Resting metabolic rate and coronary-heart-disease risk factors in aerobically and resistance-trained women[1-3]

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ABSTRACT This cross-sectional study compared physical characteristics, cardiovascular risk factors, and resting metabolic rate (RMR) in a cohort of 82 young women separated into three groups: sedentary (SED, n = 48), aerobically trained (AT, n = 21), and resistance trained (RT, n = 13). Body mass and fat-free mass (FFM) were not different between groups whereas percent body fat was lower in the AT (16.2 ± 0.7%) and RT (14.7 ± 0.8%) groups than in the SED group (21.8 ± 0.8%). There were no between-group differences for blood pressure or blood lipids. RMRs (kJ/min) for the AT (4.31 ± 0.06) and RT (4.25 ± 0.09) groups were significantly greater than those for the SED group (3.99 ± 0.05). When adjusted for differences in FFM, RMRs for the AT group (4.24 ± 0.05) were different from those of both the RT (4.13 ± 0.05) and SED (4.05 ± 0.03) groups; RMRs for the RT and SED groups were not different from each other. No differences were found in cardiovascular risk in young nonobese women of differing exercise status. Aerobic training in young women seems to increase the rate of metabolic activity of resting tissues whereas resistance training does not. *Am J Clin Nutr* 1992;56:968–74.

KEY WORDS Resting metabolic rate, thyroid hormone, insulin, exercise, cardiovascular risk factors, weight training, body composition, diet, women

Introduction

Exercise training that stresses the cardiorespiratory system (ie, aerobic exercise) is thought to reduce cardiovascular risk in part by eliciting reductions in blood pressure and by improving insulin sensitivity and blood lipid profiles (1–3). Many of these adaptations are mediated, in part, by exercise-induced reductions in body fat. Because resting metabolic rate (RMR) is increased (although less consistently in women than in men) with aerobic-exercise training (see 4 for review), this reduction in body fat may result, in part, from an increase in RMR (the largest component of daily energy expenditure).

Resistance (ie, weight) training has sometimes (5–8) but not always (9) been shown to reduce cardiovascular risk in men. Thus, it is beginning to be recognized that resistance-training exercise may provide an alternative exercise mode by which one may reduce cardiovascular risk. In light of this, we recently completed a cross-sectional study (10) in a cohort of 96 men that showed that men who regularly participate in either aerobic-type or resistance-training exercise have both a lower percent body fat and a higher RMR than do sedentary individuals. In addition, there was a modest improvement in cardiovascular risk in both exercise-trained groups compared with a sedentary control group. Therefore, it would seem that at least in young men, aerobic and resistance exercise are associated with a favorable cardiovascular risk profile. The extent to which reductions in body fat mediated by increases in RMR contribute to the reduced cardiovascular risk is not well understood.

The relationship between resistance training, cardiovascular risk profiles, and RMR in women is also not well understood. Research has shown that weight-training exercise can improve (11) or have no effect on (12, 13) the blood lipid profiles of women. To our knowledge, the effect of resistance training on the RMR of women has not been studied. Thus, the purpose of this study was to compare, by using a cross-sectional design, RMR and cardiovascular risk (body fat, blood lipids, blood pressure, and insulin and glucose concentrations) in young healthy women who regularly participate in either aerobic or resistance exercise.

Methods

Subjects

The 82 female subjects for whom data are presented in this paper had no known medical abnormalities (eg, coronary heart disease, high blood pressure, diabetes, family history of obesity) that could adversely affect the data collected, especially the RMR data. In addition, none of the subjects smoked and most were Caucasian. Two subjects (one aerobic-exercise and one resistance-exercise subject) were amenorrheic. Each subject provided written informed consent before participating in the study. The

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The subjects were assigned to the following groups based on self-reported activity data: sedentary (n = 48), in which the subjects did not exercise regularly; aerobic exercise (n = 21), in which each subject participated in activities such as running, swimming, and cycling three or more times per week (no regular resistance training); and resistance exercise (n = 13), in which subjects participated in resistance-training activities three or more times per week (no regular aerobic training). The resistance-exercise subjects generally did 3–5 sets per exercise (10–20 repetitions per set) with the resistance varying between ≈65–80% of weight that they could lift once but not twice. In general, subjects in the exercise groups had been participating regularly in that form of exercise for ≥ 2 y.

Order of testing

Each subject reported to the Clinical Research Center in the afternoon, spent the night, and underwent testing the next morning. The evening before testing, the subjects were fed a standard meal that contained ≈4200 kJ (55% carbohydrates, 15% protein, and 30% fat). In addition, an estimate of their daily food intake and daily leisure-time activity was obtained via a 3-d food record and an activity questionnaire. The RMR of each subject was determined in a fasted state (10–12 h postabsorptive) and shortly after awakening (≈0700) by indirect calorimetry. After the measurement of RMR a sample of blood was drawn to determine blood lipid, plasma insulin, plasma glucose, and plasma thyroid hormone concentrations. Body composition, peak oxygen consumption ($\dot{V}O_2$), and anthropometric measures were then made.

Resting metabolic rate

The procedures used to measure RMR as well as our test-retest reliability were reported previously (14). Briefly, on awakening, and after a 10–12-h fast, a ventilated hood was used to obtain a 45-min measurement of the subject's resting $\dot{V}O_2$. This was done in the same room in which the subject slept. Subjects did not leave the bed until the RMR measurement was completed. To minimize the possibility of the testing procedure affecting RMR, each subject practiced with the ventilated hood the night before the actual measurement. Furthermore, all subjects were tested as inpatients because these conditions yield lower values than when subjects transport themselves to the laboratory on the day of testing (15). We chose to measure RMR just after the subjects awoke because we felt that this would reduce the variability inherent in this measurement. Although RMR is technically a resting measurement, it is likely close to the basal (sleeping) metabolic rate. Energy expenditure was estimated by using the Weir equation (16). All women were tested during the follicular phase (as determined by self-report) of their menstrual cycle. Finally, to control for any residual effects of exercise, subjects refrained from exercise for 36 h before measurement of RMR, because this period has been shown to apparently eliminate the carryover effects on energy expenditure and hormonal milieu (17).

Peak $\dot{V}O_2$

Peak $\dot{V}O_2$ was measured by using an incremental treadmill running test as previously described (18). Briefly, subjects started running at a self-selected pace and the incline on the treadmill was increased by 2% every 2 min until volitional fatigue. The highest 1-min $\dot{V}O_2$ obtained was deemed to be the peak $\dot{V}O_2$. If the subject did not achieve two of the following three criteria their data were excluded from analyses: increase in $\dot{V}O_2$ of < 150 mL/min with increase in work rate, respiratory exchange ratio > 1.0, or heart rate ≥ age-predicted maximum.

Leisure-Time activity

The Minnesota Leisure Time Physical Activity Questionnaire (19) was completed by each subject. When completing the questionnaire, subjects report duration, frequency, and intensity with which they participate in various leisure-time activities. The questionnaire does not attempt to assess energy expended either at work or while doing household-related activities (eg, cleaning). The energy requirements of the various activities are then calculated and weighted for seasonal participation and finally reported as an average daily energy expenditure (kJ/d).

Body composition, anthropometry, and blood pressure

Underwater weighing with simultaneous residual-volume corrections (helium dilution) was used to determine body density. Body density was then converted into percent fat by using the equation by Keys and Brozek (20). Fat mass was calculated by multiplying body mass times percent fat, and fat-free mass was calculated by subtracting fat mass from body mass. Skinfold thicknesses were measured at nine sites (abdomen, axillary, biceps, calf, chest, subscapular, suprailiac, thigh, and triceps) and reported as the sum of nine skinfold-thickness measurements. Waist and hip circumferences were measured as the minimal circumference of the abdomen and the maximal circumference of the buttocks, respectively. A waist-to-hip ratio was then obtained by dividing the waist circumference by the hip circumference. A blood pressure machine was used to automatically measure systolic and diastolic blood pressures with reported blood pressures representing the mean of three measurements. Mean arterial blood pressure (MAP) was estimated by the following formula:

$$\text{MAP} = (0.67 \times \text{diastolic blood pressure}) + (0.33 \times \text{systolic blood pressure})$$

Energy intake

Energy intake was determined as reported previously (18) by a 3-d, self-reported diary (2 weekdays, 1 weekend day). Subjects were strongly encouraged not to change their normal dietary habits and were trained to accurately measure and report food intakes. Food records were checked for completeness by a dietitian at the time of their return by the subject.

Plasma determinations

Blood was drawn after a 10–12-h fast and after RMR was determined. A glucose analyzer (Yellow Springs Instrument, Yellow Springs, OH) was used to determine plasma glucose concentrations. A modification of the radioimmunoassay technique of Starr et al (21) was used to measure plasma concentrations of immunoreactive insulin. Thyroid hormone (total and free), as previously described (14), was determined according to the following procedures. A Clinical Assay kit (Baxter, Cambridge, MA) was used to quantify free and total concentrations of thyroxine (T_4) and the total concentration of 3,5,3-triiodothyronine

TABLE 1
Physical characteristics, age, blood pressure, and resting heart rate of sedentary (SED), aerobically trained (AT), and resistance-trained (RT) women*

	SED (n = 48)	AT (n = 21)	RT (n = 13)
Height (cm)	164.1 ± 0.9	165.4 ± 1.3	164.0 ± 1.5
Age (y)	28.7 ± 1.0	26.7 ± 1.2	27.3 ± 1.4
Body mass (kg)	60.6 ± 1.2	58.1 ± 0.9	58.4 ± 1.8
Fat-free mass (kg)	47.1 ± 0.8	48.7 ± 0.8	49.7 ± 1.4
Body fat (%)	21.8 ± 0.8	16.2 ± 0.7†	14.7 ± 0.8†
Waist-to-hip ratio	0.77 ± 0.01	0.76 ± 0.01	0.75 ± 0.01
Sum of nine skinfold thicknesses	146 ± 6	106 ± 5†	89 ± 5†
Systolic blood pressure (mm Hg)	110 ± 2	105 ± 3	107 ± 3
Diastolic blood pressure (mm Hg)	73 ± 1	74 ± 3	70 ± 2
Standing heart rate (beats/min)	67 ± 3	66 ± 2	69 ± 4

* $\bar{x}$ ± SE.
† Significantly less than SED, $P < 0.05$.

(total T_3) in plasma. An analog assay (Diagnostic Products, Los Angeles) was used to determine plasma concentrations of free T_3. An enzymatic process was used to determine total cholesterol (22) and triglycerides (23). High-density lipoproteins were measured by using a dextran sulfate procedure after low-density lipoproteins were removed (24). Low-density lipoproteins were determined by using the procedure developed by Friedewald et al (25) for use with subjects with concentrations of fasting triglycerides < 4.5 mmol/L.

Statistics

Analysis of variance was used to determine whether mean differences existed between groups (26). When a significant F ratio was present, a Neuman-Keuls post hoc test was administered to determine which means were significantly different from each other (26). RMR was also adjusted, by analysis of covariance, for differences among groups for fat-free mass (26). Finally, because few differences existed between groups with respect to coronary-heart-disease risk factors, the groups were combined and correlational analyses performed to determine the extent to which selected cardiovascular-risk variables were related to body habitus (26). All data are expressed as mean ± SE.

Results

Table 1 contains physical characteristics as well as age, blood pressure, and resting heart rate for the 82 women in the three groups. The subjects were of normal weight (overall $\bar{x}$ ± SE = 59.6 ± 1.3 kg) and height (overall 164.4 ± 1.4 cm) and were young (28.0 ± 1.1 y), with no statistically significant differences between groups for these variables.

The lack of a profound exercise effect on body mass for normal-weight females has been consistently found in both animal (27) and human models (28) and should not be surprising. Similarly, exercise training did not affect (ie, $P > 0.05$) fat-free mass (47.8 ± 0.9 kg). In contrast, participation in regular exercise had a marked effect on percent body fat and sum of nine skinfold thickness with the aerobic-exercise and resistance-exercise groups having 40–45% less body fat than the sedentary group (percent body fat = 21.8%, sum of nine skinfold thicknesses = 146 mm). Thus, both aerobic- and resistance-exercise groups had less total percent body fat and subcutaneous (sum of nine skinfold thicknesses) body fat than did sedentary women. However, no differences were noted among groups for waist-to-hip ratio (0.76 ± 0.01).

Resting systolic (108 ± 3 mm Hg) and diastolic (73 ± 2 mm Hg) blood pressure as well as resting heart rate (67 ± 3 beats/min) were all within normal ranges for this population. There were no statistically significant differences between groups for these variables.

Table 2 presents resting, activity, and peak energy expenditure data for the three groups. As expected, energy expenditure during leisure-time activity was significantly greater for the aerobic-exercise group than for the sedentary group (2530 vs 1693 kJ/d). The leisure-time activity energy expenditure for the resistance-exercise group (2180 kJ/d) was approximately midway between that of the other two groups and not significantly different from either group. In contrast, peak $\dot{V}O_2$ was significantly different between all groups with the aerobic-exercise and resistance-exercise groups having peak $\dot{V}O_2$s ≈30% and 13% higher, respectively, than the sedentary group (42 mL $O_2 \cdot min^{-1} \cdot kg^{-1}$).

Regular exercise, regardless of type (ie, aerobic or resistance), was associated with a higher RMR compared with the sedentary group. As can be seen in Table 2, the RMRs for the exercise-trained groups were ≈7% higher ($P < 0.05$) than those for the sedentary group (3.99 kJ/min). Because RMR has been shown to be strongly correlated with fat-free mass (29), it is of interest to know the extent to which the increase was greater than that which could be accounted for by an increase in fat-free mass alone. An adjusted RMR was therefore calculated by using analysis of covariance in which fat-free mass was the covariate. When adjusted for fat-free mass, the RMR of the aerobic-exercise group (4.25 kJ/min) was significantly greater than that of either the sedentary (6% higher) or resistance-exercise (3% higher) groups; RMRs of the sedentary and resistance-exercise groups were not significantly different from each other. Thus, the increase in RMR for the aerobic-exercise group was greater than can be accounted for by differences in tissue composition alone (ie,

TABLE 2
Energy expenditure during rest and activity for sedentary (SED), aerobically trained (AT), and resistance-trained (RT) women*

	SED (n = 48)	AT (n = 21)	RT (n = 13)
RMR (kJ/min)	3.99 ± 0.05	4.31 ± 0.06†	4.25 ± 0.09†
Adjusted RMR‡ (kJ/min)	4.05 ± 0.03	4.24 ± 0.05§	4.13 ± 0.05
Leisure-time activity (kJ/d)	1693 ± 148	2530 ± 215†	2180 ± 254
Peak $\dot{V}O_2$ ($mL \cdot min^{-1} \cdot kg^{-1}$)	42.1 ± 0.7	54.5 ± 0.9§	47.6 ± 1.8†

* $\bar{x}$ ± SE. RMR, resting metabolic rate; $\dot{V}O_2$, oxygen volume.
† Significantly greater than SED, $P < 0.05$.
‡ Resting metabolic rate that has been adjusted by analysis of covariance with fat-free mass as a covariant.
§ Significantly greater than SED and RT, $P < 0.05$.

TABLE 3
Self-reported 3-d dietary-intake data for sedentary (SED), aerobically trained (AT), and resistance-trained (RT) women*

	SED (n = 48)	AT (n = 21)	RT (n = 13)
Intake (kJ/d)	7883 ± 276	7928 ± 457	8551 ± 759
Carbohydrate (g/d)	256 ± 10	275 ± 17	318 ± 38†
Carbohydrate (%)‡	54 ± 1	58 ± 1	60 ± 2†
Protein (g/d)	71 ± 3	71 ± 5	89 ± 8§
Protein (%)‡	15 ± 1	15 ± 1	18 ± 1§
Fat (g/d)	65 ± 4	56 ± 6	49 ± 4
Fat (%)‡	30 ± 1‖	26 ± 2	22 ± 2
Alcohol (%)‡	2 ± 1	1 ± 1	1 ± 1
Cholesterol (mg/d)	190 ± 20	147 ± 21	162 ± 22

* $\bar{x}$ ± SE.
† Significantly greater than SED, $P < 0.05$.
‡ Expressed as a percentage of daily dietary intake (kJ/kJ).
§ Significantly greater than SED and AT, $P < 0.05$.
‖ Significantly greater than RT, $P < 0.05$.

metabolic rate is higher per kg of fat-free mass). In contrast, the higher RMR (unadjusted) for the resistance-exercise group was likely due to a tendency to have a larger fat-free mass.

Table 3 reports the results of a 3-d prospective dietary record for the three groups done just before in-house testing. There were few meaningful differences between groups and in general the groups exhibited prudent dietary habits. The percentages of dietary intake as carbohydrate (range 54–60%), protein (range 15.1–17.5%), fat (range 21.9–29.9%), and alcohol (range 0.8–1.0%) were within recommended ranges and reflect currently recommended dietary practices (30). The resistance-exercise group did consume more ($P < 0.05$) carbohydrate (g/d and % of diet) than did the sedentary group and more ($P < 0.05$) protein (g/d and % of diet) than did either the sedentary or aerobic-exercise groups. The sedentary group, although within recommended percentile limits, ate a significantly higher percentage (30%) of their dietary intake as fat compared with the resistance-exercise group.

Table 4 denotes fasting plasma lipid concentrations for the three groups. There were no significant differences between groups for total cholesterol (4.3 ± 0.1 mmol/L), triglycerides (0.96 ± 0.06 mmol/L), low-density lipoproteins (2.61 ± 0.09 mmol/L), or high-density lipoproteins (1.35 ± 0.05 mmol/L).

Table 5 characterizes the fasting thyroid hormone, insulin, and glucose concentrations in the three groups. Again, participation in regular exercise had little effect (except for free T_3) on the concentrations of these fasting hormones within the blood plasma compared with sedentary individuals. There were no significant differences between groups for T_3 (1.7 ± 0.07 mmol/L), T_4 (100 ± 4.5 mmol/L), free T_4 (20 ± 0.08 mmol/L), insulin (46 ± 3.9 pmol/L), glucose (5.2 ± 0.1 mmol/L), or insulin-glucose ratio (0.48 ± 0.04). The free T_3 plasma concentration for the resistance-exercise group was 23% higher than that of the aerobic-exercise group (3.2 mmol/L).

Because few differences existed between groups with respect to blood pressure (Table 1), dietary intake (Table 3), blood lipids (Table 4), or plasma hormone concentrations (Table 5), the groups were combined and correlational analyses were performed for the total group. The purpose of these analyses was to determine the extent to which coronary-heart-disease risk factors are related to activity, body composition, diet, RMR, and cardiorespiratory capacity in young nonobese women. **Table 6** presents the results of the selected correlations.

TABLE 4
Fasting plasma lipids in sedentary (SED), aerobically trained, (AT) and resistance-trained (RT) women*

	SED (n = 48)	AT (n = 21)	RT (n = 13)
Cholesterol (mmol/L)	4.63 ± 0.10	4.42 ± 0.13	4.37 ± 0.16
Triglycerides (mmol/L)	1.00 ± 0.05	0.91 ± 0.05	0.90 ± 0.09
Low-density lipoproteins (mmol/L)	2.59 ± 0.08	2.66 ± 0.10	2.61 ± 0.10
High-density lipoproteins (mmol/L)	1.37 ± 0.05	1.34 ± 0.05	1.27 ± 0.08

* $\bar{x}$ ± SE.

It was surprising to find the extent to which some degree of significant correlation existed between variables in a population in whom the prevalence of risk factors is quite low. The amount of body fat was positively correlated ($P < 0.05$) with total cholesterol (%fat, $r = 0.27$; sum of nine skinfold thicknesses, $r = 0.41$), fasting triglycerides (%fat, $r = 0.21$; sum of nine skinfold thicknesses, $r = 0.43$), insulin concentrations (sum of nine skinfold thicknesses, $r = 0.21$), and insulin-glucose ratio (sum of nine skinfold thicknesses, $r = 0.22$). The waist-to-hip ratio was negatively ($P < 0.05$) correlated with high-density lipoproteins ($r = -0.34$) and positively correlated ($P < 0.05$) with triglyceride concentrations ($r = 0.21$). Percent body fat was significantly correlated with the following variables: RMR ($r = -0.22$), peak $\dot{V}O_2$ ($r = -0.57$), leisure-time activity ($r = -0.32$), percentage of dietary intake as fat ($r = 0.22$), and sum of nine skinfold thicknesses ($r = 0.78$). These results can be generalized by noting that as body fat increases, so do cholesterol and triglyceride concentrations, and that body fat is inversely related to leisure-time energy expenditure. Although not shown in the table there was also a significant correlation ($P < 0.01$) between peak $\dot{V}O_2$ and RMR ($r = 0.39$).

Discussion

We assessed the extent to which resting energy metabolism and cardiovascular risk differed with exercise status (ie, sedentary

TABLE 5
Fasting thyroid hormone and insulin and glucose plasma concentrations in sedentary (SED), aerobically trained (AT), and resistance-trained (RT) women*

	SED (n = 48)	AT (n = 21)	RT (n = 13)
T_3 (nmol/L)	1.68 ± 0.05	1.65 ± 0.09	1.74 ± 0.08
T_4 (nmol/L)	99.1 ± 2.6	101.7 ± 6.4	103.0 ± 6.4
Free T_3 (pmol/L)	3.69 ± 0.14	3.17 ± 0.22	3.92 ± 0.29†
Free T_4 (pmol/L)	20.2 ± 0.6	19.0 ± 0.6	20.7 ± 1.2
Insulin (pmol/L)	47.0 ± 3.5	48.5 ± 4.3	38.2 ± 5.3
Glucose (mmol/L)	5.19 ± 0.06	5.31 ± 0.12	5.35 ± 0.08
Insulin-glucose ratio	0.49 ± 0.03	0.51 ± 0.05	0.40 ± 0.05

* $\bar{x}$ ± SE. T_3, 3,5,3′-triiodothyronine; T_4, thyroxine.
† Significantly greater than AT, $P < 0.05$.

TABLE 6
Correlations between coronary-heart-disease risk factors, energy expenditure, and body-composition variables*

	TC	TG	HDL	LDL	Ins	I:G	MAP	%Fat
RMR	−0.15	−0.04	−0.11	−0.03	−0.09	−0.06	−0.09	−0.22†
Peak $\dot{V}O_2$	−0.20	−0.08	−0.06	−0.06	−0.10	−0.10	0.04	−0.57‡
LTA	0.08	0.06	−0.01	−0.01	−0.14	−0.14	0.02	−0.32‡
Percent fat intake (%)§	−0.06	−0.05	0.08	−0.17	0.01	0.02	0.23†	0.22†
Body fat (%)	0.27†	0.21†	0.04	0.08	0.10	0.11	−0.14	1.0
Sum 9 SF	0.41‡	0.43‡	0.01	0.13	0.21†	0.22†	−0.10	0.78‡
W/H	0.09	0.21†	−0.34‡	0.11	0.08	0.06	−0.01	0.19

* TC, total cholesterol; TG, triglycerides; HDL, high-density lipoprotein; LDL, low-density lipoprotein; Ins, insulin; I:G, insulin-glucose ratio; MAP, mean arterial pressure, %Fat, percent body fat; RMR, resting metabolic rate; LTA, leisure-time activity; sum 9 SF, sum of nine skinfold thicknesses; W/H, waist circumference divided by hip circumference; and $\dot{V}O_2$, oxygen volume.
† $P < 0.05$.
‡ $P < 0.01$.
§ Percentage of daily dietary intake as fat.

and aerobic or resistance exercise) among relatively young, healthy women. Subjects who participated in regular exercise had a higher RMR than did sedentary individuals. When corrected for fat-free mass, the RMR for the aerobic-exercise group remained elevated compared with the resistance-exercise and sedentary groups. Although exercise did not appear to change cardiovascular risk (ie, blood lipid profiles, blood pressure, or insulin concentrations), statistically significant (albeit low) correlations were found between body fat and cholesterol, triglyceride, insulin concentrations, and insulin-glucose ratios.

The exercise-induced (aerobic and resistance training) increase in RMR in women with regular training (Table 2) is interesting especially because aerobic-exercise training in females does not consistently result in increases in RMR (4). It has been suggested that changes in RMR may in some way be linked to changes in peak $\dot{V}O_2$ with a statistically significant correlation existing between RMR and peak $\dot{V}O_2$ (18). A statistically significant relationship ($r = 0.39$) was also found between RMR and peak $\dot{V}O_2$ for the subjects in this study. This confirms previous data from our laboratory (18).

When one compares the aerobic-exercise and resistance-exercise groups, it appears that the mechanism of exercise-induced increase in RMR may be different between groups. As shown in Table 2, when adjusted by analysis of covariance for differences in fat-free and fat mass, the RMR of the aerobic-exercise group remained higher ($\approx$5%) than that of the sedentary group, whereas the resistance-exercise group's RMR was now significantly lower than the aerobic-exercise group and not different ($P > 0.05$) from that of the sedentary group. Thus, whereas the increase in RMR with resistance training can be accounted for by changes in fat-free mass, increases in RMR with aerobic-type training appear to be partially mediated by an increase in the rate of activity per kilogram of tissue (ie, energy expenditure per kg fat-free mass is greater than that for resistance-exercise or sedentary groups).

These results differ somewhat from those found in a recent cross-sectional study from our laboratory, which used male subjects and a similar design (10). When adjusted for differences in fat-free mass by using analysis of covariance, the RMR of males undergoing regular aerobic exercise was significantly higher than the RMR of males participating in resistance exercise who in turn had a significantly higher RMR than sedentary males. These data (10) suggest that for males, resistance training also results in increases in resting metabolism that are greater than can be accounted for by changes in fat-free mass alone. The reasons for the sexually divergent responses to resistance-training exercise are not clear and may be multifactorial. Potential mechanisms include exercise-training volume or intensity, and inherent differences between sexes with respect to anabolic hormones.

Many theories have been proposed as to the mechanism of an exercise-induced increase in RMR per kilogram of fat-free mass, including increased activity of various enzymatic reactions and shuttle systems (futile cycling), increased substrate (ie, food) flux, repair of exercise-induced trauma, increased concentrations of metabolic hormones (eg, thyroid hormones, catecholamines, and cortisol), increased protein synthesis, and replenishment of glycogen stores (4, 31). We recently showed that the increase in RMR is associated with an increase in the appearance rate of norepinephrine into the circulation (32). This suggests that elevations in RMR in response to chronic exercise are sympathetically mediated. The data (and experimental design) presented here do not warrant extended discussions of these issues but these potential mechanisms can be briefly examined in conjunction with dietary intake, thyroid hormone, and body-composition data, and the 36–48-h abstinence from exercise before ascertainment of RMR.

Any exercise-induced mechanism that would potentially exert its effects over relatively short time periods (ie, < 36–48 h) is likely not a mechanism by which exercise training increased RMR for these subjects (ie, exercise-induced trauma and replenishment of glycogen stores). Consistent with this we have also shown that exercise-induced effects on RMR increase with weeks of training (33, 34), an adaptation that seems inconsistent with trauma and recovery mechanisms for elevated RMR. Because the subjects were 10–12-h postabsorptive, substrate flux is also not likely a mechanism by which exercise training increased RMR in these subjects. Likewise, the concentrations of thyroid hormones (Table 5) were generally not different between groups. However, Herring et al (35) recently reported that both RMR and urinary catecholamines decreased in exercise-trained women who suspended their aerobic-exercise routines, suggesting that there is an acute component to exercise-induced changes in RMR. However, the adaptation may be both acute and chronic in that it takes a period of training before the effect of

exercise on RMR manifests itself (ie, chronic) and that this effect disappears relatively rapidly after the training ends (ie, acute). Of particular interest is that with detraining, the RMR for Herring et al's (35) subjects dropped 0.37 kJ/min. This is very close to the 0.33-kJ/min difference found between the sedentary and aerobic-exercise groups in this study.

Although the mechanism by which resistance training increases RMR may be different from that by which aerobic training increases RMR, the increase is meaningful nonetheless. A higher RMR, regardless of how obtained, allows one to eat more and still maintain body mass at desired levels. For example, the 0.33-kJ/min difference between the sedentary and aerobic-exercise groups represents a difference of 475 kJ/d in resting energy expenditure.

It is likely that the absence of differences between groups with respect to cardiovascular risk factors such as blood pressure (Table 1), fasting plasma lipids (Table 4), and insulin concentrations (Table 5) are due, in part, to the initial healthy concentrations of these variables within the sedentary group. However, of the four studies we could find that examined the effect of resistance training on cardiovascular risk (including this one), three suggest that resistance training does not alter (present study, 12, 13) lipid profiles compared with those in control subjects. Goldberg et al (11) reported a statistically significantly lower blood cholesterol, triglycerides, and high-density lipoproteins after resistance training. This study also appeared to have the most rigorous exercise program, with the subjects going until they could not complete another repetition (ie, failure) in each of three sets on 7–8 exercises. Thus, it may be that resistance-training exercise needs to exceed some threshold for adaptations in blood lipid profiles to occur. Future studies need to be done in subjects with riskier blood lipid profiles because all of the available studies examined subjects with fairly healthy blood lipid profiles (ie, cholesterol < 5.2 mmol/L).

The fact that statistically significant correlations (Table 6) exist between cardiovascular risk factors and other variables, even though they are quite low, are notable in a low-risk population. Furthermore, even in women who are quite lean (overall $\bar{x}$ = 19.2% body fat), body fat is significantly correlated with total cholesterol, triglyceride, and insulin concentrations. These cardiovascular-disease-risk links, if continued, may lead to problems later in life and are thus worthy of further investigation. It would be especially interesting if these correlations could help identify those healthy individuals who may be predisposed to increased cardiovascular risk with age-related changes in body composition and physical activity.

Finally, the lack of an exercise effect as suggested by the absence of differences between groups for variables such as blood lipids, thyroid hormones, insulin, etc, should be examined in the following context. First, one should not necessarily expect exercise training to exert an effect on a variable that is already near a physiological limit (ie, ceiling effect) and many of the variables studied are well into the desired range. Second, exercise training may not necessarily exert an effect or the training may not be sufficient (ie, duration, intensity, frequency) to elicit changes (although differences between groups for peak $\dot{V}O_2$ and percent fat are consistent with those that occur after exercise training). Third, the absence of differences between groups with respect to body mass and fat-free mass (as well as other variables) may be due, in part, to the cross-sectional design of this study, in which a large within-group variability can make it more difficult to achieve statistical significance.

In summary, this cross-sectional study suggests that resistance-exercise training in young women results in reductions in body fat and increases in RMR similar to those found with aerobic-exercise training. The mechanism by which exercise increases the RMR seems to be different between aerobic and resistance exercise. Exercise, regardless of type, did not seem to affect blood lipid, thyroid hormone, or insulin-glucose profiles.

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