Lifestyle Intervention of Hypocaloric Dieting and Walking Reduces Abdominal Obesity and Improves Coronary Heart Disease Risk Factors in Obese, Postmenopausal, African-American and Caucasian Women

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Background. There are few empirical data to support the claim that weight loss improves coronary heart disease (CHD) risk factors in postmenopausal women; nor is it known if there are racial differences in changes of body fat distribution, lipids, glucose tolerance, and blood pressure with weight loss. This study determined the efficacy of a lifestyle weight loss intervention in reducing total and abdominal obesity and improving CHD risk factors in obese Caucasian and African-American postmenopausal women.

Methods. Body composition (dual-energy x-ray absorptiometry), abdominal fat areas (computed tomography scan), lipoprotein lipids, insulin, glucose tolerance, and blood pressure were measured before and after 6 months of hypocaloric diet and low-intensity walking in 76 overweight or obese (body mass index $> 25$ kg/m$^2$), Caucasian (72%) or African-American (28%), postmenopausal (age = 60 ± 5 years) women who completed the study.

Results. Absolute amount of body weight lost was similar in Caucasians (−5.4 ± 3.6 kg) and African Americans (−3.9 ± 3.6 kg), but Caucasian women lost relatively more fat mass ($p < .05$). Both groups decreased their subcutaneous abdominal fat, and Caucasian women decreased their visceral fat area, but there were no racial differences in the magnitude of abdominal fat lost. The intervention decreased triglyceride and increased high-density lipoprotein and high-density lipoprotein 2 cholesterol in both races, and it decreased total and low-density lipoprotein cholesterol in Caucasian women ($p < .05$). Fasting glucose and glucose area during the oral glucose tolerance test decreased ($p < .0001$) in Caucasian women, whereas insulin area decreased in both Caucasian ($p < .01$) and African-American ($p < .05$) women. Blood pressure decreased the most in women with higher blood pressures at baseline. Changes in lipids, fasting glucose and insulin, their responses during the oral glucose tolerance test, and blood pressure were not different between racial groups.

Conclusions. Weight loss achieved through a lifestyle intervention of energy restriction and increased physical activity is an equally effective therapy in African-American and Caucasian obese, postmenopausal women for improving glucose and lipid CHD risk factors.

Obesity is an independent, modifiable risk factor for coronary heart disease (CHD), the leading cause of morbidity and mortality in middle-aged and older women (1). Overweight and obesity are also strongly related to other major risk factors for CHD, including hypertension, hypercholesterolemia, low high-density lipoprotein (HDL) cholesterol, hyperinsulinemia, and impaired glucose tolerance (2,3). The prevalence of obesity increases with aging and, in 1998, nearly two thirds of women aged 50–69 years were either overweight (body mass index, or BMI = 25–29.9 kg/m$^2$) or obese (BMI $> 30$ kg/m$^2$) (4). Besides an increase in total body weight, there is a redistribution of fat to the abdominal region that occurs in women with age and menopause (5). An excess of stored fat in the abdomen, especially around the visceral organs, independently increases risk for CHD (6), type 2 diabetes (7), and premature mortality (8). As a result, the majority of women in this age group are at a greater risk of morbidity and mortality than women of other age groups.

There are racial differences in the prevalence of obesity and the distribution of body fat that may contribute to racial disparities in the incidence of CHD and diabetes. African-American women have a twofold greater incidence of obesity than Caucasian women (4) and, on average, they gain more weight during the menopausal years than Caucasian women (9). In contrast, African-American women have a lower amount of visceral adipose tissue, despite total adiposity and waist–hip ratios that are comparable with Caucasian women (10–12). However, there are little data regarding whether there are racial differences in the effects of weight loss on body composition, body fat distribution, and CHD risk factors. This study provides data for the preliminary comparison of changes in abdominal adipose tissue and CHD risk factors in response to a diet intervention in Caucasian and African-American women.

Weight loss, achieved through hypocaloric dieting and increases in physical activity, is advocated as an effective, nonpharmacological method of reducing risk of CHD.
Dietary control.—As a way to establish dietary control prior to metabolic testing, and to eliminate changes in dietary composition as a confounder of metabolic changes with weight loss, all women met weekly with a registered dietitian for 8–10 weeks for instruction in the principles of the American Heart Association (AHA) Step 1 diet (17). Subjects were weight stable and compliant to this diet for at least 2 weeks prior to and throughout the period of baseline data collection. The dietitian monitored compliance by weekly review of 7-day food records and 24-hour dietary recalls.

Experimental protocol.—Measurements of body composition, body fat distribution, maximal aerobic capacity (VO₂max), lipoprotein lipids, and glucose tolerance were performed on two mornings after at least 2 weeks of weight stability before the lifestyle intervention and again after at least 2 weeks of weight maintenance following the intervention. A fasting blood sample was drawn on both testing days to provide a duplicate measurement of lipoprotein lipids, and the values reported are the average of these 2 days. If the total cholesterol and triglyceride values differed by more than 5%, or the HDL cholesterol values differed by more than 10%, a third sample was drawn, and the values reported are the average of three samples.

Lifestyle intervention.—During the 6-month intervention, all subjects met weekly in a group with a registered dietitian for instruction in the principles of a hypocaloric diet (250–350 kcal/d deficit) that followed the AHA guidelines. The dietary instruction focused on eating behavior, stress management, control of portion sizes, and modification of binge eating and other adverse habits, and it also encouraged low-intensity walking 3 days/wk for 30–45 minutes. The women walked 1 d/wk on a treadmill at our exercise facility at 50–60% heart rate reserve under the supervision of an exercise physiologist, and they were instructed to walk 2 d/wk on their own. This amount of exercise remained constant throughout the 6-month intervention. After the intervention, the women were weight stabilized (<0.5 kg change) on a eucaloric diet for a period of 2 weeks prior to retesting. They had to be weight stable on this diet for at least 2 weeks before they were allowed to be tested. They maintained their 3 d/wk of walking during this period, and the metabolic testing was performed at least 36 hours after a bout of walking.

Testing procedures.—For body composition, waist (minimal circumference) and hip (maximal gluteal protuberance) circumferences were measured in duplicate. Percent body fat, fat-free (bone and lean tissue) mass, and fat mass were measured by using dual energy x-ray absorptiometry (Model DPX-L, Lunar Radiation Corporation, Madison, WI). A single-slice computed tomography (CT) scan taken midway between L4 and L5 was performed by using a GE Hi-Light CT scanner (General Electric, Milwaukee, WI) to measure abdominal visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) areas as previously described (18).

For maximal aerobic capacity, VO₂max was measured on a motor-driven treadmill (Quinon) during a progressive exercise test to voluntary exhaustion as previously described.
A valid VO2 max was obtained when at least two of these three criteria were met: (i) maximal heart rate greater than 90% of age-predicted maximal heart rate (220 bpm [beats per minute] − age), (ii) respiratory exchange ratio of at least 1.10, and (iii) plateau in VO2 (<200 ml/min change) with increasing work rate.

For the oral glucose tolerance test (OGTT), after the subjects were fasted overnight, a 20-gauge polyethylene catheter was placed in an antecubital vein to facilitate blood sampling. Samples were drawn at 10 and 5 minutes prior to oral ingestion of 75 g of glucose. Subsequent samples were drawn at 30, 60, 90, 120, 150 and 180 minutes after the ingestion of the glucose. The plasma was separated by centrifugation and glucose and insulin concentrations were measured in duplicate by using the glucose oxidase method (Beckman Glucose Analyzer, Fullerton, CA) and radioimmunoassay (RIA) with an insulin-specific antibody (cross-reactivity with proinsulin less than 0.2%) (Linco, St. Louis, MO), respectively. Total 3-hour glucose and insulin areas under the curve were calculated by the trapezoidal method (19).

For lipoprotein lipids, leptin, and sex-hormone binding globulin (SHBG), venous blood samples for the measurement of lipoprotein lipids were collected in chilled tubes containing 1 mg of ethylene diamine tetra-acetic acid per cubic centimeter of blood. Plasma was separated by centrifugation at 4°C and lipoprotein lipids were measured as previously described (20). Plasma leptin (Linco Research, Inc., St. Louis, MO) and serum SHBG (Diagnostics Systems Laboratories, Inc., Webster, TX) concentrations were measured in duplicate by RIA. The intra-assay and inter-assay coefficients of variation for leptin were 5.2% and 3.5%, and for SHBG were 2.0% and 8.3%, respectively.

For the statistics, statistical analyses were performed with a Macintosh Statview program (Abacus Concepts Inc., Berkeley, CA). Distribution of the data was first tested for departures from normality by using the Shapiro–Wilk test. Triglyceride and HDL2 cholesterol data were not normally distributed, so the logarithm of each was used for parametric statistical analyses. Student’s unpaired t tests were used to compare baseline values between groups. Within-group differences between preintervention and postintervention measures of all variables were determined by using a paired t test. Change values (after-before) were compared between groups by using a one-way analysis of variance adjusted for the initial value. Correlation analyses were used to determine statistically significant relationships between variables, and partial correlation analyses were performed to determine independent predictors of the changes in metabolic risk factors. All data are presented as mean ± standard deviation, and the level of significance was set at p < .05 for all analyses.

RESULTS

Program Compliance

The 124 women who entered the study ranged in age from 50 to 73 years (mean = 60 ± 5 years), all had been at least 1 year without menses (mean = 11 ± 8 years), and all had a serum follicle-stimulating hormone concentration greater than 30 IU/l (mean = 54 ± 17 IU/l). The BMI of these women ranged from 25 to 44 kg/m² (mean = 34 ± 4 kg/m²). A total of 111 (of 124) women (72% Caucasian, 27% African American) completed the 10-week dietary control phase of the study (AHA Step 1 diet) and entered the 6-month hypocaloric diet and walking intervention. Of these 111 women, 76 (74% Caucasian, n = 57; 26% African American, n = 19) completed the weight loss phase of the study. The 48 women who dropped out of the study did so primarily because of time constraints or unexpected personal situations such as change in job status, relocation, or family death or illness. The age, years past menopause, BMI, and race of the 13 women who dropped out during the AHA dietary control phase were not different from those who completed the study (data not shown). Likewise, the age, years past menopause, BMI, race, body composition, and VO2 max were not different in the 35 women who dropped out during the weight loss intervention (data not shown) compared with those who completed this phase of the study (Table 1). Average attendance at the weekly weight loss and exercise classes was 74 ± 14% among the women who completed the entire study, and there were no racial differences in the number of classes attended (Caucasian = 74 ± 15%; African American = 72 ± 14%).

Body Composition, Fat Distribution, and Aerobic Fitness

Baseline.—At baseline, the 19 African-American women who completed the study were of similar age and years past menopause, but weighed more (p < .01) and had a greater BMI (p = .01) than the 57 Caucasian women who completed the study. Both fat mass (p < .05) and lean mass (p < .01) were greater in the African-American women, but there was no difference in percent body fat or waist and hip girths. VAT area was lower (p < .05) and abdominal SAT area was greater (p < .01) in African-American women than Caucasian women. Thus, the VAT/SAT ratio was lower in African-American women (0.38 ± 0.13 vs 0.26 ± 0.09; p < .01). Baseline VO2 max was not different between African-American and Caucasian women.

Post-treatment.—There were no racial differences in the absolute amount of weight lost (Caucasian, −5.4 ± 3.6 kg; African American, −3.9 ± 3.6 kg), but Caucasian women lost a relatively greater amount of body weight (−6.6 ± 4.2% vs −4.3 ± 4.3%; p < .05). Likewise, Caucasian women lost more fat mass than African-American women (−5.5 ± 2.9 vs −3.4 ± 3.4%; p < .05), resulting in a greater reduction in their percentage of body fat (% body fat: −4.1 ± 2.6% vs −1.9 ± 2.7%; p < .01). There were no significant changes in lean mass. By a paired t test, Caucasian, but not African-American, women significantly decreased their waist and hip circumferences (p < .0001), and the change values were significant between groups (p < .01). Although both groups reduced their SAT area (p < .01), only Caucasian women showed a significant decrease in VAT (p < .0001). However, there were no racial differences in changes in the amount of VAT or SAT lost. VO2 max increased similarly in both Caucasian (5.5 ±
8.8%) and African-American (6.7 ± 10.9%) women, but the increase reached statistical significance only in the Caucasian women.

**Relationships between changes in adiposity, VO₂max, and compliance.—** We analyzed the relationships between changes in adiposity, body fat distribution, and aerobic fitness as a result of the intervention with class attendance as well as the initial level of these variables. These analyses were conducted in all women combined because there was no interaction with race and there were no racial differences in compliance. The amount of weight lost was indirectly related to the number of weight loss classes attended (r = −.48, p < .01), such that women who attended more classes lost more weight; however, changes in VO₂max did not correlate with exercise class attendance. Changes in measures of total body adiposity (BMI, % fat, fat mass, and SAT) did not correlate with baseline values of these variables. However, changes in VAT and VAT/SAT were indirectly related to their baseline values (r = −.35, r = −.32, p < .01, respectively), suggesting that women with a greater proportion of VAT at baseline lost more VAT with the intervention.

**Lipoprotein Lipids, SHBG, and Leptin Concentrations**

At baseline, African-American women had higher HDL cholesterol (p < .05) and lower triglyceride (p < .0001) concentrations compared with the Caucasian women. Triglyceride (−12 ± 17%), total cholesterol (−2 ± 10%), and low-density lipoprotein (LDL) cholesterol levels (−3 ± 14%) decreased, whereas HDL cholesterol (7 ± 14%) and HDL₂ cholesterol levels increased (57 ± 114%) with 6 months of hypocaloric diet and walking in Caucasian women. In the African-American women there were statistically significant increases in HDL cholesterol (7 ± 11%) and HDL₂ cholesterol (43 ± 98%), and decreases in triglyceride levels (−10 ± 14%) after the intervention, but there were no racial differences in the magnitude of any lipid change. Except for HDL₂ cholesterol, the baseline value predicted changes in all lipoprotein lipids (change vs baseline for triglyceride, r = −.53, p < .0001; total cholesterol, r = −.29, p < .05; LDL cholesterol, r = −.34, p < .01; HDL cholesterol, r = −.34, p < .01).

Baseline leptin concentrations were higher in African-American women (p < .05), but they were similar when leptin was adjusted for racial differences in fat mass (Table 2). As a result of the intervention, leptin decreased (p < .05) in African-American women but did not change significantly in Caucasian women. SHBG concentrations were similar between races at baseline and did not change with weight loss in either race.

**Glucose Tolerance and Blood Pressure**

At baseline, Caucasian and African-American women had comparable fasting glucose and insulin levels, as well as responses during the OGTT. The intervention resulted in significant (p < .0001) decreases in fasting glucose (−4 ± 8%) and glucose area (−7 ± 14%) during the OGTT in Caucasian women, but the decrease in glucose area in African-American women only approached significance (−5 ± 15%; p = .12). There was a trend toward a statistically significant decline in fasting insulin in African-American women (−15 ± 22%; p = .06), but no change in fasting insulin in Caucasian women. Insulin area during the OGTT decreased in both Caucasian (−12 ± 29%, p < .01) and African-American (−17 ± 31%, p < .05) women. The changes in fasting glucose and insulin, and their responses during the OGTT, were not different between racial groups. Changes in fasting glucose (r = −.58, p < .0001), glucose area (r = −.53, p < .0001), fasting insulin (r = −.31, p < .05), and insulin area (r = −.31, p < .05) all correlated with the initial value for these variables, suggesting that women with the highest values improved the most.

African Americans had a higher initial systolic and diastolic blood pressure compared with Caucasian women (p < .05). Blood pressure did not change with weight loss in either Caucasian or African-American women; however, changes were related to initial blood pressure (systolic blood
changes in the glucose and insulin areas and changes of fat cholesterol. There were significant relationships between VAT (cholesterol concentrations correlated only with changes in pressure, \( r = -0.58, p < 0.0001 \); diastolic blood pressure, \( r = -0.51, p < 0.0001 \)).

**Predictors of Changes in Metabolic Risk Factors for CHD**

Bivariate and partial correlation analyses were used to determine the contribution of changes in VO2max, body composition, and body fat distribution to the changes in CHD risk factors with 6 months of hypocaloric diet and walking in these women. Because there were no racial differences in the responses of these risk factors, and because we found no racial differences in the slopes of the relationships between changes in the risk factors and changes in physical characteristics, data for all women are combined for these analyses.

**Bivariate correlations.**—There was no relationship between changes in any of the CHD risk factors and changes in VO2max in these women. However, changes in triglyceride, total cholesterol, and LDL cholesterol were related to the loss of total adiposity as measured by body weight, % body fat, and total fat mass (\( r = 0.27–0.39, p < 0.01 \) and \( 0.05 \)). In addition, triglyceride concentrations decreased more in women who lost more VAT (\( r = 0.23, p < 0.05 \)) and SAT (\( r = 0.34, p < 0.01 \)). Changes in HDL cholesterol concentrations correlated only with changes in VAT (\( r = -0.24, p < 0.05 \)) such that women who decreased their VAT levels more showed greater increases in HDL cholesterol. There were significant relationships between changes in the glucose and insulin areas and changes of fat mass (glucose area, \( r = 0.26, p < 0.05 \); insulin area, \( r = 0.30, p < 0.05 \)), but not for the fasting levels. Changes in blood pressure were not related to changes in any of the measured anthropometric variables.

**Multivariate analyses (Table 4).**—A multiple regression model that included changes in total fat mass, VAT, and the initial value for each risk factor was used to determine the independent predictors of the changes in lipid and glucose metabolic risk factors. The change in fat mass was the major independent predictor of changes in triglyceride, total cholesterol, and LDL cholesterol concentrations, and it contributed independently to the variance in changes in glucose and insulin areas after initial glucose and insulin areas. The change in VAT was the best independent predictor of changes in HDL cholesterol, and it was an independent predictor of changes in glucose area and fasting insulin after their initial values.

**DISCUSSION**

This study was conducted to determine the efficacy of a 6-month behavioral lifestyle intervention in reducing total and abdominal adipose tissue and improving CHD risk factors in overweight and obese, Caucasian and African-American, postmenopausal women. Overall, our results showed that the total amount of weight lost was composed mostly of adipose tissue because there was no overall loss of lean body mass. Total weight loss was indirectly related to attendance to the weight loss classes, but was not influenced by the race, age, or initial body weight of the women. The intervention reduced both SAT and abdominal VAT in Caucasian women, and the decline in visceral fat was greatest in women with a higher visceral fat area at baseline. In general, there were significant improvements in lipid and glucose metabolic risk factors for CHD, and, except for HDL cholesterol and triglyceride, the initial value of each CHD risk factor was an independent predictor of the change in that risk factor. On average, blood pressure did not change, but decreases in blood pressure also were related to the baseline values. Thus, in general, women with the worst metabolic profile at baseline improved the most with treatment, regardless of how much total or abdominal adipose tissue they lost. Although maximal aerobic fitness increased with the intervention, there was no relationship between changes in CHD risk factors and the improvement in VO2max.

The majority of previously conducted studies examining the efficacy of behavioral dietary and exercise intervention studies in women have been conducted in premenopausal women, and to our knowledge our study is one of only a few to report the effects of weight loss on CHD lipid risk factor outcomes in a racially mixed population of postmenopausal women. With the exception of one small study (21), most of the data in postmenopausal women indicate that weight loss by means of caloric restriction and exercise or drug treatment decreases circulating concentrations of triglyceride, total cholesterol, and LDL cholesterol; however, there are conflicting results about whether weight loss increases HDL cholesterol (22–26). This may be from the observation that HDL cholesterol either does not change or decreases

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**Table 2. Fasting Lipoprotein Lipid, Leptin, and SHBG Concentrations**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Before</th>
<th>After</th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total chol. (mmol/l)</td>
<td>5.30 ± 0.96</td>
<td>5.16 ± 0.94*</td>
<td>5.21 ± 0.81</td>
<td>5.15 ± 0.86</td>
</tr>
<tr>
<td>LDL chol. (mmol/l)</td>
<td>3.36 ± 0.89</td>
<td>3.22 ± 0.85*</td>
<td>3.31 ± 0.65</td>
<td>3.21 ± 0.65</td>
</tr>
<tr>
<td>HDL chol. (mmol/l)</td>
<td>1.25 ± 0.31</td>
<td>1.32 ± 0.29**</td>
<td>1.43 ± 0.33†</td>
<td>1.52 ± 0.34*</td>
</tr>
<tr>
<td>HDL2 chol. (mmol/l)</td>
<td>0.16 ± 0.15</td>
<td>0.21 ± 0.17**</td>
<td>0.23 ± 0.23†</td>
<td>0.28 ± 0.32†</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>1.56 ± 0.52</td>
<td>1.35 ± 0.50***</td>
<td>0.94 ± 0.32*</td>
<td>0.96 ± 0.32*</td>
</tr>
<tr>
<td>Leptin (ng/ml)</td>
<td>26 ± 17</td>
<td>22 ± 13</td>
<td>39 ± 32*</td>
<td>30 ± 14*</td>
</tr>
<tr>
<td>SHBG (nmol/l)</td>
<td>122 ± 61</td>
<td>133 ± 83</td>
<td>111 ± 59</td>
<td>116 ± 58</td>
</tr>
</tbody>
</table>

*Notes: Values are for before and after 6 months of hypocaloric diet and walking in postmenopausal women; they are given as mean ± SD. LDL = low-density lipoprotein; HDL = high-density lipoprotein; SHBG = sex-hormone binding globulin.

\*p < 0.05; **p < 0.01; ***p < 0.001 indicates significance from baseline.

†p < 0.05; ‡p < 0.01 between Caucasian and African-American women.
during restriction of energy and fat intake in younger women, and then increases once weight loss is stabilized (27). In the current study, HDL increased in both Caucasian and African-American women, and this was most likely due to our study design in which dietary composition was standardized before the weight loss treatment and women were weight stabilized for at least 2 weeks before post-testing.

There are even fewer studies regarding the effects of weight loss on glucose and insulin risk factors for CHD in postmenopausal women. Two studies showed an effect of weight loss in reducing fasting glucose and insulin (23,26), but another study reported that weight loss did not decrease insulin or glucose levels. Similarly, there are conflicting results in studies that directly measured glucose disposal by using a hyperinsulinemic–euglycemic clamp (24,28). In the current study, weight loss decreased insulin responses to a glucose load in both races, whereas fasting glucose and glucose area were reduced only in Caucasian women. However, because there were no racial differences in the magnitude of changes in glucose or lipid variables, the study may have been underpowered to detect a significant improvement in fasting glucose or glucose area in African Americans.

Effects of Changes in Abdominal Obesity
The anatomic location of fat loss may be an important factor contributing to the magnitude of CHD risk factor improvement with weight loss, and the selective loss of visceral fat may be the best predictor of weight-loss-induced improvements in CHD risk factors (29). In younger women, decreases in visceral fat with weight loss are more predictive of improvements in HDL cholesterol, triglyceride, and insulin sensitivity than the total amount of fat lost (30–32). Our findings in postmenopausal women show that the loss of visceral fat is an independent predictor of increases in HDL cholesterol and of decreases in glucose area and fasting insulin. This suggests that interventions tailored toward the selective loss of visceral fat could be more effective for reducing overall CHD risk in middle-aged and older women.

Effects of Changes in Aerobic Fitness
In younger individuals, aerobic exercise training is beneficial for increasing HDL cholesterol and insulin sensitivity and decreasing abdominal fat, hypertension, triglyceride levels, and glucose tolerance (33–37). Our finding that there were no significant associations between changes in CHD risk factors and improvement in maximal aerobic capacity is not surprising, given the low level of fitness in these women at baseline and the magnitude of the exercise training stimulus. The walking was combined with the dietary component of this weight loss program to encourage increases in energy expenditure, and the exercise was not designed to elicit a large aerobic training stimulus. Studies are needed to assess whether exercising at a higher intensity and frequency will result in comparable improvements in CHD risk factors in older women as those seen in younger women, and whether there are racial differences in CHD risk factor responses to exercise training.

Effects of Race
Our baseline data show that African-American women have a greater body mass, including fat and lean tissue, than Caucasian women, but no difference in % body fat. There also was a racial difference in body fat distribution, with African-American women having less visceral, but greater subcutaneous, abdominal fat than Caucasian women. These findings are consistent with studies of racial differences in body composition and fat distribution in younger women (10,12,38,39). Our findings that postmenopausal African-American women had higher blood pressure, but also higher HDL cholesterol and lower triglyceride concentrations, than Caucasian women are also in agreement with findings from larger, epidemiological studies in both young and older women (39–42).

Despite these well-known racial differences in body composition, body fat distribution, and CHD risk factors, there are very few studies that report whether body composition, body fat distribution, and risk factor responses to a dietary or exercise intervention are affected by race. Although the current study was not designed to definitively determine whether there are racial differences in these outcomes in response to this intervention, we did enroll enough African-American women to perform an initial analysis of this question. In our study, the absolute amount of weight loss was similar between Caucasian and African-American women, but, because of their lower baseline body weight, Caucasian women lost relatively more body fat during the intervention than African-American women. The

Table 3. Glucose, Insulin, and Blood Pressure

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Caucasian Before</th>
<th>Caucasian After</th>
<th>African American Before</th>
<th>African American After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting glucose (mmol/l)</td>
<td>5.31 ± 0.50</td>
<td>5.09 ± 0.38***</td>
<td>5.30 ± 0.33</td>
<td>5.27 ± 0.55</td>
</tr>
<tr>
<td>Glucose area (mmol/l × 180 min)</td>
<td>7.17 ± 1.51</td>
<td>6.59 ± 1.26***</td>
<td>7.01 ± 1.04</td>
<td>6.59 ± 1.21</td>
</tr>
<tr>
<td>Fasting insulin (pmol/l)</td>
<td>176 ± 33</td>
<td>72 ± 33</td>
<td>91 ± 36</td>
<td>78 ± 38***</td>
</tr>
<tr>
<td>Insulin area (pmol/l × 180 min)</td>
<td>417 ± 194</td>
<td>376 ± 225**</td>
<td>562 ± 396</td>
<td>412 ± 248*</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>123 ± 14</td>
<td>122 ± 15</td>
<td>135 ± 18†</td>
<td>127 ± 10</td>
</tr>
<tr>
<td>Diastolic</td>
<td>78 ± 7</td>
<td>75 ± 9</td>
<td>83 ± 9†</td>
<td>83 ± 6</td>
</tr>
</tbody>
</table>

Notes: Values are for before and after 6 months of hypocaloric diet and walking in postmenopausal women; they are given as mean ± SD.

*p < .05; **p < .01; ***p < .001; † indicates significance from baseline; ****p = 0.06.

†p < .05; between Caucasian and African-American women.
majority of the small number of previous studies examining the effects of race on weight loss responses to behavioral dietary programs show that, in general, African-American women are less compliant and lose less weight as a result (43–46). However, in one study, black and white women experienced similar reductions in body weight in response to a 13-week diet and exercise behavioral modification program (47). There are many possible reasons for lack of adherence to a hypocaloric diet by African-American women, including lack of readiness to lose weight, feasibility of following dietary recommendations as a result of ethnic habits, or different perceptions of body weight (45). The relatively short intervention (6 months) of the current study, the lack of a racial difference in the socioeconomic status of the women we studied (data not shown), or both may be some of the reasons why there were no racial differences in program compliance or total weight loss.

Our results show that there were no racial differences in the absolute or relative amounts of abdominal VAT or SAT lost with this intervention. This finding is consistent with another study of a small sample of young women (48). However, our findings conflict with those of a well-controlled diet intervention study in which all meals were prepared in a metabolic kitchen and were designed to achieve a 10-kg weight loss in young Caucasian and African-American women (11). In that study, the mean duration of dietary restriction to achieve this goal was not different between races, and, despite this similar loss of total weight, Caucasian women lost more VAT and less subcutaneous abdominal fat than African-American women. These were younger women, and the amount of total weight loss was twice as much as in the current study, which could account for the different results. Studies are needed to confirm whether there are racial differences in the location of fat loss in response to weight loss interventions.

In the current study, there were no racial differences in the magnitude of improvements in CHD risk factors, despite the fact that Caucasian women lost relatively more body fat. This suggests either that African-American women exhibit greater improvements in CHD risk factors with less relative fat loss, or that changes in risk factors are more closely related to the absolute amount of fat lost. To our knowledge, there are only a few prior studies that compared weight-loss-induced improvements in CHD risk factors between African Americans and Caucasians, and none of these showed racial differences in lipoprotein lipid, or glucose and insulin responses to a similar amount of weight loss (47–49). One study with a small sample size showed a race by diet interaction for blood pressure in that only Caucasian women decreased their diastolic blood pressure following a 17-kg loss of body weight in both races (48). It is, of course, difficult to prove a “negative” finding. Failure to show a difference in response by race can be the result of no true difference or a lack of power. In order to put our findings in perspective, it may be helpful to examine the number of subjects that would be necessary to show a significant difference, assuming that the differences and standard deviations found are “true.” We would need approximately 200 Caucasians and 200 African Americans to detect a racial difference in the amount of VAT lost, and we would need approximately 150 of each group for detection of a racial difference in fasting glucose and insulin. Even more subjects would be needed to detect racial differences in lipid responses to the intervention (HDL cholesterol = 1000 per group and LDL cholesterol = 500 per group). Adequately powered studies utilizing tightly controlled dietary and exercise interventions will be necessary to definitely determine whether there are racial differences in the magnitude of CHD risk factor improvement in response to a similar reduction of total or abdominal fat.

**Conclusions**

The findings of this study showing that a relatively small loss of body weight (4–6 kg on average) combined with a walking program improves glucose and lipid CHD risk factors in both Caucasian and African-American postmenopausal women lends support to the public health recommendations of the National Heart, Lung, and Blood Institute (2), the American College of Sports Medicine (13), and the National Cholesterol Education Program (14). As in younger men and women, our findings show that weight loss achieved through energy restriction and increased physical activity can be advocated as an effective therapy for improving glucose, lipid, and blood pressure CHD risk factors in higher-risk middle-aged and older women, and that this type of therapy is equally effective in Caucasians and African Americans.

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### Table 4. Independent Predictors of Changes in Lipid and Glucose CHD Risk Factors

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Coefficient</th>
<th>SEM</th>
<th>Cumulative ( r^2 )</th>
<th>( p ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Delta ) Triglyceride</td>
<td>( \Delta ) fat mass</td>
<td>2.40</td>
<td>1.06</td>
<td>.10</td>
</tr>
<tr>
<td>( \Delta ) Total cholesterol</td>
<td>( \Delta ) fat mass</td>
<td>2.38</td>
<td>0.86</td>
<td>.11</td>
</tr>
<tr>
<td></td>
<td>Initial chol.</td>
<td>-0.16</td>
<td>0.08</td>
<td>.18</td>
</tr>
<tr>
<td>( \Delta ) LDL cholesterol</td>
<td>( \Delta ) fat mass</td>
<td>1.94</td>
<td>0.73</td>
<td>.10</td>
</tr>
<tr>
<td></td>
<td>Initial LDL</td>
<td>-0.18</td>
<td>0.08</td>
<td>.18</td>
</tr>
<tr>
<td>( \Delta ) HDL cholesterol</td>
<td>( \Delta ) VAT</td>
<td>-0.05</td>
<td>0.03</td>
<td>.10</td>
</tr>
<tr>
<td>( \Delta ) Glucose</td>
<td>Initial glucose</td>
<td>-0.58</td>
<td>0.10</td>
<td>.37</td>
</tr>
<tr>
<td></td>
<td>Glucose area</td>
<td>-0.46</td>
<td>0.08</td>
<td>.30</td>
</tr>
<tr>
<td></td>
<td>( \Delta ) VAT</td>
<td>-0.01</td>
<td>0.01</td>
<td>.35</td>
</tr>
<tr>
<td></td>
<td>( \Delta ) fat mass</td>
<td>0.08</td>
<td>0.04</td>
<td>.40</td>
</tr>
<tr>
<td>( \Delta ) Insulin</td>
<td>Initial insulin</td>
<td>-0.36</td>
<td>0.08</td>
<td>.24</td>
</tr>
<tr>
<td></td>
<td>( \Delta ) VAT</td>
<td>0.22</td>
<td>0.10</td>
<td>.31</td>
</tr>
<tr>
<td>( \Delta ) Insulin area</td>
<td>Initial ins. area</td>
<td>-0.39</td>
<td>0.08</td>
<td>.24</td>
</tr>
<tr>
<td></td>
<td>( \Delta ) fat mass</td>
<td>17.10</td>
<td>6.85</td>
<td>.33</td>
</tr>
</tbody>
</table>

*Notes: Model included the initial value of each risk factor, change in fat mass, and change in visceral adipose tissue (VAT) area; CHD = coronary heart disease; LDL = low-density lipoprotein; HDL = high-density lipoprotein.*
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REFERENCES


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