



Comparison of risk factors for obesity in young, nonobese African-American and Caucasian women

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OBJECTIVE: To determine whether specific risk factors for obesity were more evident in young, normal-weight African-American (AA) compared to Caucasian-American (CA) women.

DESIGN: Cross-sectional age-matched study.

SUBJECTS: Young, nonobese, sedentary AA ($n = 13$, 22.5 y of age, 23.6% body fat) and CA women ($n = 11$, 21.5 y of age, 24.0% body fat).

MEASUREMENTS: Aerobic physical fitness (peak VO_2), resting metabolic rate (RMR), resting and submaximal exercise fat oxidation rates, total daily energy expenditure (TDEE) by the doubly-labeled water method, physical activity energy expenditure (PAEE), skeletal muscle glycolytic (phosphofructokinase activity (PFK)) and β -oxidative (β -hydroxy-acyl CoA dehydrogenase (β -HADH)) activity, and insulin sensitivity estimated by the insulin-augmented frequently sampled intravenous glucose tolerance test.

RESULTS: The AA and CA subjects were similar in age, body mass index and body composition, but the AA women exhibited lower peak VO_2 . There were no group differences in RMR adjusted for body composition, or in the rates of submaximal exercise energy expenditure or fat oxidation, and no difference in skeletal muscle β -HADH or PFK activity. The AA women exhibited lower insulin sensitivity and greater acute insulin response to glucose. The mean TDEE for the AA women was only 74% that of the CA women, primarily due to a lower physical activity energy expenditure (AA group: $x \text{ PAEE} = 1246 \pm 438 \text{ kJ/day}$; CA group: $x = 3310 \pm 466 \text{ kJ/day}$).

CONCLUSION: These data indicate that PAEE and its correlates of peak aerobic capacity and insulin sensitivity are lower in young, nonobese AA women compared to their CA counterparts.

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Introduction

African-American (AA) women in the United States have a higher prevalence of obesity than do Caucasian-American (CA) women.¹ Despite the high prevalence of obesity and its untoward sequelae in this group, few studies have sought to identify reasons for the excessive rate of obesity in AA women and, at present, the reasons for the higher prevalence of obesity among AA women are not well understood.²

Low rates of resting energy expenditure,³ spontaneous physical activity,⁴ and 24 h fat oxidation⁵ have been identified as risk factors for subsequent weight

gain in Caucasians and Pima Indians, but it is unclear whether these risk factors hold true for African Americans. Several recent studies^{6–10} report AA women to exhibit lower resting metabolic rates (RMR) in comparison to their Caucasian counterparts. Also, there are numerous studies that have suggested that AA women exhibit low levels of physical activity.^{11–15}

While reports of low RMR and physical activity energy expenditure from questionnaire data in AA women point toward low total daily energy (TDEE),^{12,13} few studies have actually examined TDEE using the doubly labeled water technique in free-living AA women. Carpenter *et al*¹⁶ found that TDEE was approximately 10% lower in older (age > 55 y) AA men and women compared with Caucasians. In a study of obese women, Kushner *et al*¹⁷ found that AA women exhibited lower TDEE values than their CA counterparts, but the difference was not statistically significant. Little is known about TDEE measured by doubly labeled water in young, nonobese AA women.

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Despite reports that lower rates of fat oxidation^{5,18,19} are associated with increased obesity risk, few studies have addressed this issue in AA women. Chitwood *et al*²⁰ found lower resting and submaximal exercise fat oxidation, as determined by respiratory gas exchange, in 11 young AA women compared with 11 CA women living in Mississippi. They concluded that the young AA women had a greater metabolic propensity for obesity than did the white women. In a recent report, Weyer *et al*¹⁰ found that 24 h respiratory exchange ratio (RER) values were significantly higher (indicative of proportionately greater carbohydrate and lower fat oxidation) in AA men compared with CA men, but were not different between AA and CA women. These studies suggest the possibility of ethnic differences in macronutrient partitioning that must be considered when examining reasons for the higher prevalence of obesity in AA women.

Other factors related to obesity development in several studies include high insulin sensitivity and a low capacity for fatty acid oxidation in skeletal muscle. Swinburn *et al*²¹ have identified higher insulin sensitivity as a predictor of subsequent weight gain. The high prevalence of insulin resistance and type 2 diabetes is well documented in obese AA women.^{22,23} However, little is known about insulin sensitivity/resistance in young, normal-weight AA women. Even less information is available about the possible role of skeletal muscle metabolism in contributing to the racial differences in obesity. Ama *et al*²⁴ compared skeletal muscle types, and various phosphagenic, glycolytic and oxidative enzymes in sedentary African males and sedentary Caucasian Canadians. They found the Africans to exhibit a lower percentage of type I fibers and greater activity of phosphagenic and glycolytic enzymes than the Caucasians, suggesting that glucose may be a preferred substrate in these AA males. Duey *et al*²⁵ reported no significant racial differences in the percentages of type I, IIa, or IIb fibers in untrained, college-aged AA and CA females. Nevertheless, with so few studies undertaken to examine skeletal muscle morphology and metabolism in AA and CA women, it is important to pursue this line of investigation.

Given that the aforementioned risk factors for obesity may be more prevalent in those individuals from groups with a higher prevalence of obesity, we examined behavioral and metabolic characteristics related to obesity risk in AA and CA women. Specifically, we sought to determine whether or not young sedentary AA women exhibit lower resting, physical activity and total daily energy expenditure, lower resting and exercise fat oxidation, and whether any racial differences in fat oxidation could be related to insulin sensitivity or skeletal muscle characteristics. We chose to study normal-weight young women, given the possibility that characteristics may be present in nonobese subjects that predispose them to later obesity, but may be 'normalized' as obesity develops.

Methods

Subjects

Volunteers for this study were recruited from the Colorado State University campus and the surrounding Fort Collins area. After screening, 13 young, sedentary African-American (AA) and 12 sedentary Caucasian American (CA) women between the ages of 18 and 28 qualified for participation in the study. In order to qualify for participation, the subjects were required to be able to trace their ethnicity back to the four grandparents. Reasons for exclusion from the study included: pregnancy; tobacco use; insulin-treated diabetes mellitus; ≥ 2 kg weight fluctuation during the previous year, body mass index (BMI) > 29 kg/m²; history of any eating disorders; participation in formal exercise more than twice a week during the past 6 months; history of menstrual irregularities; a history of hypo or hyperthyroidism; and use of any medications that could influence energy or substrate metabolism. After initial screening, one CA study participant with an initial BMI of 27.8 gained > 2 kg during the data collection period, and was thus excluded from the study, leaving only 11 CA study subjects. The study protocol was reviewed and approved by the Colorado State University Human Research Committee and by the Colorado Multiple Institution Review Board at the University of Colorado Health Sciences Center. Subjects underwent the following tests to determine possible group differences in variables related to body weight regulation. Only a subset of subjects from each racial group completed the measures of insulin sensitivity and energy expenditure using the doubly labeled water technique. There were no group differences in age or body composition in these subsets of AA and CA women.

Height and weight

Height was measured using a stadiometer, to the nearest 1.0 mm, with subjects standing without shoes. Weight was measured to the nearest 0.1 kg, with subjects lightly clothed and without shoes. Each subject's BMI was calculated using the equation: weight (kg)/height (m)².

Body density

Body density and percentage body fat were measured by hydrostatic weighing, using the Siri equation.²⁶ Dry weight was measured on land with a Detecto Platform scale to the nearest 0.1 kg, and submerged weight was measured in water, adjusted for water temperature using an overhead spring scale, to the nearest 25 g. Each subject performed 7–10 trials to ensure accurate measurements. The average of the three heaviest trials was used to obtain underwater weight. Residual lung volume was determined by the method of Wilmore.²⁷

Peak oxygen consumption

Peak aerobic capacity was measured using a CardiO₂ cycle ergometer (Medical Graphics Corporation, St Paul, MN). Using a standard protocol, subjects performed a graded exercise test to volitional exhaustion. Subjects warmed up at 25 W for 3 min, then the work load was increased by 25 W every minute until subjects could not continue pedaling. During the test, heart rate was recorded using a Burdick E350 3-lead ECG (Siemens Burdick, Milton, WI). Respiratory exchange ratio (RER), VO₂ and VCO₂ were determined via respiratory gases collected on a Sensor-medics MMC Horizon metabolic cart (Sensor-medics, Yorba Linda, CA). The test was considered maximal if two of three criteria were satisfied. The criteria were: RER greater than 1.05; no increase in oxygen consumption even with increased workload; and reaching a maximum heart rate greater than 90% of the age-predicted maximum.

Resting metabolic rate

Resting metabolic rate (RMR) was measured by indirect calorimetry using a Sensor-medics MMC Horizon metabolic cart (Sensor-medics, Yorba Linda, CA). Subjects reported to the laboratory early in the morning following an overnight fast of 12 h. Each subject lay supine on a bed with her head enclosed in a ventilated hood for 45 min during which time she rested quietly with a minimum of movement and without falling asleep. The first 15 min were used as an adaptation period to the hood, and the last 30 min for data collection. The expired air was analyzed for oxygen and carbon dioxide. Metabolic rate was then calculated using the deWeir equation.²⁸ The metabolic cart was calibrated before each RMR test using known concentrations of calibration gases.

Submaximal exercise test

Once the RMR test was completed, the metabolic cart was changed to exercise mode and was recalibrated using gases of known oxygen and carbon dioxide concentration (16% O₂ and 4% CO₂). Subjects exercised on a CardiO₂ cycle ergometer (Medical Graphics Corporation, St Paul, MN) for 5 min at 25 W, to warm up, followed by three 15 min periods at progressively higher power outputs (25, 50 and 75 W). Subjects exercised for 15 min at each intensity level to help ensure that a steady-state condition was achieved at these low absolute exercise intensities. RER and VO₂ values from the last 10 min of each 15 min period were used in calculating substrate oxidation rates. To familiarize the subjects to this testing procedure, all study participants completed a trial run of this exercise protocol prior to the actual test.

To minimize the confounding effects of antecedent diet on substrate utilization during the exercise tests, subjects consumed a controlled diet for 3 d prior to the submaximal exercise tests. The diet consisted of 55%

of the energy as carbohydrate, 15% protein and 30% fat. Total energy intake varied for each subject based on individual energy requirements. After consuming the diet for 3 days, subjects reported to the laboratory on the fourth day after an overnight fast to perform the submaximal exercise test.

Muscle biopsy

All subjects underwent a muscle biopsy, performed at University Hospital, Denver, Colorado. The muscle tissue was analyzed for activity of 3-hydroxyacyl-CoA dehydrogenase (HADH), and phosphofructokinase (PFK). The tissue sample was obtained from the right vastus lateralis muscle. An incision (8 mm long), was made about 12–16 cm above the patella, and 1–2 inches from midline of the midlateral thigh. The subject arrived at the hospital after an overnight (12 h) fast. The biopsy was performed with the subject lying in a supine position. A Bergstrom needle was inserted at a 90° angle to the leg. Approximately 50–100 mg of muscle tissue was obtained from each subject. Samples were immediately frozen in liquid nitrogen then wrapped in foil and frozen at –70°C until assayed.

Skeletal muscle enzyme assays

The skeletal muscle tissue obtained from the biopsy was homogenized in a 0.1 M K₂HPO₄ buffer, 1:10 wt/vol then diluted to a 1% (PFK) or a 5% (HADH) vol/vol concentration. The dilution mixture was 40 mM K₂HPO₄, glycerin and mercaptoethanol in solution. The HADH assay was performed according to the methods described by Gayles *et al.*²⁹ A 0.1 ml homogenate was added to 0.9 ml reaction mixture containing 500 mM EDTA, 2 mM NADH, 1.67 M triethanolamine, and 1 mM acetoacetyl CoA. A spectrophotometer was used to measure the disappearance of NADH for 5 min at 340 nm. All samples were measured in duplicate to ensure reliability. Phosphofructokinase activity from the vastus lateralis muscle was also measured using an assay described by Gayles *et al.*²⁹ An aliquot (0.03 ml) of homogenate was added to 0.87 ml of reaction mixture containing 1 M Tris (hydroxymethyl) aminomethane–HCl, 0.1 M MgCl₂, 2 mM NADH, fructose-6-phosphate (F6P), 5.5 U aldolase, 53 U triose phosphate isomerase, and 6.7 U alpha-glycerophosphate dehydrogenase. The reaction was initiated by adding 0.1 ml of 20 mM ATP to three concentrations of F6P (0.1, 0.4 and 2 mM). The reaction was followed for 17 min as the disappearance of NADH at 340 nm. Data from the enzyme analyses are presented as absolute reaction rates (μmol/(g (min))) and as the ratio of PFK/HADH, used to define glycolytic vs beta-oxidative capacity.

Frequently sampled intravenous glucose tolerance test

A subset of study subjects (*n* = 9 AA, *n* = 8 CA) volunteered for measures of insulin sensitivity. The

groups were similar in age and body composition, as shown in Table 3. An insulin-augmented frequently sampled intravenous glucose tolerance test (IVGTT) was administered to these subjects following an overnight, 12 h fast. All glucose tolerance tests were performed at the General Clinical Research Center at the University of Colorado Health Sciences Center in Denver. The day prior to the test, subjects consumed a standardized diet, which included at least 200 g of carbohydrate. The test was performed with the subject in a supine position, after a 30 min relaxation period. An intravenous (i.v.) catheter was placed in each antecubital vein, one for the administration of insulin and glucose, and one for collecting frequent blood samples. Four blood samples were obtained at time = -5, -2, -1 and 0 min, with the mean of these four samples used as the baseline insulin and glucose levels. At time zero, a bolus of glucose (0.3 g/kg in a of 50% dextrose solution) was infused over a 1 min period. At $t = 20$ min, intravenous insulin (0.03 U/kg) was infused over 30 s. Blood samples were obtained at $t = 2, 3, 4, 5, 6, 8, 10, 12, 14, 16, 19, 22, 23, 24, 25, 27, 30, 40, 50, 60, 70, 90, 100, 120, 140, 160$ and 180 min and immediately centrifuged at 4°C then stored at -20°C until assayed. Serum glucose concentrations were measured by the glucose oxidase method using a glucose autoanalyzer (Beckman Instruments, Fullerton, CA). Serum insulin levels were measured by standard, double-antibody, radioimmunoassay techniques. Insulin and glucose values from the IVGTT were entered into the MINMOD program (R Bergman, University of Southern California) for determination of insulin sensitivity, acute insulin response to glucose and glucose effectiveness. This model uses measurements of plasma glucose and insulin concentrations to deduce *in vivo* insulin sensitivity.³⁰ The parameters are representative of the efficiency of glucose removal independent of the effect of insulin (glucose effectiveness, S_g), and of sensitivity of tissues to the effect of insulin (insulin sensitivity index, S_i).

Total daily energy expenditure measured by doubly labeled water

Ten AA and 8 CA study participants volunteered to participate in measurement of energy expenditure by the doubly labeled water procedure. One AA subject had a TDEE value that was more than 3 standard deviations from the group mean when her data were included, and 9 standard deviations from the mean value when her data were not included in determination of the group mean and standard deviation; thus the doubly labeled water results for this subject were not used in the analysis. On day 1, each subject completely emptied her bladder before reporting to the testing location. A baseline urine sample was collected before the loading isotope was given. Subjects were then weighed as accurately as possible. Isotope doses were measured and administered to

subjects based on the following calculations: 0.20 g²H₂/kg body weight and 0.07 g ¹⁸O/kg body weight. A 0.5 g sample of the final isotope mixture was stored for later analysis. Subjects stayed in the study center for the next 6 h (isotope equilibration period), during which time no food or drink, other than water, was ingested and subjects remained sedentary. The first urine sample was collected at 6 h post-loading dose. Subjects departed from the testing center and resumed normal daily activities. The second urine sample was collected 24 h after first sample was collected. A subsequent urine sample was collected on day 14 at approximately the same time of day. Samples were analyzed by mass spectrometry at the University of Alabama-Birmingham Department of Physiology and Metabolism. Samples were analyzed in triplicate for ²H₂O and H₂¹⁸O using the off-line zinc reduction method and a modification of the CO₂ equilibration technique. In the modified CO₂ equilibration technique 1.0 ml aliquots were equilibrated with 0.5 ml 99.9% pure CO₂ gas. The samples were allowed to equilibrate overnight at room temperature by shaking. The CO₂ gas was analyzed by injecting the gas into a packed gas chromatography column before introduction to the isotope ratio mass spectrometer. Isotope turnover rates and zero-time enrichments were calculated as previously described³¹ and converted to energy expenditure using the food quotients calculated from each subject's dietary record.²⁸ Total body water was determined for body composition measurements. There were no group differences in total body water, percentage body fat and fat-free weight in this subset of study subjects.

Those subjects who were dosed with doubly labeled water were asked to record all food and beverage intake (except water) for four consecutive days, which encompassed two weekdays and two weekend days, during the 14 d of the doubly labeled water test. They were instructed to complete the diet record with as much accuracy as possible, recording serving sizes, name brands) and method of preparation. Energy and macronutrient intake was determined by the Food Intake Analysis System (University of Texas Health Sciences Center, Houston).

Physical activity energy expenditure

Physical activity energy expenditure (PAEE) for each subject was based on subtraction of the measured RMR and the estimated thermic effect of food from the measured total energy expenditure. The thermic effect of food for each subject was estimated to be 10% of daily energy expenditure. The specific formula used for the calculation was:³¹

$$\begin{aligned} \text{PAEE} &= \text{TEE} - (\text{RMR} + \text{TEF}), \text{ where TEF} \\ &= \text{TEE} * 0.10 \end{aligned}$$

Additionally, a physical activity index (PAI) was calculated as TEE/RMR.

Statistical analyses

All data were analyzed using the Statistical Package for Social Sciences (SPSS, Chicago, IL). Data were initially analyzed for normality using the Kolmogorov–Smirnov test. The only variable not passing the normality test was the PFK/HADH enzyme ratio, which was then analyzed using the Mann–Whitney test for nonparametric data. Unpaired *t*-tests were used to analyze all anthropometric, fitness, REE and enzyme data comparing AA and CA groups. Unpaired *t*-tests were also used to analyze differences in resting fat oxidation, oxygen consumption and respiratory exchange ratios, and for total daily and physical activity energy expenditure. Changes in submaximal exercise, and post-exercise fat oxidation, oxygen consumption and RER, for both AA and CA women, were analyzed using an analysis of variance with repeated measures with fitness level serving as a covariate (ANCOVA). *Post-hoc* analyses were used to determine significant group mean differences. Student's unpaired *t*-tests were also used to compare insulin and glucose data from the IVGTT. Where appropriate, simple and partial correlational analyses were used to examine relationships among variables including measures of insulin sensitivity, TDEE, PAEE, PAI and peak VO_2 . Values are presented as mean values \pm s.e.m. Statistical significance was set at $P < 0.05$.

Results

Physical characteristics

Table 1 reveals that the AA and CA study participants were young, nonobese women of similar age and anthropometric characteristics, including body weight, body mass index (BMI), percentage body fat, and fat-free weight (FFW). The AA women exhibited a significantly lower mean peak VO_2 (both absolute and relative to body weight or fat-free weight) compared with the CA women (Table 2). The CA women also had a lower mean heart rate at peak VO_2 , and attained a lower percentage of predicted maximal heart rate than the CA women. There were no group differences in skeletal muscle enzyme activity for either β -HADH or PFK. The ratio of PFK

Table 1 Physical characteristics of African-American (AA) and Caucasian-American (CA) women^a

Anthropometric and skeletal muscle characteristics	AA woman (n = 13)	CA women (n = 11)	P value
Age (y)	22.5 \pm 0.7	21.5 \pm 0.9	0.37
Weight (kg)	60.1 \pm 2.4	59.3 \pm 2.2	0.82
BMI (kg/m ²)	22.5 \pm 0.8	23.0 \pm 0.8	0.66
Body fat (%)	23.56 \pm 1.41	23.96 \pm 1.98	0.87
FFW (kg)	45.66 \pm 1.61	45.07 \pm 1.21	0.78

Abbreviations: BMI, body mass index; FFW, fat-free weight.
^a $\bar{x} \pm$ s.e.m.

Table 2 Peak exercise test variables and skeletal muscle enzyme activity in African-American (AA) and Caucasian-American (CA) women^a

Measurement	AA women (n = 13)	CA women (n = 11)	P value
VO_2 peak (l/min)	1.59 \pm 0.09	1.90 \pm 0.06	0.011
(ml/(kg min))	26.02 \pm 0.91	32.24 \pm 1.21	0.001
(ml/(kgFFW min))	34.77 \pm 1.24	42.21 \pm 1.34	0.001
Heart rate at VO_2 peak	168.6 \pm 3.2	179.2 \pm 2.6	0.02
RER at VO_2 peak	1.17 \pm 0.02	1.18 \pm 0.01	0.68
HADH ($\mu\text{mol}/(\text{g min})$)	1.46 \pm 0.18	1.47 \pm 0.29	0.97
PFK ($\mu\text{mol}/(\text{g min})$) ^b	62.69 \pm 3.32	65.33 \pm 4.84	0.65
PFK/HADH ^b	58.90 \pm 10.49	66.27 \pm 17.66	0.72

Abbreviations: RER, respiratory exchange ratio; PFK, phosphofructokinase; HADH, β -hydroxy-acyl-CoA dehydrogenase.

^a $\bar{x} \pm$ s.e.m.

^bPFK and PFK/HADH values based on 11 AA.

to β -HADH was also not significantly different between groups.

Resting metabolic rate and resting fat oxidation

The mean RMR was about 3–4% lower in the AA women compared with CA women, but this difference did not reach statistical significance, either expressed in absolute terms (AA = 5069 \pm 132, CA = 5236 \pm 175 kJ/day) or standardized for fat-free weight using ANCOVA (AA = 5052 \pm 118, CA = 5256 \pm 127 kJ/day). The mean RER obtained under resting conditions was significantly lower for the AA women than the CA women (AA = 0.87 \pm 0.01, CA = 0.90 \pm 0.01). The mean rates of resting fat oxidation were calculated from the resting VO_2 and RER data, and were significantly higher ($P < 0.05$) in the AA women (AA = 38.4 \pm 3.6 mg/min, CA = 28.21 \pm 3.07 mg/min).

Submaximal exercise and post-exercise data

A repeated measures analysis of covariance was used to examine oxygen uptake (VO_2) during the exercise and post-exercise periods, with group and time as the independent variables, and peak $\dot{\text{V}}\text{O}_2$ as the covariate. There was no significant group by time interaction, indicating that the AA and CA women had similar VO_2 values during the three different submaximal exercise intensities, and recovery period (Figure 1). As shown in Figure 1, the subjects' rates of oxygen consumption had returned to baseline values by the 45th min post-exercise.

A similar analysis was used to examine the RER values during the time periods of exercise and post-exercise recovery. As previously indicated, the RER values were significantly lower for the AA compared with CA women under resting conditions. During exercise there were no group differences in RER values, but the AA women exhibited significantly lower mean RER values during the last 30 min of the recovery period (Figure 2).

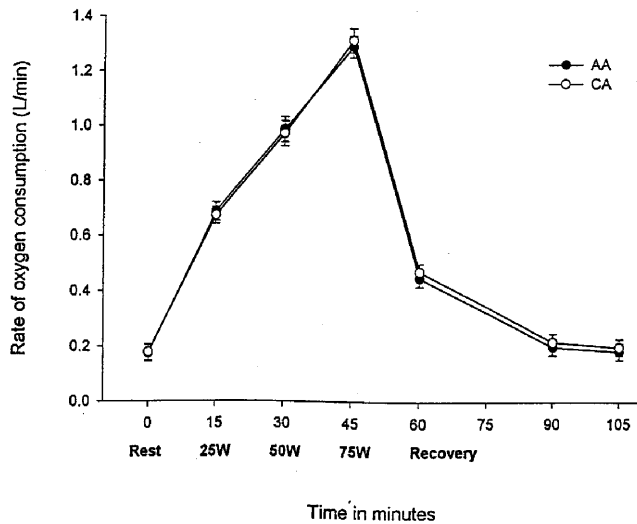


Figure 1 Resting, exercise (25, 50, 75 W), and post-exercise oxygen consumption in African-American (AA, $n = 13$) and Caucasian-American (CA, $n = 11$) women. There were no statistically significant group differences in oxygen consumption during any of the measured time periods.

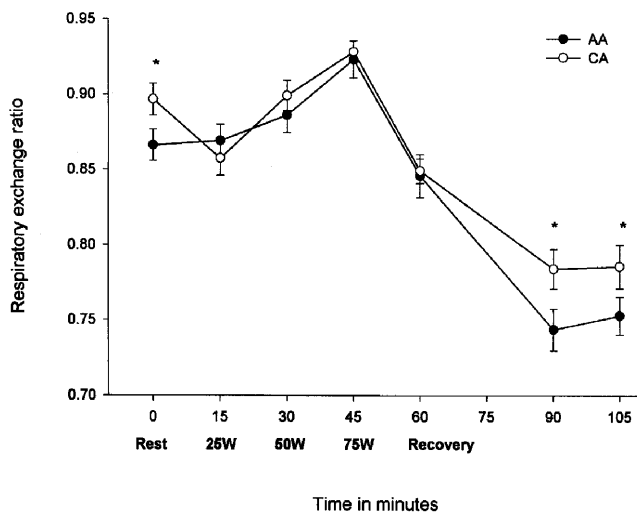


Figure 2 Resting, exercise (25, 50, 75 W), and post-exercise respiratory exchange ratio (RER) in African-American (AA, $n = 13$) and Caucasian-American (CA, $n = 11$) women. *RER for AA < CA ($P < 0.05$).

Insulin sensitivity

Table 3 shows the physical characteristics and the results of the insulin-augmented frequently sampled intravenous glucose tolerance test conducted in a subset of study participants. None of the women had S_i values of zero. Despite group similarities in anthropometric characteristics, the AA women had significantly lower insulin sensitivity values than CA women, indicating greater insulin resistance. The AA women also had a significantly higher acute phase insulin response to glucose (AIR_g) and lower glucose effectiveness (S_g) values compared with the CA women. There were no significant group differences in fasting insulin and glucose. Insulin sensitivity was unrelated to resting and exercise substrate oxidation rates when examined by correlational analysis.

Energy expenditure

Table 4 shows the physical characteristics and the components of energy expenditure in those young African American and Caucasian women who were studied using doubly labeled water. Total daily energy expenditure and PAEE were significantly lower ($P < 0.05$) in African Americans than Caucasians. The calculated physical activity index [$PAI = (PAEE/RMR)$] was also lower in the AA women compared with CA women (1.39 vs 1.8).

Subjects who participated in the doubly labeled water portion of this study remained weight stable during the 14 d measurement period. Despite sizeable differences in TDEE, the AA and CA women reported similar energy intakes (6801 ± 519 and 6910 ± 310 kJ/day, data not shown in table format), suggesting that the CA women underreported dietary energy intake more than did the AA.

The relationships between peak VO_2 and various measures of energy expenditure were examined in the entire subsample of AA and CA women combined using correlation analyses. Because body weight and FFW are related to both peak VO_2 and to energy expenditure, partial correlation coefficients were calculated controlling for body weight and FFW. Peak

Table 3 Insulin sensitivity, glucose effectiveness and acute insulin response to glucose based on the frequently sampled intravenous glucose tolerance test in a subsample of African-American (AA) and Caucasian-American (CA) women^a

Measurement	AA women ($n = 9$)	CA women ($n = 8$)	<i>P</i> value
Age (y)	22.7 ± 0.9	21.9 ± 1.2	0.60
Weight (kg)	58.1 ± 3.2	59.7 ± 2.8	0.70
BMI (kg/m ²)	21.7 ± 1.0	22.5 ± 0.8	0.55
Body fat (%)	23.15 ± 1.5	24.6 ± 2.59	0.63
VO_2 peak (ml/(kg min))	26.63 ± 0.96	31.29 ± 1.22	0.01
Insulin sensitivity, $S_i \times 10^4$ (μU /(ml min))	3.26 ± 0.34	4.60 ± 0.47	0.03
Glucose effectiveness, $S_g \times 10^2$ (min^{-1})	2.13 ± 0.31	1.40 ± 0.23	0.08
Acute insulin response to glucose (pmol/(mL min))	643.56 ± 67.89	296.56 ± 48.45	0.001
Fasting insulin (μU /ml)	7.8 ± 0.97	6.0 ± 0.50	0.14
Glucose (mmol/l)	5.07 ± 0.14	4.84 ± 0.14	0.25

^a $\bar{x} \pm$ s.e.m.

Table 4 Total daily and physical activity energy expenditure in a subset of the larger sample of African-American (AA) and Caucasian-American (CA) women^a

Measurement	AA women (n = 9)	CA women (n = 8)	P value
Age (y)	22.5 ± 0.9	21.9 ± 1.2	0.65
Weight (kg)	58.4 ± 2.1	59.8 ± 2.7	0.70
BMI (kg/m ²)	21.8 ± 0.8	22.5 ± 0.8	0.56
Body fat (%)	23.2 ± 1.5	24.6 ± 2.6	0.64
VO _{2 peak} (ml/(kg min))	25.8 ± 1.2	31.4 ± 1.2	0.005
RMR (kJ/24 h) ^b	5052 ± 118	5257 ± 127	0.35
TDEE (kJ/24 h) ^c	6963 ± 330	9474 ± 847	0.02
TEF (kJ/24 h)	696 ± 33	948 ± 85	0.01
PAEE (kJ/24 h) ^c	1246 ± 438	3310 ± 466	0.006
PAI	1.39 ± 0.05	1.80 ± 0.12	0.006

Abbreviations: TDEE, total daily energy expenditure; TEF, thermic effect of food; PAEE, physical activity energy expenditure; PAI, physical activity index based on (PAEE/REE).

^ax ± s.e.m.

^bRMR values after adjustment for fat-free weight and fat weight.

^cTDEE and PAEE values after adjustment for body weight.

VO₂ was significantly positively correlated with TDEE (partial $r=0.57$, $P<0.05$), PAEE (partial $r=0.57$, $P<0.05$), and the PAI (partial $r=0.53$, $P<0.05$).

Discussion

In the present study, specific metabolic and behavioral characteristics were compared in young, normal-weight women, prior to any anthropometric manifestations of obesity to determine whether specific risk factors for obesity were more evident in AA than CA women. The major finding was that the PAEE was significantly lower in AA compared to CA women. Additionally, the PAEE correlates of peak aerobic capacity and insulin sensitivity were lower in young, nonobese AA women compared with their CA counterparts.

Total and physical activity energy expenditure

Based on questionnaire data, many studies have suggested that African American women exhibit low levels of exercise^{10–13} which probably contributes to low TDEE. Only a few studies have examined TDEE and PAEE in free-living AA using the doubly labeled water approach. Kushner *et al*¹⁷ found that obese AA women exhibited lower TDEE values than their white counterparts, but the difference was not statistically significant. Carpenter *et al*¹⁶ reported TDEE in older African Americans (age ≥ 55 y) to be about 10% lower than older Caucasians, and concluded that the lower TDEE in older AAs was the result of lower RMR and PAEE. Neither of these studies examined differences in total energy expenditure among young, nonobese African American and Caucasian women. In our study, the TDEE of the AA women was more than 30% lower than that of the CA women, primarily the

result of lower PAEE. The CA group was more active than might be expected of very sedentary individuals (total daily energy expenditure/RMR = 1.8 compared to values of 1.4–1.7 typical of sedentary individuals). Still it is apparent that the AA women expended little energy beyond that required of their resting needs, based on their calculated PAI of 1.39.

The lower PAEE in the AA women was associated with lower cardiorespiratory fitness (peak VO₂) despite the low levels of purposeful leisure-time exercise in both groups. We cannot rule out the possibility of selection bias, wherein CA women were recruited who were relatively more physically active than the AA women, despite the fact that purposeful leisure-time exercise was low in both groups and there were no group differences in reported levels of such exercise. It appears that, within our definition of sedentary, AA women expend substantially less PAEE, most likely the result of less nonexercise activity thermogenesis (NEAT, which refers to fidgeting, maintenance of posture, and other physical activities of daily life). Levine *et al*³² recently found the magnitude of NEAT to be quite variable in overfed men and women. For some individuals, NEAT contributed significantly to TDEE and high levels of NEAT provided a substantial buffer against weight gain during the overfeeding period. Our data suggest the strong possibility that NEAT is lower in nonexercising AA compared to CA women, a phenomenon that could place AA women at higher risk for weight gain. This issue needs further exploration.

Estimates of insulin sensitivity

It is clear that obesity among AA women is correlated with insulin resistance and with the higher prevalence of type 2 diabetes compared with CA women.²³ In the present study, the AA women exhibited lower insulin sensitivity and greater acute phase insulin response to glucose, despite similarities in body mass and body composition in the two groups. These findings are similar to those of Jiang *et al*³³ and Svec *et al*,³⁴ in which young black girls displayed lower insulin sensitivity than their white counterparts. Despite these findings, it is unclear that the lower insulin sensitivity in young black women contributes to greater risk for obesity. In contrast, it has hypothesized that insulin resistance helps limit weight gain.²¹

Resting and exercise metabolism

There were no group differences in resting and sub-maximal exercise energy metabolism that shed light on the higher risk for obesity in AA compared to CA women. Several previous studies^{6–10} found African-American women to exhibit lower RMR values than Caucasian women. In the present study, although the mean adjusted RMR was about 4% (204 kJ/day).

lower in the AA compared with CA women, this difference did not reach statistical significance. Lack of adequate statistical power could explain lack of significant group differences, although our sample size was similar to that of Chitwood *et al*,²⁰ who found lower RMR values in young AA compared with CA women. Likewise, in our study there were no group differences in VO_2 and RER during exercise at 25, 50 and 75 W and following exercise. The lack of group differences in the activity of skeletal muscle enzymes involved in carbohydrate utilization and oxidation of fatty acids coincides with lack of differences in exercise substrate oxidation rates. These findings do not support the hypothesis that AA women oxidize less fat than CA women during submaximal exercise and recovery from exercise, and are in opposition to those of Chitwood *et al*,²⁰ who reported AA women to exhibit lower rates of fat oxidation during and after submaximal exercise. Reasons for the discrepant findings are not readily identifiable. In the present study we controlled diet during the several days prior to the exercise bout in an attempt to eliminate any confounding influences on substrate oxidation rates of group differences in energy balance and macronutrient intake.

There are two caveats to the present study that should be mentioned. First, menstrual cycle phase may have a small effect on energy expenditure and substrate oxidation rates.³⁵ Given the large number of tests performed by each subject, we were unable to standardize each test for the same menstrual cycle phase for all subjects. We assumed that the distributions of women in the pre- and post-ovulatory phases were similar across ethnic groups. However, random differences in the menstrual cycle phase between the two groups could have occurred. Even with this possible caveat, it should be noted that Jakicic and Wing⁸ found no effect of menstrual cycle phase on either RMR or resting RER in AA or CA women. A second issue is the possibility that AA women who are able to maintain normal weight and body composition into early adulthood (as was true of our study participants) exhibit metabolic characteristics that are different from those of their counterparts who gain weight during adolescence and early adulthood. Our study of normal weight women would then tend to potentially minimize group differences in measured metabolic characteristics. Nevertheless, given the use of a study design that would potentially minimize group differences, it is even more striking that AA women were found to exhibit lower aerobic capacity, TDEE, PAEE and insulin sensitivity than their CA counterparts.

Conclusions

Data from this study indicate that PAEE and its correlates of peak aerobic capacity and insulin sensi-

tivity are lower in young, nonobese AA women compared to their CA counterparts. Longitudinal studies are necessary to identify whether or not these specific behavioral and metabolic characteristics of African-Americans predict weight gain, so that appropriate strategies can be devised to lower the prevalence of obesity and its untoward sequelae in this population.

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