Alterations in resting metabolic rate as a consequence of 20 wk of endurance training: the HERITAGE Family Study[1–3]

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ABSTRACT This study determined the effects of endurance exercise training on the resting metabolic rate (RMR). It was hypothesized that the RMR would be increased posttraining, but that this increase would reflect the influence of the last exercise bout, not a chronic adaptation to exercise training. Seventy-four subjects (40 men and 37 women) aged 17–63 y participated in a 20-wk endurance training program. RMR and maximal oxygen uptake ($\dot{V}O_2$max) were each measured on 2 separate days both pre- and posttraining; the posttraining RMR measurements were taken 24 and 72 h after the last exercise bout. There were small but significant changes posttraining in relative body fat (−1.0%), fat mass (−0.6 kg), and fat-free mass (0.7 kg) and a 17.9% increase in $\dot{V}O_2$max. The RMR remained unchanged posttraining, both 24 and 72 h after the last exercise bout, even when the data were adjusted to account for the potential confounding effects of age, sex, body composition, and $\dot{V}O_2$max. In conclusion, 20 wk of endurance exercise training had no effect on the RMR even in the presence of small changes in body composition and a large increase in $\dot{V}O_2$max. *Am J Clin Nutr* 1998; 68:66–71.

KEY WORDS Resting metabolic rate, RMR, endurance exercise training, resting heart rate, maximal oxygen uptake, body composition

INTRODUCTION

The resting metabolic rate (RMR) accounts for 60–75% of a normally active individual's total daily energy expenditure (1), although values < 60% have been reported. With the dramatic increase in the United States over the past decade in prevalence of overweight and obesity in adults (2), as well as in children and adolescents (3), there is increasing interest in interventions that increase the RMR. Both resistance and endurance exercise training have been proposed as potential interventions. It has been suggested that resistance exercise training could increase the RMR by increasing fat-free body mass, in recognition of the significant relation between fat-free mass and the RMR (4, 5). Endurance exercise training has been proposed to elevate the RMR through its possible effects on increasing the activity of thyroid hormones and the sympathetic nervous system and by increasing substrate flux and elevating protein synthesis (1).

The research literature is mixed regarding the effects of resistance and endurance training, separately or in combination, on elevating the RMR. There exist both cross-sectional (6–16) and longitudinal studies (8, 17–19) suggesting that exercise training, without dietary restriction, increases the RMR. However, there are also many cross-sectional (7, 20–22) and longitudinal studies (4, 23–30) in which changes in the RMR with endurance or resistance training in the absence of dietary restriction were not observed. The differences in the results of these studies appear to be independent of differences in sex, age, or training volume.

One possible explanation for these discrepant results could be the timing of the RMR measurement in relation to the time of the last exercise bout. For example, Tremblay et al (31) reported a 6.6% reduction in the RMR after a 3-d interruption in the training schedule of 8 highly trained male long-distance runners and cross-country skiers compared with an initial measurement obtained 16 h after their last training bout. Herring et al (32) reported similar findings in a group of highly trained female runners. They found that the RMR decreased 8% below baseline 39 h after the last exercise training bout and was maintained at this reduced level for up to 87 h postexercise. Finally, Broeder et al (4) found the RMR to be elevated 14 h after the last exercise bout after 12 wk of high-intensity aerobic training, but found that it returned to pretraining levels within 48 h of the last exercise bout. The results from these 3 studies suggest that any increase in the RMR as a consequence of exercise training is transitory, most likely lasting < 24 h. Thus, the effect of exercise on the RMR might be similar to the effect of exercise on insulin action

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and glycemia (33), in that most of the effect is in response to an acute bout of exercise and is only marginally associated with chronic adaptations to exercise training. This, in turn, could be the result of a high energy flux associated with a high energy intake, matching a high volume of exercise training to maintain energy balance, as suggested by the studies by Goran et al (34) and Bullough et al (35).

Thus, the purpose of the present study was to investigate the effects of a 20-wk, laboratory-monitored endurance exercise training program on the RMR in a large group of previously untrained men and women. To test the hypothesis that any change in the RMR is the direct result of the previous bout of exercise (ie, an acute effect) and not a chronic adaptation to exercise training, RMR measurements were obtained on 2 separate days pretraining to establish reliability and 24 and 72 h after the last exercise training bout. This study is part of a larger multicenter clinical trial, the HERITAGE Family Study, investigating possible genetic bases for variability in the response of physiologic measures and cardiovascular disease and diabetes risk factors to endurance exercise training. This study was described in detail in a previous publication (36).

SUBJECTS AND METHODS

Subjects

The HERITAGE Family Study subject population is composed of families, including the natural father and mother and ≥2 (African American families) or ≥3 (white families) offspring aged ≥17 y. Subjects for this study were recruited from the University of Texas at Austin HERITAGE Family Study Clinical Center because it was the only site involved in the RMR ancillary study. Whereas all subjects were recruited from families participating in the HERITAGE Family Study, not all members of the family necessarily participated in this ancillary study because participation was voluntary. Thus, familial analyses of these data were not possible. A total of 77 subjects participated in this RMR study, 37 women and 40 men aged from 17 to 63 y. Subject characteristics are presented in **Table 1**. Before they were accepted for participation in the HERITAGE Family Study, all subjects had to meet a set of inclusionary criteria, explained in detail in a previous publication (36), and pass a physician-administered physical examination that included taking a 12-lead electrocardiogram tracing during a maximal exercise test (36). All subjects were untrained before the start of the study. Because of financial constraints, it was not possible to include a nonexercising control group in the full HERITAGE Family Study design. The study protocol was approved previously by The University of Texas at Austin Institutional Review Board and informed consent was obtained from each subject.

Experimental design

Each subject completed an extensive battery of tests, including measurement of the RMR, before starting the 20-wk endurance training program (36). The training program was conducted on cycle ergometers (Universal Aerobicycle; Universal, Cedar Rapids, IA) interfaced with a computer system (Universal Gym Mednet) that controlled the power output of the ergometer to maintain a constant training heart rate. Use of this computer system in combination with an exercise leader who was present at each training session ensured precise subject compliance with the prescribed exercise program. Subjects started training at the heart rate equivalent of 55% of their maximal oxygen uptake ($\dot{V}O_2$max) for 30 min/d and gradually progressed to the heart rate equivalent of 75% of $\dot{V}O_2$max for 50 min/d at the end of 14 wk and maintained this intensity and duration throughout the remaining 6 wk. Frequency was maintained at 3 sessions/wk. Heart rate was plotted against $\dot{V}O_2$ for each submaximal power output during the initial graded exercise test to exhaustion to determine the heart rate equivalents of a given percentage of $\dot{V}O_2$max. Use of the heart rate equivalent of a set percentage of the subject's $\dot{V}O_2$max ensures a constant relative training intensity. As $\dot{V}O_2$max increases during the training program, it takes a higher absolute exercise intensity to attain a given heart rate response. Further details of the training program were published previously (36). At the end of the 20-wk training program, the subjects completed the posttraining battery of tests.

Methodology

The RMR was defined as the subject's metabolic rate in the early morning after a 12-h fast and a restful night's sleep in accordance with the suggestion of Berke et al (37). The pretest conditions were identical to those imposed on subjects before assessment of the basal metabolic rate, with the exception that the subjects slept at home and were transported to the laboratory in the early morning as soon as possible after awakening. RMR measurements were obtained on 2 visits before the start of the exercise training program on different days and 24 ± 2 and 72 ± 2 h after the last exercise bout of the 20-wk training program. All measurements were completed by 1000. All premenopausal women were tested during the follicular phase of their cycle for both pretraining measurement periods and all but 11 were tested during the follicular phase for both posttraining measurement periods. It was more difficult to schedule posttraining measurements to coincide with the follicular phase because these 2 measurement periods had to correspond with 24 and 72 h after the subject's final (60th) exercise training session. Subsequent analyses of these 11 women showed that their RMR training responses did not differ from those of the other women. Women taking hormone replacement therapy maintained the same regimen for the 2 pretraining and the 2 posttraining measurement periods.

Subjects maintained a record of all food intake for the 36-h period before the start of the first RMR test. They were then instructed to follow a similar dietary regimen before all subsequent tests, which included fasting for 12 h before the start of the measurement period. Only water was allowed ad libitum during this period. Subjects also refrained from all strenuous physical activity for 36 h before the 2 pretraining RMR tests. They were provided a heart rate monitor (Polar Vantage XL; Polar USA Inc, Montvale, NJ) and instructed on its use the afternoon or evening before each day of measurement. The heart rate monitor was worn

TABLE 1
Descriptive characteristics of the subject population[1]

Subjects	Age	Height	Weight
	y	*cm*	*kg*
Men (*n* = 40)	34.5 ± 15.0	177.3 ± 6.7	83.6 ± 15.0
Women (*n* = 37)	35.5 ± 14.6	163.1 ± 6.0	71.9 ± 15.3
All subjects (*n* = 77)	34.9 ± 14.7	170.4 ± 9.5	78.0 ± 16.1

[1] $\bar{x}$ ± SD.

from the time they went to bed the evening before the day of testing until the end of the RMR measurement period on the test day. Subjects were instructed to go to bed early and to try to get a normal night's sleep. They were instructed to limit their physical activity after awakening the morning of the test to include only grooming and slow movement. Bathing or showering was done the night before, before they attached the heart rate monitor. Subjects drove by car to the laboratory and were not allowed to walk > 100 m from their residence to the car or from the car to the laboratory. On arrival at the laboratory, they were allowed to void their bladder and were then placed in the measurement room and allowed to rest for 30 min in a recliner chair in a semirecumbent position. The room was isolated from any disturbing noise and was dimly lit and the room temperature was maintained between 22 and 24 °C.

A metabolic measurement cart (SensorMedics 2900; SensorMedics, Yorba Linda, CA) was used to measure the RMR with either a dilution mask (no. 803923-03; Scott-O-Vista Facepiece Assembly, SensorMedics) or a face mask (no. 7900; Hans Rudolph, Kansas City, MO). The type of mask used was kept constant for each subject. Furthermore, we found close agreement between these 2 masks when used on the same individuals (ie, within 20 mL/min). The 30-min period of rest was followed by a 30-min measurement period, after which the heart rate monitor was removed and the heart watch receiving unit was downloaded into a computer to obtain an analysis and printout of the previous night's sleeping heart rate (HR_{sleep}) and the heart rate obtained during the RMR measurement period (HR_{RMR}). The RMR and HR_{RMR} were defined as the mean value over the 30-min measurement period and HR_{sleep} was defined as the mean heart rate over 60 min when the heart rate was at its nadir during sleep.

Maximal exercise tests were conducted on a cycle ergometer (Ergo-Metrics 800S, SensorMedics) twice before and twice after the training program with a graded exercise test protocol. The results of these tests were used to establish the endurance training program work rates and to quantify the magnitude of the training response (36). $\dot{V}O_2$, the rate of carbon dioxide production ($\dot{V}CO_2$), and the respiratory exchange ratio (RER = $\dot{V}CO_2/\dot{V}O_2$) were determined by using the metabolic measurement cart; submaximal and maximal heart rates were determined from the electrocardiogram (SensorMedics). $\dot{V}O_2$max was defined as the peak $\dot{V}O_2$ obtained during the test, providing that the RER was > 1.10 and the subject was within 10 beats/min of his or her age-predicted maximum heart rate. The average of the 2 pretraining and the 2 posttraining tests was used to determine the relative (%) increase in $\dot{V}O_2$max as a consequence of training. When one of the pre- or posttraining tests did not meet the criteria for a maximal test, a single value was used.

Body composition was assessed by densitometry with hydrostatic weighing (38). Ten trials were obtained for each subject and the highest 3 underwater weights were averaged. Residual volume was determined by using the nitrogen dilution technique (39). Relative body fat was estimated from body density by the equation of Siri (40) for white men, Lohman (41) for white women, Schutte et al (42) for black men, and Ortiz et al (43) for black women. Fat mass and fat-free mass were obtained by the following equations:

$$\text{Fat mass} = (\text{mass} \times \text{relative fat})/100 \quad (1)$$

$$\text{Fat-free mass} = \text{mass} - \text{fat mass} \quad (2)$$

A resting blood sample was obtained early in the morning on a separate day after a 12-h fast. All samples were kept frozen at −21 °C until the end of the study, at which time they were to have been analyzed for concentrations of plasma thyroxine, free thyroxine, 3,3,3′-triiodothyronine, and free 3,3,3′-triiodothyronine. Because changes in the RMR were not observed posttraining, the decision was made to not analyze these samples for thyroid hormones.

Statistical analysis

All data were analyzed by using the SAS statistical package (version 6.12; SAS Institute Inc, Cary, NC). Except where noted, data are expressed as means ± SDs. Intraclass correlations were computed to estimate reliability by using the 2 pretraining trials (44). A multiple-testing analysis of variance (ANOVA) was implemented by using the general linear models procedure to assess whether there were differences across the 4 trials. Tukey's studentized range (honestly significant differences) test was used to determine between which trials there were significant differences. The multiple-testing ANOVA used controls for all potential sources of variation, such as age, sex, fat-free mass, and $\dot{V}O_2$max. Statistical significance was set at the 0.05 level.

RESULTS

Reliability across the 2 pretraining trials according to intraclass correlations was high: for resting oxygen consumption (in mL/min), $R = 0.91$; for energy expenditure (in kJ/d), $R = 0.94$; for HR_{sleep}, $R = 0.89$; for HR_{RMR}, $R = 0.90$; and for $\dot{V}O_2$max, $R = 0.97$. The intraclass correlation for the resting RER was significant, but considerably lower ($R = 0.50$). This was expected, however, because of the known small intersubject variability in resting RER values. There were small but significant changes in relative body fat, fat mass, and fat-free mass for the total sample, which reflected significant changes in the men but not the women (**Table 2**). Furthermore, the endurance training program resulted in an increase in $\dot{V}O_2$max of 17.9% (Table 2).

The RMR data across the 4 trials are presented in **Table 3**. There was no change in the RMR as a consequence of endurance training either 24 or 72 h posttraining, irrespective of whether values were expressed in mL/min, MJ/d, or $kJ \cdot kg$ fat-free $mass^{-1} \cdot d^{-1}$. Statistical adjustments for age, sex, fat-free mass, and pretraining $\dot{V}O_2$max did not alter these findings. The RER decreased slightly from pretraining trial 2 to the 24-h posttraining trial 3 (0.82 to 0.80), but then returned to pretraining values by 72 h posttraining. There was a small but significant decrease posttraining in both HR_{sleep} (from 61.0 to 59.3 beats/min) and HR_{RMR} (from 60.7 to 58.7 beats/min).

DISCUSSION

The purpose of the present study was to determine whether the RMR is altered after a 20-wk endurance training program. It was hypothesized that there would be an acute increase in the RMR as measured 24 h after the final exercise bout, but that this increase would dissipate over time and would not be evident at 72 h. The results of this study did not support this hypothesis: there was no evidence of an increase in the RMR at either test period posttraining. Use of the SAS general linear models procedure to control for differences in age, sex, fat-free mass, and pre-training $\dot{V}O_2$max did not change these results.

Although the small decreases in resting heart rate after training would suggest that the training program did not result in a significant training effect, the mean increase in $\dot{V}O_2$max of 17.9% supported a substantial cardiorespiratory training effect, com-

TABLE 2
Alterations in body weight, body composition, and maximal oxygen uptake ($\dot{V}O_2$max) as a consequence of a 20-wk endurance training program[1]

Variable	Pretraining	Posttraining
Body weight (kg)		
Men (n = 40)	83.6 ± 15.0	83.6 ± 15.1
Women (n = 37)	71.9 ± 15.3	71.9 ± 15.3
Total (n = 77)	78.0 ± 16.1	78.0 ± 16.2
Relative body fat (%)		
Men (n = 39)	24.0 ± 7.8	22.7 ± 8.1[2]
Women (n = 32)	35.4 ± 8.9	35.0 ± 8.8
Total (n = 71)	29.2 ± 9.9	28.2 ± 10.4[2]
Fat mass (kg)		
Men (n = 39)	20.8 ± 9.9	19.8 ± 10.1[2]
Women (n = 32)	26.0 ± 10.9	25.7 ± 10.9
Total (n = 71)	23.1 ± 10.6	22.5 ± 10.8[2]
Fat-free mass (kg)		
Men (n = 39)	62.4 ± 7.7	63.4 ± 7.6[2]
Women (n = 32)	44.5 ± 5.2	44.9 ± 5.4
Total (n = 71)	54.3 ± 11.2	55.0 ± 11.4[2]
$\dot{V}O_2$max (mL/min)		
Men (n = 40)	2946 ± 584	3446 ± 606
Women (n = 37)	1779 ± 362	2126 ± 363
Total (n = 77)	2385 ± 762	2812 ± 831
$\dot{V}O_2$max (mL · kg^{-1} · min^{-1})		
Men (n = 40)	36.3 ± 9.3	42.4 ± 10.0
Women (n = 37)	25.8 ± 7.6	30.8 ± 8.1
Total (n = 77)	31.3 ± 10.0	36.8 ± 10.8

[1] $\bar{x}$ ± SD.
[2] Significantly different from pretraining, $P < 0.05$.

parable with effects seen after other endurance training programs of similar durations and intensities (45). The subjects in this study cycled for 50 min each day at 75% of their initial $\dot{V}O_2$max during the last 6 wk of training. The lack of a substantial change in resting heart rate was reported previously by others and was addressed in a previous HERITAGE Family Study paper (46). The magnitude of the change in body composition also agrees with the results of previous studies in which similar amounts of training and training programs of similar duration were used (47).

The quality-control procedures instituted in the present study strongly support the veracity of the resulting data. Few investigators have implemented replication of the RMR assessment both pre- and posttraining. The high intraclass correlation for the RMR measured in both kJ/d and mL/min attests to the reproducibility of the data and agrees with the intraclass correlation (R = 0.92) reported in a previous study from this laboratory (4). In fact, replicate RMR measures in the present study (ie, trials 1 and 2 pretraining) were within 10 mL/min for 67.8% and 20 mL/min for 91.2% of the sample tested. The use of the heart rate monitor for the 8–10-h period of time before the measurement period was an important feature of the protocol to ensure true resting measurements. There was excellent agreement between the HR_{sleep} and the HR_{RMR} (r = 0.83), suggesting that the RMR measurement was obtained under true resting conditions. Furthermore, in a previous study, we found no significant difference between the RMR measured when subjects underwent an identical protocol to that used in the present study compared with a protocol in which subjects slept overnight at the measurement site (48). Thus, it is assumed that the values obtained in this study accurately represented the true RMR for our 4 measurement periods, ie, the 2 pretraining and the 2 posttraining measurements.

Although the results of the present study agree with those of previous longitudinal training studies (4, 23–30), other longitudinal studies showed an increase in the RMR with exercise training (8, 17–19). It is important to look closely at each of these longitudinal studies that have reported increases in the RMR with training to see whether it is possible to explain the differences in results compared with the present study. The mean age of the subjects in 3 of these studies ranged from 58 to 66 y (17–19), the total number of subjects per study ranged from 13 to 19, and 1 of the studies used only strength training, which resulted in an increase in fat-free mass of 1.6 kg. In the 2 studies by Poehlman et al (17, 18), the mode of training was stationary cycling 3 times/wk for 8 wk, which resulted in gains in the RMR and $\dot{V}O_2$max of 10% and 15%, respectively, in their first study and 7% and 11%, respectively, in their second study. In the fourth study, Tremblay et al (8) studied 8 moderately obese women (age not specified) who trained 5 h/wk for 11 wk through stationary cycling, jogging, swimming, and aerobic dance. In this study, the RMR increased by 8% but the change in $\dot{V}O_2$max was not reported.

Thus, when compared with the present study, the age of the

TABLE 3
Alterations in resting metabolic rate as a consequence of a 20-wk endurance training program[1]

	Pretraining			Posttraining		
Resting metabolic rate	Trial 1	Trial 2	Mean	Trial 3: 24 h	Trial 4: 72 h	Mean
($\dot{V}O_2$, mL/min)						
Men	253 ± 34	249 ± 32	252 ± 33	255 ± 34	253 ± 34	255 ± 33
Women	194 ± 29	196 ± 29	196 ± 31	198 ± 33	200 ± 32	199 ± 31
Total	226 ± 43	224 ± 42	225 ± 42	227 ± 44	228 ± 42	228 ± 42
(MJ/d)						
Men	7.32 ± 0.97	7.25 ± 0.94	7.31 ± 0.95	7.37 ± 0.98	7.35 ± 0.98	7.38 ± 0.97
Women	5.62 ± 0.83	5.69 ± 0.91	5.70 ± 0.89	5.74 ± 0.95	5.80 ± 0.92	5.78 ± 0.89
Total	6.56 ± 1.24	6.51 ± 1.21	6.54 ± 1.22	6.56 ± 1.26	6.61 ± 1.23	6.61 ± 1.23
(kJ · kg FFM^{-1} · d^{-1})						
Men	118.0 ± 11.7	117.6 ± 14.2	118.0 ± 12.6	117.1 ± 12.1	116.7 ± 14.6	117.2 ± 13.0
Women	125.9 ± 13.4	126.8 ± 14.2	127.2 ± 13.4	125.5 ± 13.4	128.9 ± 13.4	127.2 ± 12.1
Total	121.3 ± 13.0	121.3 ± 14.6	121.8 ± 13.8	121.3 ± 13.4	121.8 ± 15.1	121.8 ± 13.4

[1] $\bar{x}$ ± SD. n = 40 men, 37 women; however, data were not obtainable for 6 subjects for trial 1, 3 subjects for trial 2, 4 subjects for trial 3, and 1 subject for trial 4. There were no significant differences between pre- and posttraining. $\dot{V}O_2$, oxygen uptake; FFM, fat-free mass.

subjects, the training mode (endurance compared with strength), and the obese state of the subjects appear to be major distinguishing factors of these 4 studies that reported increased RMR with exercise training. In those previous studies that failed to find changes in the RMR or sleeping metabolic rate with training, several used strength training (4, 28, 29), 1 studied older subjects (26), and 1 studied moderately overweight subjects (25). Furthermore, statistical adjustments for age and body composition in the present study did not alter our findings.

It is important to acknowledge that high-volume endurance training over periods of years might lead to increases in the RMR. Tremblay et al (15) reported higher RMRs in endurance-trained athletes than in untrained control subjects, but this difference was removed by β-adrenergic blockade (propranolol, 80-mg single dose). This suggests that sympathetic nervous system activity is increased, or β-adrenergic receptors are up-regulated, in chronically trained endurance athletes. Moreover, Poehlman et al (17, 18) reported increased sympathetic nervous system activity in elderly subjects after 8 wk of endurance training, and this increase was associated with an increase in the RMR, suggesting that this response can also occur in the short term. Unfortunately, it was not possible to evaluate norepinephrine kinetics in the present study.

Therefore, we conclude that endurance exercise training of 20 wk duration had no effect on the RMR in men and women 17–63 y of age even when the data were adjusted to account for the potential confounding effects of age, sex, body composition, and pretraining $\dot{V}O_2$ max. This study is the largest of its kind, the only study that obtained duplicate RMR measures both pre- and post-training and was able to show no difference between HR_{sleep} and HR_{RMR}, and the only study in which all subjects performed exactly the same exercise prescription under carefully monitored conditions. The reasons for the disagreement between those studies that observed increases in RMR with exercise training and the present study, and others that observed no changes, are not immediately obvious.

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