Insulin Sensitivity and Serum Triglyceride Level in Obese White and Black Women: Relationship to Visceral and Truncal Subcutaneous Fat

Matthew A. Marcus, Laura Murphy, F. Xavier Pi-Sunyer, and Jeanine B. Albu

Although independent associations of visceral fat with the insulin resistance syndrome were previously reported in obese women, the importance of truncal subcutaneous fat with regard to insulin sensitivity is still controversial. We measured the insulin sensitivity index (S_I), serum triglyceride (TG) level, and regional fat by two methods: (1) the sum of five truncal and four peripheral skinfolds (TrSUM and PerSUM) in 38 white and black obese nondiabetic premenopausal women, and (2) abdominal visceral (VFM) and subcutaneous fat mass (AbdSCFM) by a combination of magnetic resonance imaging (MRI) and dual x-ray absorptiometry (DXA) in a subset of 26 of these women. After adjusting for the total body fat mass, TrSUM and VFM were independently and negatively related to S_I ($n = 38$, $P < .012$ and $n = 26$, $P < .035$, respectively), whereas PerSUM and AbdSCFM were not related ($P > .50$). Based on multiple regression modeling, TrSUM significantly predicted S_I independently of the VFM ($n = 26$, $P < .001$). Black women had lower S_I at all levels of TrSUM ($n = 38$, $P = .061$ for the slope and $P = .03$ for the intercept of the regression lines). After adjusting for the total body fat mass, only VFM showed an independent positive relation to serum TG, and race did not affect this relationship ($n = 26$, $P < .001$). In conclusion, (1) we confirmed the independent association of the VFM with insulin resistance and elevated TG in obese women; (2) the AbdSCFM measured by a combination of MRI and DXA did not show an independent association with S_I in obese women; and (3) the independent association of TrSUM with S_I suggests that truncal subcutaneous fat depots contribute to insulin resistance in obese women independently of the degree of visceral fat.

PREVIOUS REPORTS have demonstrated that women with a greater proportion of upper-body (truncal/abdominal) fat tend to be more insulin-resistant, hyperinsulinemic, glucose-intolerant, and dyslipidemic than women with a greater proportion of lower-body (gluteal/femoral) fat.[1-7] An enlarged visceral fat mass (VFM) resulting in an increased portal free fatty acid (FFA) flux delivered to the liver has been proposed as one of the mechanisms underlying this association.[8] When imaging techniques such as magnetic resonance imaging (MRI) and computed tomography (CT) were used, visceral fat accumulation was found to be specifically associated with the metabolic alterations of obesity both in men and in women.[9-14] We previously reported that the visceral fat area measured at the midwaist relates best to increased glucose, insulin, and triglyceride (TG) levels in black and white premenopausal nondiabetic women.[15] In our study, both the visceral and subcutaneous fat areas were negatively related to insulin sensitivity measured by the minimal model (S_I), but only the visceral fat area was independently and negatively related to S_I after adjusting for percent body fat. Our findings were consistent with data reported by other investigators measuring insulin sensitivity and visceral and subcutaneous fat areas in adolescent girls,[16] premenopausal women,[14,17] and postmenopausal women and men.[12,13] When visceral and abdominal subcutaneous fat volume and mass were measured in premenopausal women by Ross et al,[18] only the VFM had independent associations with insulin and glucose areas during an oral glucose tolerance test (OGTT).

In contrast, the contribution of subcutaneous fat patterning (upper body *v* lower body or trunk *v* periphery) to the metabolic disturbances of upper-body obesity is still controversial. Population-based studies have demonstrated a trend toward greater accumulation of truncal subcutaneous fat measured as the sum of truncal skinfolds in hyperglycemic and hyperinsulinemic versus normal subjects.[19-21] Moreover, women with a greater proportion of upper-body (truncal/abdominal) fat have increased systemic FFA flux[22] and a higher rate of lipolysis in the upper-body subcutaneous adipocytes[23-24] than women with a greater proportion of lower-body (gluteal/femoral) fat. Still, associations between the amount of subcutaneous fat on the trunk and metabolic disturbances, more specifically insulin sensitivity, have been reported so far only in men.[25-27] Furthermore, despite previously described differences between white and black obese women in visceral versus subcutaneous fat distribution[28] and in the relation of this distribution to insulin resistance and blood lipids,[15,17] it is unclear whether the metabolic impact of subcutaneous fat patterning varies with race. This comparison may be especially important, considering prior observations of a greater tendency for accumulation of truncal subcutaneous fat in black women compared with white women.[4-5,21,29-30]

To test these hypotheses and as an improvement over our previous study, the associations of the truncal and peripheral skinfolds and the AbdSCFM and VFM measured by a combination of MRI and dual x-ray absorptiometry (DXA) with insulin sensitivity and serum TG were determined in a sample of obese nondiabetic black and white women. The homogeneity of these associations was compared between races.

SUBJECTS AND METHODS

Subjects

Thirty-eight white ($n = 20$) and black ($n = 18$) women were evaluated. This group was a subset of women who participated in a study

From the Obesity Research Center and Department of Medicine, St. Luke's-Roosevelt Hospital, College of Physicians and Surgeons, Columbia University, New York, NY.

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Address reprint requests to Jeanine B. Albu, MD, Obesity Research Center, St. Luke's-Roosevelt Hospital, 1111 Amsterdam Ave, New York, NY 10025.

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previously reported by us.[14] Whites were non-Hispanic Caucasians and blacks were African-Americans, with race defined according to the ancestry of all four grandparents. All subjects were in good health and were not taking any medications. Normal glucose tolerance according to National Diabetes Data Group criteria[31] was based on a screening OGTT. All subjects reported a regular menstrual cycle and stable body weight (±2 kg) for the 6 months preceding the study. Body composition and metabolic measurements were obtained with subjects in the fasting state. The study was approved by the Institutional Review Board of St. Luke's-Roosevelt Hospital Center. All subjects provided written consent prior to participation.

Body Composition Measurements

All 38 subjects had anthropometric and MRI measurements, but in only 26 subjects were DXA measurements possible.

Anthropometric and skinfold measurements. Fasting weight and height were measured with the subjects wearing only undergarments. Skinfold thickness was measured to ±2 mm with a Lange caliper (Cambridge Scientific Industries, Cambridge, MD) at five sites on the thorax (subscapular, midaxillary, suprailiac, umbilicus, and abdomen [between the umbilicus and pubic rami]) and at four sites on the extremities (triceps, biceps, thigh, and calf) according to the methods of Harrison et al.[32] The average of two readings was recorded. The sums of truncal (TrSUM) and peripheral (PerSUM) skinfolds were then calculated, respectively.

Hydrodensitometry (underwater weighing). Total body fat mass (FM_{UWW}) and fat-free mass (FFM_{UWW} = weight (kilograms) − FM_{UWW}) were determined by hydrodensitometry using the Siri equation. In calculating percent body fat with the Siri equation, different densities of the FFM were used for black (1.106 g/cm^3) and white (1.100 g/cm^3) women, in accordance with a report from our center that quantified the density of FFM using a four-compartment model of body composition and found FFM density to be slightly greater in black versus white women, primarily due to heavier bone mass.[33]

MRI measurements. The areas of visceral (VAT), subcutaneous (SAT), and total (TAT) adipose tissue in the abdomen were measured by MRI as described by Seidell et al[34] and van der Kooy et al.[35] With the subject in the supine position, the midpoint between the last rib and the iliac crest (midwaist) was determined in millimeters on a scout coronal image (scans by General Electric System Signa Advantage 5.3 scanner; General Electric Medical Systems, Milwaukee, WI). This midwaist point corresponds to the minimal waist and the L2-L3 spinal level.[36] Four 1.5-mm thickness axial images 5 mm apart were obtained above and below the midwaist. The images were analyzed by a single investigator on a General Electric independent display console using an "automatic boundary detection program" (5.3 software; General Electric Medical Systems). VAT, SAT, and TAT were determined by an image-masking technique. For each area, specific pixel ranges were determined that produced the best fat/nonfat delineation. The pixel ranges for VAT and SAT were similar to those reported by other investigators.[35,37] The reported values represent the average of three readings for each image. The intraclass correlation for repeated VAT determination in our laboratory is .99.

DXA measurements. DXA (Lunar, Madison, WI; software version 3.6) measurements of the central fat mass in the abdomen ($AbdFM_{DXA}$) were obtained from whole-body scans in a subset of 26 subjects (14 white and 12 black) as previously described.[4,38] Although all 38 subjects underwent DXA measurements, the proper delineation of the abdominal region to avoid inclusion of arm fat was possible only in these 26 subjects. Unlike CT and MRI, DXA-measured fat mass is not solely adipose tissue but is the sum concentration of fatty elements in soft tissue. The region of interest in the abdomen comprised all tissue below the first and above the fifth lumbar vertebrae (L1-L2 to L4-L5), similar to regional measurements described previously.[7,39] DXA total truncal fat measurements were not used, since these contain an excessive amount of gluteal fat and the pelvic fat in its entirety.

VFM and AbdSCFM. Using calculations similar to those described by Jensen et al,[40] the absolute quantities of visceral and subcutaneous abdominal fat mass (VFM and AbdSCFM) were calculated from the total central abdominal fat mass ($AbdFM_{DXA}$) as measured by DXA, based on the relative proportions of visceral and subcutaneous fat areas (VAT, SAT, and TAT) as measured by MRI, respectively: VFM (g) = [VAT (cm^2)/TAT (cm^2)] × $AbdFM_{DXA}$ (g), and AbdSCFM (g) = [SAT (cm^2)/TAT (cm^2)] × $AbdFM_{DXA}$ (g).

Metabolic Measurements

All metabolic measurements were performed after a 12-hour overnight fast.

OGTT. Plasma glucose and insulin levels were measured in blood samples taken at 30-minute intervals for 2 hours after glucose ingestion (75 g dextrose; Baxter Healthcare, Valencia, CA). The plasma glucose level was measured with a glucose analyzer (Beckman, Fullerton, CA; coefficient of variation [CV] <4%), and the plasma insulin level was measured by radioimmunoassay using the charcoal extraction technique (CV, 12%).[41] This method does not distinguish insulin from proinsulin levels. Glucose and insulin areas under the curve (AUCs) were estimated by the trapezoid method.

Frequently sampled intravenous glucose tolerance test. Peripheral insulin sensitivity was measured in vivo according to the tolbutamide-modified frequently sampled intravenous glucose tolerance test (FSIGT) of Bergman et al.[42] For all subjects, this measurement was made within 10 days of the onset of the menstrual cycle. Glucose (0.3 g/kg, 50% dextrose injection; Abbott, North Chicago, IL) was administered intravenously at time 0 minutes, followed by injection of tolbutamide (Orinase Diagnostic; Upjohn, Kalamazoo, MI) at 20 minutes. Subjects with a body mass index (BMI) less than 30 kg/m^2 received 300 mg tolbutamide, and subjects with a BMI greater than 30 kg/m^2 received 500 mg. Frequent blood sampling (taken before glucose injection at −20, −15, −10, and −5 minutes and after glucose injection at 2, 3, 4, 5, 6, 8, 10, 12, 14, 16, 19, 22, 24, 25, 27, 30, 40, 60, 70, 90, 100, 120, 140, 160, and 180 minutes) was performed through a catheter in the contralateral arm. Plasma glucose and insulin levels were measured on all samples, and S_I was calculated from these values with the nonlinear mathematical model of glucose disappearance (MINMOD program; copyright R.N. Bergman, 1986).

Fasting serum TG. Fasting blood samples were taken for the measurement of total TG (assay kit 210; Diagnostic Chemicals, Monroe, CT).

Data Analysis

Unless otherwise noted, data are presented as the mean ± SD. Between-race comparisons of body composition and metabolic measurements were performed using *t* tests for independent samples. Pearson product-moment correlation coefficients and partial correlation coefficients controlling for FM_{UWW} were calculated for the entire sample to determine univariate and partial relationships between body composition measurements and metabolic parameters. Multiple regression analysis with backward elimination was performed to determine the combination of body composition variables that were most predictive of S_I and TG. For deriving predictive equations, backward elimination closely replicates the outcome of trying all possible combinations of independent variables to determine the best single model.[43] When appropriate, slope and intercept terms of regression models were compared between races. Since only 26 of 38 subjects included in this study had DXA abdominal fat measurements, all analyses (correlations, *t* tests, and regressions) involving the total abdominal fat mass ($AbdFM_{DXA}$) or derivatives thereof (VFM and AbdSCFM) reflect this subset of the entire sample. A significance level of .05 was used for all

statistical tests. All statistical analyses were performed using SPSS version 6.1 for Windows (Chicago, IL).

RESULTS

The 20 white and 18 black women included in this study were similar with respect to age, weight, height, BMI, FM_{UWW}, FFM_{UWW}, percent fat, PerSUM, and TrSUM (all $P \geq .17$; Table 1). This was also true for the subset of 14 white and 12 black women with DXA measurements. Table 1 also presents metabolic measurements for all subjects. Although no significant racial differences were noted for S_I ($P = .65$), insulin AUC ($P = .64$), or TG ($P = .06$), white women did have a significantly larger glucose AUC than blacks ($P < .001$). Again, these measurements were similar for the subset of women with DXA measurements. Table 2 shows the abdominal adipose tissue areas measured by MRI (VAT, SAT, and TAT), abdominal fat mass measured by DXA ($AbdFM_{DXA}$), and calculated values for VFM and AbdSCFM for the 26 subjects with DXA measurements. There were no significant between-race differences in any abdominal fat measurements for the subjects evaluated (all $P > .27$). Table 3 shows Pearson correlation coefficients for body composition measurements and skinfolds in all subjects (N = 38) and for body composition measurements and abdominal fat measurements for 26 subjects with DXA. TrSUM was significantly correlated with FM_{UWW}, $AbdFM_{DXA}$, VFM, and AbdSCFM (all $P \leq .05$). However, AbdSCFM explained only about 16% of the variance in TrSUM.

Univariate and partial (controlling for FM_{UWW}) correlation coefficients between selected body composition measurements and S_I are presented in Table 4. FM_{UWW}, $AbdFM_{DXA}$, VFM, TrSUM, and PerSUM were negatively correlated with S_I (all $P \leq .022$), but AbdSCFM was not correlated with S_I ($P = .23$). After controlling for FM_{UWW}, only VFM ($P = .035$) and TrSUM ($P = .002$) were significantly related to S_I. Among 26 subjects with DXA abdominal fat mass measurements, multiple regression analysis with backward elimination was performed to determine the relationship between S_I and the body composition measurements FM_{UWW}, VFM, TrSUM, and PerSUM. Only the relation to TrSUM remained significant after modeling ($P < .001$). When a similar modeling procedure was performed using all 38 subjects after race stratification, FM_{UWW} and PerSUM dropped out of the equation, leaving TrSUM as the only significant predictor of S_I in blacks ($P = .03$) and whites ($P < .001$) (Fig 1). When the regression of S_I on TrSUM was compared between races, it was found that while the slope of the regression line did not differ ($P = .06$), the intercept for whites was higher than for blacks ($P = .032$). Thus, at all levels of truncal subcutaneous fat, black women were significantly more insulin-resistant than white women.

Univariate and partial (controlling for FM_{UWW}) correlation coefficients between selected body composition measurements and TG are presented in Table 5. FM_{UWW}, $AbdFM_{DXA}$, VFM, and TrSUM were negatively correlated with TG (all $P \leq .011$), while AbdSCFM was not correlated with TG ($P = .38$). After controlling for FM_{UWW}, only $AbdFM_{DXA}$ ($P = .013$) and VFM ($P < .001$) were significantly related to TG. For 26 subjects with DXA abdominal fat mass measurements, multiple regression analysis with backward elimination was performed to determine the relationship between TG and the body composition measurements FM_{UWW}, VFM, TrSUM, and PerSUM. Only the relation to VFM remained significant after modeling ($P < .001$). Furthermore, for 26 subjects evaluated, the relationship between TG and VFM did not differ by race ($P \geq .35$).

Table 1. Subject Characteristics

Characteristic	White Women (n = 20)	Black Women (n = 18)
Age (yr)	34 ± 6	36 ± 6
Weight (kg)	95 ± 15	92 ± 11
BMI (kg/m²)	35 ± 5	34 ± 3
FM_{UWW} (kg)	44 ± 10	41 ± 8
%Fat	45 ± 5	44 ± 6
TrSUM (mm)	268 ± 44	245 ± 55
PerSUM (mm)	161 ± 24	155 ± 28
Insulin AUC (pmol/2 h)	2,679 ± 1,673	2,437 ± 1,467
Glucose AUC (mmol/2 h)*	29.3 ± 5.1	23 4 ± 3.7
S_I (10^{-4} min^{-1} · mU · mL^{-1})	2.6 ± 2.2	2.3 ± 2.1
TG (mmol/L)	1.25 ± 0.52	0.94 ± 0.47

NOTE. Values are the mean ± SD. %Fat = 100 · FM_{UWW} (kg)/weight (kg).

*$P < .001$. All other between-race comparisons, $P > .06$.

Table 2. Abdominal Fat Measurements

Parameter	White Women (n = 14)	Black Women (n = 12)
TAT (cm²)	497 ± 88	478 ± 121
VAT (cm²)	112 ± 50	105 ± 43
SAT (cm²)	385 ± 72	373 ± 107
$AbdFM_{DXA}$ (g)	3,150 ± 525	2,936 ± 569
VFM (g)	709 ± 304	646 ± 296
AbdSCFM (g)	2,441 ± 490	2,290 ± 429

NOTE. Results are the mean ± SD for 26 subjects (14 whites and 12 blacks) with DXA abdominal fat measurements. For all between-race comparisons, $P \geq .33$.

DISCUSSION

The clustering of upper-body and abdominal obesity, insulin resistance, and dyslipidemia has received particular attention with regard to defining the metabolic hazards of obesity.[44-46] In this study, we sought to examine the relationship of different components of upper-body obesity to insulin sensitivity and serum TG.

We and others previously reported independent associations of VAT but not of SAT areas measured at L_4-L_5 or L_2-L_3 with insulin sensitivity and TG levels in obese women.[12,14-16] As a methodological improvement over these studies, we combined MRI and DXA abdominal measurements to evaluate the contributions of $AbdFM_{DXA}$, VFM, and AbdSCFM and TrSUM and PerSUM measurements to the trunk versus peripheral subcutaneous fat mass.

We found that the $AbdFM_{DXA}$ and VFM were significantly associated with insulin sensitivity, whereas the AbdSCFM was not. Previous studies using DXA methodology also found a strong relationship of total abdominal fat mass measurements by DXA with insulin sensitivity in lean and mildly obese women,[39] as well as more severely obese women.[47] However, in these studies, the separate identification of visceral versus subcutaneous abdominal fat mass was not made. When we separated the two depots using their relative ratio by MRI, we

Table 3. Pearson Correlation Coefficients Between Body Composition Measurements

Parameter	FM_{UWW}	FFM_{UWW}	TrSUM	PerSUM	VAT	SAT	$AbdFM_{DXA}$	VFM
FM_{UWW}	—							
FFM_{UWW}	.38*	—						
TrSUM	.64‡	.27	—					
PerSUM	.52‡	00	.64‡	—				
VAT	.56‡	34	.59‡	.21	—			
SAT	.79‡	.20	.41*	.40*	.29	—		
$AbdFM_{DXA}$	.63‡	36	.74‡	.54†	.46*	.54†	—	
VFM	.34	.19	.74‡	.50†	.95‡	−.10	.53†	—
AbdSCFM	53†	.31	.40‡	.32	−.06‡	.70‡	.84‡	−.01

*$P < .05$.
†$P < .01$.
‡$P \leq .001$.

found that in obese women, only the VFM, not the AbdSCFM, was significantly related to insulin sensitivity independently of total body fat mass. This finding is in contrast to results reported in obese men.[25,26] In these studies, the AbdSCFM measured by MRI from the dome of the diaphragm to the symphysis pubis was a better predictor of decreased insulin sensitivity than the VFM. These contrasting findings may reflect methodological differences in the determination of fat distribution in the abdomen and/or may be a result of different adverse effects of specific subcutaneous fat depots on the trunk in women compared with men.

The measurement of subcutaneous abdominal fat as a single area by MRI and measurement of subcutaneous abdominal fat by a combination of DXA and MRI have limitations. The amount of subcutaneous abdominal fat in a single area may be dependent on the level at which it is measured. For example, in men, lower-lumbar and sacral MRI slices contain nearly twice the area of posterior subcutaneous fat compared with more rostral slices in the abdomen.[48] The measurement of total abdominal fat mass by DXA in our subjects was consistent and compares well with earlier reports using DXA methodology.[7,39,49] However, the region of interest we measured by DXA corresponds to a significantly smaller area of the abdomen (top of L_2 to bottom of L_4) than is typically included in studies using multiple slice imaging by CT or MRI in women[18] or men.[25,26] We chose this region because, in previous studies using DXA methodology, it was shown that in women the central abdominal fat region relates much better to the degree of insulin sensitivity than the total amount of trunk fat.[39] The total trunk fat mass by DXA may have included an excessive amount of gluteal and pelvic fat. Indeed, when we analyzed the effect of trunk subcutaneous fat mass by DXA (trunk fat mass minus the VFM) in our subjects, we again did not find an independent relationship with insulin sensitivity (results not shown). Therefore, the method by which upper-body subcutaneous fat is measured may influence the evaluation of its relationship to insulin sensitivity.

However, we found that in our sample of obese women, the sum of a series of skinfold thickness measured on the trunk (TrSUM) was a better predictor of insulin sensitivity than the sum of skinfolds measured on the extremities (PerSUM). Even more importantly, TrSUM was a better predictor of insulin sensitivity than VFM. The finding of a relationship between the individual truncal skinfolds or their sum and indices of carbohydrate metabolism is not new. Feldman et al,[19] 30 years ago, found that diabetic women had a greater absolute truncal skinfold thickness but showed no differences in the skinfold thickness of the upper and lower extremities compared with nondiabetic women. Despres et al[11] found a significant correlation between the sum of several truncal skinfolds and several parameters of glucose metabolism in both the fasting state and during glucose tolerance testing in 52 premenopausal obese women. Furthermore, the role of truncal subcutaneous fat with regard to these obesity-associated health risks is supported by the finding of increased rates of lipolysis in upper-body

Table 4. Univariate and Partial (controlling for $TBFM_{UWW}$) Correlations Between Selected Body Composition Measurements and S_I

Parameter	Univariate *r*	*P*	Partial *r*	*P*
FM_{UWW}, kg (N = 38)	−.56	<.001	—	
TrSUM, mm (N = 38)	−.62	<.001	−.41	.012
PerSUM, mm (N = 38)	−37	.022	−.11	.51
$AbdFM_{DXA}$, g (n = 26)	−.49	.012	−.26	.20
VFM, g (n = 26)	−.51	.008	−.42	.035
AbdSCFM, g (n = 26)	−.25	.23	.014	.95

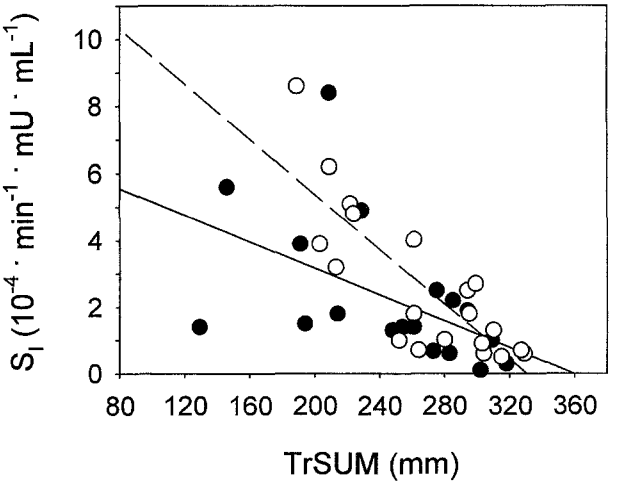

Fig 1. S_I versus TrSUM for black (●) and white (○) women. Blacks: S_I (10^{-4} min^{-1} · mU · mL^{-1}) = −0.020 · TrSUM (mm) + 7.1 (R^2 = .26, SEE = 1.9, P = .031); whites: S_I (10^{-4} min^{-1} · mU · mL^{-1}) = −0.041 · TrSUM (mm) + 13.6 (R^2 = .67, SEE = 1.3, $P < .001$).

Table 5. Univariate and Partial (controlling for FM_{UWW}) Correlations Between Selected Body Composition Measurements and Serum TG

Parameter	Univariate *r*	*P*	Partial *r*	*P*
FM_{UWW}, kg (N = 38)	.41	.011	—	
TrSUM, mm (N = 38)	.49	.002	.32	.053
PerSUM, mm (N = 38)	.30	.065	.11	.50
$AbdFM_{DXA}$, g (n = 26)	.56	.003	.49	.013
VFM, g (n = 26)	.76	<.001	.73	<.001
AbdSCFM, g (n = 26)	.18	.38	.01	.96

subcutaneous adipocytes,[23-24] which contribute to higher systemic FFA flux in women with a greater proportion of upper-body (truncal/abdominal) fat compared with women with a greater proportion of lower-body (gluteal/femoral) fat.[22]

It should be noted that we are unaware of any previously published report that validates the sum of truncal skinfolds versus cadaveric-, CT-, or MRI-derived measurements of total truncal subcutaneous fat in women. However, for subjects in the present study, abdominal subcutaneous fat measured by a combination of MRI and DXA explained only about 16% of the variance in TrSUM, while VFM was significantly correlated with TrSUM even after adjustment for abdominal subcutaneous fat ($P < .001$). In women, this may reflect the heterogeneity of subcutaneous fat on the trunk and perhaps an inherent association between certain truncal subcutaneous fat depots and visceral fat accumulation that is largely independent of the quantity of abdominal subcutaneous adipose tissue. In women, whether there is lipolytic heterogeneity among truncal subcutaneous adipocytes, ie, those in the upper trunk versus the abdominal region, is not known. Thus, the adverse effects of subcutaneous fat on the trunk may reflect fat deposition on the upper trunk, as well as the abdominal region. Indeed, accumulation of subcutaneous fat specifically on the chest and subscapular region has been associated with impaired glucose tolerance in Japanese-American women.[21] In this respect, in women, TrSUM may be a better representation of undesirable truncal subcutaneous fat distribution than just abdominal subcutaneous fat.

Previous reports from our laboratory[15] and others[17] have suggested that the association between visceral adiposity and insulin resistance is affected by race. Specifically, black women tend to be more insulin-resistant than white women at all levels of fatness.[17] In the present study, an identical trend was observed when comparing the association between insulin sensitivity and truncal subcutaneous fat across race. After adjustment for TrSUM, black women were significantly more insulin-resistant than white women ($P = .032$) (Fig 1). This finding is particularly important given the greater tendency for upper-body subcutaneous fat accumulation in black women.[28-30]

A strong association has been noted previously between the absolute or relative quantity (visceral to subcutaneous ratio) of visceral fat and the serum TG level.[15,50-52] In the present study, VFM was the strongest individual predictor of serum TG, even after controlling for total body fat mass. Neither the TrSUM nor the AbdSCFM were significantly related to serum TG after controlling for total body fat mass. Thus, these findings confirm the contribution of visceral fat to serum lipids in obese women.[9,15-17] With respect to blood lipids, we observed racial homogeneity in the relationship between the serum TG level and VFM.

Taken together, the results of this study indicate that the health risks of upper-body obesity in women may reflect specific contributions of different compartments of central adipose tissue. That is, insulin sensitivity appears to be a function of both visceral and subcutaneous fat on the trunk, while the serum TG level appears largely dependent on visceral fat accumulation. Rosenfalck et al,[53] using DXA-derived measures of regional adipose tissue in 89 women, found that abdominal fat was most predictive of serum TG, while fasting insulin was most related to the degree of truncal adipose tissue. While neither of the measurements used in that study directly reflect distribution of fat between subcutaneous versus visceral depots, the results indicate a potential heterogeneity of metabolic risk in relation to different regions of upper-body fat.

In conclusion, the present study indicates that specific subcutaneous and visceral components of central adiposity are differentially related to individual parameters of carbohydrate and lipid metabolism in obese premenopausal women. Further studies are needed to clarify the mechanism of these associations in various high-risk obese groups.

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