Resting Metabolic Rate after Endurance Exercise Training

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ABSTRACT

LEE, M., D. A. SEDLOCK, M. G. FLYNN, and G. H. KAMIMORI. Resting Metabolic Rate after Endurance Exercise Training. *Med. Sci. Sports Exerc.*, Vol. 41, No. 7, pp. 1444–1451, 2009. **Purpose**: 1) To examine the effect of a 12-wk endurance exercise training program on RMR and 2) to provide insight into the mechanisms responsible for alterations in RMR that may occur after exercise training. **Methods**: Male participants (19–32 yr) in an exercise group (EX; $n = 9$) performed jogging and/or running 3–4 $d \cdot wk^{-1}$, 25–40 min per session, at 60%–80% $\dot{V}O_{2max}$, whereas subjects in a control group (CON; $n = 10$) maintained their normal activity patterns. Body composition, $\dot{V}O_{2max}$, RMR, epinephrine, norepinephrine, total thyroxine, free thyroxine, insulin, free fatty acids, and glucose were measured before and after the intervention. **Results**: Training resulted in a significant increase in $\dot{V}O_{2max}$ in EX (46.2 ± 1.2 to 51.0 ± 1.3 $mL \cdot kg^{-1} \cdot min^{-1}$, $P < 0.001$). Absolute and relative values for RMR did not significantly change in EX after training. Mean values for epinephrine, norepinephrine, total thyroxine, insulin, and glucose did not significantly change in either group; however, free thyroxine decreased significantly after training in EX ($P = 0.04$). Training also resulted in a significant increase in free fatty acid concentration in EX (0.37 ± 0.03 to 0.48 ± 0.04 $mmol \cdot L^{-1}$, $P < 0.001$). RMR in CON decreased significantly when expressed as an absolute value ($P < 0.01$) and relative to body weight ($P < 0.01$), fat-free mass ($P < 0.01$), and fat mass ($P = 0.04$). **Conclusions**: The mechanism for the decrease in CON is unknown, but it may be related to seasonal variations in RMR. Training may have prevented a similar decline in RMR in EX and may be related to a training-induced increase in fat oxidation. **Key Words**: ENERGY EXPENDITURE, AEROBIC EXERCISE, HORMONES, FREE FATTY ACID, GLUCOSE

RMR is the minimum energy required for maintenance of critical body functions, i.e., internal work, and represents the largest proportion of total daily energy expenditure. It is usually estimated by measuring energy expenditure at rest in a postabsorptive state. Because RMR accounts for 60%–70% of daily energy expenditure, any intervention or lifestyle behavior that chronically alters RMR may have important implications for energy balance and weight control (25).

Exercise training is a lifestyle behavior that has the potential to alter RMR. However, results of cross-sectional studies regarding RMR and exercise training/fitness status are equivocal. RMR has been reported as being higher (11,26), lower (3), or the same (5) in trained or highly fit persons than in untrained or low-fit individuals. Notwithstanding limitations of cross-sectional research, prospec-

tive studies also have produced inconsistent results. Researchers have reported increases ranging from 3% to 7.7% (4,14,18,21), no change (15,22,36), or an 8% decrease (31) in RMR with exercise training.

Part of the conflicting results of training effects on RMR may be because of discrepancies in methods among the studies. For example, intensity and duration of exercise training programs often vary between studies, as does the time elapsed between the last exercise session and the RMR measurement. Insufficient sample sizes and inconsistencies in the reported units of RMR are other possible reasons for the contradictory findings, i.e., some authors have reported RMR in absolute terms and/or relative to body weight (15,18,21,31), whereas others have also included RMR expressed relative to body composition (4,14,22,36). In addition, not all studies have included a control group (14). Lack of a control group could confound interpretation of the results because of seasonal variations in RMR, which reportedly explained as much as 17% of intraindividual variation in SMR (19).

Mechanisms responsible for a higher RMR in trained individuals remain to be elucidated; however, several have been suggested. They include, but are not limited to, alterations in thyroid hormone concentrations [i.e., thyroxine (T_4) and triiodothyronine (T_3)], which are major regulators of RMR (28), increased norepinephrine (NOR)-induced lipolysis (1), protein synthesis (17), glycogen

resynthesis (33), and/or increased muscle mass or fat-free weight (FFW) (4). Genetic variation in RMR is also known to account in part for discrepancies in RMR adaptations after an exercise program (2), which makes this issue more complex. Clearly, more research is needed regarding the interaction between exercise training and RMR.

The purpose of this investigation was to further examine the effects of an endurance exercise training intervention on RMR. The study was designed to minimize some of the aforementioned shortcomings, i.e., it was conducted in a longitudinal manner to eliminate intersubject variability associated with cross-sectional designs and used a control group to minimize intrasubject variability and seasonal variation. In addition, pertinent hormones and substrates were measured to provide insight into the mechanisms responsible for alterations in energy metabolism that may occur after exercise training.

METHODS

Subjects. Twenty apparently healthy, untrained male volunteers, aged 18–32 yr, were randomly assigned to one of two groups, i.e., exercise (EX; $n = 10$) or nonexercise control (CON; $n = 10$). All were nonsmokers, had no significant changes in body weight for 6 months before enrolling in the study, and were not previously engaged in any systematic exercise training. In addition, all subjects had to refrain from using any prescription and nonprescription drugs for 5 d preceding the testing. Each subject completed a medical history questionnaire at the start of the study, and those who met the criteria for inclusion and wished to participate were provided written consent. The study was approved by the Purdue University Institutional Review Board.

Experimental procedures. Subjects completed two sets of testing sessions, one before and one after a 12-wk exercise (or inactive control) intervention period. Pre-intervention testing (PRE) was performed in the early spring (~May), and postintervention testing (POST) was performed in late summer (~August). Each testing set consisted of two visits to the laboratory. Indices of body size (i.e., height and weight), body composition, and maximal oxygen consumption ($\dot{V}O_{2max}$) were measured during the first visit. The second visit was scheduled approximately 48 h later and consisted of the RMR measurement and blood sampling.

Subjects reported to the laboratory in a postabsorptive state wearing shorts and a tee shirt. Body weight (BW) and height were determined using a balance beam scale with an attached stadiometer. Body density (D_b) was determined by hydrostatic weighing performed in the shallow end of a swimming pool using a chair suspended from a Chatillon scale. Ten weighing trials were performed, and the average of three weights that were within 0.1 kg was used in the equation. Residual volume (RV) was measured using the closed-circuit oxygen dilution method of Wilmore et al.

(37). Relative body fat (BF) was determined from D_b using the equation of Siri (23), fat weight (FW) was calculated from BF and BW, and FFW was recorded as the difference between BW and FW. All body composition and RV measurements were performed by the same technician.

Subjects performed a continuous, graded, multistage treadmill exercise test to determine $\dot{V}O_{2max}$. The protocol for the test was developed on the basis of suggestions by Howley et al. (12) and included changes in both speed and grade. The test began with a warm-up at 3.5 mph and 0% grade for 5 min followed by a 1-min rest. For minutes 1 and 2, speed and grade were 4.0 mph and 0%, respectively. Speed and grade were increased to 4.3 mph and 2%, respectively, for minutes 3 and 4. After minute 4, speed and/or grade were increased every minute until minute 9 when speed was 5.0 mph and grade was 10%. Thereafter, grade remained constant and speed was increased by 0.3 mph every minute until termination of the test. $\dot{V}O_{2max}$ was assumed to have been achieved when two of the following three criteria were met: 1) <150-mL·min^{-1} increase in $\dot{V}O_2$ with an increase in work rate, 2) HR > 90% of age-predicted maximum, and 3) RER > 1.10.

Subjects were transported to the laboratory by motor vehicle at 0550 h after a restful night's sleep ($\geq$7 h), a 12-h fast, and refraining from vigorous physical activity for 48 h. After BW and height were measured, subjects inserted a rectal temperature (T_{re}) probe (YSI 400; Yellow Springs Instrument Co., Inc., Yellow Springs, OH) to a depth of 12–15 cm and were fitted with a telemetric HR monitor and a ventilated hood, and a catheter was inserted into an antecubital vein. A 20-min period of quiet rest in a supine position then preceded the ensuing RMR measurement, which began at ~0630 h. For the RMR measurement, $\dot{V}O_2$, RER, and pulmonary minute ventilation ($\dot{V}_E$) were monitored for 45 min. Ambient room temperature was maintained at ~24 $\pm$ 1°C, and the testing site was screened from other areas of the laboratory. The room was darkened, and noise was kept at a minimum during the testing. Subjects were instructed to remain awake but as quiet as possible before and throughout the entire RMR session.

$\dot{V}O_2$ and RER values measured during the last 30 min of the 45-min measurement period were averaged and used to calculate RMR. HR was recorded every 15 min, with the mean value reported as resting HR. T_{re} was recorded at the end of the RMR measurement. This was followed by collection of a 10-mL blood sample used for subsequent determination of epinephrine (EPI), NOR, total thyroxine (TT$_4$), free thyroxine (FT$_4$), insulin (INS), free fatty acids (FFA), and glucose (GLU). Insulin resistance was calculated from GLU and INS values using the homeostasis model assessment of insulin resistance (HOMA-IR) (16). Metabolic data were collected using a ventilated hood and a metabolic measurement system (Truemax 2400 Metabolic Measurement System; ParvoMedics, Salt Lake city, UT). The instrument was calibrated immediately before each test with room air and calibration gases of known concentration

(16% O_2 and 4% CO_2) guaranteed by the supplier to be within 0.1% of the stated values. $\dot{V}O_2$ was converted to energy expenditure (kcal) using the Weir equation (10): kilocalories (kcal) = [(1.1 × RER) + 3.9] × $\dot{V}O_2$. Kilocalories were converted into kilojoules (kJ), where 1 kcal = 4.184 kJ. RMR was expressed on an absolute basis (kJ·min^{-1}) and relative to body weight (kJ·kg^{-1} BW·h^{-1}), fat-free weight (kJ·kg^{-1} FFW·h^{-1}), and fat weight (kJ·kg^{-1} FW·h^{-1}).

Exercise training program. Subjects assigned to EX completed a 12-wk endurance exercise training program, which consisted of jogging and/or running. In addition to the prescribed exercise, each training session included a 10-min warm-up and a 5- to 10-min cool-down. Initially, frequency, intensity, and duration were three times per week at an intensity calculated to elicit 60% $\dot{V}O_{2max}$ for 25 min per session. Training was gradually increased, so that by the 12th wk, subjects were exercising four times per week at 80% $\dot{V}O_{2max}$ for 40 min per session. All training sessions were supervised by of one of the investigators, and compliance was 100%. Subjects in CON were asked to maintain their normal activity patterns.

Blood collection and storage. Five milliliters of blood was collected in a tube containing sodium heparin and 100 μL of glutathione and EGTA (ethyleneglycerol-bis-(β-aminoethylether) *N*, *N*, *N*, *N*-tetraacetic acid) for subsequent determination of catecholamines (NOR and EPI). Additional blood samples were collected in 7-mL plain tubes for subsequent determination of TT_4, FT_4, INS, FFA, and GLU. After being allowed a minimum of 30 min to clot in an ice bath, samples were centrifuged at 4°C and 960*g* for 15 min then stored at −80°C for subsequent analyses.

Blood analysis. Isolation of catecholamines from the prepared plasma sample was accomplished by alumina extraction using a Chromsystems reagent kit (Alko Diagnostics, Holliston, MA). After extraction, concentrations of EPI and NOR were quantified by high-performance liquid chromatography. Intra-assay coefficient of variation (CV) was less than 1%, interassay CV was less than 3%, the correlation coefficient from 9 to 1000 pg was 0.9989, and sensitivity was 5 pg with a signal-to-noise ratio of 5:1. TT_4, FT_4, and INS were determined using a solid-phase radioimmunoassay (Coat-A-Count; Diagnostic Products Co., Los Angeles, CA). FFA was determined using an enzymatic method (Wako, Richmond, VA). GLU was also determined using an enzymatic method (Sigma Diagnostics, St. Louis, MO). Intra-assay CV for TT_4, FT_4, FFA, INS, and GLU were 2.1%, 1.9%, 1.4%, 1.0%, and 2.0%, respectively. Interassay CV for TT_4, FT_4, FFA, INS, and GLU were 4.2%, 5.6%, 0.7%, 5.4%, and 1.5%, respectively.

Statistical analysis. A power analysis indicated that eight subjects were necessary to find a 10% change in RMR with 90% power at α = 0.05. One subject in EX withdrew from the study because of personal matters after completing the training program but none of the postintervention testing. Therefore, data from 9 subjects in EX and 10 subjects in CON were used in the final analyses.

Data were analyzed using the Statistical Analysis System (SAS) for Windows V8. All data are reported as mean ± SEM. ANOVA using a split–plot design was used to test for differences between EX and CON (group) and between PRE and POST tests (test). *Post hoc* multiple comparisons using Bonferroni's adjustments were performed where appropriate. The relationship between RMR and $\dot{V}O_{2max}$ was analyzed using the correlation analysis, and the Pearson's *r* was calculated. Statistical significance was accepted for all tests at $P < 0.05$.

RESULTS

Physical characteristics. Physical characteristics of subjects and changes in variables related to body composition and aerobic capacity after the intervention are shown in Table 1. There were no significant differences in any of the pretest variables between EX and CON. After the intervention, significant ($P < 0.001$) decreases were seen in BF and FW in EX, whereas CON showed no significant changes. This resulted in a significant ($P < 0.001$) interaction effect. Although not statistically significant, there was a trend for BW to decrease ($P = 0.06$) and FFW to increase ($P = 0.08$) in EX. Training resulted in a significant increase ($P < 0.001$) in $\dot{V}O_{2max}$ in EX when expressed both in absolute and relative terms. $\dot{V}O_{2max}$ expressed as liters per minute, milliliters per kilogram per minute, and milliliters per kilogram FFW per minute increased by 9.4%, 10.3%, and 8.9%, respectively. There was no significant change in $\dot{V}O_{2max}$ in CON, resulting in a significant interaction effect ($P < 0.001$).

RMR. Preintervention and postintervention RMR values for EX and CON are shown in Table 2. Absolute values for RMR remained unaltered in EX after training, but decreased by 4.5% in the CON ($P < 0.01$). Results were similar when RMR was expressed relative to BW ($P < 0.01$), FFW ($P < 0.01$), and FW ($P = 0.04$), i.e., a significant decrease in CON, and no significant change in EX. T_{re} was also significantly lower in CON after the intervention. Because both RMR and T_{re} decreased in CON, we correlated changes in RMR with changes in T_{re} in CON. No significant relationship was found ($r = -0.08$, $P > 0.05$). There was a significant interaction effect for resting HR ($P < 0.01$). HR in EX after training was significantly lower when compared with EX before training ($P < 0.001$) and CON both before ($P = 0.02$) and after ($P < 0.01$) the intervention period. Resting RER in EX tended to be lower from PRE to POST ($P = 0.07$), although the change was not statistically significant.

The relationship between RMR and $\dot{V}O_{2max}$ was examined using data for 19 subjects during PRE when they were untrained. Significant positive correlations were found

TABLE 1. Physical characteristics, body composition, and aerobic capacity in the exercise training (EX) and control (CON) groups before (PRE) and after (POST) a 12-wk intervention.

Variables	Group	Test PRE	Test POST	Group × Test Interaction (P)
Age (yr)	EX	26.2 ± 1.4		
	CON	26.2 ± 0.9		
Height (cm)	EX	174.8 ± 1.2		
	CON	174.8 ± 1.7		
BW (kg)	EX	73.8 ± 2.1	73.3 ± 2.0	NS
	CON	71.2 ± 2.7	71.3 ± 2.6	
BF (%)	EX	16.4 ± 1.7	15.2 ± 1.8*	<0.001
	CON	17.7 ± 1.1	18.1 ± 1.0	
FW (kg)	EX	12.1 ± 1.4	11.2 ± 1.4*	<0.001
	CON	12.8 ± 1.2	13.1 ± 1.1	
FFW (kg)	EX	61.7 ± 2.0	62.1 ± 2.0	NS
	CON	58.4 ± 1.7	58.2 ± 1.7	
$\dot{V}O_{2max}$ (L·min^{-1})	EX	3.41 ± 0.11	3.73 ± 0.11†	<0.0001
	CON	3.20 ± 0.12	3.16 ± 0.12	
$\dot{V}O_{2max}$ (mL·kg^{-1} BW·min^{-1})	EX	46.2 ± 1.2	51.0 ± 1.3†	<0.0001
	CON	45.1 ± 1.4	44.5 ± 1.3	
$\dot{V}O_{2max}$ (mL·kg^{-1} FFW·min^{-1})	EX	55.3 ± 1.1	60.2 ± 0.9†	<0.0001
	CON	54.8 ± 1.3	54.3 ± 1.2	

Values are mean ± SEM. $n = 9$ in EX. $n = 10$ in CON.
* Significantly different from EX PRE ($P < 0.001$).
† Significantly different from EX PRE, CON PRE, and CON POST ($P < 0.001$).
BF, percent body fat; BW, body weight; FW, fat weight; FFW, fat-free weight; NS, not significant.

between RMR and $\dot{V}O_{2max}$ when data were expressed relative to BW ($r = 0.646$, $P < 0.01$) and FFW ($r = 0.459$, $P < 0.05$). Data are shown in Figures 1 and 2, respectively.

Blood variables. Results of the blood analyses are presented in Table 3. There were no significant group differences before the intervention. Mean values for EPI and NOR did not significantly change in either group. No significant change was found in TT_4; however, FT_4 decreased significantly after training in EX ($P = 0.04$). Training resulted in a significant increase in resting FFA in EX ($P < 0.001$), whereas values remained relatively unchanged in CON. No significant changes were found in resting INS and GLU concentrations, although some trends were noted in the data. Resting INS decreased by 34.2% ($P = 0.07$) and resting GLU declined by 8.6% ($P = 0.06$) in EX. Although INS also decreased by 35.2% in CON

($P = 0.09$), there was no change in GLU. No significant group effect or interaction was found for HOMA-IR; however, there was a significant decrease from preintervention to postintervention ($P < 0.001$).

DISCUSSION

Physical characteristics. A group of 19 healthy, young, and untrained male subjects participated in this study. There were no significant differences between EX ($n = 9$) and CON ($n = 10$) about age, height, BW, BF, FFW, FW, and $\dot{V}O_{2max}$ before the intervention, indicating that the groups were well matched for body composition and aerobic capacity. This is an important consideration because metabolic rate is known to be related to body composition. Cunningham (7) reported that FFW could account for

TABLE 2. RMR, HR, and rectal temperature (T_{re}) in the exercise training (EX) and control (CON) groups before (PRE) and after (POST) a 12-wk intervention.

Variables	Group	Test PRE	Test POST	Group × Test Interaction (P)
RMR (L·min^{-1})	EX	0.22 ± 0.01	0.22 ± 0.01	0.042
	CON	0.22 ± 0.01	0.21 ± 0.01*	
RMR (mL·kg^{-1}·min^{-1})	EX	3.03 ± 0.08	3.04 ± 0.08	0.028
	CON	3.12 ± 0.11	2.94 ± 0.06*	
RMR (kcal·min^{-1})	EX	1.10 ± 0.03	1.09 ± 0.04	NS
	CON	1.09 ± 0.04	1.03 ± 0.03*	
RMR (kJ·min^{-1})	EX	4.58 ± 0.13	4.55 ± 0.16	NS
	CON	4.56 ± 0.16	4.31 ± 0.14*	
RMR (kJ·kg^{-1}·h^{-1})	EX	3.73 ± 0.09	3.73 ± 0.09	NS
	CON	3.87 ± 0.13	3.64 ± 0.08*	
RMR (kJ·kg^{-1} FFW·h^{-1})	>EX	4.47 ± 0.11	4.41 ± 0.12	NS
	CON	4.69 ± 0.11	4.44 ± 0.06*	
RMR (kJ·kg^{-1} FW·h^{-1})	EX	25.53 ± 3.38	28.16 ± 4.12	0.019
	CON	22.66 ± 2.63	20.89 ± 1.70*	
HR (beats·min^{-1})	EX	56.1 ± 1.8	51.2 ± 1.9†	0.002
	CON	56.8 ± 1.0	57.7 ± 1.3	
T_{re} (°C)	EX	35.94 ± 0.07	35.90 ± 0.05	NS
	CON	36.10 ± 0.04	35.94 ± 0.04*	
RER	EX	0.90 ± 0.02	0.87 ± 0.02	NS
	CON	0.90 ± 0.01	0.90 ± 0.01	

Values are mean ± SEM. $n = 9$ in EX. $n = 10$ in CON.
* CON POST: significantly different from CON PRE ($P < 0.01$).
† EX POST: significantly different from EX PRE, CON PRE, and CON POST ($P < 0.01$).
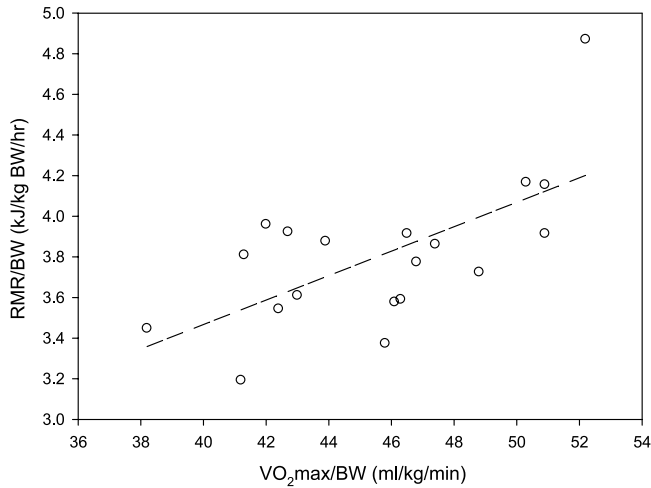
FIGURE 1—Correlation between maximal oxygen consumption per body weight and RMR per body weight at pretest (PRE; $r = 0.646$, $P = 0.003$).

60%–90% of RMR, and Smith et al. (24) found that FFW was a significant predictor of RMR and accounted for most of the differences in RMR between subjects of varying $\dot{V}O_{2max}$. The similarity in body composition between EX and CON in this study suggests that this variable can be excluded as potentially confounding interpretation of the results.

In the present study, a significant positive correlation was found between $\dot{V}O_{2max}$ and RMR at PRE, especially when the variables were expressed in the same relative units (Figs. 1 and 2). This relationship emerged despite the subjects' lack of systematic exercise training on entry into the study. This finding indicates that cross-sectional studies examining the association between RMR and training and/or fitness level are limited in the ability to clarify this relationship because it is not possible to determine whether a higher RMR is related to a genetically higher $\dot{V}O_{2max}$ or if RMR is higher because of an increase in $\dot{V}O_{2max}$ from training. The significant relationship between $\dot{V}O_{2max}$ and RMR found in the untrained individuals in this study implies that $\dot{V}O_{2max}$ *per se* rather than training state is linked to RMR.

Significant decreases were seen in BF and FW in EX after training. Although not statistically significant, there was a trend for BW ($P = 0.06$) to decrease and FFW to increase ($P = 0.08$) in EX. These changes in body composition agree with results from previous endurance exercise training studies (18,36). Although it is well accepted that endurance exercise training decreases FW, effects of exercise training on FFW are inconsistent. Several researchers reported a significant increase in FFW from endurance exercise training (21), whereas others report no change (31).

The endurance training program used in the present study effectively increased aerobic fitness of EX. The significant increase in $\dot{V}O_{2max}$ of EX after training is consistent with

observed changes in $\dot{V}O_{2max}$ after other endurance training programs of similar duration and intensity (8) and support a substantial cardiorespiratory training effect.

RMR. Contrary to our hypothesis that RMR would increase as a result of endurance training, neither absolute nor relative (to BW and FFW) values for RMR were altered in EX. In addition, there was no significant change in T_{re} in EX. In contrast, RMR and T_{re} significantly decreased in CON; however, these changes were not significantly related.

If our study design did not include a control group, as in a previous study (14), the logical conclusion would be that training had no effect on RMR. Similar results were reported for RMR when exercise training was combined with energy restriction, although energy intake was not restricted in the current study. Van Dale et al. (34) reported that diet groups showed a significant decline in RMR, whereas exercise training groups showed either no change or a smaller decline in RMR, suggesting that a decline in RMR from caloric restriction could be offset by exercise training. Our results are consistent with several longitudinal studies in which no effects of exercise training on RMR were found (15,36) but inconsistent with other longitudinal studies in which RMR significantly increased (4,14,18,21,31). There are several possible reasons for the discrepancy between our results and results of others who reported a significant increase in RMR. These include elapsed time between the last exercise session and the measurement of RMR, variability associated with indirect calorimetry measurements, insufficient sample size, and inconsistency in the units of expressing RMR. In addition, differences in age and initial fitness level of subjects, as well as training mode may be other reasons for the contradictory findings.

RMR has been reported to remain elevated for a period of 14–18 h after aerobic exercise training (30). In the present study, as well as in other studies where RMR significantly
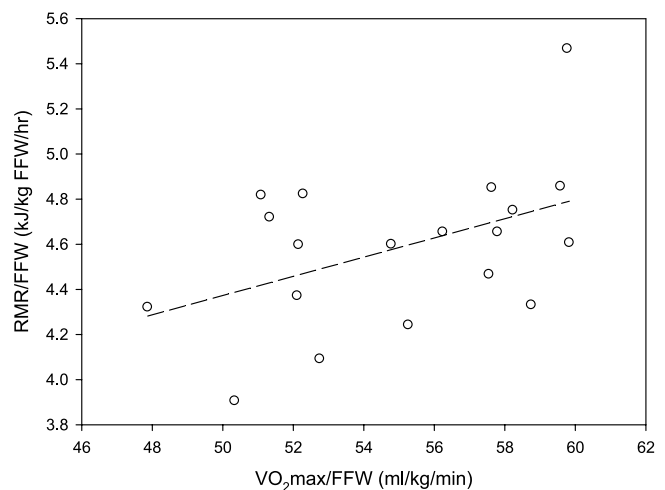

FIGURE 2—Correlation between maximal oxygen consumption per fat-free weight and RMR per fat-free weight at pretest (PRE; $r = 0.459$, $P = 0.048$).

TABLE 3. Blood analytes and nutrients in the exercise training (EX) and control (CON) groups before (PRE) and after (POST) a 12-wk intervention.

Variables	Group	Test PRE	Test POST	Group × Test Interaction (P)
EPI (pg·mL^{-1})	EX	41.0 ± 12.3	23.9 ± 1.9	NS
	CON	29.7 ± 4.4	18.7 ± 1.6	
NOR (pg·mL^{-1})	EX	196.3 ± 35.9	202.3 ± 19.0	NS
	CON	165.0 ± 19.2	129.4 ± 9.8	
TT$_4$ (nmol·L^{-1})	EX	87.1 ± 2.7	83.1 ± 2.3	NS
	CON	85.3 ± 2.8	89.2 ± 4.2	
FT$_4$ (pmol·L^{-1})	EX	15.5 ± 0.6	13.8 ± 0.5*	NS
	CON	15.3 ± 0.7	15.5 ± 0.4	
FFA (mmol·L^{-1})	EX	0.37 ± 0.03	0.48 ± 0.04†	0.049
	CON	0.40 ± 0.04	0.41 ± 0.04	
INS (μIU·mL^{-1})	EX	7.9 ± 0.8	5.2 ± 0.9	NS
	CON	8.8 ± 0.8	5.7 ± 0.4	
GLU (mmol·L^{-1})	EX	5.5 ± 0.2	5.0 ± 0.1	NS
	CON	5.4 ± 0.1	5.4 ± 0.1	
HOMA-IR‡	EX	1.95 ± 0.24	1.18 ± 0.22	NS
	CON	2.12 ± 0.21	1.38 ± 0.10	

Values are mean $\pm$ SEM. $n = 9$ in EX. $n = 10$ in CON.
* Significantly different between EX PRE and EX POST ($P < 0.05$).
† Significantly different between EX PRE and EX POST ($P < 0.001$).
‡ Significantly different between PRE and POST ($P < 0.001$).

increased after training (20,29), measurements were made at least 24 h after the last exercise bout. Therefore, this factor is not likely to account for differences between results of the present study and previous studies showing an increase in RMR. In addition, it is unlikely that variability associated with indirect calorimetry measurements contributed to the inconsistent findings because either a low coefficient of variation (<4.3%) or a high correlation coefficient ($r > 0.88$) for reliability in RMR measurements was reported in those studies. Adequately high reliability for RMR measurements in the present study was confirmed in a pilot study where the intraclass correlation coefficient for RMR test–retest reliability in five subjects was $r = 0.98$ (unpublished data). The same technician, instruments, and protocol were used for the pilot test and during data collection for the present study. Insufficient sample size does not seem to be responsible for the contradictory findings, i.e., a power analysis indicated that eight subjects were necessary to find a statistically significant difference in RMR with 90% power. Regarding inconsistency in the units of expressing RMR, we found no significant differences between pre-training and posttraining RMR values in EX regardless of whether RMR was expressed in absolute terms, relative to BW, or relative to body composition (i.e., FFW or FW). Therefore, the units of expressing RMR do not seem to account for any discrepant findings.

Several other possible reasons for the discrepant results exist in addition to the four listed above. First, young male subjects (~26 yr) participated in the present study. Some studies reporting a training-induced increase in RMR were conducted with the elderly (18,21) with mean values for age ranging from 57 to 69 yr. Second, it is possible that training will impact RMR when aerobic capacity is either relatively low or relatively high but not when it is in a more moderate range such as in the subjects of the present study. This interpretation is supported by studies showing an increase in RMR after training when mean values for $\dot{V}O_{2max}$ varied

from 23 to 29 mL·kg^{-1}·min^{-1} (18,21) and by a cross-sectional study (5) in which no significant difference in RMR between aerobically trained and control subjects was found, but there was a trend for a higher RMR in highly trained subjects when they were grouped by intensity of training, i.e., highly trained, moderately trained, and un-trained. Finally, although not likely, differences in training mode may account for the discrepant findings between the present study and previous studies. Whereas the aerobic exercise mode in this study was treadmill jogging and running, previous studies reporting training-induced increases in RMR used cycling, jogging, and swimming as their modes of aerobic training. Resistive exercise training has also been shown to impact RMR (14,21).

The reason for the significant decline in RMR of CON is unclear, but the most likely explanation may be related to seasonal variations in RMR. RMR was reported as being highest (4.85 ± 0.55 kJ·min^{-1}) in winter ($6 \pm 3°$C) and lowest (4.56 ± 0.53 kJ·min^{-1}) in summer ($21 \pm 3°$C), indicating a distinct seasonal variation in RMR (19). In the present study, preintervention testing was performed in May (approximately 15°C) as the cool weather season was ending, and postintervention testing was performed in August (approximately 30°C), which is normally the hottest month of the year. It is possible that the difference in environmental temperature between the two testing periods will account for the ~6% decrease in RMR of CON. When the decrease in RMR in CON is considered along with the lack of an increase in RMR in EX, it could be suggested that a decline in RMR in EX was prevented by endurance training.

Biochemical variables. Resting FFA concentration significantly increased in EX after training, suggesting that training may have increased FFA oxidation. This is supported by Kiens (13) who reported that plasma FFA concentration correlated fairly well with the rate of FFA turnover and oxidation. Friedlander et al. (9) found that training increased FFA oxidation by increasing both the

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appearance and disappearance rate of FFA. Further support for an increase in FFA oxidation in our EX subjects is evidenced by the trend ($P = 0.07$) toward a decline in their RER after training. Neither INS nor catecholamines (i.e., NOR and EPI) mediated the change in FFA concentration because they did not change in response to training. Other possible mechanisms suggested for a training-induced increase in FFA oxidation are increased number and/or activity of plasma membrane fatty acid binding protein in muscle (32), improved delivery of fatty acids to the interstitial space by a training-induced increase in muscle capillarization (35), and decreased inhibitory effect of malonyl CoA on carnitine acyltransferase I (13), an enzyme that assists in the transport of fatty acids through mitochondrial inner membrane by converting acyl CoA to acyl carnitine.

Significant increases in both NOR and RMR have been reported after exercise training (20). This is in contrast with results of the present study in which no significant training-induced changes were observed in RMR, EPI, and NOR. It should be noted that the previous study was performed using elderly subjects. Other possible reasons for the discrepancy between the aforementioned study and the present study are unclear; however, it may be partially explained by the fact that plasma concentrations of EPI and NOR vary widely and their secretions are extremely labile. For example, resting concentrations of EPI averaged 160 $pmol \cdot L^{-1}$ and ranged from less than 50 $pmol \cdot L^{-1}$ to 620 $pmol \cdot L^{-1}$ (6). Inter-subject variations of plasma EPI and NOR were also found to be fairly large in the present study (Table 3).

No significant change was found in TT_4, whereas FT_4 decreased in EX after training in the present study. Tremblay et al. (31) reported significant decreases in thyroid hormones (total and free T_3 and TT_4) after a cycle ergometer training intervention. However, they also observed a significant reduction in RMR. The decrease in FT_4 observed in EX of the present study did not seem to affect RMR. It could be that the concentration of serum TT_4 or FT_4 may not represent the activity of the thyroid hormones.

For example, T_4 secretion increased during the initial stage of exercise training; however, the rate of T_4 breakdown increased at a greater rate as training continued, leading to a reduction in the concentration of circulating T_4 (27). It seems that the concentration as well as appearance and disappearance rates of the thyroid hormones should be determined to more clearly elucidate the relationship between thyroid hormones and RMR in response to exercise training.

There was a trend for resting INS (34.2%, $P = 0.07$) and GLU (8.6%, $P = 0.06$) concentrations to be lower in EX after training. INS in CON also tended to be lower (35.2%, $P = 0.09$) but with no concomitant change in resting GLU (Table 3). Although the subjects were not considered to be insulin-resistant on entry into the study, these data suggest that insulin resistance in EX may have been reduced after training. However, results of the HOMA-IR analysis do not support a training-induced change in insulin resistance because HOMA-IR was reduced in both groups after the intervention period. The significantly lower postintervention HOMA-IR values were likely a result of the lower INS concentrations. It is unclear why INS concentration tended to be lower in both groups after training.

CONCLUSION

No significant change in RMR was found in EX, whereas RMR decreased significantly in CON after a 12-wk intervention period. Thus, it was suggested that training may have prevented a decline in RMR. This preventive effect may have been related to a significant training-induced increase in fat oxidation that was not explained by INS, EPI, or NOR because there were no significant changes in these variables. The mechanism for the decreased RMR in CON is unknown, but it may be related to seasonal variations in RMR.

Results of the present study do not constitute endorsement by ACSM.

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