See corresponding editorial on page 1348.

Influence of distribution of lean body mass on resting metabolic rate after weight loss and weight regain: comparison of responses in white and black women[1–4]

Nuala M Byrne, Roland L Weinsier, Gary R Hunter, Renee Desmond, Mindy A Patterson, Betty E Darnell, and Paul A Zuckerman

ABSTRACT

Background: Little is known about the effect of weight change on regional lean body mass (LBM) distribution or on racial differences in resting metabolic rate (RMR).

Objective: The study compared total and regional LBM patterns in white and black women after weight loss and regain and assessed the influence of regional LBM on variances in RMR.

Design: Eighteen white and 22 black women who did not differ in age, weight, and height were studied 3 times: in the overweight state, after weight reduction to the normal-weight state, and after 1 y without intervention. Total and regional lean and fat masses were assessed by dual-energy X-ray absorptiometry.

Results: White and black women did not differ significantly in mean (±SD) weight loss (13.4 ± 3.6 and 12.7 ± 3.2 kg, respectively) and regain (6.1 ± 5.5 and 6.4 ± 5.4 kg, respectively). Black subjects had significantly less trunk LBM and significantly more limb LBM at each time point ($P < 0.05$). In both races, weight regain was associated with significant increases in limb LBM ($P < 0.05$) but not in trunk LBM ($P = 0.21$). RMR, adjusted for total LBM and fat mass, was significantly higher in white women after weight loss ($P < 0.01$) and regain ($P < 0.01$). However, no racial difference was found when RMR was adjusted for LBM distribution.

Conclusions: In both races, trunk LBM decreased with weight loss and remained lower, despite significant weight regain, which potentially reflected decreased organ mass. Regional LBM distribution explained the racial difference in RMR. *Am J Clin Nutr* 2003;77:1368–73.

KEY WORDS Overweight, obesity, weight loss, body composition, resting metabolic rate, lean body mass, fat mass, regional distribution, African American women

INTRODUCTION

The prevalence of overweight and obesity is increasing (1, 2), and recidivism after weight loss is common (3, 4). The prevalence of obesity is greater in black women than in white women (5), and black women reputedly lose less weight with a range of treatment modalities (6–10). There remains considerable debate regarding the degree to which cultural, behavioral, physiologic, and metabolic factors are responsible for racial differences in weight gain, response to weight-loss treatment, and posttreatment recidivism. Some research suggests that obese black women have a lower resting metabolic rate (RMR) than do obese white women, even after adjustment for variation in body composition (11–13), and that weight loss results in a greater reduction in RMR (adjusted for body composition) in black women (10).

Racial differences in the distribution of fat and lean tissue have been found in both normal and overweight conditions. Black persons tend to have greater amounts of skeletal muscle (14, 15) and greater appendicular bone lengths (14–16) across all ages in adulthood than do white subjects. Gallagher et al (17) found that, although there was no racial difference in height, the black subjects in their study had greater leg length relative to their height than did the white subjects. Further, after adjustment for stature and body weight, age, sex, and ethnicity are all significant independent determinants of appendicular skeletal muscle mass (17). These racial differences may have some effect on energy expenditure. Given the differences in the energetics of the different components of lean body mass (LBM; 18–20), variation in the proportion of organ tissue to skeletal muscle tissue may explain some of the residual variability in RMR beyond that explained by total LBM (21, 22).

In a sample of 40 normal-weight white adults, Sparti et al (23) found that regression models based on organ size did not improve the estimation of RMR beyond that obtained with the use of the traditional model based on LBM and fat body mass. In contrast, we have recently shown cross-sectionally that differences in sleeping energy expenditure and RMR between black and white women may be mediated by proportionately lower trunk LBM (and thus lower organ mass) in black women (24). However, little is known about the effect of weight loss and weight regain on

[1] From the Departments of Nutrition Sciences (NMB, RLW, MAP, and PAZ) and Human Studies (GRH), the Biostatistics Unit (RD), the General Clinical Research Center (BED), and the Clinical Nutrition Research Center (RLW, GRH, and RD), University of Alabama at Birmingham, and the School of Human Movement Studies, Faculty of Health, Queensland University of Technology, Brisbane, Australia (NMB).

[2] RL Weinsier is deceased.

[3] Supported by NIH grants R01-DK49779 and R01-DK51684, General Clinical Research Center grant M01-RR00032, and Clinical Nutrition Research Unit grant P30-DK56336. Stouffer's Lean Cuisine entrées were generously supplied by the Nestlé Food Co, Solon, OH.

[4] Address reprint requests to NM Byrne, School of Human Movement Studies, Faculty of Health, Queensland University of Technology, Victoria Park Road, Kelvin Grove, Brisbane Q4059, Australia. E-mail: n.byrne@qut.edu.au.

Received March 7, 2002.

Accepted for publication December 16, 2002.

changes in regional lean mass and fat mass distributions in different races or about whether regional lean tissue distribution may explain race differences in RMR.

Dual-energy X-ray absorptiometry (DXA) provides the opportunity to study both total and regional fat and lean tissue in vivo (17). Earlier research (25–29) supports the validity of DXA estimates of regional lean tissue. With the use of DXA, the mass of skeletal muscle may be approximated by measurement of the lean mass of the extremities (17). The purposes of the current study were to compare total and regional lean mass and fat mass patterns in white and black women in 3 weight states (overweight, normal-weight, and at 1-y follow-up) with the use of DXA and, if racial differences were found, to assess the degree to which regional lean tissue mass explains variance in RMR in different weight states.

SUBJECTS AND METHODS

Study subjects

Subjects were 18 white and 22 black premenopausal women aged 20–46 y who had a baseline body mass index (BMI; in kg/m^2) of 27–30 (chosen to increase the likelihood that subjects could attain a normal weight in a reasonable time frame) and a family history of obesity (BMI > 27) in at least one first-degree relative. Classification of subjects as black or white was based on self-reporting. Normal glucose tolerance was documented by measurement of fasting blood glucose concentrations and 2-h postprandial blood glucose concentrations after an oral glucose load. Subjects were nonsmokers, were sedentary, and had normal menstrual cycles. The study protocol was approved by the Institutional Review Board of the University of Alabama at Birmingham. Written informed consent was obtained from all subjects before study participation, in compliance with Department of Health and Human Services Regulations for Protection of Human Research Subjects. The cohort studied features a number of subjects [15 white and 20 black women (30); 14 white and 19 black women (31)] who were included in data previously reported from our laboratory [the General Clinical Research Center (GCRC) at the University of Alabama at Birmingham]; however, the outcome variables in these previous studies were different from those in the current investigation.

Study design

Subjects were evaluated at 3 time points: in the overweight state, in the normal-weight state, and after 1-y follow-up. Study variables were assessed under weight-stable, diet-controlled conditions through the GCRC. Before each evaluation, subjects were maintained in a weight-stable state for 4 wk, during the final 2 wk of which meals were provided through the GCRC to maintain macronutrient intake within the range of 20–22% of energy as fat, 16–23% as protein, and 55–64% as carbohydrate. Subjects were then admitted to the GCRC for 4 d, during the follicular phase of the menstrual cycle. After their discharge, the GCRC prepared all meals for weight reduction, providing 3350 kJ/d (800 kcal/d) and including frozen entrées twice daily (Stouffer's Lean Cuisine; Nestlé Food Co, Solon, OH). Dietary adherence and body weight were monitored twice a week until subjects lost > 10 kg and reached a normal weight, defined as a BMI < 25. Although they were sedentary, no attempt was made to alter their physical activity patterns. On reaching a normal BMI, subjects repeated the protocol of energy balance for 4 wk and GCRC admission for 4 d. After their discharge in the normal-weight state, no intervention was provided, and the subjects were contacted < 10 mo later to schedule a follow-up evaluation according to the same protocol of weight maintenance for 4 wk before GCRC admission.

Study variables

Measurements of body height (stretch stature) to the nearest 0.1 cm with the use of a stadiometer and of body weight to the nearest 5 g recorded on a digital scale were taken when subjects were in a fasted state and immediately after they voided in the morning. Whole-body and regional (trunk, arm, and leg) lean and fat tissue were determined with the use of DXA (DPX-L; Lunar Radiation Corp, Madison, WI). The scans were analyzed with the use of ADULT software, version 1.33 (Lunar Radiation Corp). The calculation of appendicular lean and fat mass was made according to the approach described by Heymsfield et al (25). With the use of specific anatomic landmarks, the legs and arms are isolated on the skeletal X-ray planogram (anterior view). The arm encompasses all soft tissue extending from the center of the arm socket to the phalange tips, and contact with the ribs, pelvis, or greater trochanter is avoided. The leg consists of all soft tissue extending from an angled line drawn through the femoral neck to the phalange tips. The system software provides the total mass, ratio of soft tissue attenuations, and bone mineral mass for the isolated regions. The ratio of soft tissue attenuation for each region was used to divide bone mineral–free tissue of the extremities into fat and lean components. Limb fat and lean tissue were calculated from summed arm and leg fat and lean tissues, respectively.

Subjects spent 23 h in a whole-room respiration calorimeter (3.38-m long, 2.11-m wide, and 2.58-m high) for measurement of total energy expenditure and RMR. The design characteristics and calibration of the calorimeter were described previously (32). Oxygen consumption and carbon dioxide production were continuously measured with the use of a magnetopneumatic differential oxygen analyzer (Magnos 4G; Hartmann & Braun, Frankfurt, Germany) and a nondispersive infrared industrial photometer differential carbon dioxide analyzer (Uras 3G, Hartmann & Braun. The calorimeter was calibrated before each subject entered the chamber. The zero calibration was carried out simultaneously for both analyzers. The full scale was set for 0–1% for the carbon dioxide analyzer and for 0–2% for the oxygen analyzer. Each subject entered the calorimeter at 0800. Although metabolic data were collected throughout the 23-h stay, only RMR data are reported here. Each subject was awakened at 0630 the next morning in the calorimeter. RMR was then measured for 30 min before the subject left the calorimeter at ≈0700. Energy expenditure was calculated by the Weir equation (33). The RMR data were extrapolated over 24 h and expressed as kJ/d.

Statistical analysis

Statistical analyses were performed with the use of SPSS software, version 10.0 (SPSS Inc, Chicago) and SAS software, version 8.0 (SAS Institute, Inc, Cary, NC). Descriptive statistics for the outcome variables were calculated for the total sample and for both racial groups at each study state. Two-way repeated-measures analysis of variance was performed to determine whether there were any statistically significant differences in outcome variables between the races across the 3 study phases. Post hoc tests were run to examine the separate effects of weight loss on the mean values of body composition in each race, with the use of Bonferroni

TABLE 1
Resting metabolic rate (RMR) by race of women in the overweight state (baseline), after weight reduction to a normal body weight, and at follow-up after an average of 1 y without intervention[1]

	Overweight state		Normal-weight state		At follow-up after 1 y		P^2	
RMR	White	Black	White	Black	White	Black	Weight change	Race
Unadjusted (kcal/d)	1522 ± 50	1428 ± 48	1343 ± 33	1274 ± 31	1456 ± 35	1343 ± 35	<0.001	0.05
Adjusted for LBM and FM (kcal/d)	1450 ± 45	1338 ± 43	1387 ± 33	1325 ± 31	1401 ± 32	1306 ± 31	0.70	0.01
Adjusted for trunk LBM, limb LBM, and FM (kcal/d)	1451 ± 48	1340 ± 46	1366 ± 33	1336 ± 31	1376 ± 32	1343 ± 33	0.52	0.94

[1] x ± SEM. LBM, lean body mass; FM, fat mass.
[2] Repeated-measures ANOVA (unadjusted) and analysis of covariance (adjusted) examining independent effects of weight change, race, and their interaction. No race-by-weight change interactions were significant.

corrections for additive alphas. To determine whether RMR, adjusted for body composition variables, was altered with weight loss and weight regain and whether racial differences existed with altered weight states, repeated-measures analysis of covariance (ANCOVA) was performed. A 2 × 2 (time × race) ANCOVA was performed to determine whether there were any statistically significant differences in outcome variables (RMR; raw and adjusted for body composition variables) between the races, between subjects in the overweight and normal-weight states, and between subjects in the normal-weight state and those at 1-y follow-up. Two analyses were required to deal with lack of linearity for change in the adjusting variables (LBM and fat mass) between the 3 time points. Mean values for the normal-weight state are reported in **Table 1**. Bonferroni corrections were made to correct for additive alphas. Significance was set at $P < 0.05$ for all tests. Although a 3 × 2 (time × race) repeated-measures ANCOVA would allow analysis of all 3 time points in one analysis without the use of Bonferroni corrections, a fundamental flaw exists in repeated-measures ANCOVA when ≥3 time points are being examined and the covariate or covariates do not change linearly across time. This flaw occurs because repeated-measures ANCOVA adjustments on the dependent variable mean are made at each time point on the basis of a linear regression of the different covariates across time points. No problem exists with the adjustments when only 2 time points exist, when the covariates do not change across time, or when they change in a linear manner. However, repeated-measures ANCOVA is not capable of appropriately adjusting ≥3 time points when the adjusting variable follows a nonlinear pattern across time points. The covariates in the analysis of RMR (LBM and fat mass) did not change linearly across time in this study (ie, fat mass starts at 31.7 kg, drops to 20.5 kg, and then rises to 27 kg; LBM follows the same pattern). Therefore, two 2 × 2 repeated-measures ANCOVAs with Bonferroni corrections for additive alpha were required for the repeated-measures ANCOVA of RMR.

RESULTS

At baseline (overweight state), there were no significant differences between the white and black women in mean (±SEM) body weight (78.7 ± 5.3 and 78.0 ± 9.0 kg, respectively), BMI (29.1 ± 1.6 and 28.8 ± 1.7, respectively), or percentage body fat (44.7 ± 3.4 and 43.5 ± 3.7, respectively). Furthermore, the white and black women did not differ significantly in age (37.4 ± 5.9 and 35.4 ± 6.0 y, respectively). The duration of weight-loss treatment averaged 0.42 ± 0.11 y and 0.48 ± 0.26 y in the white and black women, respectively ($P = 0.44$). The magnitude of weight loss from baseline did not differ significantly between the races, averaging 13.4 ± 3.6 kg in the white women and 12.7 ± 3.2 kg in the black women (17% and 16%, respectively). The duration of follow-up after assessment in the normal-weight state averaged 0.99 ± 0.48 y and 0.96 ± 0.23 y in the white and black women, respectively ($P = 0.50$). Weight regain averaged 6.1 ± 5.5 kg in the white women and 6.4 ± 5.4 kg in the black women (9% and 10%, respectively). Body weight, percentage body fat, LBM, and fat mass changed significantly (all $P < 0.001$), and, at each of the 3 measurement time points, the white and black women did not differ significantly in these variables (**Table 2**).

Whereas the races did not differ in trunk or limb fat mass, there were significant racial differences in the regional distribution of LBM. Trunk LBM was significantly lower in blacks than in whites, as shown in **Figure 1**. In contrast, limb LBM was significantly greater in blacks than in whites. With weight loss, trunk and limb LBM and fat mass decreased significantly in both groups (Table 2, Figure 1). When the data for black and white women were evaluated collectively, total fat mass but not total LBM increased significantly from the normal-weight state to the 1-y follow-up. Differences were also noted for regional tissue distributions during weight regain. Whereas trunk and limb fat mass and limb LBM increased significantly from the normal-weight state to the 1-y follow-up, trunk LBM remained lower despite the average body weight regain of 6.2 kg.

Absolute RMR values decreased significantly as a function of weight change (Table 1). However, the influence of weight change on RMR was not significant after adjustment for total LBM and fat mass or after adjustment for regional LBM. Racial differences in absolute RMR were of borderline significance ($P = 0.05$), but differences in RMR were evident when adjustments were made for total LBM and fat mass ($P = 0.01$): white women had a significantly higher adjusted RMR. However, this racial difference in RMR was no longer present ($P = 0.94$) after adjustment for regional LBM and fat mass.

DISCUSSION

As we (24) and others (14, 15, 17) found previously, we found in the present study that, matched for height and weight, premenopausal black women have more limb LBM and less trunk LBM than do premenopausal white women. To our knowledge, this is the first study to test the effects of weight loss and regain on the distribution of LBM in premenopausal black and white women. Trunk and limb LBM decreased proportionately in black

TABLE 2
Body composition of 40 women (18 white and 22 black) measured in the overweight state (baseline), after weight reduction to a normal body weight, and at follow-up after an average of 1 y without intervention[1]

Variables	Overweight state	Normal-weight state	At follow-up after 1 y	P^2	
				Weight change	Race
Weight (kg)	78.3 ± 7.5[a]	65.4 ± 6.4[b]	71.6 ± 8.3[c]	<0.001	0.94
BMI (kg/m^2)	29.0 ± 1.7[a]	23.9 ± 1.0[b]	26.3 ± 2.5[c]	<0.001	0.79
Percentage body fat					
(including bone mass)	42.6 ± 3.5[a]	33.1 ± 4.6[b]	39.0 ± 5.3[c]	<0.001	0.70
(soft tissue only)	44.1 ± 3.6[a]	34.4 ± 4.8[b]	37.2 ± 6.5[c]	<0.001	0.27
Total LBM (kg)	40.1 ± 4.0[a]	38.8 ± 4.0[b]	39.2 ± 4.3[b]	<0.001	0.88
Total FM (kg)	31.7 ± 4.4[a]	20.5 ± 4.0[b]	27.0 ± 5.9[c]	<0.001	0.74
Trunk LBM (kg)	19.4 ± 2.2[a]	18.9 ± 2.3[b]	18.7 ± 2.4[b]	<0.001	<0.04
Trunk FM (kg)	14.4 ± 2.9[a]	9.0 ± 2.3[b]	11.8 ± 3.1[c]	<0.001	0.14
Limb LBM (kg)	19.3 ± 2.4[a]	18.5 ± 2.2[b]	19.0 ± 2.4[a]	<0.001	0.02
Limb FM (kg)	17.3 ± 3.1[a]	11.4 ± 2.6[b]	15.2 ± 3.5[c]	<0.001	0.40

[1] x ± SD. LBM, lean body mass; FM, fat mass. Values in the same row with different superscript letters are significantly different, $P < 0.05$ (post hoc tests).
[2] Repeated-measures ANOVA examining independent effects of weight change, race, and their interaction. No race-by-weight change interactions were significant.

and white women with weight loss. However, with weight regain 1 y after the achievement of normal-weight status, the women had regained limb LBM, whereas trunk LBM remained reduced. Thus, an interesting feature of this study was that, despite the absolute differences between the races in the distribution of regional body composition, the black and white women had the same temporal pattern in regional tissue changes during weight loss and weight regain. Further, organ mass is contained within the trunk LBM; by contrast, limb LBM is primarily muscle and represents <75% of total skeletal muscle mass (27). Consequently, the results of the current study suggest that, for both races, a delay may exist in the regain of metabolically active organ mass during the first year of weight regain after weight loss.

LBM is known to be a function of stature and body weight (25, 34). Because the average heights of the black and white women in the current study did not differ by >1 cm and their average body weights were within 1 kg at each time point, it is not surprising that no racial difference was found in total LBM at any measurement point. However, racial differences were found in LBM distribution: trunk LBM was significantly lower and limb LBM was significantly higher in the black women. Previous research noted that, compared with white subjects, black subjects have greater amounts of skeletal muscle (14, 15) and greater appendicular bone lengths (14–16) at any given age across adulthood. Gallagher et al (17) compared black and white adults and found that, whereas there was no racial difference in height, the black subjects had greater leg length relative to their height than did the white subjects. Using the same measurement technique adopted in the current study, Gallagher et al (17) found that, after adjustment for stature and body weight, ethnicity independently determined a person's appendicular skeletal muscle mass and that the black women had more limb skeletal tissue than did the white women. In a study that used DXA to measure racial differences in body composition, Aloia et al (28) found that, after adjustment for height, weight, and age, the black women had significantly greater skeletal muscle mass than did the white women. It was further

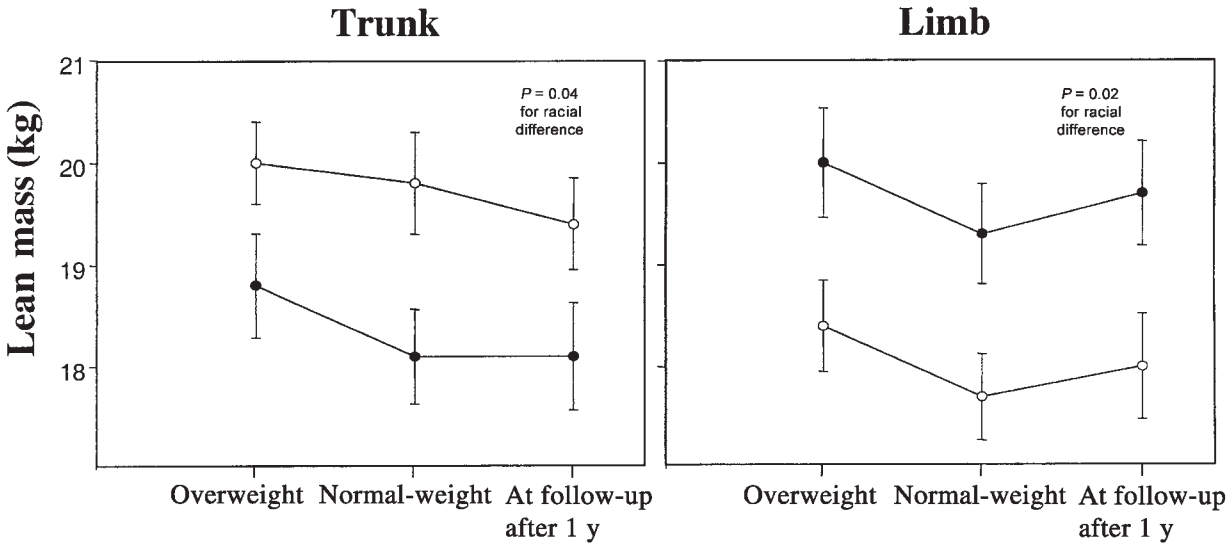

FIGURE 1. Mean (± SEM) trunk and limb lean mass among 18 white (○) and 22 black (●) women measured in the overweight state (baseline), after weight reduction to the normal-weight state, and after an average of 1 y without intervention.

shown that muscle mass as a proportion of total lean tissue was significantly greater in the black women than in the white women. However, the subjects from these studies were of normal weight, and the racial groups differed significantly in age and weight. Data from the current study extend the findings of these previous studies by showing that black and white women who do not differ in stature, weight, and age have significant differences in limb LBM as well as trunk LBM and that these racial differences appear to be inherent and persistent, because they exist throughout the overweight, normal-weight, and weight-regain states.

It has been shown (10, 35, 36) that RMR is lower in blacks than in whites after adjustment for total LBM and fat mass. Consequently, the secondary aim of this study was to investigate what role a racial difference in the regional LBM distribution in weight-matched women might have in explaining possible differences in RMR. Basal, sleeping, and resting metabolic rates are highly correlated with body weight in general and with LBM in particular, because its metabolic rate is higher than that of fat tissue (37). Consequently, the results of the current study have implications for analyses of metabolic rate, especially in comparisons of racial groups. In the current study, the black and white women did not differ in total LBM. However, the lower trunk LBM and higher limb LBM in the black women suggest that they have less organ tissue and more skeletal muscle tissue than do the white women. Organ tissue is more metabolically active than is skeletal muscle tissue during resting conditions, which explains a greater proportion of the variances in RMR (20). These findings suggest that, even when RMR is adjusted for differences in LBM, previously observed racial differences in metabolic rate may still exist because of the divergence in the ratio of organ to skeletal tissue. Research by Sparti et al (23) in 40 normal-weight adults did not support the hypothesis that the composition of the fat-free mass was the main determinant of RMR. However, we have shown cross-sectionally that differences in sleeping energy expenditure and RMR between black and white women may be mediated by proportionately lower trunk LBM (and thus lower organ mass) in the black women (24). This is the first study to test this hypothesis longitudinally.

The data from the current study concur with the findings of Foster et al (10, 11) that racial differences in RMR are evident in weight-matched white and black women even after adjustment for total LBM. However, the current study shows longitudinally for the first time that these racial differences in RMR during weight loss and weight regain can be explained by regional LBM distribution.

To our knowledge, no study has measured regional changes in LBM through a cycle of weight loss and total weight regain. There are a few reports of changes in *total* lean and fat mass and regional fat mass during a weight cycle. Wadden et al (38) studied changes in fat mass, fat-free mass, and waist-to-hip ratio in 12 obese women of unreported ethnicity after the loss of 18.9 ± 0.6 kg (80% fat mass, 20% fat-free mass) and the regain of an average of 19 kg over 2–3 y. Their results indicated that the weight cycle did not increase the deposition of upper body fat or alter the ratio of total fat to lean tissue. Similarly, van der Kooy et al (39), using underwater weighing to calculate percentage body fat and magnetic resonance imaging to measure body-fat distribution, found in obese women that a single cycle of losing 12.1 ± 3.8 kg and then gaining 11.4 ± 5.8 kg did not result in greater body fatness after weight regain than at baseline. Although no data were provided for changes in lean LBM, because body weight and fat mass at follow-up did not differ significantly from those values at baseline, it is reasonable to assume that LBM after weight regain also did not different from baseline values. The length of follow-up varied; it averaged 67 wk, which, again, is longer than that in the current study. Despite the differences in experimental designs, these studies concur that, compared with baseline values, neither total fat mass nor LBM is altered after a period of weight loss and weight regain. Thus, our data support these findings of changes in total lean and fat masses; however, in evaluating regional changes in LBM, we found that trunk LBM did not return to the baseline overweight level, at least on partial weight regain. It is important to note, however, that in the current study design, weight loss was induced by a diet-only intervention. Exercise training was not prescribed, and no attempt was made to alter patterns of physical activity. The alterations in regional body-composition patterns that accompany weight loss in this study may differ from those seen when exercise is prescribed as a treatment modality (40).

In conclusion, whereas racial differences were found in regional body-composition distribution, temporal changes in LBM and fat mass with weight loss and regain did not differ significantly between the races. In both blacks and whites, trunk LBM decreased with weight loss and remained lower at the 1-y follow-up, despite the fact that body weight and body fat mass both rebounded. Furthermore, although racial differences were seen during weight loss and weight regain when RMR was adjusted for total LBM and fat mass, these differences were no longer evident after adjustment for regional LBM distribution. Consequently, these results show that, in comparing energy expenditure between races, adjustment for differences in distribution of LBM may have to be considered.

We express our appreciation to Harry Vaughn and Robert Petri, who provided invaluable technical assistance in the conduct of this study.

All authors contributed to various stages of the study including the design of the experiment, collection of data, analysis of data, and writing of the manuscript. No author had any financial or personal relations with the company or organization sponsoring the research, other than as employees of the University of Alabama at Birmingham.

REFERENCES

1. World Health Organization. Obesity: preventing and managing the global epidemic. Geneva, Switzerland: World Health Organization, 1998.
2. National Heart, Lung, and Blood Institute, National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. Washington, DC: Government Printing Office, 1998.
3. Franz MJ. Managing obesity in patients with comorbidities. J Am Diet Assoc 1998;98:S39–43.
4. Williamson DF, Serdula MK, Anda RF, Levy A, Byers T. Current weight loss attempts in adults: goal, duration, and rate of weight loss. Am J Public Health 1992;82:1251–7.
5. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys, 1960 to 1991. JAMA 1994;272:205–11.
6. Kumanyika SK, Obarzanek E, Stevens VJ, Hebert PR, Whelton PK. Weight-loss experience of black and white participants in NHLBI sponsored clinical trials. Am J Clin Nutr 1991;53(suppl):1631S–8S.
7. Darga LL, Holden JH, Olson SM, Lucas CP. Comparison of cardiovascular risk factors in obese blacks and whites. Obes Res 1994;2:239–45.
8. Yanovski SZ, Gormally JF, Lesser MS, Gwirtsman HE, Yanovski JA. Binge eating disorder affects outcome of comprehensive very low-calorie diet treatment. Obes Res 1994;2:205–12.
9. Sugerman HJ, Londrey GL, Kellum JM. Weight loss with vertical

banded gastroplasty and Roux-Y gastric bypass with selective vs. random assignment. Am J Surg 1989;157:93–102.
10. Foster GD, Wadden TA, Swain RM, Anderson DA, Vogt RA. Changes in resting energy expenditure after weight loss in obese African American and white women. Am J Clin Nutr 1999;69:13–7.
11. Foster GD, Wadden TA, Vogt RA. Resting energy expenditure in obese African American and Caucasian women. Obes Res 1997;5:1–8.
12. Albu J, Shur M, Curi M, Murphy L, Heymsfield SB, Pi-Sunyer FX. Resting metabolic rate in obese, premenopausal black women. Am J Clin Nutr 1997;66:531–8.
13. Jakicic JM, Wing RR. Differences in resting energy expenditure in African-American versus Caucasian overweight females. Int J Obes Relat Metab Disord 1998;22:236–42.
14. Gerace L, Aliprantis A, Russell M, et al. Skeletal differences between black and white men and their relevance to body composition estimates. Am J Hum Biol 1994;6:255–62.
15. Malina RM. Regional body composition: age, sex, and ethnic variation. In: Roche AF, Heymsfield SB, Lohman TG, eds. Human body composition. Champaign, IL: Human Kinetics, 1996:217–55.
16. Aloia JF, McGowan DM, Vaswani AN, Ross P, Cohn SH. Relationship of menopause to skeletal muscle mass. Am J Clin Nutr 1991;53:1378–83.
17. Gallagher D, Visser M, De Meersman RE, et al. Appendicular skeletal muscle mass: effects of age, gender, and ethnicity. J Appl Physiol 1997;83:229–39.
18. Brozek J, Grande F. Body composition and basal metabolism in man: correlation analysis versus physiological approach. Hum Biol 1955;27:22–31.
19. Elia M. The inter-organ flux of substrates in fed and fasted man, as indicated by atrio-venous balance studies. Nutr Res Rev 1991;4:3–31.
20. Elia M. Organ and tissue contribution to metabolic rate. In: Kinney JM, Tucker HN, eds. Energy metabolism: tissue determinants and cellular corollaries. New York: Raven, 1992:61–80.
21. Garby L, Lammert O. Between-subjects variation in energy expenditure: estimation of the effect of variation in organ size. Eur J Clin Nutr 1994;48:376–8.
22. Weinsier RL, Schutz Y, Bracco D. Reexamination of the relationship of resting metabolic rate to fat-free mass and to the metabolically active components of fat-free mass in humans. Am J Clin Nutr 1992;55:790–4.
23. Sparti A, DeLany JP, de la Bretonne JA, Sander GE, Bray GA. Relationship between resting metabolic rate and the composition of the fat-free mass. Metabolism 1997;46:1225–30.
24. Hunter GR, Weinsier RL, Darnell BE, Zuckerman PA, Goran MI. Racial differences in energy expenditure and aerobic fitness in premenopausal women. Am J Clin Nutr 2000;71:500–6.
25. Heymsfield SB, Smith R, Aulet M, et al. Appendicular skeletal muscle mass: measurement by dual-photon absorptiometry. Am J Clin Nutr 1990;52:214–8.
26. Jebb SA, Goldberg GR, Elia M. DEXA measurements of fat and bone mineral in relation to depth and adiposity. In: Ellis KJ, Eastman JD, eds. Human body composition: in vivo methods, models and assessment. New York: Plenum, 1993:115–9.
27. Wang Z, Visser M, Ma R, et al. Skeletal muscle mass: evaluation of neutron activation and dual-energy X-ray absorptiometry methods. J Appl Physiol 1996;80:824–31.
28. Aloia JF, Vaswani A, Mikhail M, Flaster ER. Body composition by dual-energy X-ray absorptiometry in black compared with white women. Osteoporosis Int 1999;10:114–9.
29. Aloia JF, Vaswani A, Ma R, Flaster E. Comparison of body composition in black and white premenopausal women. J Lab Clin Med 1997;129:294–9.
30. Lara-Castro C, Weinsier RL, Hunter GR, Desmond R. Visceral adipose tissue in premenopausal black and white women: longitudinal study of the effects of fat gain, time, and race. Obes Res 2002;10:868–74.
31. Weinsier RL, Hunter GR, Desmond RA, Byrne NM, Zuckerman PA, Darnell BE. Free- living activity energy expenditure in women who are successful and unsuccessful at maintaining a normal body weight. Am J Clin Nutr 2002;75:499–504.
32. Nelson KM, Weinsier RL, Long CL, Schutz Y. Prediction of energy expenditure from fat-free mass and fat mass. Am J Clin Nutr 1992;56:848–56.
33. Weir JB. New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol 1949;109:1–9.
34. Forbes GB. Lean body mass-body fat interrelationships in humans. Nutr Rev 1987;45:225–31.
35. DeLany JP, Bray GA, Harsha DW, Volaufova J. Energy expenditure in preadolescent African American and white boys and girls: the Baton Rouge Children's Study. Am J Clin Nutr 2002;75:705–13.
36. Gannon B, DiPietro L, Poehlman ET. Do African Americans have lower energy expenditure than Caucasians? Int J Obes Relat Metab Disord 2000;24:4–13.
37. Hunter GR, Weinsier RL, Gower BA, Wetzstein C. Age-related decrease in resting energy expenditure in sedentary white women: effects of regional differences in lean and fat mass. Am J Clin Nutr 2001;73:333–7.
38. Wadden TA, Foster GD, Stunkard AJ, Conill AM. Effects of weight cycling on the resting energy expenditure and body composition of obese women. Am J Physiol 1996;270:E363–6.
39. Van der Kooy K, Leenen R, Seidell JC, Deurenberg P, Hautvast JG. Effect of a weight cycle on visceral fat accumulation. Am J Clin Nutr 1993;58:853–7.
40. Janssen I, Ross R. Effects of sex on the change in visceral, subcutaneous adipose tissue and skeletal muscle in response to weight loss. Int J Obes Relat Metab Disord 1999;23:1035–46.