

The effects of either high-intensity resistance or endurance training on resting metabolic rate¹⁻³

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ABSTRACT The effects of either 12-wk of high-intensity endurance or resistance training on resting metabolic rate (RMR) were investigated in 47 males aged 18–35 y. Subjects were randomly assigned to either a control (C), resistance-trained (RT) or endurance-trained (ET) group. After training both exercise groups showed significant declines in relative body fat either by reducing their total fat weight and maintaining fat-free weight (ET) or by reducing their total fat weight and increasing fat-free weight (RT). RMR did not significantly change after either training regimen although a small decline in energy intake was observed along with an increase in energy expenditure [ET, 2.721 MJ (650 kcal) per training day]. These results suggest that both endurance and resistance training may help to prevent an attenuation in RMR normally observed during extended periods of negative energy balance (energy intake < expenditure) by either preserving or increasing a person's fat-free weight. *Am J Clin Nutr* 1992;55:802–10.

KEY WORDS Resting metabolic rate, resistance training, endurance training

Introduction

For individuals who are overweight or obese, it is well-established that to lose weight energy expenditure must exceed energy intake by bringing about a negative energy balance. Dieting has been the most popular method used to create a negative energy balance for weight loss. However, the long-term results of dieting for weight loss have not been very encouraging, ie, there is a considerable regain of the weight lost. This has led investigators to reconsider the energy-expenditure side of the energy-balance equation, especially factors affecting a person's resting metabolic rate (RMR) (1, 2). For example, although dieting induces a negative energy balance, it also was shown to produce a subsequent decline in RMR of as much as 30% (3), which may occur to help a person maintain a set point for body weight (1, 2, 4).

RMR accounts for ≈60–75% of a person's total daily energy needs. Thus, small changes that either attenuate or potentiate RMR can have a dramatic effect on a person's total daily energy expenditure. Several cross-sectional studies have suggested that endurance training may potentiate RMR (5–7) whereas other studies have been unable to confirm these results (8–11). However, cross-sectional studies are limited because they do not allow us to determine if a relationship between RMR and aerobic fitness is the result of exercise training or is due to other covariates, such as genotype (12, 13).

The effects of endurance training on RMR were also studied longitudinally (14–19). Longitudinal studies observing the responses of an untrained group of subjects to a period of training can provide valuable information about the sequence of adaptations occurring during a particular training period. The results of previous studies regarding the effects of endurance training on RMR are inconsistent. Some studies suggested that endurance training can potentiate RMR (14, 15) whereas other studies indicate that RMR is unaltered (16, 17, 19) or may decline slightly (18) after extended training periods.

No studies have investigated the effects of resistance training on RMR in nondieting individuals. However, heavy resistance training promotes skeletal-muscle development, which could potentiate a person's RMR by increasing the total amount of metabolically active tissue [ie, fat-free weight (FFW)]. The purpose of this study was to investigate the effects of 12 wks of either heavy resistance or high-intensity endurance training on RMR in 64 previously active but untrained men aged 18–35 y.

Methods

Subject recruitment and treatment-group descriptions

Sixty-four male volunteers (62 white, 2 black) aged 18–35 y were recruited from the student population at the University of Texas at Austin and from the surrounding community. All methods and procedures had been approved by the Human Subjects Committee at the University of Texas at Austin. All subjects read and signed the subject-consent form and completed a medical history form before the study began. Information provided by the medical-history reports indicated that all subjects had no significant changes in body weight 6 mo before the study began. Subjects were randomly assigned to one of three study groups including a control group (C, $n = 20$), a resistance-trained group (RT, $n = 22$), and an endurance-trained group (ET, $n = 22$). Of these 64 original volunteers, 17 did not complete the study making the total sample size 47 (C, $n = 19$; RT, $n = 13$;

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ET, $n = 15$). In the exercise-trained groups each subject completed $\geq 90\%$ of his 48 scheduled workout sessions. Of those 17 subjects not completing the study, 14 subjects dropped out because of lack of time and/or commitment to the program whereas 3 subjects dropped out as a result of injury.

Pre-, mid-, and posttreatment measurements

Before and after the control or training period, each subject completed a body composition analysis by hydrostatic weighing with an estimation of residual volume, two submaximal steady-state cycle-ergometer tests, two graded treadmill tests for the determination of maximal oxygen consumption ($\dot{V}O_{2\max}$); two one-repetition maximum (1-RM) strength test for six different lifts, and two determinations of RMR by indirect calorimetry. In addition, a 3-d dietary and activity log was obtained for each subject before, between weeks 6 and 7 of the treatment period, and after the training or control period (Fig 1).

Body composition analysis

Hydrostatic weighing was performed as described by Behnke and Wilmore (20) to determine body density. Relative body fat was calculated by using the equation of Siri (21), except for two blacks, for whom the equation of Schutte et al (22) was used. Residual lung volume was estimated using the oxygen dilution method according to Wilmore et al (23). Skinfold-thickness measurements were taken to help verify each subject's relative body fat, FFW, and fat weight (FW) determined by the hydrostatic-weighing procedures. The following skinfold sites were used: subscapular, tricep, chest, suprailium, abdomen, and thigh (20). Before and after each treatment each subject's body mass index (BMI) and waist-to-hip ratio were also determined.

Resting metabolic rate

RMR was measured for all subjects before and after either the training or control period. All subjects had a restful night's sleep (≥ 8 h) and did not eat or consume any liquids, except water, for ≤ 12 hours before each testing session. Subjects did not perform any exercise training for ≥ 48 h before each testing session and were at complete rest in a semirecumbent position for ≥ 30 min before each testing session. The ambient room temperature was maintained at $\approx 23 \pm 1$ °C. The mean of each subject's two RMR trials was used unless the difference between the two trials exceeded 25 mL oxygen/min, in which case a third RMR trial was performed. The final RMR value was determined by taking the mean of the two closest trials. In addition, each subject in the ET group had his RMR measured during weeks 11 and 12 of the training period to determine the acute effects on RMR of exercise within 24 h. This RMR was determined ≈ 14 h after a 50-min exercise bout performed at an intensity of $\approx 82\%$ of each subject's pretraining $\dot{V}O_{2\max}$. A linear regression equation was developed from pretraining heart rate and oxygen-consumption values on the graded exercise test. The average heart rate during the 50-min exercise bout was then used to estimate an average oxygen consumption. The mean group caloric energy expenditure for this exercise was ≈ 2.721 MJ (650 kcal).

To ensure that each subject was well-rested the night before each RMR determination, a telemetric heart-rate monitor by CIC, Inc (Polar Electro Oy, Kempele, Finland) was used to record sleeping heart rates continuously throughout the night. A set of instructions was provided and explained carefully to each person.

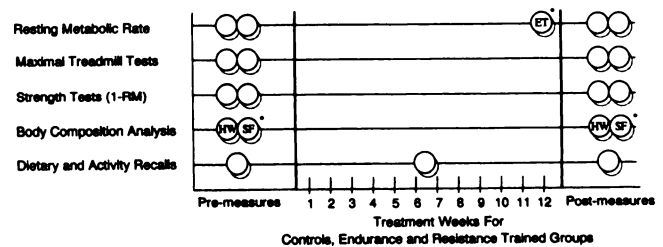


FIG 1. Data collection summary. HW, hydrostatic weighing; SF, skinfold measurements; ET, endurance-trained group.

The heart rate monitor was activated by the subject immediately before going to sleep. The monitor was set so that heart rates would be recorded each minute for ≤ 17 h. The heart rate monitor was not removed until the next morning at the end of the RMR trial.

Each subject was transported by motor vehicle to the testing site to ensure minimal activity before RMR determination. After entering the laboratory, the subject was seated in a semirecumbent position for ≥ 30 min. A Hans-Rudolph (Kansas City, MO) face mask was positioned on the subject to assure an air tight seal. Ventilation, oxygen consumption, and respiratory exchange ratio were then monitored continuously for 30 min. Each subject was instructed to remain as quiet as possible before and throughout the entire RMR measurement period. The room was darkened and noise was kept to a minimum during testing. Metabolic data were collected using either a SensorMedics (Yorba, CA) Horizon or a SensorMedics 2900 metabolic cart, two automated computerized analysis systems. Both systems were calibrated before each RMR test by using a calibration gas of a known concentration determined by Scholander analysis. Immediately after each test a postcalibration verification test was performed to determine sensor stability throughout the test period.

Maximal treadmill exercise tests

Before and after the training and control periods, all subjects performed two graded treadmill tests to exhaustion to determine $\dot{V}O_{2\max}$. Before each treadmill test subjects performed an 8-min submaximal cycle-ergometer trial to establish steady-state conditions at 100 W. The results of the submaximal exercise trials will not be discussed here. During the graded treadmill trials, $\dot{V}O_{2\max}$ was defined as that point at which 1) oxygen consumption reaches a plateau or 2) the peak $\dot{V}O_{2\max}$ value if the respiratory exchange ratio was > 1.15 . The graded treadmill test to exhaustion for determining $\dot{V}O_{2\max}$ began with a 4-min warm-up period. For minutes 1 and 2, the speed and grade were set at 5.6 km/h and 0%, respectively. For minutes 3 and 4, the speed remained the same and grade was increased to 2.5%. Beginning at minute 4 either speed or grade was increased every minute until a treadmill grade of 10% was achieved. Thereafter, only the treadmill speed was increased until each subject reached volitional fatigue.

Metabolic measurements during exercise were obtained by using a two-way low-resistance breathing valve with mouthpiece interfaced with a SensorMedics 2900 metabolic cart. Both the carbon dioxide and the oxygen analyzers were calibrated before and after each test by using a calibration gas of a known concentration determined by Scholander analysis. Exercising heart

rates during submaximal and maximal tests were determined by using both a standard electrocardiograph (SensorMedics Horizon System) and a telemetric system (CIC, Inc).

Determination of maximal strength

Each subject underwent pre- and posttraining strength testing using a one-repetition maximum method (1-RM) after a brief warm-up period. Each subject was tested twice before the control or training periods for maximum strength on the following weight-lifting movements: bench press, barbell curl, tricep pressdown, leg press, leg extension, and leg curl. The mean value was used to represent each person's 1-RM. This protocol was repeated after the training and control periods.

Three-day dietary and activity recalls

Each subject completed a 3-d dietary recall for determining mean daily total caloric intake and percentage of energy nutrients before, during weeks 6 and 7, and after the control or exercise-training period. Subjects were provided with written guidelines and a record booklet for keeping track of daily food intake. All subjects were shown how to record dietary intake in the record-keeping booklet. Recording days were randomly assigned; however, these recalls always included 1 weekend day and 2 weekdays for each 3-d dietary-recall period.

Each subject also completed a 3-d activity recall to estimate the time spent in low-, moderate-, or high-intensity activities before, during weeks 6 and 7, and after the control or exercise-training period. Activity levels were defined with the Borg scale of perceived exertion as follows: low-intensity activities had a value of < 11; moderate-intensity activities, 11–15; and high-intensity activities, ≥ 16 (24). Recording days were identical to those for the dietary recalls.

Training program

After completing all pretreatment tests, each subject participated for 12-wk in one of the activity groups: C, RT, or ET. C subjects were instructed not to change activity and dietary habits.

The program for subjects in RT group was specifically designed to increase strength and fat-free mass. Each subject performed heavy resistance training with a combination of free weights and Nautilus machines (Nautilus Sports/Medical Indus, Inc, Independence, VA), 4 d/wk (Monday, Tuesday, Thursday, and Friday). All training sessions were monitored by a trained exercise leader. The strength-training program included the following exercise movements: bench press, parallel dip, behind-neck press, upright rows, tricep pressdown, leg press, leg extension, leg curl, lat pulldown, barbell curl, and abdominal crunch. These exercises were divided into six movements per training day and took approximately 1 h. Upper-body exercises were performed on Monday and Thursday and lower-body exercises were performed on Tuesday and Friday. Abdominal crunches were performed at the end of every workout day. For each subject's safety while performing free-weight exercises, subjects were divided into groups so that each person lifting had at least one spotter.

During the first 2 wk of the resistance-training program, subjects performing 10–12 repetitions per set, three sets per exercise movement, after a brief stretching and warm-up period. The resistance was set on each exercise so that each subject became fatigued between 10 and 12 repetitions. This 2-wk period was designed to prepare each subject for the remaining 10 wk of the program. During the next 10 wk upper- and lower-body exercises

were performed with the weight established on each set so that failure to lift the weight occurred between 10 and 12 repetitions on the first set, 8 and 10 repetitions on the second set, and 6 and 8 repetitions on the third set. Resistance was increased for each exercise movement during the program based on the number of repetitions required to promote fatigue in each subject. Abdominal crunches were performed for one set until failure or until the subject reached 50 repetitions. When a subject could do 50 repetitions of the abdominal crunch, weight was added by having the subject hold a 2.27–11.33-kg weight over his chest while performing each crunch. Training make-up sessions were on Wednesday and Saturday.

Each subject in ET group participated in a walk and/or jog program 4 d/wk (Monday, Tuesday, Thursday, and Friday) for 12 wk. All exercise-training sessions were monitored by a trained exercise leader and each subject was instructed to gradually increase exercise duration and intensity so that a new training goal was reached every 4 wk. By the end of week 4, each person was exercising for 40 min at a minimum intensity of 70% $\dot{V}O_2\text{max}$. After week 8, each person was exercising for 50 min at an intensity between 70% and 85% $\dot{V}O_2\text{max}$. In weeks 8–12 each subject also included fartlek-type intervals in his training regimen: 2–5 min at $\geq 90\%$ $\dot{V}O_2\text{max}$. Over the 12-wk training program six ET subjects who completed the study exercised on the Schwinn (Chicago) Airdyne for 1–3 d and one exercised for 2 wk because of injury or symptoms suggesting injury. These subjects were placed back in the walk-jog program as soon as they became symptom free. Training make-up session times were provided on Wednesday and Saturday.

To accurately determine that each subject maintained a predetermined target heart rate, subjects periodically wore a telemetric heart-rate monitor. The determination of each subject's target heart rate was estimated from a linear-regression model by using steady-state oxygen-consumption and heart rate values achieved during each stage of the pretest determinations of $\dot{V}O_2\text{max}$.

Statistical analysis

Linear regression was used to assess the relationship between RMR and age, height, total body weight (TWT), relative body fat, FFW, FW, and energy intake for both the pre- and post-treatment measurement periods. Data were analyzed with subjects classified by treatment group. An analysis of variance (ANOVA) with a repeated measure was used to analyze the differences between each of the groups for RMR expressed as kJ/min, $\text{kJ} \cdot \text{kg TWT}^{-1} \cdot \text{h}^{-1}$, and $\text{kJ} \cdot \text{kg FFW}^{-1} \cdot \text{h}^{-1}$. An ANOVA with a repeated measure was also used to determine the differences between treatment groups for all other dependent variables (eg, age, height, and weight). When the *F* ratio was significant at $P < 0.05$, multiple-contrast procedures were performed to identify the significant difference. Finally, both an intrasubject correlation and an ANOVA with a repeated measure were performed to determine the between-trial reliability and the significance of differences between the repeat trials for RMR, $\dot{V}O_2\text{max}$, treadmill times to exhaustion, maximum heart rate (HR_{max}), maximum ventilation rate ($\dot{V}E_{\text{max}}$), maximum respiratory exchange ratio (RER_{max}), and 1-RM and between each sleeping and RMR heart rate, pairing for both the pre- and posttreatment measurement periods. All values are expressed as mean \pm SE. *Super ANOVA* (Abacus Concepts, Inc, Berkeley, CA) was used for statistical analyses.

TABLE 1
Reliability for the measurements of RMR and maximal tests*

Variable	Pretreatment			Posttreatment		
	Trial 1	Trial 2	Intrasubject correlation	Trial 1	Trial 2	Intrasubject correlation
RMR (kJ/min)	5.40 ± 0.17	5.40 ± 0.17	0.92	5.48 ± 0.08	5.44 ± 0.13	0.94
$\dot{V}O_2$ max (mL · kg ⁻¹ · min ⁻¹)	48.8 ± 1.1	49.4 ± 1.2	0.99†	51.2 ± 1.2	51.4 ± 1.2	0.99
Treadmill time (min)	8.7 ± 0.2	9.0 ± 0.2	0.96†	9.2 ± 0.2	9.3 ± 0.2	0.93†
HRmax (bpm)	199.0 ± 1.0	199.0 ± 1.0	0.89	196.8 ± 1.5	195.7 ± 1.4	0.94†
$\dot{V}E$ max (L/min)	133.9 ± 2.9	140.2 ± 2.4	0.88†	144.4 ± 2.6	146.0 ± 2.7	0.92†
RERmax	1.17 ± 0.01	1.17 ± 0.01	0.56	1.21 ± 0.01	1.21 ± 0.01	0.57
1-RM (kg)						
Upper body‡	103.7 ± 3.0	104.6 ± 3.2	0.98	110.0 ± 3.2	110.5 ± 3.1	0.98
Lower body§	141.2 ± 4.6	147.0 ± 4.3	0.95†	156.7 ± 4.7	158.0 ± 4.5	0.96

* $\bar{x} \pm SE$.

† $P < 0.05$.

‡ Mean values for bench press, bicep curl, and tricep press-down.

§ Mean values for leg press, leg extension, and leg curl.

Results

The pretreatment intrasubject correlations to establish reliability for RMR, $\dot{V}O_2$ max, treadmill time to exhaustion, HRmax, $\dot{V}E$ max, RERmax, upper-body 1-RM, and lower-body 1-RM varied between 0.56 and 0.99 (Table 1). The ANOVA procedures indicated that there were no significant differences between the two pretreatment measurements for RMR, HRmax, RERmax, and upper-body 1-RM. In contrast, significant differences were found for $\dot{V}O_2$ max, treadmill time to exhaustion, $\dot{V}E$ max and lower-body 1-RM. However, because the differences between the two values for $\dot{V}O_2$ max, treadmill times to exhaustion, $\dot{V}E$ max, and lower-body 1-RM obtained were only 1.2%, 3.5%, 4.7%, and 4.1%, respectively, the representative pretreatment values were obtained by averaging these two trials.

The posttreatment intrasubject correlations to establish reliability for RMR, $\dot{V}O_2$ max, treadmill time to exhaustion, HRmax, $\dot{V}E$ max, RERmax, and upper- and lower-body 1-RM ranged between 0.57 and 0.99 (Table 1). The differences between the two posttreatment trials for these variables were not statistically significant, except for treadmill times and HRmax. There

was a significant difference observed between trials 1 and 2 for treadmill times and HRmax but these differences were only $\approx 1\%$, so the final posttreatment treadmill time and HRmax were obtained by averaging the two trials.

$\dot{V}O_2$ max, treadmill time to exhaustion, HRmax, $\dot{V}E$ max, and RERmax were not significantly different between groups during the pretreatment measurements (Table 2). For $\dot{V}O_2$ max, comparisons between pre and posttreatment measurement periods indicated that only the ET group showed a significant increase in $\dot{V}O_2$ max (11.3%). The RT group showed an increase in $\dot{V}O_2$ max of 3.3%. This value approached significance the 12-weeks of resistance training ($P < 0.06$). Treadmill times increased significantly from pre- to posttreatment for both the RT and ET groups. There were no significant differences pre- to posttreatment in the C group for either $\dot{V}O_2$ max or treadmill time. HRmax declined significantly only in the ET group whereas $\dot{V}E$ max increased significantly in the ET and RT groups after the 12-wk training period. RERmax was significantly higher in all three groups during the posttreatment period.

At the start of the study, there were no statistically significant differences observed between groups for each 1-RM test (Table

TABLE 2
Maximal treadmill data for pre- and posttreatment*

Variable	Pretreatment			Posttreatment		
	Control (n = 19)	Resistance (n = 13)	Endurance (n = 15)	Control (n = 19)	Resistance (n = 13)	Endurance (n = 15)
$\dot{V}O_2$ max (mL · kg ⁻¹ · min ⁻¹)	49.1 ± 2.2	48.1 ± 1.5	49.6 ± 2.2	49.2 ± 2.1	49.7 ± 1.5†	55.2 ± 2.2‡§
Treadmill time (min)	8.9 ± 0.3	8.7 ± 0.2	8.9 ± 0.4	8.9 ± 0.3	9.1 ± 0.2§	9.8 ± 0.3‡§
HRmax (bpm)	195.6 ± 2.3	201.3 ± 1.9	201.1 ± 2.6	194.9 ± 2.4	198.9 ± 2.3	195.2 ± 2.6§
$\dot{V}E$ max (L/min)	139.1 ± 3.8	141.7 ± 5.2	131.8 ± 4.3	141.3 ± 3.6	150.8 ± 5.1§	145.2 ± 4.9§
RERmax	1.18 ± 0.02	1.18 ± 0.02	1.18 ± 0.02	1.20 ± 0.01§	1.20 ± 0.01§	1.21 ± 0.01§

* $\bar{x} \pm SE$.

† Almost reached significance for difference from pretreatment.

‡ Significantly different from posttreatment resistance and control, $P < 0.05$.

§ Significantly different from pretreatment for same group, $P < 0.05$.

TABLE 3
1-RM values before and after training and control periods*

Variable	Pretreatment			Posttreatment		
	Control (n = 19)	Resistance (n = 13)	Endurance (n = 15)	Control (n = 19)	Resistance (n = 13)	Endurance (n = 15)
Bench press (kg)	63.8 ± 3.0	68.2 ± 4.4	68.8 ± 4.6	63.2 ± 2.8	79.9 ± 4.4†‡	71.6 ± 4.6
Bicep curl (kg)	40.1 ± 1.5	40.5 ± 2.4	42.5 ± 1.9	42.0 ± 1.4	49.0 ± 2.0†‡§	42.5 ± 1.8
Tricep press (kg)	32.6 ± 1.7	33.3 ± 1.9	34.0 ± 1.7	36.2 ± 1.3‡	41.3 ± 1.8†‡§	34.3 ± 1.7
1-RM upper body (kg)	45.5 ± 2.0	47.3 ± 2.7	48.5 ± 2.5	47.1 ± 1.6	56.6 ± 2.5†‡§	49.5 ± 2.7
Leg press (kg)	66.8 ± 3.0	68.7 ± 5.5	66.7 ± 3.4	63.5 ± 3.0‡	74.2 ± 4.7†‡§	61.8 ± 2.5‡
Leg curl (kg)	42.2 ± 2.6	42.4 ± 3.7	44.5 ± 1.9	50.4 ± 2.5‡	57.9 ± 3.5‡	51.9 ± 2.0‡
Leg extension (kg)	86.4 ± 4.8	82.8 ± 6.3	86.0 ± 4.9	91.3 ± 4.9	106.5 ± 6.0‡	90.5 ± 5.2
1-RM lower body (kg)	65.1 ± 2.7	64.7 ± 4.7	63.9 ± 3.1	68.4 ± 3.0	79.5 ± 4.4†‡§	68.0 ± 3.0

* $P < 0.05$.

† Significantly different from posttreatment control, $P < 0.05$.

‡ Significantly different from pretreatment for same group, $P < 0.05$.

§ Significantly different from posttreatment endurance, $P < 0.05$.

3). In addition, when all upper- and lower-body 1-RM tests were averaged, there were also no statistically significant differences observed between groups. After the treatment period only the RT group showed significant gains in strength over the pretreatment values for all 1-RM resistance tests performed, with a significant mean increase in upper- and lower-body measurements by 19.6% and 10.3%, respectively. The percent changes in both aerobic fitness and strength in all groups are summarized in Figure 2.

The body composition results for the pre- and posttreatment measurement periods are presented in Table 4. For the pretreatment period there were no significant differences observed between groups for TWT, relative body fat, FFW, FW, BMI, and the waist-hip ratio. RT and ET groups showed a significant decline in their relative body fat, FW, and waist-hip ratio from pre- to posttreatment measurements. In the RT group, FFW increased significantly by 3.3% and FW declined 11.8%. The ET group experienced a significant decline in FW of 9.6% but there was not a significant increase in FFW. For the C group there were no significant pre- to posttreatment differences TWT, relative body fat, FFW, and FW. There was a strong correlation for the relative body fat determinations between hydrostatic

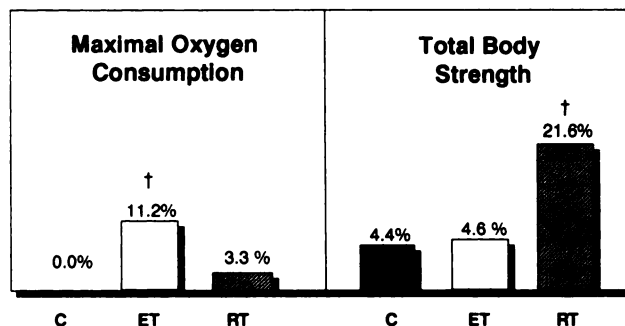


FIG 2. Percent changes in maximal oxygen consumption ($\dot{V}O_{2max}$) and strength. † $P < 0.05$ for a significant gain from pre- to posttreatment measurement. C, control group; ET, endurance-trained group; and RT, resistance-trained group.

weighing and the skinfold-thickness measurements for both the pre- ($r = 0.92$, $P < 0.0001$) and posttreatment periods ($r = 0.92$, $P < 0.0001$). However, skinfold-thickness measurements underestimated relative body fat by 2.4% and 2.1% for the pre- and posttreatment periods, respectively, for all subjects combined.

Pretreatment values for RMR in kJ/min , $\text{kJ} \cdot \text{kg TWT}^{-1} \cdot \text{h}^{-1}$, $\text{kJ} \cdot \text{kg FFW}^{-1} \cdot \text{h}^{-1}$; RER; sleeping heart rate (SHR), and resting heart rate (RHR) were not statistically significant between groups (Table 5). After the control and training periods no significant changes were found in RMR for any group. However, significant differences were found between the pre- and posttreatment periods for RER, SHR, and RHR but only in the ET group (RER was 2.6% higher whereas RHR and SHR were 10.0% and 9.8% lower, respectively, posttreatment).

Regarding the acute effects of endurance exercise on RMR in the ET group (Figure 3), there were no significant differences observed between the posttraining RMR not preceded by exercise and the RMR measurement made within 14 h after a 50-min bout of endurance exercise (14-h RMR). However, there was a significant difference between the pretreatment RMR and the 14-h RMR expressed in kJ/min (pretreatment RMR without prior exercise 5.236 vs 14-h RMR 5.487 kJ/min) and in $\text{kJ} \cdot \text{kg TWT}^{-1} \cdot \text{h}^{-1}$ (pretreatment RMR without prior exercise 4.060 vs 14-h RMR 4.273 $\text{kJ} \cdot \text{kg TWT}^{-1} \cdot \text{h}^{-1}$).

Linear regression analysis showed that there was a significant relationship in the pretreatment period between RMR in L/min and FFW ($r = 0.62$), TWT ($r = 0.57$), and FW ($r = 0.37$) and in the posttreatment period between RMR in L/min and FFW ($r = 0.82$), TWT ($r = 0.82$), FW ($r = 0.56$), and relative body fat ($r = 0.39$).

Energy intakes were not significantly different between groups before the control and training periods (Table 6). There were no significant changes found between the pre- and posttreatment measurements for all groups. However, energy intake expressed in $\text{MJ} \cdot \text{kg TWT}^{-1} \cdot \text{d}^{-1}$ was found to be significantly lower mid-treatment than during pre- or posttreatment periods. During the pretreatment period the control group consumed significantly fewer calories from carbohydrates than did either the RT or ET

TABLE 4
Body composition values before and after training and control periods*

Variable	Pretreatment			Posttreatment		
	Control (n = 19)	Resistance (n = 13)	Endurance (n = 15)	Control (n = 19)	Resistance (n = 13)	Endurance (n = 15)
TWT (kg)	79.6 ± 3.1	81.6 ± 3.9	79.0 ± 3.8	79.9 ± 3.1	81.6 ± 3.8	77.9 ± 3.4
Relative body fat (%)	18.6 ± 2.1	21.8 ± 1.7	18.5 ± 1.9	18.5 ± 2.1	19.3 ± 1.7†	17.0 ± 1.5†
FFW (kg)	64.8 ± 1.3	63.8 ± 2.4	64.4 ± 2.3	65.0 ± 1.3	65.9 ± 2.6†‡	64.7 ± 2.2
FW (kg)	14.8 ± 2.0	17.8 ± 2.0	14.6 ± 2.0	14.9 ± 2.4	15.7 ± 1.9†	13.2 ± 1.7†
BMI	25.3 ± 1.0	25.5 ± 1.1	25.1 ± 1.1	25.3 ± 1.0	25.4 ± 1.0	24.8 ± 1.0
Waist-hip ratio	0.90 ± 0.01	0.90 ± 0.02	0.88 ± 0.02	0.89 ± 0.01	0.88 ± 0.02†	0.87 ± 0.01†

* $\bar{x} \pm SE$.

† Significantly different from pretreatment for same group, $P < 0.05$.

‡ Significantly different from posttreatment control and endurance, $P < 0.05$.

groups. Calories from fat were significantly higher in the control group than in the other two groups before training. No significant differences were found between the mid- and posttreatment periods for the percentage of calories derived from carbohydrates, fat, or protein between groups.

Three-day activity recalls indicated there were no statistically significant differences between groups before the study began regarding the amount of time they spent performing low-, moderate-, and high-intensity activities. In addition, there were no significant changes in each group's normal daily activity patterns between measurement periods (pre-, mid-, or posttreatment) aside from changes induced by the training protocols. When all pre-, mid-, and posttreatment measurements were averaged together for all three groups, low-intensity activities accounted for 88.6%, moderate-intensity activities accounted for 10.5%, and high-intensity activities accounted for 0.92% of the total daily living activities.

Discussion

The results of this study agree well with other studies showing that when exercise training was the primary determinant in producing a negative energy balance, RMR remained unchanged

after an extended period of training (17, 19). When RMR was expressed in either absolute terms or relative to FFW and TWT, there were no significant changes in RMR after either exercise-training regimen. In a study by Bingham et al (17), the effects of a combination 9-wk jogging and isometric exercise-training program on basal metabolic rate (BMR) was investigated in six normal-weight volunteers. All subjects lived in a metabolic ward, allowing the investigators to tightly control dietary intake and caloric expenditure throughout the entire experimental period. However, because energy intake was held constant throughout the study and energy expenditure was increased by a daily exercise training program (jogging 1 h/d, 5 d/wk), energy expenditure exceeded intake by 20% in these subjects by weeks 8 and 9, producing an exercise-induced negative energy balance. Yet, as in the present study, BMR did not decline in this group of subjects. In a study by Tremblay et al (19), subjects performed cycle-ergometer exercise at approximately 50% $\dot{V}O_2\max$ for 6 d/wk over a 100-d period, producing 4.2 MJ/d negative energy balance. Both caloric intake and exercise training were strictly controlled by housing subjects in an experimental facility. Thus, the long-term energy deficit was totally generated by exercise training. After the training period, there were again no significant declines in RMR expressed in absolute terms or relative to FFW.

TABLE 5
RMR data pre- and posttreatment*

Variable	Pretreatment			Posttreatment		
	Control (n = 19)	Resistance (n = 13)	Endurance (n = 15)	Control (n = 19)	Resistance (n = 13)	Endurance (n = 15)
RMR						
(kJ/min)	5.53 ± 0.17	5.36 ± 0.17	5.23 ± 0.13	5.48 ± 0.13	5.53 ± 0.21	5.32 ± 0.13
(kJ · kg TWT ⁻¹ · h ⁻¹)	4.23 ± 0.17	4.02 ± 0.13	4.06 ± 0.17	4.14 ± 0.08	4.11 ± 0.08	4.19 ± 0.13
(kJ · kg FFW ⁻¹ · h ⁻¹)	5.19 ± 0.13	5.11 ± 0.13	4.98 ± 0.17	5.11 ± 0.08	5.07 ± 0.13	5.02 ± 0.13
RER	0.79 ± 0.01	0.79 ± 0.01	0.78 ± 0.01	0.81 ± 0.01	0.80 ± 0.01	0.83 ± 0.01†
SHR (bpm)	56.0 ± 1.5	55.0 ± 1.7	57.0 ± 3.0	57.3 ± 1.4	54.2 ± 2.1	51.4 ± 2.3†‡
RHR (bpm)	59.2 ± 1.5	61.0 ± 2.5	61.9 ± 2.7	60.2 ± 1.5	59.8 ± 2.2	55.7 ± 2.4†‡

* $\bar{x} \pm SE$.

† Significantly different from pretreatment for same group, $P < 0.05$.

‡ Significantly different from posttreatment control and resistance, $P < 0.05$.

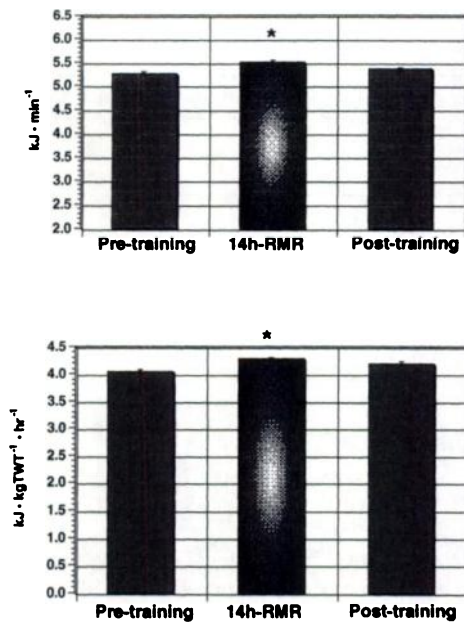


FIG 3. Acute effects of endurance training on RMR. *Significantly different from pretraining value, $P < 0.05$.

In the present study when the small declines in dietary intake (C, 11.8%; RT, 7.3%; and ET, 7.7%) are considered in conjunction with the calories that were being expended by the subjects in both training groups, the observation that RMR did not decline is important. For example, between weeks 8 and 12, subjects in the ET group were running for 50 min/d at 82% $\dot{V}O_2$ max, which was equivalent to ≈ 2.7 MJ (650 kcal) per exercise training session. When the calories that were being expended during each exercise session are combined with the ET group's 24-h RMR value of 7.5 MJ/d (1792 kcal/d), the thermic requirements for digesting and absorbing their food ($\approx 10\%$ of the total calories ingested or 1.1 MJ/d) (252 kcal/d), and caloric expenditure for other physical activities such as walking to class, standing, etc [10% of the total energy expenditure or 1.3 MJ/d) (305 kcal/d)], the mean total number of calories required by the ET group per training day was estimated to be ≈ 12.6 MJ (3000 kcal). From this daily total energy expenditure value and the posttreatment

value for energy ingested per day, the estimated negative energy balance for the endurance group was ≈ 2.1 MJ (500 kcal) for each of the scheduled training days. Thus, although the ET subjects were in a negative energy balance for at least 4 of 7 d of the week as a result of their exercise training, there was not a subsequent decline in RMR as might be expected.

Our results as well as those from Bingham et al (17) and Tremblay et al (19) described above agree well but are in conflict with others (14, 15, 18). For example, in an earlier study by Tremblay et al (14), eight moderately obese women participated in 11 wk of aerobic exercise 5 h/wk at an exercise intensity of $\approx 50\%$ $\dot{V}O_2$ max. Subjects were instructed to maintain their preexperimental dietary intake practices. After training there was a significant increase in RMR, expressed in kJ/min or relative to FFW, of $\approx 8\%$. Although, no values were reported regarding the dietary intake of these subjects, a significant decline in both TWT and FW did occur, suggesting that a negative energy balance was most likely brought about by the exercise-training program. In a study by Lennon et al (15), a statistically significant increase in RMR (10.1%) expressed in mL · kg TWT⁻¹ · min⁻¹ was reported after training in subjects who had participated in a 12-wk jogging program. However, there was also a 10.7% decline in this group's TWT after the 12-wk of endurance training. This decline in TWT occurred primarily because of a loss in FW (26.8 to 18.2 kg, pre- to posttraining) with only a small change in FFW (63.5 to 62.4 kg, pre- to posttraining). Thus, by normalizing these data by using TWT, posttraining RMR values were actually overestimating metabolic size after training in this group. In fact, when RMR is expressed in kJ · kg FFW⁻¹ · h⁻¹, pre- to posttraining values were identical (5.023 kJ · kg FFW⁻¹ · h⁻¹). Finally, in a study by Poehlman et al (18), RMR was reported to decline by $\approx 8\%$ after 22 d of prolonged (116 min/d) cycle-ergometer training at 58% $\dot{V}O_2$ max in six pairs of monozygote twins.

The exact cause of the discrepancy among longitudinal studies investigating the effects of exercise training on RMR is unknown. Factors suggested as playing a role include 1) the method used to normalize the RMR data when differences in body-composition variables are found between comparison groups or between two different measurement periods in the same group of subjects, 2) differences in sample size and statistical power, 3) the timing of the RMR measurement in relationship to the last bout of

TABLE 6
Dietary analysis results for pre- and posttreatment*

Variable	Pretreatment			Posttreatment		
	Control (n = 16)	Resistance (n = 11)	Endurance (n = 15)	Control (n = 16)	Resistance (n = 11)	Endurance (n = 15)
Energy intake						
(MJ/d)	11.5 ± 0.9	10.9 ± 0.9	11.5 ± 0.8	10.1 ± 0.8	10.1 ± 0.8	10.6 ± 1.0
(MJ · kg FFW ⁻¹ · d ⁻¹)	0.178 ± 0.012	0.176 ± 0.016	0.180 ± 0.012	0.158 ± 0.013	0.158 ± 0.014	0.166 ± 0.014
(MJ · kg TWT ⁻¹ · d ⁻¹)	0.146 ± 0.013	0.140 ± 0.015	0.149 ± 0.012	0.130 ± 0.013	30.9 ± 11.4	33.1 ± 3.1
Carbohydrates (%)	43.6 ± 1.7	48.9 ± 2.0†	51.0 ± 1.7†	45.8 ± 2.1	47.2 ± 2.3	49.8 ± 1.9
Fat (%)	38.6 ± 1.3	33.5 ± 1.4†	32.9 ± 1.3†	34.9 ± 1.6	34.0 ± 1.8	31.7 ± 1.2
Protein (%)	17.8 ± 1.0	17.6 ± 1.5	16.1 ± 1.0	19.3 ± 1.4	18.8 ± 1.4	18.5 ± 1.4

* $\bar{x} \pm SE$.

† Significantly different from pretreatment control, $P < 0.05$.

exercise, 4) genetics-related factors; 5) measurement errors, 6) mode of exercise training (resistance training vs jogging), 7) intensity, duration, and frequency of exercise, 8) total training load difference between the pre- and posttraining measurements; and 9) initial fitness level of study participants.

In this study regardless of the method used to express RMR, there were no significant differences between the pre- and post-treatment values for each of the groups. Thus, the method used to express the RMR data cannot account for any differences between the present study and those studies previously reported. Interestingly, in the RT group, RMR in kJ/min increased by $\approx 3\%$ as did the total amount of FFW after the resistance-training program. Only the RT group showed a significant correlation between the changes in pre- to posttreatment values for FFW and RMR in L/min ($r = 0.64$, $P < 0.02$) (Figure 4). In contrast, the ET group's RMR in kJ/min increased 1.6% from pre- to posttraining but FFW increased only 0.5%. Only the RT group showed both a significant increase in FFW and significant decrease in FW after the 12 wk of training. Thus, these results suggest that resistance training may play an important role in maintaining or increasing metabolic rate when a person is in a state of negative energy balance.

In regards to sample size and statistical power, a power analysis was conducted according to the methods of Kraemer and Thiemann (25) before the study began. The minimum number of subjects necessary to find a statistically significant difference was determined to be eight subjects per group to give a statistical power level of 0.90 and $P < 0.05$. Thus, a lack of statistical power cannot account for why a significant difference in RMR was not found between pre- and posttreatment measurements for any of the three groups.

Another possible reason for the discrepancy in results between studies may be related to the timing of the RMR measurement in regards to the last exercise-training bout. RMR was measured 48 h after any strenuous physical activity in the pre- and post-treatment periods and was also measured in the endurance group within 14 h after a 50-min exercise bout during weeks 11 and 12 at an intensity of $\approx 82\%$ pretraining $\dot{V}O_2\max$ (14-h RMR). The results from these RMR trials in the ET group showed that the 14-h RMR was significantly higher than the pretraining RMR value expressed in kJ/min and $\text{kJ} \cdot \text{kg} \cdot \text{TWT}^{-1} \cdot \text{h}^{-1}$ (Figure 3). These results agree with studies suggesting that RMR may be elevated for periods of 14–18 h after exercise training (26, 27) but disagree with other studies (28, 29). If previous physical activity was not controlled for before the RMR measurement during both the pre- and posttreatment periods, the conclusion might have been drawn that endurance training could significantly elevate RMR whereas resistance training had little or no effect. Thus, it appears that controlling for previous physical activity, especially endurance activities, is critical when comparing the effects of exercise training on RMR in longitudinal studies.

Measurement errors may also account for differences in the results observed among the studies investigating the effects of exercise training on RMR. In the present study a minimum of two measurements for RMR and maximal exercise variables were made during both the pre- and posttreatment measurement periods. In addition, a strict set of criteria for accepting the two pre- and posttreatment trials was established for RMR and $\dot{V}O_2\max$. Using telemetric heart-rate monitoring of SHRs and RHRs provided additional confirmation of the time each subject

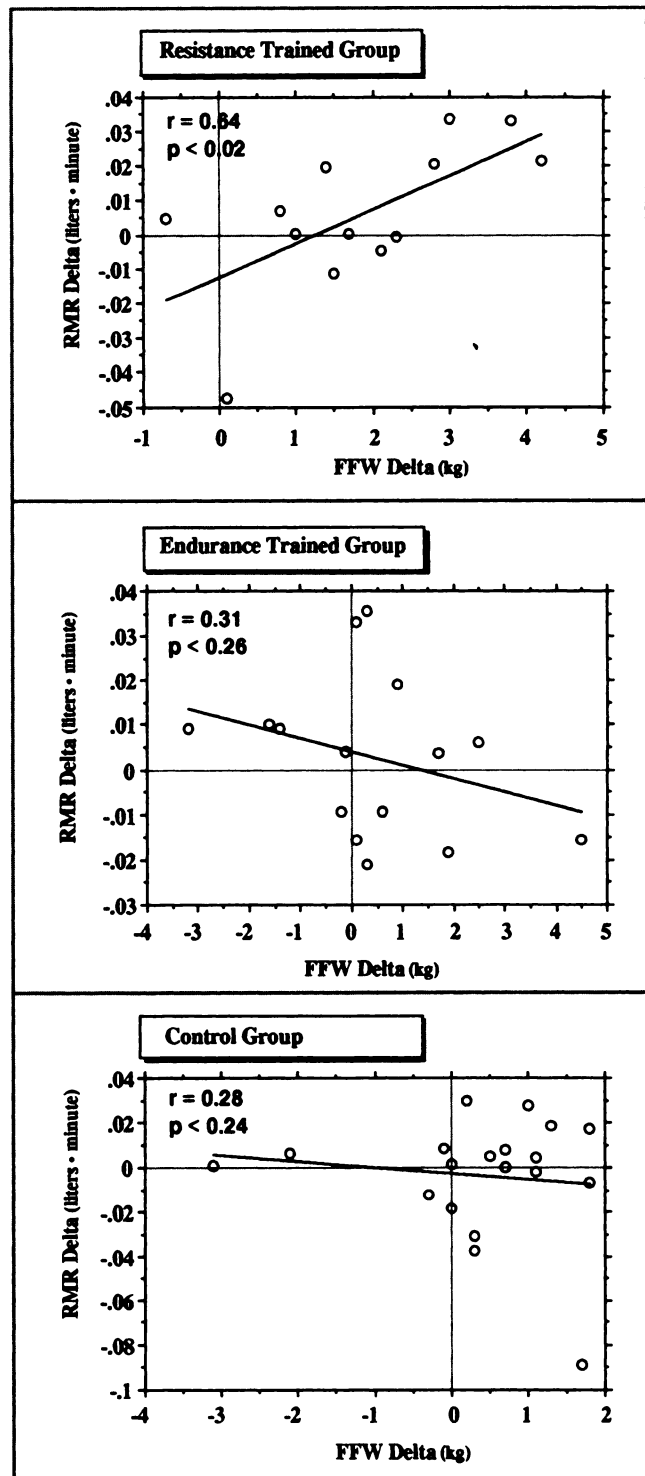



FIG 4. Changes in RMR vs FFW.

went to sleep on the night preceding the RMR measurement and the state of the subject at the time of the RMR measurement relative to his state during deep sleep. As a result, the differences for the two pretreatment trials for RMR, $\dot{V}O_2\max$, TMT, HRmax, $\dot{V}Emax$, RERmax, upper-body 1-RM, and lower-body 1-RM were $< 0.5\%$, 1.2%, 3.5%, $< 0.5\%$, 4.7%, $< 0.5\%$, 0.9%, and 4.1% respectively. The differences for the two posttreatment trials were 0.8%, $< 0.5\%$, 1.0%, 0.6%, 1.1, $< 0.5\%$, 0.5%, and

0.8%, respectively. Thus, it is unlikely that measurement error contributed to our not finding statistically significant differences in RMR after training.

Finally, differences in training mode, exercise intensity, duration, frequency, and total training load may also account for some of the discrepancies among studies. Both the RT and ET groups exhibited significant changes that strongly reflected each of their training programs. For example, a statistically significant increase in $\dot{V}O_2\text{max}$ was found only in the ET group whereas a statistically significant gain in strength was observed only in the RT group after the 12-wk training programs. In the ET group the mean exercise intensity between weeks 8 and 12 was $\approx 82\%$ $\dot{V}O_2\text{max}$ for 50 min/d, 4 d/wk. As a result, each endurance training bout was equivalent to ≈ 2.7 MJ/d (650 kcal/d) in the ET group, which is within the caloric expenditure range reported in previous studies (14–19). In the RT group between weeks 8 and 12, relative exercise-training intensity was $\approx 86\%$ of the overall pretraining mean for all 1-RM measurements. Because this is the first study investigating the effects of resistance training on RMR in weight-stable, nondieting individuals, further investigations will be needed before conclusions and study comparisons can be made regarding the effects resistance-training intensity, duration, and frequency may have on RMR.

In conclusion, after both forms of exercise training, RMR did not significantly change although a small decline in energy intake was observed along with an increase in energy expenditure [for the ET group, 2.7 MJ (650 kcal) per training day]. These results suggest that both endurance and resistance training may help to prevent an attenuation in RMR normally observed during extended periods of negative energy balance by either preserving or increasing FFW. Thus, for the person interested in reducing excessive relative body fat without a consequent attenuation in RMR during extended periods of negative energy balance, the best approach may be an exercise program that includes either or both endurance and resistance training. Because a significant correlation was found in the RT group between the change in FFW and this change in RMR, well-controlled studies are needed to determine the effects of a long-term resistance-training program (ie, studies lasting 6 mo–1 y) as well as the combined effects of a resistance- and endurance-training program (ie, endurance and resistance training 3 d/wk each) on RMR. 

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References

1. Woo R, Daniels-Kush R, Horton ES. Regulation of energy balance. *Annu Rev Nutr* 1985;5:411–33.
2. Sims EH. Storage and expenditure of energy in obesity and their implications for management. *Med Clin North Am* 1989;73:97–110.
3. Molé PA. Impact of energy intake and exercise on resting metabolic rate. *Sports Med* 1990;10:73–87.
4. Keeseey RE. Physiological regulation of body weight and the issue of obesity. *Med Clin North Am* 1989;73:15–27.
5. Poehlman ET, Melby CL, Bradylak SF. Resting metabolic rate and postprandial thermogenesis in highly trained and untrained males. *Am J Clin Nutr* 1988;47:793–8.
6. Poehlman ET, Melby LM, Badylak SF, Calles J. Aerobic fitness and resting energy expenditure in young adult males. *Metabolism* 1989;38:85–90.
7. Tremblay A, Fontaine E, Nadeau A. Contribution of postexercise increment in glucose storage to variations in glucose-induced thermogenesis in endurance athletes. *Can J Physiol Pharmacol* 1985;63:1165–9.
8. LeBlanc J, Diamond P, Côté J, Labrie A. Hormonal factors in reduced postprandial heat production of exercise-training subjects. *J Appl Physiol* 1984;3:772–6.
9. Hill JO, Heymsfield SB, McMannus C III, DiGirolamo M. Meal size and thermic response to food in male subjects as a function of maximum aerobic capacity. *Metabolism* 1984;33:743–9.
10. Davis JR, Tagliaferro AR, Kertzer R, Gerardo T, Nichols J, Wheeler J. Variations in dietary-induced thermogenesis and body fatness with aerobic capacity. *Eur J Appl Physiol* 1983;50:319–29.
11. Ravussin E, Borgardus C. Relationship of genetics, age, and physical fitness to daily energy expenditure and fuel utilization. *Am J Clin Nutr* 1989;49:968–75.
12. Bouchard C, Tremblay A, Nadeau JP, et al. Genetic effect in resting and exercise metabolic rates. *Metabolism* 1989;38:364–70.
13. Borgardus C, Lillioja S, Ravussin E, et al. Familial dependence of the resting metabolic rate. *N Engl J Med* 1986;315:96–100.
14. Tremblay A, Fontaine E, Poehlman ET, Mitchell D, Perron L, Bouchard C. The effect of exercise training on resting metabolic rate in lean and moderately obese individuals. *Int J Obes* 1986;10:511–7.
15. Lennon D, Nagle F, Stratman F, Shrago E, Dennis S. Diet and exercise training effects on resting metabolic rate. *Int J Obes* 1984;9:39–47.
16. Tagliaferro AR, Kertzer R, Davis JR, Janson C, Tse SK. Effects of exercise training on the thermic effect of food and body fatness of adult women. *Physiol Behav* 1986;38:702–10.
17. Bingham SA, Goldberg GR, Coward WA, Prentice AM, Cummings JH. The effect of exercise and improved physical fitness on basal metabolic rate. *Br J Nutr* 1989;61:155–73.
18. Poehlman ET, Tremblay A, Nadeau A, Dussault J, Thériault G, Bouchard C. Heredity and changes in hormones and metabolic rates with short-term training. *Am J Physiol* 1986;250:E711–7.
19. Tremblay A, Nadeau A, Després JP, St-Jean L, Thériault G, Bouchard C. Long-term exercise training with constant energy intake. 2: Effect on glucose metabolism and resting energy metabolism. *Int J Obes* 1990;14:75–84.
20. Behnke AR, Wilmore JH. Evaluation and regulation of body fluid and composition. Englewood Cliffs, NJ: Prentice-Hall, Inc, 1974.
21. Siri WE. Body composition from fluid spaces and density. In: Brozek J, Henschel A, eds. *Techniques for measuring body composition*. Washington, DC: National Academy of Sciences, 1961:223–44.
22. Schutte JE, Townsend EJ, Hugg J, Shoup RF, Malina RM, Blomqvist CG. Density of lean body mass is greater in blacks than whites. *J Appl Physiol* 1984;56:1647–9.
23. Wilmore JH, Vodak PA, Parr RB, Girandola RN, Billing JE. Further simplification of a methods for determination of residual lung volume. *Med Sci Sports Exerc* 1980;12:216–8.
24. Pollock ML, Wilmore JH. Exercise in health and disease: evaluation and prescription for prevention and rehabilitation. Philadelphia: WB Saunders Co, 1990.
25. Kraemer HC, Thiemann S. How many subjects? Statistical power analysis in research. Newbury Park, CA: SAGE Publications, Inc, 1987.
26. Bielski R, Schutz Y, Jéquier E. Energy metabolism during the postexercise recovery in man. *Am J Clin Nutr* 1985;42:69–82.
27. Tremblay A, Nadeau A, Fournier G, Bouchard C. Effect of a three-day interruption of exercise training on resting metabolic rate and glucose induced thermogenesis in trained individuals. *Int J Obes* 1988;12:163–8.
28. Freedman-Akabas S, Colt E, Kissileff HR, Pi-Sunyer X. Lack of sustained increase in $\dot{V}O_2$ following exercise in fit and unfit subjects. *Am J Clin Nutr* 1985;41:545–9.
29. Pacy PJ, Barton N, Webster J, Garrow JS. The energy cost of aerobic exercise in fed and fasted normal subjects. *Am J Clin Nutr* 1985;42:764–8.