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ScienceDirect
Metabolism Clinical and Experimental 55 (2006) 1358–1364
Metabolism
Clinical and Experimental
www.elsevier.com/locate/metabol

Effects of exercise training on resting metabolic rate in postmenopausal African American and Caucasian women

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Received 23 March 2005; accepted 7 June 2006

Abstract

Ambiguous findings have been reported in previous studies concerning the relationships between resting metabolic rate (RMR), effects of exercise, and race in postmenopausal women. The purposes of this study were (1) to determine the effect of exercise training on RMR in postmenopausal women and (2) to determine whether this effect is different by race. We analyzed data from 24 African American (AA) (age, 56.3 ± 5.0 years) and 23 Caucasian (CA) (age, 58.6 ± 6.1 years) women. RMR was measured by indirect calorimetry after an overnight fast. Subjects completed tests of maximal exercise with metabolic measurements, waist to hip ratio, and body composition. At the completion of all tests, each subject was randomly assigned to either the exercise training or the control group. Aerobic exercise was performed 3 to 4 days per week for 6 months. Intensity was set at 70% to 85% maximal heart rate for 45 to 60 minutes per session. Both AA and CA exercise groups had a significant decrease ($P < .05$) in body weight (kilograms), percent body fat, and body mass index (BMI) and an increase in aerobic capacity, whereas the control groups did not change. There were no race effects. AA women exhibited significantly lower values than CA women for measured RMR ($P < .05$). The RMR (kilojoules) of the AA women (both control and exercise groups) decreased over time ($P < .05$), whereas the RMR of the CA women did not change. Adjusting RMR for BMI or for changes in lean body mass and fat mass did not alter these results. In conclusion, this 6-month endurance-training program did not affect RMR in these postmenopausal women, as RMR did not change in CA women, but decreased significantly over time in both groups of AA women. The factors that contribute to these findings should be the focus of future studies.

1. Introduction

Resting metabolic rate (RMR) represents the largest fraction of an individual's daily energy expenditure, accounting for approximately 60% to 75% of the daily total energy expenditure [1]. Aging is associated with a progressive decline in RMR [2-6] partially due to diminished fat-free mass (FFM) [6-8].

A decline in RMR is cause for concern because a low RMR is a significant predictor of subsequent weight regain in formerly obese subjects [7,9]. Recent data have indicated obesity to be one of the nation's most serious health problems [10,11], affecting 1 in every 3 Americans [12]. The prevalence of obesity has been found to be higher in minority women [11], as nearly 50% of African American (AA) women are overweight compared with 33% of Caucasian (CA) women [13]. The excess weight in AA women has been associated with more medical problems, especially diabetes, compared with their CA counterparts [14,15]. Several studies have suggested that ethnicity may independently affect RMR [16-18], and it is generally accepted that AA women have lower RMR values than CA women [13,16-24].

We have previously found RMR to be higher in CA compared with AA premenopausal women, and fitness levels as well as lean body mass (LBM) were found to be significant predictors of RMR in both races [16]. Exercise training may attenuate the age-related decrease in RMR. Both cross-sectional and intervention studies report that RMR may increase through endurance training in young men and women [25-31]. Some exercise intervention studies have

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doi:10.1016/j.metabol.2006.06.006

also shown endurance training to increase RMR in older individuals in the absence of changes in FFM [29]. However, women have been poorly represented, as evidenced by the small numbers of women [29]. Furthermore, not all studies show an increase in RMR with endurance training. Poehlman et al [32] found no change in daily energy expenditure in young, nonobese, sedentary women after 6 months of exercise training after adjusting RMR for FFM.

The results suggest exercise training may affect RMR differently in women compared with men. Because RMR decreases and body weight increases after menopause, investigating potential mediators of RMR is especially relevant in postmenopausal women. Furthermore, even less is known about the effects exercise training may have on RMR in postmenopausal AA women. Considering the low RMR and high levels of obesity in this population, the potential for increasing RMR through exercise training needs to be investigated.

Therefore, the purpose of this study was 2-fold. First, we determined whether exercise training has an effect on RMR in postmenopausal women. The second purpose was to determine whether there were different responses in RMR due to training between AA and CA postmenopausal women.

2. Methods and materials

2.1. Human subjects

Subjects for this study included 47 [33] healthy postmenopausal women volunteers (24 AA and 23 CA) aged between 45 and 70 years. To be eligible, women had to be postmenopausal, defined as amenorrhea >6 months or older than 55 years with no bleeding for 6 months; sedentary for a minimum of 3 months before participation ("sedentary" was defined as no more than 1 day per week of participation in aerobic exercise activity lasting 20 minutes or more); and in generally good health and able to exercise at the rate described in our protocol.

Subjects were excluded if they had moderate to severe hypertension, defined as blood pressure greater than 160/100 mm Hg at entry; diabetes; history of thyroid disease or use of thyroid hormones; history of deep vein thrombosis, pulmonary embolism, cardiovascular disease, or familial hypercoagulability and if they were current cigarette smokers. Women taking hormone replacement therapy (HRT) were included in the study. There were 5 AA women and 6 CA women on HRT.

All subjects volunteered to participate in the study and provided written informed consent before participation. The study protocol was approved by the George Washington University Medical Center Institutional Review Board.

2.2. Design

This was a randomized longitudinal study examining the effects of a 6-month, moderate to vigorous-intensity, and cardiovascular exercise program on RMR in AA and CA postmenopausal women. After undergoing baseline tests (maximum oxygen consumption [$\dot{V}O_2$max], RMR, and body fat), women were randomly assigned to one of 2 groups: exercise or control. Three AA women in the control group and 2 in the exercise group were taking HRT, and 3 CA women in each group were taking HRT. The exercise group participated in a 6-month supervised exercise program, whereas the control group was instructed to maintain their current lifestyle.

3. Testing procedures

3.1. Resting metabolic rate measurements

Subjects were instructed to report to the laboratory within 60 minutes of waking and after a 12-hour fast. After 30 minutes of quiet supine rest in a dimly lit room, RMR was measured for 30 minutes through indirect calorimetry (Deltatrac II Metabolic Monitor, Sensormedics, Yorba Linda, CA) by using a ventilated hood system. The coefficient of variation for RMR measurements using this system in our laboratory is less than 4.0%. All women on HRT were tested on days when they were taking both hormones. For the exercise groups, the post-RMR was measured after 48 hours of the last exercise training session. Predicted metabolic rates were determined by using the Harris-Benedict equation.

3.2. Anthropometric measurements

After the RMR measurement, body composition was determined by an air displacement plethysmograph (BOD POD, LMI, Concord, CA). The BOD POD consists of a dual-chambered plethysmograph that provides a densitometric means of body composition analysis whereby the subject is tested in the front chamber and instrumentation is located in the back of a fiberglass capsule. The BOD POD has been proven to be highly reliable, with test-retest coefficients of variation similar to hydrostatic weighing, and valid, correlating well with hydrostatic weighing (r = 0.96) [34]. For the AA groups, the Schutte et al equation [35] was used to predict fat mass and LBM. Height and weight were measured on a standard scale to determine body mass. BMI was calculated as weight (kilograms)/height (meters)^2.

3.3. Maximal exercise test

Maximal exercise testing was conducted by using a modified Bruce protocol on a motorized treadmill with continuous electrocardiographic (ECG) monitoring. Twelve-lead ECGs, blood pressure, and ratings of perceived exertion were recorded during each exercise stage. Subjects breathed through a nonrebreathing 2-way valve, and expired air was analyzed for volume and percent expired oxygen and carbon dioxide with an automated, computerized, metabolic system (Quinton Q-Plex, Quinton, Bothell, WA). The system was calibrated with known volumes and gases before each test. Data were evaluated in 20-second

averages, and maximal (or peak) oxygen uptake ($\dot{V}O_2$peak) was defined as the highest 20-second value attained provided 2 of the following criteria were met: a maximal heart rate (MHR) within 10 beats per minute of age-predicted MHR, a plateau in $\dot{V}O_2$ with an increase in workload (an increase of <2.0 mL · kg^{-1} · min^{-1}), rating of perceived exertion of 18 or higher, and/or a respiratory exchange ratio of 1.05 or higher.

4. Exercise program

At the completion of all tests, each subject was randomly assigned to either the exercise training or the control group. The exercise-training group entered the supervised exercise program, whereas the control subjects were asked to maintain their current behaviors throughout the 6-month study period.

Exercise was prescribed according to published guidelines [36]. The exercise group participated in supervised group exercise a minimum of 3 to 4 days per week. Subjects were provided with heart rate monitors (Polar Electro, Port Washington, NY) to ensure adherence to the prescribed intensity, and a daily diary, which included the date, activity, duration of exercise, and heart rate during exercise, was kept by the investigators. Exercise included treadmill walking/jogging, stationary cycling, and rowing. Intensity was calculated by using the straight percentage of MHR method and was set at 70% to 85% (~60%-80% $\dot{V}O_2$peak). Exercise duration was gradually increased during the first 8 weeks until subjects were exercising for 45 to 60 minutes per session. All subjects were instructed to maintain current dietary patterns during the entire study. Subjects were also instructed to maintain their current vitamin/supplement/hormone therapy regimen (if any) throughout the study period. They were told to report the use of any new prescription or over-the-counter medications.

5. Statistics

All group values are reported as means ± SEM. To evaluate the effect of the 6-month training program and if AA women responded differently to training compared with CA women, a 3-way training group (exercise vs control) by race (AA vs CA) by time (pre vs post) analysis of variance with repeated measures was used. To more appropriately evaluate the effect on RMR, a 3-way training group by race by time analysis of covariance (ANCOVA) was conducted adjusting for LBM. We used the LBM both before and after exercise training as covariates in the ANCOVA. We also conducted a 3-way training group by race by time ANCOVA adjusting for changes in LBM and fat mass and for baseline BMI. We used the change in LBM, change in fat mass, and the baseline BMI as covariates in the model. Finally, we conducted a 3-way training group by race by time ANCOVA on RMR expressed as kilojoules per LBM using the baseline BMI as a covariate. We used baseline BMI as a covariate in these analyses because there were substantial differences in baseline BMI between the AA and the CA women randomized to the training group. Significance was set at the $P < .05$ level.

6. Results

Between-group means are presented in Table 1. A significant ($P < .05$) training group by time interaction was found in both AA and CA women in the exercise group for several anthropometric variables. Those in the exercise group had a significant decrease ($P < .05$) in body weight (kilograms), fat mass, percent body fat, and BMI, whereas those in the control group did not change. There were no race effects or any 3-way interactions for any of these variables. In contrast, exercise training did not affect LBM, even when adjusted for changes in fat mass,

Table 1
Anthropometric and maximal exercise data

	AA				CA			
	Control (n = 12)		Exercise (n = 12)		Control (n = 13)		Exercise (n = 10)	
Age (y)	54.9 ± 1.6		57.7 ± 1.6		59.1 ± 1.5		58.0 ± 1.8	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Body weight (kg)	77.4 ± 4.01	78.5 ± 4.25	74.7 ± 5.43	72.8 ± 5.43*	73.7 ± 4.07	73.7 ± 4.30	67.4 ± 2.76	64.4 ± 2.83*
Lean body mass (kg)†	45.5 ± 1.5	45.3 ± 1.6	43.7 ± 1.4	43.7 ± 1.6	41.7 ± 1.4	41.8 ± 1.6	40.6 ± 1.6	40.7 ± 1.7
Fat mass (kg)	30.6 ± 3.3	31.9 ± 3.3	31.1 ± 3.1	28.9 ± 3.2*	30.0 ± 3.1	30.0 ± 3.2	26.9 ± 3.4	23.6 ± 3.5*
% Body fat	39.1 ± 2.3	40.6 ± 2.0	39.7 ± 2.5	37.7 ± 2.9*	42.3 ± 2.2	41.2 ± 1.9	39.3 ± 1.2	36.1 ± 2.0*
BMI (kg/m^2)	28.4 ± 1.6	28.8 ± 1.7	28.2 ± 2.1	27.5 ± 2.1*	27.1 ± 1.4	27.1 ± 1.4	24.6 ± 1.1	23.6 ± 1.4*
Waist-hip ratio	.80 ± .02	.81 ± .02	.84 ± .03	.83 ± .03	.84 ± .03	.83 ± .03	.77 ± .03	.77 ± .02
$\dot{V}O_2$max (mL · kg^{-1} · min^{-1})	25.5 ± 1.1	25.3 ± 1.2	24.5 ± 2.2	28.3 ± 2.4*	26.5 ± 1.4	26.4 ± 1.4	28.7 ± 1.9	34.9 ± 2.8*
MHR (bpm)	167 ± 3.2	165 ± 3.4	166 ± 4.7	167 ± 4.1	165 ± 3.7	163 ± 4.0	162 ± 4.2	166 ± 3.7
MRQ	1.18 ± .02	1.16 ± .02	1.16 ± .02	1.18 ± .03	1.19 ± .03	1.20 ± .02	1.21 ± .04	1.25 ± .03

Values are mean ± SE. MRQ indicates maximal respiratory quotient.

* $P < .05$ (significant changes with exercise and significantly different from the control group).

† Race effect: CA women exhibited lower values than AA women ($P < .05$).

Table 2
Resting metabolic rate

	AA				CA			
	Control (n = 12)		Exercise (n = 12)		Control (n = 13)		Exercise (n = 10)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Predicted RMR (kJ)*	6033.2 ± 157.0	6087.6 ± 169.9	5853.1 ± 207.6	5794.5 ± 204.7	5798.7 ± 178.3	5794.5 ± 185.8	5568.4 ± 129.3	5463.7 ± 119.3
RMR (kJ)†,#	5133.1 ± 151.98	4923.6 ± 190.9	5229.3 ± 226.9	4932.5 ± 328.2	5296.3 ± 271.3	5208.3 ± 304.7	5241.8 ± 199.7	5195.2 ± 241.1
RMR adjusted for LBM (kJ)†,#	4893.9 ± 141.9	4551.0 ± 138.5	5262.8 ± 135.2	4990.6 ± 131.8	5225.1 ± 128.5	5112.0 ± 125.6	5639.62 ± 157.0	5698.2 ± 153.2
RMR adjusted for pre BMI (kJ/LBM)†,#	112.6 ± 4.1	107.6 ± 4.1	118.4 ± 3.9	110.5 ± 3.8	125.1 ± 3.8	122.2 ± 4.3	131.4 ± 4.3	131.0 ± 4.3
RQ†	.81 ± .01	.81 ± .01	.81 ± .01	.85 ± .01	.87 ± .01	.86 ± .02	.84 ± .01	.86 ± .02

Values are mean ± SE.
* $P < .05$ (change with exercise).
† $P < .05$ (race effect: CA women exhibited higher values than AA women).
# $P < .05$ (race by exercise training effect).

waist-to-hip ratio, MHR, or maximal respiratory quotient (RQ) regardless of race. However, LBM was higher in AA women ($P < .05$).

Aerobic capacity, represented by $\dot{V}O_2$max, increased significantly ($P < .05$) in both AA and CA women in the exercise training group, with no change in the control group. There was no race effect or any race by time or 3-way interactions.

There was a significant race effect for all measured RMR variables ($P < .05$), with AA women exhibiting significantly lower values than CA women (Table 2). Furthermore, expressing RMR as kilojoules of energy expenditure, we found a significant race by time interaction, as the values for AA women (both the control and exercise group) decreased significantly after the 6-month period (mean of both AA groups combined: pre 5028.3 ± 97.1 vs post 4731.0 ± 100.0, $P < .05$), whereas those for the CA women did not change (mean of both CA groups combined: pre 5384.2 ± 100.4 vs post 5346.5 ± 102.9). Adjusting RMR for baseline BMI or for changes in fat mass and LBM did not alter these results. In addition, expressing RMR as kilojoules per kilogram and kilojoules per LBM, we found the resting energy expenditure of AA women (combined mean of the exercise and control group) to decrease significantly ($P < .05$) after the 6-month period, whereas those of the CA women did not change.

There was also a race effect for resting RQ ($P < .05$). CA women exhibited a higher RQ, but there was no effect of training on RQ and no significant interactions.

7. Discussion

There were 2 main findings from this study. First, 6 months of exercise training did not affect RMR in these postmenopausal women despite a substantial training-induced increase in $\dot{V}O_2$max. Second, although RMR was unaffected in CA women, RMR decreased significantly over time in AA women, regardless of whether they received training or not. These changes were accompanied by similar changes in body weight, LBM, fat mass, percent body fat, and BMI in both AA and CA women. Consequently, exercise training had no effect on the decrease in RMR observed over time in the AA women.

Our findings seem in contrast to some of the available literature on the effects of exercise training on RMR. Several cross-sectional studies have shown higher RMR in exercise-trained compared with sedentary individuals [5,37,38]. Some longitudinal studies have shown similar results, but most of these studies involved primarily men or premenopausal women. For instance, Lawson et al [28] found that a 10-week jogging program increased RMR in 6 lean sedentary women aged 22 to 35 years without changes in body weight or body composition. The increased RMR may have been related to the increased energy intake seen throughout the study to maintain body weight. Poehlman and Danforth [29] found similar changes in older individuals after an 8-week exercise training program. Endurance training elevated RMR by 10% in the absence of changes in FFM. They also reported an increase in food intake to maintain energy balance, and a higher rate of norepinephrine appearance.

A recent report showed [32] that absolute RMR increased in resistance-trained young women, but when adjusted for FFM, this change was not significant. There was no change in absolute or relative (adjusted for FFM) RMR as an effect of endurance training. Thus, our data are different from those of the above studies, probably because of the difference in the subject populations tested.

Exercise training does not appear to have any chronic, long-term effect on sleeping metabolic rate [39-44]. Also, exercise intervention studies [30,31,45] that measured RMR after the last training period (≥36 h) or sleeping metabolic rate show no clear long-term effect of exercise on RMR, especially after adjustment for FFM. Our data support these

findings, as we observed either no change or slight decreases in RMR regardless of adjustments for changes in FFM and fat mass. Furthermore, we did not find a significant group by training interaction, demonstrating that exercise training did not affect RMR in our study.

The women in our study did not increase RMR despite a training volume of more than 3768 kJ/wk and despite showing a substantial improvement in $\dot{V}O_2$max. Because $\dot{V}O_2$max is positively related to RMR in both pre- and postmenopausal women [5], and a training volume of 3768 kJ/wk increased RMR in older individuals [29], this was an unexpected finding. However, the women in our study lost weight in response to training, whereas the women in previous studies increased their energy intake to maintain energy balance. Thus, a small decrease in RMR as a result of the weight loss might be expected in our study. However, FFM did not change in our study, although fat mass decreased, which may partially explain our findings. It is possible that exercise training affects postmenopausal women differently than premenopausal women. In contrast to our findings, Svendsen et al [46] showed that a combined diet and exercise program in postmenopausal women produced an 11% increase in RMR despite a 10-kg weight loss. A closer examination of their data, however, revealed that they expressed RMR per kilogram of body weight. Our data show that when expressed per kilogram of body weight, RMR increased by 4% in the exercise group, whereas it decreased by 2% in the control group (the CA women). This difference was significant ($P < .05$) and similar to the difference between exercise and control groups in the study by Svendsen et al [46] (6% vs 7%). Consequently, exercise training appears to maintain or slightly increase RMR depending on how RMR is expressed, even in the presence of weight loss in CA women.

We have also previously shown that RMR (adjusted for body weight) of AA women was significantly lower (9%) than that of CA women [16]. Similarly, obese AA women have been found to have a 5% lower RMR than CA women, and this difference increased to 8% when adjusted for LMB [17]. In contrast, others have found no differences in RMR between obese postmenopausal AA and CA women when RMR was adjusted for LBM [47].

Our current data are consistent with those of most previous studies showing that AA women have lower RMR than CA women [17,23,47,48]. However, the only other study on postmenopausal women showed no difference in RMR, when adjusted for LBM, between AA and CA women. Our data still showed that RMR was lower in AA women even after adjusting for LBM. This may indicate that AA LBM is not as metabolically active as that of their CA counterparts, which would be consistent with a higher bone density in AA women [49-51]. A higher bone density would make bone a proportionally greater part of LBM, which could account for the apparently lower metabolic activity of LBM in AA women. Suminski et al [52] found differences in skeletal muscle oxidative metabolism between AA and CA men, consistent with a lower RMR. However, although lower RMR is a common finding in the AA population, the effect of exercise training has not been previously investigated.

To our knowledge, no other studies have examined the effects of endurance training on RMR in postmenopausal AA women, nor have differences between CA and AA endurance-trained women been evaluated. Therefore, the decrease in RMR after the 6-month period in AA postmenopausal women was an unexpected new finding. Because AA women exhibit lower RMR to start with, which has been identified as early as childhood [48,53-56], our current findings may partially help explain the high levels of obesity in this population.

The implications of our results suggest that the impact of exercise training and physical activity are similar in AA and CA postmenopausal women. Both improved $\dot{V}O_2$max, lost body weight, and reduced relative body fat as a result of the training program. These changes are important because they may potentially improve insulin sensitivity and protect against the development of type 2 diabetes mellitus [10,14,15]. Consequently, both AA and CA postmenopausal women should be encouraged to exercise, but should not expect the exercise to affect their RMR. That RMR decreased over time in the AA group may suggest a need for other lifestyle interventions in addition to exercise training in this population. However, our finding needs to be viewed with caution because both the AA control group and the exercise training group exhibited a similar decrease in RMR and we did not find a significant 3-way interaction. Furthermore, the initial body weight was higher in the AA training group than in the CA training group, which may have affected our results. However, whether or not we adjusted for changes in FFM, fat mass, and baseline BMI, the decrease in RMR was significantly greater in AA women compared with either group of CA women. It is unlikely that HRT use affected our findings because the number of women on HRT was similar among the groups. These data may suggest a possible seasonal variation in RMR in AA women, not evident in CA women, or some other unknown effect on RMR. This needs to be further investigated in future studies.

We can only speculate on potential contributors to the differential training response we observed. Leptin [47,57] may play a possible role in the energy expenditure of AA postmenopausal women; however, leptin levels were not measured in our study. Leptin interacts with brain pathways, particularly in the hypothalamus, to reduce food intake and control energy expenditure and body weight [58] and may play a possible contributing role in the energy expenditure of AA postmenopausal women [59]. Leptin and FFM explained interindividual variation in sleeping metabolic rate, measured in 25 healthy volunteers, during an overnight stay in a respiratory chamber [60]. Thus, it is possible that training-induced changes in leptin may play a role, but this remains to be investigated.

The presence of lower RMR in AA women has been viewed as a manifestation of their genetic predisposition to obesity [22]. Recently, Walston et al [61] showed that genetic variability contributes to RMR. The RMR in Arg64 β3 adrenoreceptor allele homozygotes was significantly lower than in Trp64 homozygotes. However, it is unknown if such gene differences exist between AA and CA postmenopausal women. A recent study by Kimm et al [22] found that uncoupling proteins contribute to RMR and these uncoupling proteins were different between AA and CA young women, suggesting a genetic link to the lower RMR found in AA women. However, it is unknown if such differences also contribute to the differential response to exercise training.

In conclusion, this 6-month endurance-training program did not affect RMR in these postmenopausal women. In fact, RMR was decreased in both the control and exercise groups of AA women over the 6-month period, whereas the exercise program did not affect RMR in CA postmenopausal women. The factors that contribute to these findings should be the focus of future studies.

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