

Changes in resting energy expenditure after weight loss in obese African American and white women¹⁻³

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ABSTRACT

Background: Previous studies showed that resting energy expenditure (REE) is lower in obese African American women than in obese white women. It is unknown, however, whether there are racial differences in how REE responds to weight loss and energy restriction.

Objective: We assessed REE, body composition, and respiratory quotient before and after weight loss in obese black and white women.

Design: We measured REE by indirect calorimetry and body composition by densitometry before and after 20–24 wk of treatment with a 3870–4289-kJ/d diet. Subjects were 109 obese females (24 black, 85 white) with a mean (\pm SD) body mass index (in kg/m²) of 36.3 ± 5.0 , weight of 95.7 ± 12.6 kg, and age of 42.3 ± 8.1 y.

Results: Before treatment, REE, adjusted for body composition, was significantly lower in black than in white subjects ($P = 0.001$). Black subjects lost significantly less weight during treatment than did white subjects (13.4 ± 5.9 kg or $14.2 \pm 5.7\%$ compared with 16.4 ± 5.6 kg or $17.0 \pm 5.7\%$, respectively; $P = 0.04$). Analyses that controlled for initial REE and changes in fat mass and fat-free mass showed that blacks had significantly greater decreases in REE after treatment than did whites ($9.9 \pm 7.3\%$ compared with $6.3 \pm 7.4\%$; $P = 0.02$).

Conclusion: This study suggests that weight loss results in greater reductions in REE in obese black women than in obese white women. These data underscore the need to consider both biological and behavioral factors when setting expectations and assessing outcomes for obesity treatment in African American women. *Am J Clin Nutr* 1999;69:13–7.

KEY WORDS Obesity, ethnicity, resting energy expenditure, metabolism, body composition, respiratory quotient, weight loss, women

INTRODUCTION

Obesity is a serious public health problem that is increasingly prevalent, especially among minority women. When obesity is defined as a body mass index (BMI; in kg/m²) ≥ 27.3 , nearly 50% of African American women are obese compared with only 33% of white women (1). In addition, obese black women lose less weight than do white women with a variety of weight-loss treatments, including balanced deficit diets (2), very-low-energy diets (3, 4), and bariatric surgery (5). Explanations for these dif-

ferences have focused primarily on behavioral and cultural factors such as diet, activity, and ideals of beauty (6–9).

Recent data suggest that metabolic factors may also contribute to the observed racial differences in obesity prevalence and treatment outcome. Four studies reported within the past 2 y showed that among obese women, blacks have a significantly lower resting energy expenditure (REE) than do whites, even after weight and body composition are controlled for (10–13). A fifth study found similar racial differences in REE that were not statistically significant in a small sample (14).

Independent of any differences in REE that may exist before weight-loss treatment, it is unknown whether blacks and whites differ in how REE changes with weight loss. The finding that black women experience larger reductions in REE in response to dieting than do white women would further explain their smaller weight losses. Thus, the present study assessed measures of weight, REE, respiratory quotient, and body composition before and after weight loss in obese black and white women.

SUBJECTS AND METHODS

Subjects

Subjects were selected from participants of 2 obesity treatment studies described in the next section. Both studies were randomized clinical trials approved by the University of Pennsylvania Committee on Studies Involving Human Beings. Exclusion criteria for both studies included a recent (within 1 y) myocardial infarction or evidence of any cardiac abnormality; current (or history of) cerebrovascular, kidney, liver, or thyroid disease; cancer; anemia; bulimia nervosa or any significant psychiatric illness; and type 1 diabetes. Subjects $< 140\%$ of their recommended weight (15) were excluded. Additional exclusions for the present study were smoking and invalid REE or body-composition measurements.

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Treatment conditions

The first study ($n = 128$) from which subjects were selected was described previously (16). It compared 4 treatments: diet alone, diet plus aerobic exercise, diet plus strength-resistance training, and diet plus aerobic exercise and strength-resistance training combined. Subjects in the exercise conditions participated in 3 weekly, supervised sessions. Subjects attended weekly group sessions for 24 wk.

Subjects for the current analysis were also selected from a second clinical trial that compared the effects of various degrees of energy restriction on weight loss and eating behavior. Subjects ($n = 25$) in one of the treatment conditions in that study consumed a diet (described below) similar to that consumed by subjects in study 1 (16). Subjects in study 2 were encouraged to increase their physical activity (typically by a modest walking program), but the type of exercise was neither controlled nor supervised. Subjects in study 2 attended weekly group sessions for 20 wk.

Diet

During the first week of treatment, all subjects were asked to consume their typical diets. Thereafter, subjects in study 1 consumed a 3870-kJ/d diet for 16 wk. The diet consisted of 4 servings daily of a liquid meal replacement (628 kJ each), a portion-controlled frozen dinner entrée (1172–1255 kJ each), and 2 cups of vegetable salad. After this time, subjects were asked to consume a self-selected diet providing 5021–6276 kJ/d. Subjects in study 2 followed the same dietary protocol as those in study 1 with several minor exceptions. The diet of study 2 subjects included a fruit serving (251 kJ) and the liquid meal replacement provided 669 rather than 628 kJ, bringing the total energy intake to 4289 rather than 3870 kJ/d. In addition, study 2 subjects consumed the portion-controlled diet for 12 wk rather than 16 wk. Subjects in study 2 were also instructed to increase their intake to 5021–6276 kJ/d with self-selected foods after this time.

Exclusions

On the basis of their similar dietary treatments, 153 subjects (128 from study 1 and 25 from study 2) were eligible for participation in this study. Of these, 8 (3 black, 5 white) were excluded because of medical conditions that required treatment during the study. Of the remaining 145 subjects, 24 were excluded for the following reasons: 9 subjects (3 black, 6 white) were smokers, 1 Hispanic woman was not identified with respect to black or white race, 9 subjects (3 black, 6 white) had baseline REE measurements that could not be equilibrated after 2 separate attempts, and 5 subjects (2 black, 3 white) did not have valid baseline body-composition assessments.

During the study period, 7 of the 121 subjects (1 black, 6 white) discontinued treatment. Thus, weight-loss data were available for 114 subjects (25 black, 89 white). We were unable to obtain equilibrated REE measurements for 2 subjects (both white) and valid body-composition measurements for 3 subjects (1 black, 2 white) after treatment. Thus, 109 subjects (24 black, 85 white) were included in the present analysis, all of whom had pre- and posttreatment assessments of weight, REE, and body composition. Most of the subjects (17 black, 74 white) were from study 1 and the remainder (7 black, 11 white) were from study 2.

Dependent measures

Body weight, REE, and body composition were assessed before and after weight loss. Subjects in study 1 were assessed at

week 24 and those in study 2 at week 20. Thus, all subjects were assessed 7–8 wk after discontinuing the structured 3870–4289-kJ/d diet and when prescribed a self-selected diet of 5021–6276 kJ/d. Body weight was measured by using a balance-beam scale and height was measured with use of an attached vertical rod.

REE was measured by indirect calorimetry (DeltaTrac Metabolic Monitor; SensorMedics, Yorba Linda, CA). Gas exchange (oxygen consumption and carbon dioxide production) was measured with a canopy collection technique in the morning after subjects had fasted overnight and after they had rested in a semi-recumbent position for 30 min. Subjects were instructed to consume only water, avoid exercise, and refrain from taking any over-the-counter medication for the 12 h preceding the measurement. Subjects were interviewed before their REE measurement to assess adherence to the pretest conditions.

The metabolic cart was calibrated daily by using standard gases of known concentration (96% O₂, 4% CO₂). Continuous measurements of gas exchange were computed at 1-min intervals until 5 consecutive data points were obtained for both oxygen consumption and carbon dioxide production, which had a CV <5% (17). These equilibrated (ie, steady state) measurements were averaged and applied to the abbreviated Weir (18) formula to determine daily REE.

Body composition was assessed by densitometry. Subjects were submerged in a 1.5 × 1.5 × 1.7 m tank with a light metal chair suspended from a 15-kg scale (Chatillon, Kew Gardens, NY) as described by Goldman and Buskirk (19). Underwater measurements were made ≥6 times until 3 readings within 25 g were obtained. Residual lung volume was measured by the oxygen dilution technique (20) with a metabolic cart (MMC Horizon II; SensorMedics) while subjects rested in the same position that they had been in underwater. Body density was calculated from the formula of Brozek et al (21) and percentage body fat from the formula of Siri (22). Fat-free mass (FFM) was defined as body weight minus body fat.

Statistical analyses

Pretreatment

Analysis of variance was used to compare the dependent variables in blacks and whites before treatment. Traditionally, investigators have adjusted REE by expressing it as kcal/kg body wt or FFM. As Ravussin and Bogardus (23) noted, such adjustments are flawed because they are based on the faulty assumption of a 1:1 relation between REE and weight or FFM. Although linear, the relation is not 1:1 (ie, it does not have a 0,0 intercept) and it varies considerably at the extremes of body weight (24). As suggested, regression analyses were performed to identify the principal determinants of REE in this sample, and subsequent analyses of covariance (ANCOVAs) controlled for the relation between REE and its principal determinant or determinants.

Before treatment, univariate analyses revealed that weight was the strongest correlate of REE ($r = 0.75$, $P = 0.0001$). Because body weight consists of FFM and fat mass, we performed separate regression analyses on these variables to determine their relative contribution to REE. When FFM was entered into the regression first, it accounted for 29.7% of the variance and fat mass accounted for an additional 26.7%. When fat mass was entered into the regression first, it accounted for 36.6% of the variance and FFM accounted for an additional 19.8%. Thus, body weight explained 56% of the variance in REE, and fat mass was a stronger predictor of REE than was FFM.

Posttreatment

The posttreatment data were analyzed by ANCOVA. For ease of understanding, data are presented as absolute change or percentage change. However, because change and percentage change data were not normally distributed, analyses were performed on the absolute values after treatment with the initial (pretreatment) values as the covariates. For the REE analyses, changes in fat mass and FFM were also used as covariates. Pearson product moment correlations were used to assess the factors associated with weight loss and change in REE.

Preliminary analyses showed that there was no significant effect of the 5 treatment conditions (4 from study 1 and 1 from study 2) on changes in weight, fat, or FFM. There was, however, a treatment effect for change in REE. Subjects in the diet plus modest, unsupervised walking program had significantly greater reductions in REE than did subjects in the diet plus aerobic exercise or the diet plus aerobic exercise and strength training combined program. As described previously, the last 2 conditions were supervised on-site 3 times per week. These results support the findings of the earlier report (16) in which subjects who received aerobic training tended to have smaller reductions in REE at week 24. For the purposes of this study, analyses of post-treatment REE controlled for the observed treatment condition effects before assessing any race effects. All data are reported as means \pm SDs.

RESULTS

Before treatment

The 109 subjects (24 black, 85 white) had a mean weight of 95.7 ± 12.6 kg, a BMI of 36.3 ± 5.0 , and an age of 42.3 ± 8.1 y. There were no significant differences between blacks and whites in pretreatment values for weight, BMI, body composition, age, or respiratory quotient (**Table 1**). ANCOVA with adjustment for fat mass and FFM did show, however, that blacks had a significantly lower REE than whites.

After treatment

An ANOVA that controlled for initial weight revealed that black women lost less weight than did white women ($P = 0.04$): 13.4 ± 5.9 kg ($14.2 \pm 5.7\%$) compared with 16.4 ± 5.6 kg ($17.0 \pm 5.7\%$). FFM decreased by 2.5 ± 2.5 kg ($4.9 \pm 4.9\%$) in

blacks and by 1.5 ± 2.5 kg ($3.1 \pm 4.8\%$) in whites. An ANCOVA that controlled for initial FFM and weight loss revealed that the difference between groups in change in FFM was not significant ($P = 0.09$).

An ANCOVA that controlled for initial REE, change in FFM, change in fat mass, and treatment condition showed that blacks had significantly greater reductions in REE than did whites after weight loss ($P = 0.02$). In blacks, REE was reduced by 713.4 ± 516.7 kJ/d, or 170.5 ± 123.5 kcal/d ($9.9 \pm 7.3\%$), compared with 454.1 ± 516.7 kJ/d, or 108.7 ± 123.5 kcal/d ($6.3 \pm 7.4\%$), in whites (**Figure 1**). There were no differences between the groups in changes in respiratory quotient.

Correlates of changes in weight and REE

Two additional posttreatment analyses were conducted. The first assessed the univariate correlates of weight loss and the second examined the univariate correlates of change in REE.

For whites, there was a strong relation between weight loss and initial weight ($r = 0.43$, $P < 0.0001$); additionally, initial REE ($r = 0.34$, $P < 0.002$) and change in REE ($r = 0.38$, $P = 0.0001$) showed a significant relation to weight loss. Thus, higher initial weights and REEs and greater reductions in REE were associated with greater weight losses. For blacks, weight loss was most strongly related to change in REE ($r = 0.49$, $P = 0.02$), followed by initial REE ($r = 0.34$, $P = 0.1$). There was no relation between initial weight and weight loss in blacks ($r = 0.1$, $P = 0.66$).

For whites, change in REE was most strongly related to initial REE ($r = 0.41$, $P < 0.0001$), followed by weight loss ($r = 0.38$, $P < 0.001$) and initial weight ($r = 0.35$, $P < 0.001$). Thus, higher initial REEs, larger weight losses, and higher initial weights were associated with greater reductions in REE. For blacks, change in REE was most strongly related to initial REE ($r = 0.55$, $P = 0.005$) and weight loss ($r = 0.49$, $P = 0.02$). Initial weight, however, showed no relation to change in REE ($r = 0.05$, $P = 0.82$) in black subjects.

TABLE 1

Comparison between obese black and white women before weight-loss treatment¹

Variable	White (n = 85)	Black (n = 24)
BMI (kg/m ²)	36.1 \pm 5.1	36.8 \pm 4.4
Weight (kg)	95.1 \pm 12.9	97.8 \pm 11.4
Age (y)	43.0 \pm 7.8	39.8 \pm 8.6
REE, unadjusted (kJ/d)	6960.5 \pm 930.5	6670.6 \pm 698.7
REE, adjusted for fat-free mass (kJ/d)	6977.7 \pm 736.8	6607.8 \pm 737.9 ²
REE, adjusted for fat mass (kJ/d)	7011.1 \pm 671.2	6663.9 \pm 680.5 ³
REE, adjusted for body weight (kJ/d)	7029.1 \pm 551.5	6597.7 \pm 559.6 ⁴
RQ	0.80 \pm 0.005	0.80 \pm 0.004
Fat-free mass (kg)	50.8 \pm 6.2	51.8 \pm 6.1
Fat mass (kg)	44.3 \pm 10.1	46.0 \pm 9.4

¹ $\bar{x} \pm$ SD. REE, resting energy expenditure; RQ, respiratory quotient.

²⁻⁴Significantly different from whites (ANCOVA): ² $P = 0.03$,

³ $P = 0.02$, ⁴ $P = 0.001$.

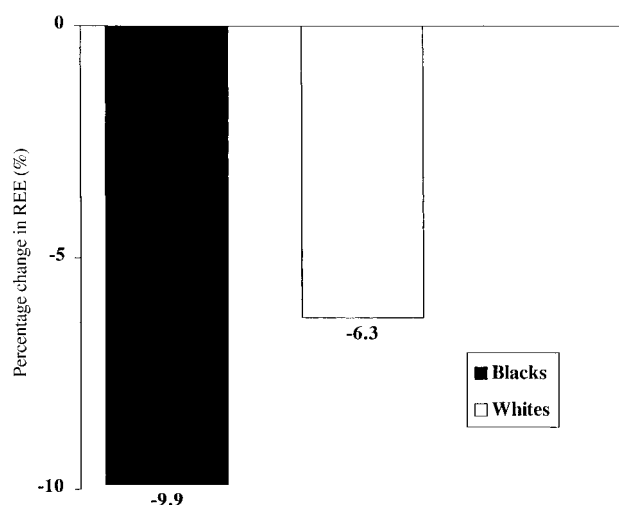


FIGURE 1. Percentage reduction in resting energy expenditure (REE) by race ($n = 24$ blacks and 85 whites) after 20–24 wk of weight-loss treatment. Mean values for percentage change in REE were adjusted for initial REE, change in fat-free mass, change in fat, and treatment condition.

DISCUSSION

There were 3 principal findings of this study. The first was that obese black women lost significantly less weight (2.2 kg, or 2.5%) than did white women when prescribed the same dietary treatment. This result was not surprising for 2 reasons. First, although the diet used in this study provided 3870–4289 kJ/d, was portion controlled, and was structured, similar data have been reported with a variety of dietary interventions (2–5). Second, given the importance of REE in determining weight loss with a fixed intake (25), the lower pretreatment REE of black subjects alone would be expected to result in smaller weight losses for these subjects.

The second and more novel finding of this study suggested an additional metabolic difference that may lead to smaller weight losses in obese black women. Specifically, black subjects in our study had significantly greater reductions in REE after energy restriction and weight loss than did white subjects, even after the smaller weight losses in the black subjects were controlled for. Thus, in addition to blacks having lower REEs than whites before starting weight-loss treatment (10–12), these new data suggest that there are also racial differences in how REE responds to energy restriction and weight loss. To our knowledge, these are the first data to compare changes in REE after weight loss between black and white subjects. Unfortunately, both pre- and posttreatment differences in REE suggest that blacks have a more difficult time losing weight than do whites. The reasons for these differences are unknown. Although blacks and whites showed differences in changes in weight and FFM, our analyses controlled for these differences and still found a significant effect of race. In addition, menopausal status of the women did not significantly affect REE (6959.2 ± 948.5 , 6975.2 ± 872.3 , and 6680.0 ± 759.4 kJ/d for premenopausal, postmenopausal, and undetermined menopausal status, respectively; $F = 1.0$, $P = 0.37$), and menstrual status categories were not significantly different between black and white women (chi-square = 4.2, $P = 0.12$).


The third principal finding of this study was that there were both similarities and differences in the determinants of weight loss and change in REE in black and white women. For example, initial REE and change in REE were similarly and significantly related to weight loss in black and white subjects. By contrast, initial weight was the strongest determinant of weight loss in whites but showed no relation to weight loss in blacks. Similarly, initial weight was significantly related to change in REE in whites but not in blacks. These data are difficult to interpret but they underscore the need for caution when extrapolating findings from studies of white populations to black and other nonwhite populations.

This study had several limitations. First, the use of an outpatient sample precluded the precise quantification of the behavioral and biological factors related to decreased weight loss in black subjects. Although our data suggest that racial differences in initial REE and changes in REE are likely explanations for the observed differences in weight loss, our study did not adequately assess the role of behavioral factors such as dietary (including alcohol) intake and physical activity. These factors would not only affect weight loss, but also may have independent effects on changes in REE. The questionable reliability and validity of self-reports of food intake make assessments of dietary adherence in an outpatient setting difficult. Future studies can better assess adherence by using the doubly labeled water method (26) to esti-

mate total daily energy expenditure or by conducting studies in a controlled environment.

A second limitation was our inability to separate the effects of weight loss and those of energy restriction on REE because our subjects were prescribed a 5021–6276-kJ/d diet at the time of their second REE measurement. Assessing patients when they are in energy balance but at a reduced weight would help to clarify whether the greater reduction in REE in blacks persists when energy restriction is fully terminated.

A third limitation was the use of densitometry to assess body composition. Densitometry does not take into account the greater bone mineral density of African Americans (27–29), which may lead to an overestimation of FFM (30). This flaw, however, seems to have little, if any, effect on body-composition estimates (29, 31), especially when using the Siri (22) formula, as we did in this study. Because densitometry was used both before and after treatment, it is less likely that the trend for blacks to lose more FFM than whites was due to the assumptions of densitometry. Nevertheless, any possible artifacts of densitometry can be remedied by using dual-energy X-ray absorptiometry measurements in future studies.

In summary, the results of this study suggest that a greater reduction in REE after weight loss or energy restriction may partially contribute to the observed differences in weight loss between obese black and white women. It would be imprudent to use these data to support a solely biological explanation for reduced weight loss by African American women. Clearly, behavioral (eg, adherence) and cultural (eg, desired body shape) factors affect weight-loss outcomes. The current data suggest that, in addition to any behavioral factors, a lower initial REE and a greater decrease in REE after energy restriction and weight loss will make weight control efforts in African American women more difficult. Thus, biological as well as behavioral differences should be considered when setting expectations and evaluating treatment outcomes for obese African American women (32). 

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