Abdominal Fat Distribution and Metabolic Risk Factors: Effects of Race

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Previous studies have shown differences between African-American and Caucasian populations in the prevalence of obesity and obesity-related diseases such as type II diabetes. The purpose of this study was (1) to compare the insulin sensitivity index (S_I) from the minimal model in 37 African-American and 22 Caucasian women matched for age and obesity, and (2) to determine whether the relationship between intraabdominal fat distribution and S_I (and other health risk factors) was similar in both races. To address the second question, intraabdominal fat distribution was assessed by computed tomographic (CT) scans in a subset of 23 African-American and 15 Caucasian women. Despite having a similar body mass index ([BMI] weight in kilograms divided by height in meters squared) and waist to hip ratio (WHR), African-American women had a mean S_I value that was approximately 36% lower than in the Caucasian women (3.45 ± 0.42 *v* $5.40 \pm 0.55 \times 10^{-5}$ $min^{-1}/pmol \cdot L$, $P = .007$). Visceral fat area was smaller in African-American women (98.0 ± 8.5 cm^2) than in Caucasian women (117.3 ± 12.4 cm^2) despite similar BMI and WHR. Visceral fat area was strongly correlated with WHR in the Caucasian women ($r = .76$, $P < .001$), as previously observed, but not in the African-American women ($r = .24$, NS). WHR was significantly correlated with fasting insulin and serum cholesterol in the Caucasian women but not in the African-Americans. Visceral fat was correlated with metabolic risk factors in both groups, but subcutaneous abdominal fat was significantly correlated with S_I and fasting insulin only in the African-American women. These results suggest that the relationship between body fat distribution and health risk factors may be different in African-Americans and Caucasians. Additionally, reduced insulin sensitivity in African-American women may in part explain the high diabetes rate in this population.

THE PREVALENCE of type II diabetes in the United States is significantly higher in a number of ethnic groups compared with Caucasians.[1-3] This ethnic difference is particularly striking among African-American women, who have twice the diabetes prevalence of Caucasians in some age groups.[1] Despite the significant cost of diabetes in the African-American population, little is known about factors that predispose this population to develop diabetes at high rates.

Insulin resistance and obesity are known risk factors for both diabetes and heart disease. Although obesity is more prevalent in African-American than in Caucasian women,[4] this difference in obesity rates does not appear to explain the increased diabetes risk in the African-American population.[5] In longitudinal assessments of a number of high-risk populations, including Pima Indians[6] and the offspring of diabetic parents,[7] insulin resistance has been shown to predict diabetes development. Recently, glucose effectiveness (S_G, from the minimal model, which reflects the ability of glucose to enhance its own disposal at basal insulin) has also been shown to be predictive of the development of diabetes in the offspring of diabetic parents.[7]

Obesity is consistently associated with insulin resistance, and many studies have shown that upper-body obesity (UBO), particularly increased visceral, abdominal fat, is most strongly associated with metabolic and cardiovascular risk.[8] The majority of studies relating fat distribution to health risk have been performed in Caucasian populations. When the relationship between anthropometric measures of fat distribution (eg, waist to hip ratio [WHR]) and health risk has been assessed in African-American populations, the correlations have been weaker than in the Caucasian studies.[9,10]

Recently, it was reported that African-American women have less visceral fat than Caucasian women with the same WHR.[11] This finding raised the possibility that the weak association between WHR and health risk in African-Americans might be due to a difference in intraabdominal fat distribution. Few studies have measured intraabdominal fat in African-Americans using imaging techniques like computed tomographic (CT) scan that allow quantification of visceral and subcutaneous fat.

The present study was undertaken to test several hypotheses. First, we sought to confirm previous reports that insulin sensitivity (by insulin sensitivity index [S_I]) is reduced in African-American women compared with Caucasian women matched for age, degree of obesity, and WHR. Second, we tested the hypothesis that the relationship between intraabdominal fat distribution and health risk factors differs in the two populations by comparing CT scan–derived measures with insulin sensitivity, blood lipids, and blood pressure.

SUBJECTS AND METHODS

Subjects

Healthy women with a range of body weight were recruited from the Baton Rouge area using newspaper and radio advertisements and word of mouth. Respondents underwent a two-step screening process to determine eligibility. To be accepted into the study, the women had to be premenopausal and taking no medications and to have laboratory values (chemistry panel, complete blood cell count, and fasting glucose) within the normal range. A total of 37 African-American and 22 Caucasian women underwent measurements of insulin sensitivity and lipid profiles. A subset of 23 African-American and 15 Caucasian women had CT scans. All subjects signed an informed-consent form before participation in

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screening or study examinations. The protocol was approved by the Louisiana State University Institutional Review Board.

Anthropometrics and Body Composition

Height and weight were measured in fasting subjects wearing light clothing. Waist circumference was measured at the smallest circumference and hip circumference at the maximum protrusion of the buttocks for calculation of the WHR. Body composition was determined by dual-energy x-ray absorptiometry (QDR2000; Hologic, Waltham, MA). CT scans were performed at the L4-L5 intervertebral space using a General Electric High-Speed Advantage helical CT scanner (Milwaukee, WI). The scan was 1 cm thick, and was analyzed for visceral and subcutaneous fat tissue (-190 to -30 Hounsfield units) by outlining the area inside the abdominal muscle wall on the computer screen with a light pen. A single abdominal scan at this level has been shown to correlate well with multiple scans in providing an estimate of visceral and subcutaneous fat area.[12]

Frequently Sampled Intravenous Glucose Tolerance Tests

An insulin-modified frequently sampled intravenous glucose tolerance test (FSIGT) was performed in each subject following 3 days of a standard diet containing at least 200 g carbohydrate. Subjects were studied after an overnight fast. An intravenous (IV) catheter was placed in each antecubital vein, one for administering glucose and insulin and one for collecting frequent blood samples. An IV bolus of glucose (300 mg/kg 50% dextrose solution, Abboject; Abbott Laboratories, Abbott Park, IL) was administered at time 0 and a bolus of insulin (0.03 U/kg Humulin; Eli Lilly, Indianapolis, IN) at 20 minutes. Frequent blood samples were collected at 0, 2, 3, 4, 5, 6, 7, 8, 10, 12, 14, 16, 19, 22, 23, 24, 25, 27, 30, 40, 50, 60, 70, 80, 90, 100, 120, 140, 160, and 180 minutes, as described previously.[13] Glucose and insulin values from the FSIGT were entered into the MINMOD program (© R. Bergman) run on a personal computer for minimal-model determination of S_I and S_G (glucose effectiveness). K_G was calculated as the slope of the regression line from 8 to 19 minutes before administering insulin. Glucose effectiveness at zero insulin, GEZI, was calculated as S_G minus the basal insulin effect (fasting insulin $\times$ S_I).

Laboratory Analyses

Glucose was analyzed on a Beckman (Brea, CA) CX7 autoanalyzer using the glucose oxidase method. Insulin was analyzed by radioimmunoassay using a commercial kit (Diagnostic Products, Los Angeles, CA). Cross-reactivity with proinsulin as reported by the manufacturer is approximately 40%.

Statistical Analysis

Group differences in the raw data were assessed using a *t* test, adjusting for equal or unequal variance as required. Additionally, an analysis of covariance with both race and body mass index (BMI) as covariates was performed, and the LSMEANS procedure was used to determine adjusted means and group differences for S_I. Other variables such as visceral fat were not included in multivariate models with race, since high correlations between these factors and race cause potential problems with multicollinearity. Thus, primarily unadjusted data are reported. All variables were tested for normality, and those that were not normally distributed were analyzed using nonparametric statistics. Nonparametric Spearman correlations (*r* values) were used to assess the relationship between metabolic variables and fat distribution. Since Spearman correlations are based on ranks rather than on absolute values, they minimize any potential distortion of the results due to a small number of subjects. Data are reported as the mean ± SE unless otherwise specified. All data were analyzed using SAS for Windows (SAS, Cary, NC) run on a personal computer. An α level of .05 was considered significant.

RESULTS

Race Differences in Anthropometric and Metabolic Variables

Comparisons between anthropometric and metabolic variables in African-American and Caucasian women are shown in Table 1. The groups did not differ significantly in age, BMI, or percentage body fat. The majority of subjects were obese, as indicated by the mean values for BMI and percent fat. Additionally, the two groups had a similar WHR of approximately 0.80, indicating only a slight degree of abdominal obesity. African-American women had less visceral fat than Caucasian women, although the difference was not statistically significant when tested by *t* test on the raw data. However, when the means were adjusted for BMI, there was significantly less visceral fat in African-American women ($P = .03$). Subcutaneous fat area did not differ between the groups, although it was slightly higher in African-American women (this difference lessened after adjusting for BMI). Total abdominal fat area also was not different between groups.

Metabolic differences between African-American and Caucasian women are shown in Table 2. After adjusting for BMI, there were no significant differences between groups in fasting glucose level, but fasting insulin levels were significantly higher in African-American women ($P = .009$). K_G, reflecting IV glucose tolerance, was not different between the groups. Using either the raw data or BMI-adjusted means, African-American women had a significantly lower S_I than Caucasian women (~ 36% lower; Table 2). In contrast to previous reports,[14] there were no race differences in S_G.

With regard to cardiovascular measures, there were no significant group differences in serum total or high-density lipoprotein cholesterol (Table 2). Triglyceride levels were significantly higher in Caucasians ($P = .03$). Both systolic and diastolic blood pressure were higher in African-American women than in Caucasian women, but the difference was only statistically significant for diastolic blood pressure ($P = .02$).

Correlations Between S_I, S_G, and Metabolic Parameters

In general, S_I was more strongly correlated with anthropometric and metabolic parameters than was S_G (Table 3).

Table 1. Characteristics of 37 African-American and 22 Caucasian Women

Characteristic	African-American	Caucasian	*P*
Age (yr)	35.0 ± 1.1	36.2 ± 1.3	.50
BMI (kg/m²)	31.3 ± 1.1	29.6 ± 1.4	.34
Body fat (%)	41.5 ± 1.1	42.3 ± 1.8	.70
WHR	0.80 ± 0.01	0.80 ± 0.02	.80
Visceral fat area (cm²)	98.0 ± 8.5	117.3 ± 12.4	.19
Subcutaneous fat area (cm²)	436.7 ± 37.1	417.2 ± 37.0	.72
Total abdominal fat area (cm²)	522.9 ± 45.3	534.6 ± 45.7	.86

NOTE. Data are the mean ± SEM; *P* values are from Student's *t* test (unadjusted values). For CT scan measures, n = 23 African-American and n = 15 Caucasian women.

Table 2. Metabolic and Cardiovascular Variables in 37 African-American and 22 Caucasian Women (mean ± SEM adjusted for BMI)

Variable	African-American	Caucasian	P*
Fasting glucose (mmol/L)	5.14 ± 0.1	5.24 ± 0.1	NS
Fasting insulin (pmol/L)	75.8 ± 5.8	50.2 ± 7.5	.009
K_G (%)	2.30 ± 0.14	2.06 ± 0.18	NS
S_I ($\times 10^{-5}$ min^{-1}/pmol · L)†	3.45 ± 0.42	5.40 ± 0.55	.007
$S_G \times 100$ (min^{-1})	2.41 ± 0.11	2.41 ± 0.15	NS
GEZI × 100 (min^{-1})	2.08 ± 0.10	2.05 ± 0.16	NS
Total cholesterol (mg/dL)	175.1 ± 5.8	160.8 ± 6.9	NS
HDL cholesterol (mg/dL)	44.5 ± 2.0	41.0 ± 2.4	NS
Triglycerides (mg/dL)	60.9 ± 7.6	87.6 ± 9.2	.03
Systolic BP (mm Hg)	116.4 ± 2.3	110.3 ± 2.8	.09
Diastolic BP (mm Hg)	74.6 ± 1.8	67.7 ± 2.2	.02

Abbreviations: GEZI, glucose effectiveness at zero insulin; HDL, high-density lipoprotein; BP, blood pressure.

*From least-square means procedure.

†To convert to traditional S_I units (10^{-4} $min^{-1}/\mu U \cdot mL$), multiply by 0.6.

The correlations between S_I and age and BMI tended to be stronger in Caucasian women than in African-American women (Table 3). The relationship between S_I and fasting insulin was hyperbolic in both African-American and Caucasian subjects (Fig 1), as previously shown for Caucasian women.[15]

S_G was correlated with age in Caucasian women but not African-American women (Table 3). S_G was correlated with BMI and WHR in both groups (not significant in Caucasians), but was only weakly correlated with percentage body fat. Interestingly, S_G was much more highly correlated with fasting glucose in African-American women ($r = -.64$, $P = .0001$) than in Caucasian women ($r = -.11$, NS).

Correlations Between Fat Distribution and Health Risk

Relationships between WHR, visceral and subcutaneous fat, and various health risk factors are shown in Table 4. In Caucasian women, strong relationships were observed between WHR and insulin sensitivity, fasting insulin, and

Table 3. Correlations (Spearman r values) Between S_I or S_G and Anthropometric and Metabolic Variables in 37 African-American and 22 Caucasian Women

	All	African-American	Caucasian
S_I *v* age	.00	.14	−.46*
BMI	−.69‡	−.60‡	−.83‡
WHR	−.40†	−.41†	−.45*
% body fat	−.40†	−.43†	−.49*
Basal glucose	−.37†	−.43†	−.45*
Basal insulin	−.74‡	−.69‡	−.73‡
S_G *v* age	−.19	−.05	−.46*
BMI	−.44†	−.40*	−.40
WHR	−.37†	−.33*	−.41
% body fat	−.19	−.12	−.28
Basal glucose	−.47‡	−.64‡	−.11
Basal insulin	−.44†	−.43†	−.28

*$P < .05$.

†$P < .01$.

‡$P < .0001$.

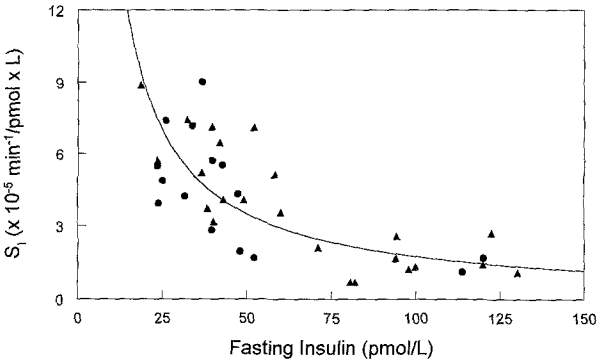

Fig 1. Relationship between S_I from the minimal model and fasting insulin level in (▲) 37 African-American and (●) 22 Caucasian women.

serum cholesterol, as seen previously.[8] The relationships between WHR and these risk factors were weaker in African-Americans, particularly for insulin and cholesterol levels. WHR was significantly associated with visceral fat, as expected, in Caucasian women, but only weakly related to visceral fat in African-Americans (Fig 2). Significant relationships were seen between visceral fat and measures of insulin action, glucose disposal, and fasting insulin in both Caucasian and African-American women (Table 4). Interestingly, visceral fat was not significantly associated with serum total or high-density lipoprotein cholesterol or blood pressure in either population. Finally, subcutaneous abdominal fat was significantly associated with S_I and fasting insulin in African-Americans. Relationships between subcutaneous fat and metabolic risk factors were weaker in Caucasian women.

DISCUSSION

The reasons for the significantly increased prevalence of type II diabetes in African-American women have remained elusive. The results presented herein confirm that African-American women are more insulin-resistant (reflected as a lower S_I value) than Caucasian women even when they are matched for age, degree of obesity, and WHR. Somewhat paradoxically, we also confirmed that African-American women have a lower visceral fat area,

Table 4. Spearman Correlations Between Intraabdominal Fat and Risk Factors in 23 African-American and 15 Caucasian Women

	WHR		VFAT		SQFAT	
Risk Factor	AA	C	AA	C	AA	C
WHR	—		.24	.76‡	.03	.53*
S_I	−.47*	−.45*	−.47*	−.54*	−.47*	−.44
S_G	−.33*	−.72†	−.59†	−.64†	−.36	−.29
Fasting insulin	.20	.59†	.49*	.48*	.53†	.41
Total cholesterol	−.05	.62†	.31	.38	.30	.36
HDL cholesterol	−.34	−.31	−.29	−.26	−.33	.49
Systolic BP	.10	.01	.39	.03	.24	.28
Diastolic BP	−.28	.24	.25	−.07	.23	−.22

Abbreviations: VFAT, visceral fat area by CT scan; SQFAT, subcutaneous fat area by CT scan; BP, blood pressure.

*$P < .05$.

†$P < .01$.

‡$P < .001$.

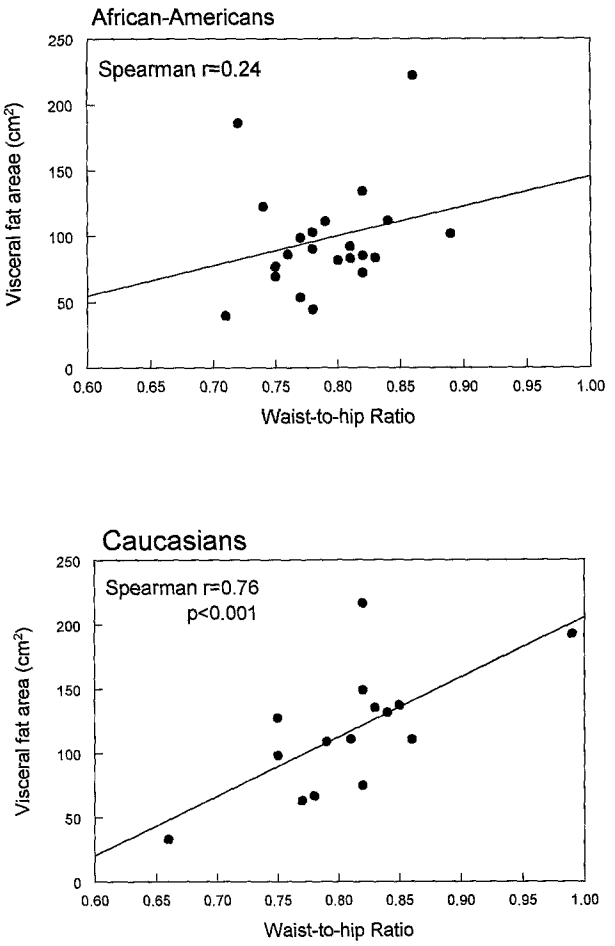

Fig 2. Relationship between intraabdominal visceral fat area and WHR in African-American (n = 23) and Caucasian (n = 15) women.

which would be expected to be associated with a higher, rather than a lower, S_I compared with Caucasians. However, within each ethnic group, relationships between visceral fat and health risk factors (S_I and blood lipids) were similar.

Several previous studies support the idea of diminished insulin sensitivity in healthy African-Americans. Osei et al[14,16,17] have reported that S_I was diminished in healthy African-Americans compared with Caucasians independently of family history of diabetes. Dowling and Pi-Sunyer[10] also found that peripheral insulin sensitivity was generally lower in African-American women compared with Caucasian women. Furthermore, in their study, S_I in Caucasian women was influenced by body fat distribution (with reduced S_I in UBO), but S_I in African-American women did not differ in those with UBO versus lower-body obesity (LBO). Recently, these investigators also reported that fat cells from UBO or LBO African-American women were equally responsive to insulin, whereas cells from UBO Caucasian women were less insulin-responsive than those from their LBO counterparts.[18]

The data from our study using abdominal CT scan to assess the relationships between visceral fat and insulin action are generally supportive of these findings. Although we found that S_I and WHR were similarly correlated in both populations, WHR was much more weakly correlated with S_G and fasting insulin in African-American women than in Caucasian women. The relationships between visceral fat area and S_G and fasting insulin are also weaker in African-Americans than in Caucasians. Similar relationships between visceral fat and serum lipids were found in the two groups.

Comparing the CT scan results with the metabolic differences raises some interesting questions. It is puzzling that African-American women have less visceral fat yet are more insulin-resistant than Caucasians, since this contradicts the expected positive association between visceral fat and insulin resistance. However, within the African-American group, those with more visceral fat were more insulin-resistant (Table 4), consistent with the expected relationship. These results suggest that either (1) there is a bimodal distribution of visceral fat in the overall population, but within each ethnic group the expected associations between visceral fat and health risk hold true, or (2) insulin resistance in African-Americans is more strongly influenced by factors other than excess visceral fat (ie, there is a different mechanism explaining insulin resistance in the two groups). Our finding that subcutaneous abdominal fat is significantly associated with insulin sensitivity and fasting insulin in African-Americans suggests that there may be other factors, perhaps related to total adiposity rather than intraabdominal adiposity, that influence metabolic risk in this population. One possibility is that the hyperinsulinemia actually drives the relative expansion of intraabdominal adipose tissue depots, and that there are differential sensitivities of visceral and subcutaneous adipose tissue to this insulin stimulus in different ethnic groups.

In addition to observing race differences in S_I, previous reports from our laboratory[19] and from Osei and Cottrell[14] also showed an increased S_G in African-Americans, indicating improved non–insulin-mediated glucose disposal. The importance of a reduced S_G as a diabetes risk factor is beginning to be more widely recognized. Bergman[20] has shown through computer simulations that an 80% defect in either S_I or S_G can lead to a diabetic glucose tolerance curve. In the present study, we did not observe any race difference in S_G, for reasons that are unclear. The populations studied previously were located in different areas of the United States than in the current study and thus may have had different genetic or dietary factors that might influence S_G. Given the importance of S_G in predicting the development of diabetes in Caucasians, this issue needs further exploration in African-Americans, ideally with longitudinal follow-up evaluation.

The hyperbolic relationship between S_I and fasting insulin originally postulated by Bergman et al[21] was confirmed in 93 healthy Caucasians studied by Kahn et al.[15] In their study using a tolbutamide-modified FSIGT, they were able to develop percentile plots based on the interaction of S_I and β-cell function that allowed prediction of the likelihood

to develop diabetes. Although the hyperbolic relationship between S_I and β-cell function was thought to be characteristic, the present study is the first (to our knowledge) to report a similar hyperbolic relationship between these variables using the insulin-modified FSIGT and to show that the relationship holds in both Caucasians and African-Americans.

One limitation of this study is the relatively small number of subjects, although this group is larger than those reported in several other such studies.[10,11,14] Power analysis indicated that considerably larger subject numbers would have been needed (>60 per group) to detect significant differences in BMI or visceral fat in the unadjusted data at the level of difference reported in Table 1. Nevertheless, these differences, as well as differences in cholesterol and blood pressure, are fairly substantial and are similar in magnitude to those reported by others that are considered clinically significant. The small subject numbers also influence our correlational data, increasing the risk of one or two points unduly influencing the regression line. However, regression diagnostics were performed, which indicated that even points that were statistical outliers did not significantly alter the slopes or intercepts of our regression lines. Furthermore, some of these correlations (eg, the relationship between visceral fat and WHR in Caucasians, Fig 2) are consistent with data reported by others, and thus it is unlikely that the relatively small subject numbers have produced spurious correlations. Nevertheless, some caution should be used in interpreting these data until the results can be confirmed in a larger number of subjects.

It should be noted that interpretations of our data should not be extrapolated to African-American men. Although African-American men have a higher prevalence of diagnosed diabetes than Caucasian men, we have chosen to focus on African-American women, since they have the highest diabetes prevalence and the number of cases diagnosed from 1963 to 1990 appears to be growing at the fastest rate in this population.[22] However, African-American men may represent a unique population for study, since previous reports indicate that approximately 33% of African-American men with type II diabetes have a unique insulin-sensitive variant of the disease.[23] Whether this relative insulin sensitivity is also found in healthy obese or lean African-American men is unknown, and should be the focus of future studies.

In summary, these data confirm that African-American women are more insulin-resistant than Caucasian women matched for age, degree of obesity, and WHR. Additionally, African-American women in general have less visceral fat than Caucasian women and WHR does not correlate with visceral fat area in African-Americans, whereas these variables are strongly correlated in Caucasians. Finally, relationships between body fat distribution and metabolic and cardiovascular risk factors were generally weaker in African-American women, although the relationships between insulin sensitivity and visceral fat were strong and significant in both populations.

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