\ddagger$	$0.4 \pm 1.8\§$
Fat, %	$-1.3 \pm 1.7^*$	$-2.6 \pm 2.0\ddagger$	$-1.6 \pm 1.5^*$

Values are means \pm SD. T₈, T₁₈, weeks in program. *Significantly different from T₀, $P < 0.05$; $\ddagger P < 0.001$ and from T₈, $\ddagger P < 0.05$. $\§$ Significantly different between groups, $P < 0.01$.

mass increased at T₈ with 1.1 ± 1.5 kg ($P < 0.05$) returning to preintervention values at T₁₈.

ADMR. In ADMR subjects, $n = 12$, at T₈, the initial ADMR increased from 12.4 ± 1.2 to 13.5 ± 1.3 MJ/day (9.3%, $P < 0.001$). At T₁₈, ADMR was 13.5 ± 1.9 MJ/day, revealing no further increase. The mean relative increase in ADMR over the 18-wk period was 9.5%.

SMR. There were no changes in SMR in EXER (4.8 ± 0.5 , 4.9 ± 0.5 , 4.8 ± 0.5 kJ/min at T₀, T₈, and T₁₈, respectively) and Con (4.7 ± 0.4 and 4.8 ± 0.4 kJ/min, T₀ and T₁₈, respectively). In EX12, however, SMR increased significantly at T₈ ($P < 0.05$); but at T₁₈, SMR did not differ from T₀ or T₈ (4.6 ± 0.4 , 4.9 ± 0.4 , 4.7 ± 0.3 kJ/min, respectively). The increase in SMR at T₈ was not correlated with changes in body mass or fat-free mass ($P = 0.14$).

Food intake. As shown in Fig. 2, average daily energy intake did not differ between T₀, T₈, and T₁₈ in EXER (10.1 ± 1.8 , 9.7 ± 1.8 , 9.2 ± 1.9 MJ/day) or in Con (10.2 ± 2.6 , 9.4 ± 1.8 , 10.1 ± 1.5 MJ/day, respectively). Food composition did not differ between the groups at the start of the study and did not change during the study. The average ($n = 26$) contribution to daily energy intake of the macronutrients carbohydrate, fat, protein, and alcohol was 48 ± 7 , 35 ± 7 , 15 ± 3 , and $4 \pm 4\%$, respectively. In EX12, energy intake was significant lower than ADMR at all three moments of data collection (-2.6 ± 1.8 , -3.9 ± 2.6 , -4.6 ± 2.0 MJ/day, $P < 0.001$). This underreporting in absolute and relative terms (-21 ± 14 , -28 ± 18 , $-34 \pm 14\%$) increased

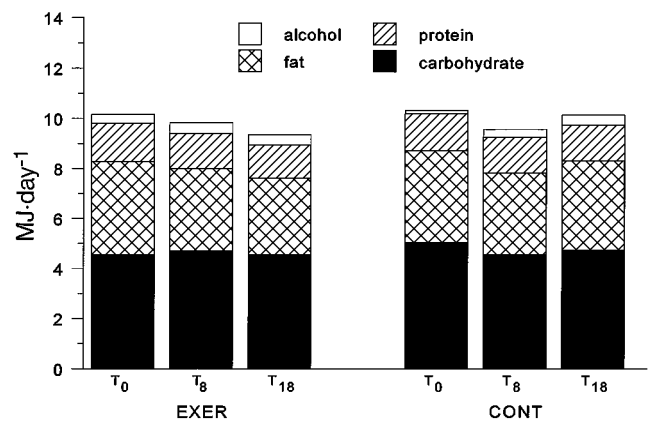


Fig. 2. Energy and macronutrient intake as measured with a 3-day dietary record before (T₀) and after 8- and 18-wk weight training (T₈, and T₁₈, respectively) for EXER and CONT.

during the study but was only significantly different between T_0 and T_{18} ($P < 0.01$).

Workout at the fitness club. Average training compliance, including the six training sessions in weeks 1–3, was $95 \pm 7\%$. The average number and duration of the workouts did not differ between T_8 and T_{18} (1.9 ± 0.5 vs. 1.9 ± 0.5 sessions/wk, 71 ± 11 vs. 73 ± 13 min/workout). To check whether the EE/HR-relationship was affected by the training program, both pre- and postintervention equations were used to predict EE_{ex} at T_8 and T_{18} . Preintervention equations always resulted in a significantly lower EE_{ex} compared with postintervention equations, revealing a change in the exercise EE/HR relationship (at T_8 , 28.7 ± 5.6 vs. 32.7 ± 6.5 kJ/min; at T_{18} , 26.7 ± 5.1 vs. 30.8 ± 5.4 kJ/min, $P < 0.001$). Hence, the preintervention equations were used to estimate EE_{ex} at T_8 , whereas postintervention equations were applied at T_{18} . Average EE_{ex} increased significantly in EXER (28.7 ± 5.6 vs. 30.8 ± 5.4 kJ/min equal to $2,014 \pm 446$ vs. $2,271 \pm 607$ kJ/session, $P < 0.05$) but did not change in EX12 (29.8 ± 4.9 vs. 30.9 ± 6.0 kJ/min equal to $1,952 \pm 348$ vs. $2,151 \pm 616$ kJ/session). Net daily energy cost of the workouts ($EE_{ex} - SMR$, MJ/day) was not different between T_8 and T_{18} in EXER (0.47 ± 0.20 vs. 0.50 ± 0.18 MJ/day) or EX12 (0.43 ± 0.20 vs. 0.48 ± 0.15 MJ/day). At T_8 and T_{18} , only 38 and 41% of the increase in ADMR could be attributed to the net energy cost of the workouts performed at the fitness club.

The components of ADMR. The following components of ADMR (see Fig. 3) were calculated: 24-h SMR (MJ/day), diet-induced thermogenesis (DIT), and the energy cost of physical activity (ADMR – SMR – DIT, MJ/day). The latter was subsequently split up into the net cost of weight training and the cost of nontraining physical activity. The DIT was assumed to be 10% of the measured ADMR. The energy cost of physical activity increased significantly ($P < 0.01$) at T_8 but showed no further increase at T_{18} (4.4 ± 0.9 , 5.3 ± 0.9 , 5.5 ± 1.6 MJ/day, respectively). However, the energy cost of the

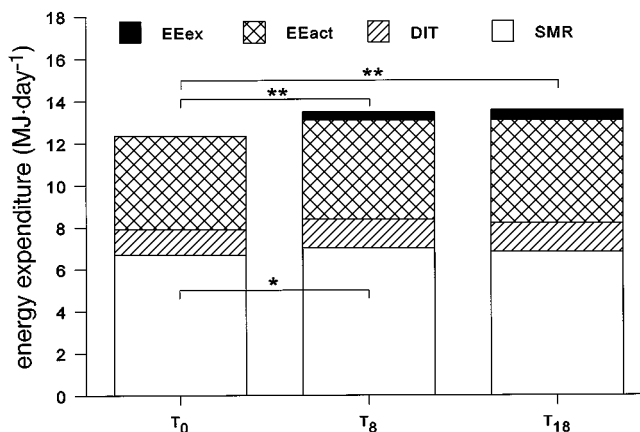


Fig. 3. Mean values of ADMR ($n = 12$) and its components: 24-h SMR, diet-induced thermogenesis (DIT), net energy cost of the exercise ($EE_{ex} = EE_{workout} - SMR$) and energy expenditure for nontraining physical activity (EE_{act}) ($EE_{act} = ADMR - SMR - DIT - EE_{ex}$), before and after 8 and 18 wk of weight training. * $P < 0.05$; ** $P < 0.001$ compared with connected bars.

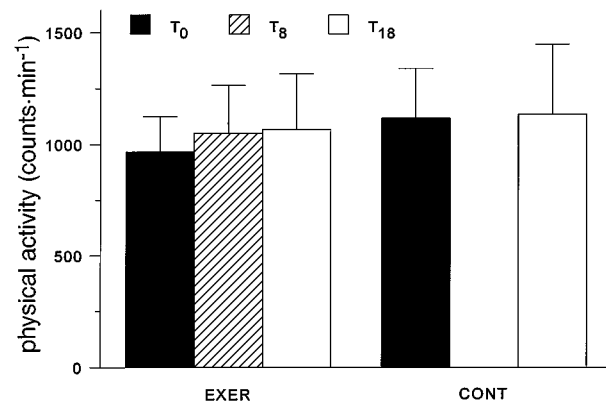


Fig. 4. Nontraining physical activity measured with a triaxial accelerometer, before and after 8 and 18 wk of weight training for EXER and CONT (T_0 , and T_{18}).

nontraining physical activity (ADMR – DIT – SMR – net EE_{ex}) did not change (4.4 ± 0.9 , 4.9 ± 0.8 , and 5.0 ± 1.6 MJ/day, respectively)

Nontraining physical activity. As depicted in Fig. 4, physical activity as measured with the triaxial accelerometer did not change between T_0 , T_8 , and T_{18} in the exercise group (967 ± 158 , $1,052 \pm 214$, $1,068 \pm 249$ counts/min) or the control group ($1,119 \pm 222$, $1,137 \pm 311$ counts/min). Also, activity time did not change in both groups (EXER: 14.0 ± 1.7 , 12.9 ± 2.5 , 13.3 ± 2.3 h/day; Con: 14.3 ± 1.1 , 12.7 ± 2.3 h/day). Average weekly variation in accelerometer output ($n = 26$), expressed as coefficient of variation (individual $SD/x \times 100$), was $14 \pm 8\%$. The daily coefficient of variation decreased gradually during the study and was significantly different between T_0 and T_{18} (28.0 ± 9.9 vs. $20.5 \pm 10.3\%$, $P < 0.01$).

DISCUSSION

Changes in physical characteristics. Although the change in fat mass in the exercise group is in concordance with weight training-induced changes in fat mass found in the literature (-0.1 kg/wk; Ref. 38), the change in fat mass in the control group is an indication that seasonal changes in food habits or spontaneous activity, although not detected by the accelerometer output, could have played a role. The increase in fat-free mass was most likely due to the exercise, because no change was found in the controls. Assuming an energy equivalent of 38.9 and 6.3 MJ/kg fat mass and fat-free mass, respectively (34), it can be calculated that the subjects in both the EXER and Con groups were in a small negative energy balance over the total period of 18 wk (0.5 and 0.4 MJ/day, respectively).

ADMR. This study examined the effect of an 18-wk weight-training program on average daily energy expenditure and found a 1.1 MJ/day (9.3%) and 1.2 MJ/day (9.5%) increase after 8 and 18 wk, respectively. To our knowledge, only four other studies used doubly labeled water to measure the effect of an exercise program on ADMR. The interventions, however, consisted of endurance training instead of weight training. Meijer et al. (19) studied the effect of 20-wk endurance training in a

group of 32 untrained subjects preparing to run a half marathon. ADMR was determined in a subgroup of four men and three women. After 8 and 20 wk, ADMR was significantly increased with ~ 1.6 and 2.2 MJ/day (15 and 20%, respectively). To examine the effect over a prolonged period, Westerterp et al. (35) measured ADMR in a subgroup of 13 subjects who continued the previously mentioned training program for another 20 wk and found no further increase in ADMR after 40 wk (2.3 MJ/day, 21%). Bingham et al. (4) studied a group of three men and two women who followed a 9-wk running program (60 min, 5 day/wk) and found an average increase of 2.8 MJ/day (28%). Blaak et al. (5) found an average increase of 1.3 MJ/day (21%) in a group of 10 obese boys, ages 10–11 yr, after a cycling program (4 wk; 5 sessions/wk, 45 min/session, 50–60% maximal O_2 consumption). In contrast with the above-mentioned studies that found an exercise-induced increase in ADMR, a study by Goran and Poehlman (14) revealed no change in ADMR in a group of 11 elderly subjects who followed an 8-wk cycling program.

Campbell et al. (11) examined the effect of weight training on ADMR, but this study did not use the doubly labeled water technique. The required energy intake to maintain body weight was used to estimate ADMR in a study on the effect of a 12-wk weight-training program in older adults ($n = 12$). An average 1.1 MJ/day ($\approx 15\%$) increase in daily energy intake was required to compensate the cost of weight-training exercise and the increase in resting metabolic rate.

Because the above-mentioned interventions not only differed in the mode of activity but also varied widely in intensity, duration, and frequency of the training program, additional information on the average net energy cost of the extra exercise and changes in the remaining components of ADMR is required to compare and interpret the effect of the various training programs on ADMR.

Net energy cost of the training intervention. Because the net energy cost of a training program depends on the intensity, duration, and frequency of the training sessions, it is evident that studies can differ considerably in exercise-induced changes in ADMR. After 18 wk of weight training, the net EE_{ex} was 0.48 MJ/day. The net EE_{ex} in the study of Campbell et al. (11) as well as the increase in ADMR were similar with the present study (0.42 and 1.1 MJ/day, respectively). Although the energy cost of the training program in the studies of Blaak et al. (5) and Goran and Poehlman (14) was $\sim 50\%$ higher (± 0.63 MJ/day) compared with the present study, ADMR only increased in the study of Blaak et al. (5) that used a group of young boys (1.3 MJ/day). In the elderly group, ADMR did not change. This was explained by a 60% reduction in spontaneous and/or voluntary physical activity. The net EE_{ex} of the added exercise in the study of Meijer et al. (19) was almost twice as high. The increase in ADMR in the latter study was also almost twice the increase of the present study (0.87 and 2.2 MJ/day, respectively). In the study by Bingham et al. (4), no values on net EE_{ex} were presented. From calculations based on the aver-

age time (37 min), speed (11.5 km/h), and body mass (63 kg), net EE_{ex} was estimated to be 1.9 MJ/day (1). This higher EE_{ex} also induced a larger increase in ADMR (2.8 MJ/day).

In general, interventions with higher EE_{ex} showed a higher increase in ADMR. The increase in ADMR, however, always exceeded the energy expenditure due to exercise. As shown in the study by Goran and Poehlman (14), exercise could also affect the remaining components of ADMR (i.e., spontaneous activity). Therefore, other components of ADMR like SMR and nontraining (spontaneous) physical activity should always be included in the design.

SMR. Studies on the effect of exercise on SMR are somewhat controversial (22). The unchanged SMR in the entire exercise group ($n = 18$) over a period of 18 wk was in concordance with a previous weight-training intervention that did not reveal a change in SMR after 12 wk of weight training (33). In EX12, however, SMR showed a small increase at T_8 (0.25 ± 0.27 kJ/min, 5%) but returned to preintervention values after 18 wk of training. Body mass and fat-free mass, two important determinants of SMR, also increased at T_8 . The change in SMR, however, could not be explained by the increase in body mass or fat-free mass at T_8 , probably due to the small changes in SMR and the anthropometric data.

Two of the previously mentioned studies (11, 14), both using elderly subjects, reported a change in resting metabolic rate, whereas the present study and the three remaining studies (4, 5, 19) showed no change in SMR. Besides differences in average age, intervention, changes in body mass, and level of training, the protocol used to determine resting energy expenditure (SMR vs. resting metabolic rate) might affect the outcome of the study. In some studies that measured both SMR and resting metabolic rate after a weight-training intervention (31) or a bout of endurance exercise (3), resting energy expenditure increased, whereas sleeping energy expenditure did not change. On the other hand, Bingham et al. (4) measured also both parameters but found no change in resting metabolic rate or SMR.

Differences in training intervention (type of exercise) are not likely to explain the inconsistency in exercise-induced changes in SMR. Cross-sectional (30) as well as intervention studies (10) that compared the effect of either high-intensity resistance or endurance training revealed no exercise-specific change in resting metabolic rate.

Although some studies reveal a substantial training-induced increase in resting metabolic rate (up to 10%), the increasing effect on ADMR would be at most 5%.

Dietary intake. A 3-day food record was considered to provide information on the food quotient (FQ). The FQ was supposed to reflect the respiratory exchange ratio required to calculate oxygen consumption during the doubly labeled water period. Unfortunately, the results of the 3-day food record revealed a substantial, and gradually increasing, underreporting. Although underreporting is also found in other studies (25, 29, 36), the

increasing magnitude of the underreporting made the data unusable for the calculation of DIT and FQ. Because the changes in body composition revealed only a small negative energy balance, DIT was set at 10% of the measured ADMR (26). An average FQ of 0.85 was assumed (6, 35) during the three measurement periods, because changes in FQ will be relatively small and therefore will not greatly affect the calculation of ADMR (39).

Energy cost of physical activity. The net energy cost (ADMR – SMR – DIT) of physical activity increased significantly by 0.9 and 1.1 MJ/day, equivalent to 20 and 25%. Like other studies that revealed an increase in ADMR (5, 11, 19), the net EE_{ex} explains only partly (± 40 –50%) the increase in the net energy cost of physical activity. In the present study and the study of Meijer et al. (19), the remaining discrepancy could not be attributed to a change in nontraining activity as recorded by an accelerometer. The unchanged accelerometer output in the present study is in line with the unchanged net energy cost of physical activity at T_8 and T_{18} . Blaak et al. (5) also found no change in spontaneous activity measured by HR recording. As indicated by the large weekly and daily coefficients of variation, it is possible, although not likely, that the accelerometer method is not sensible enough to measure small changes in physical activity. The decreased daily variation in physical activity at the end of the study was probably due to seasonal and/or weather changes (study started in winter and ended in summer).

Because the energy cost of physical activity is not directly measured but the residue of subtracting SMR, DIT, and net EE_{ex} from ADMR, the discrepancy could also be due to measurement errors in one of these components. Part of the discrepancy could be attributed to a slight underestimation of the energy cost of EE_{ex} because it does not include the residual energy expenditure associated with postexercise recovery or a short-term effect on SMR. However, the magnitude of postexercise recovery is assumed to be small after both endurance exercise [38–125 kJ per exercise bout (24)] as well as weight training [± 80 kJ (20)]. This small contribution (76–250 kJ/wk) of postexercise recovery to the net energy cost of exercise can hardly explain the remaining part of the increased energy expenditure (a total of 4.4 MJ/wk). On the other hand, a small underestimation of SMR due to a short-term effect (SMR was measured >30 h postexercise), could largely explain the unaccountable increase in energy cost of physical activity and the unchanged physical activity. Although the effect of weight training on the separate components of ADMR remains partially unclear, for the determination of the efficacy of weight training in weight-control programs, the absolute change in ADMR is the main value of interest.

In summary, the unchanged accelerometer output revealed that there was no change in (nontraining) physical activity in both the exercise as well as the control group. Hence, we assume that the change in daily energy expenditure could be attributed to the weight-training program. Therefore, a weight-training

program that consisted of 2 sessions/wk resulted in an average increase in daily energy expenditure of $\sim 10\%$ after 8 wk, with no further increase after 18 wk. Only 40% of the increase could be attributed to the net energy cost of the program. The unexplained part of the increase in ADMR could be due to an underestimation of the net EE_{ex} or measurement errors in other components of ADMR, although the results on SMR revealed no significant change. Weight training did not change body mass, and there was a 2.1 kg increase in fat-free mass in the exercise group. The exercise group as well as the control group showed a decrease in fat mass. From the change in daily energy expenditure, it can be concluded that weight training, although of modest energy cost compared with endurance training, induces a significant increase in ADMR and, therefore, can be applied as an effective and safe adjunct to exercise based weight-control programs.

This study was supported by a research grant from the Netherlands Heart Foundation.

Address for reprint requests: L. M. L. A. Van Etten, Dept. of Human Biology, Univ. of Limburg, PO Box 616, 6200 MD Maastricht, The Netherlands.

Received 26 March 1996; accepted in final form 5 September 1996.

REFERENCES

1. **American Council on Sports Medicine.** *Guidelines for Exercise Testing and Prescription* (4th ed.). Philadelphia, PA: Lea & Febiger, 1991.
- 1a. **Åstrand, P.-O.** Why exercise? *Med. Sci. Sports Exercise* 24: 153–162, 1992.
2. **Berlin, J. A., and G. A. Colditz.** A meta-analysis of physical activity in the prevention of coronary heart disease. *Am. J. Epidemiol.* 132: 612–628, 1990.
3. **Bielinski, R., Y. Schutz, and E. Jequier.** Energy metabolism during the postexercise recovery in man. *Am. J. Clin. Nutr.* 42: 69–82, 1985.
4. **Bingham, S. A., G. R. Goldberg, W. A. Coward, and J. H. Cummings.** The effect of exercise and improved physical fitness on basal metabolic rate. *Br. J. Nutr.* 61: 155–173, 1989.
5. **Blaak, E. E., K. R. Westerterp, O. Bar-Or, L. J. M. Wouters, and W. H. M. Saris.** Total energy expenditure and spontaneous activity in relation to training in obese boys. *Am. J. Clin. Nutr.* 55: 777–782, 1992.
6. **Black, A. E., A. M. Prentice, and W. A. Coward.** Use of food quotients to predict respiratory quotients for the double labelled water method of measuring energy expenditure. *Hum. Nutr. Clin. Nutr.* 40C: 381–391, 1986.
7. **Blair, S. N., H. W. Kohl, R. S. Paffenbarger, D. G. Clark, K. H. Cooper, and L. W. Gibbons.** Physical fitness and all-cause mortality. A prospective study on healthy men and women. *J. Am. Med. Assoc.* 262: 2395–2401, 1989.
8. **Bouten, C. V. C., W. P. H. G. Verboeket-Van de Venne, K. R. Westerterp, M. Verduin, and J. D. Janssen.** Daily physical activity assessment: comparison between movement registration and doubly labeled water. *J. Appl. Physiol.* 81: 1019–1026, 1996.
9. **Bouten, C. V., K. R. Westerterp, M. Verduin, and J. D. Janssen.** Assessment of energy expenditure for physical activity using a triaxial accelerometer. *Med. Sci. Sports Exercise* 26: 1516–1523, 1994.
10. **Broeder, C. E., K. A. Burrhus, L. S. Svanevik, and J. Wilmore.** The effects of either high-intensity resistance or endurance training on resting metabolic rate. *Am. J. Clin. Nutr.* 55: 802–810, 1992.
11. **Campbell, W. W., M. C. Crim, V. R. Young, and W. J. Evans.** Increased energy requirements and changes in body composition with resistance training in older adults. *Am. J. Clin. Nutr.* 60: 167–175, 1994.

12. **De Weir, J. B.** New methods for calculating metabolic rate with special reference to protein metabolism. *J. Physiol. Lond.* 109: 1–9, 1949.
13. **Fuller, N. J., S. A. Jebb, M. A. Laskey, W. A. Coward, and M. Elia.** Four-component model for the assessment of body composition in humans: comparison with alternative methods, and evaluation of the density and hydration of fat-free mass. *Clin. Sci.* 82: 687–693, 1992.
14. **Goran, M. I., and E. T. Poehlman.** Endurance training does not enhance total energy expenditure in healthy elderly persons. *Am. J. Physiol.* 263 (*Endocrinol. Metabol.* 26): E950–E957, 1992.
15. **Hill, D. W., and S. D. Butler.** Haemodynamic responses to weightlifting exercise. *Sports Med.* 12: 1–7, 1991.
16. **Hurley, B. F., R. A. Redmond, R. E. Pratley, M. S. Treuth, M. A. Rogers, and A. P. Goldberg.** Effects of strength training on muscle hypertrophy and muscle disruption in older men. *Int. J. Sports Med.* 16: 378–384, 1995.
17. **McArdle, W. D., and G. F. Foglia.** Energy cost and cardiorespiratory stress of isometric and weight training exercises. *J. Sports Med.* 9: 23–30, 1969.
19. **Meijer, G. A. L., G. M. E. Janssen, K. R. Westerterp, F. Verhoeven, W. H. M. Saris, and F. ten Hoor.** The effect of a 5-month endurance-training programme on physical activity: evidence for sex-difference in the metabolic response to exercise. *Eur. J. Appl. Physiol. Occup. Physiol.* 62: 11–17, 1991.
20. **Melby, C. L., T. Tincknell, and W. D. Schmidt.** Energy expenditure following a bout of non-steady state resistance exercise. *J. Sports Med. Phys. Fitness* 32: 128–135, 1992.
21. **Miller, W., W. Sherman, and J. Ivy.** Effect of strength training on glucose tolerance and post-glucose insulin response. *Med. Sci. Sports Exercise* 16: 539–543, 1984.
22. **Poehlman, E. T.** A review: exercise and its influence on resting energy metabolism in man. *Med. Sci. Sports Exercise* 21: 515–525, 1989.
23. **Poehlman, E. T., A. W. Gardner, P. A. Ades, S. M. Katzman-Rooks, S. M. Montgomery, O. K. Atlas, D. L. Ballor, and R. S. Tyzhir.** Resting energy metabolism and cardiovascular disease risk in resistance-trained and aerobically trained males. *Metabolism* 41: 1351–1360, 1992.
24. **Poehlman, E. T., C. L. Melby, and M. I. Goran.** The impact of exercise and diet restriction on daily energy expenditure. *Sports Med.* 11: 78–101, 1991.
25. **Schoeller, D. A., and C. R. Fjeld.** Human energy metabolism: what have we learned from the doubly labeled water method? *Annu. Rev. Nutr.* 11: 355–373, 1991.
26. **Schutz, Y., T. Bessard, and E. Jequier.** Diet-induced thermogenesis measured over a whole day in obese and nonobese women. *Am. J. Clin. Nutr.* 40: 542–552, 1984.
27. **Stone, M. H., S. J. Fleck, N. Travis Triplet, and W. J. Kraemer.** Health and performance-related potential of resistance training. *Sports Med.* 11: 210–231, 1991.
28. **Stone, M. H., and D. G. Wilson.** Resistive training and selected effects. *Med. Clin. North Am.* 69: 109–122, 1985.
29. **Thompson, J. L., M. M. Manore, J. S. Skinner, E. Ravussin, and M. Spraul.** Daily energy expenditure in male endurance athletes with differing energy intakes. *Med. Sci. Sports Exercise* 27: 347–354, 1995.
30. **Toth, M. T., and E. T. Poehlman.** Resting metabolic rate and cardiovascular disease risk in resistance- and aerobic-trained middle aged women. *Int. J. Obesity* 9: 691–698, 1995.
31. **Treuth, M. S., G. R. Hunter, R. L. Weinsier, and S. H. Kell.** Energy expenditure and substrate utilization in older women after strength training: 24-h calorimeter results. *J. Appl. Physiol.* 78: 2140–2146, 1995.
32. **Van Dale, D., P. F. M. Schoffelen, F. ten Hoor, and W. H. M. Saris.** Effects of addition of exercise to energy restriction on 24-hour energy expenditure, sleeping metabolic rate and daily physical activity. *Eur. J. Clin. Nutr.* 43: 441–451, 1989.
33. **Van Etten, L. M. L. A., K. R. Westerterp, and F. T. J. Verstappen.** Effect of weight-training on energy expenditure and substrate utilization during sleep. *Med. Sci. Sports Exercise* 27: 188–193, 1995.
34. **Westerterp, K. R.** Balance between energy intake and energy expenditure. In: *Food Intake and Energy Expenditure*, edited by M. S. Westerterp-Plantenga, E. W. H. M. Fredrix, and A. B. Steffens. Boca Raton, FL: CRC, 1994, p. 291–309.
35. **Westerterp, K. R., A. Meijer, E. Janssen, W. Saris, and F. ten Hoor.** Long term effect of physical activity on energy balance and body composition. *Br. J. Nutr.* 68: 21–30, 1992.
36. **Westerterp, K. R., W. P. H. G. Verboeket-van de Venne, G. A. L. Meijer, and F. ten Hoor.** Self-reported intake as a measure for energy intake, a validation against doubly labelled water. In: *Obesity in Europe 91*, edited by G. Ailhaud, B. Guy-Grand, M. Lafontan, and D. Ricquier. London: Libbey, 1992, p. 17–22.
37. **Westerterp, K. R., L. Wouters, and W. D. Marken Lichtenbelt.** The Maastricht protocol for the measurement of body composition and energy expenditure with labeled water. *Obesity Res.* 3, *Suppl.* 1: 49–57, 1995.
38. **Wilmore, J. H.** Variations in physical activity habits and body composition. *Int. J. Obesity* 19, *Suppl.* 4: S107–S112, 1995.
39. **Wolfe, R. R.** Measurement of total energy expenditure using the doubly-labeled water method. In: *Radioactive and Stable Isotope Tracers in Biomedicine: Principles and Practice of Kinetic Analysis*. New York: Wiley-Liss, 1992, p. 207–233.