Published in final edited form as:

Eur J Clin Nutr. 2015 March ; 69(3): 329–336. doi:10.1038/ejcn.2014.227.

DEXA MEASURED VISCERAL ADIPOSE TISSUE PREDICTS IMPAIRED GLUCOSE TOLERANCE AND METABOLIC SYNDROME IN OBESE CAUCASIAN AND AFRICAN AMERICAN WOMEN

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Abstract

Background and Aims—New methods to measure visceral adipose tissue (VAT) by DEXA may help discern sex, race and phenotype differences in the role of VAT in cardiometabolic risk. This study was designed to: a) compare relationships between cardiometabolic risk factors and DEXA-VAT, anthropometric and body composition measures; b) determine thresholds for DEXA-VAT by race; and c) determine the most robust predictors of impaired glucose tolerance (IGT) and metabolic syndrome (MetSx) in obese women.

Methods—VAT area (cm^2) was measured using Lunar iDXA scanner in 229 obese (BMI 30-49.9) women age 21–69 years of European American (EA = 123) and African American (AA = 106) descent. Linear regression modeling and areas under the curve (AUC) compared relationships with cardiometabolic risk. Bootstrapping with LASSO regression modeling determined thresholds and predictors of IGT and MetSx.

Results—DEXA-VAT explained more of the variance in triglycerides, blood pressure, glucose and HOMA-IR compared to anthropometric and body composition variables. DEXA-VAT had the highest AUC for IGT (0.767) and MetSx (0.749). Including race and interactionXrace terms in modeling did not significantly change results. Thresholds at which probability was ≥ 50% for IGT or MetSx were lower in AA women (IGT: 2120cm^2 AA *vs* 2550cm^2 EA; MetSx: 1320cm^2 AA *vs* 1713cm^2 EA). The odds for IGT or MetSx was 3-fold greater with each standard deviation increase in DEXA-VAT.

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DISCLOSURES:

None of the authors have financial interests to disclose.

Conclusion—DEXA-VAT provides robust clinical information regarding cardiometabolic risk in AA and EA women and has great potential in risk reduction efforts.

Keywords

obesity; visceral; DEXA; body composition; cardiovascular; metabolic syndrome

INTRODUCTION

The accumulation of body fat in the visceral depot is mechanistically related to insulin resistance and development of type 2 diabetes, hypertension, dyslipidemia and cardiovascular disease (1-3). More specifically, intra-abdominal or visceral adipose tissue (VAT) promotes hepatic inflammation, steatosis, insulin resistance and dyslipidemia by releasing free fatty acids, hormones, and inflammatory chemokines and cytokines into the portal circulation that functionally impair insulin sensitivity and action (4, 5). While VAT is particularly pathogenic (6-8) and independently predicts all-cause mortality (9), mounting evidence suggests abdominal subcutaneous adipose tissue (SAT) also impacts the development of obesity related insulin resistance in certain phenotypes by first releasing fatty acids into the venous circulation and later into the portal vein (10, 11). With the epidemic prevalence of overweight and obesity (BMI ≥ 25) reaching 70% of U.S. adults (12), there is need to reliably differentiate and quantify VAT and SAT to discern differences in the role and function of VAT and SAT by sex, race and metabolically at risk phenotypes.

Although the prevalence of abdominal adiposity has increased two-fold in U.S. women over the past four decades (13), men tend to have greater VAT accumulation than (pre-menopausal) women (9, 14). In fact, women have about five times more SAT than VAT in the intra-abdominal compartment (15). This sexual dimorphism is apparent in other cardiovascular risk biomarkers (16) as well as relationships between abdominal fat and cardiometabolic risk. Racial and ethnic differences are also evident as people of south Asian descent have a greater proportion of VAT compared to those of European descent (17, 18) and European Americans have more VAT than African Americans, even when controlled for total body fat (19, 20). Despite less VAT, African Americans have greater prevalence of insulin resistance, type 2 diabetes and cardiovascular disease than other population subgroups (21-23). While prior investigations indicate functional roles for both VAT and SAT in the disparities associated with genotype (24), more information is needed further elucidating sex and race differences in the relationship between VAT and SAT and cardiometabolic risk to enable design of subgroup specific interventions.

In clinical practice and public health settings, anthropometric measures such as body mass index (BMI), waist circumference (WC), hip circumference (HC), waist-hip ratio (WHR), and more recently waist-height ratio (WHtR), are used as surrogates for intra-abdominal adiposity since they require little expense, time or technical expertise. Notably, these indicators are predictive of cardiometabolic outcomes in large population groups (25). However, anthropometric measures are unable to distinguish fat *versus* lean mass or the amount, type and distribution of adipose tissue. For example, BMI, WC, WHR and WHtR do not discriminate whether a higher value is due to increased total abdominal fat or the relative proportion of VAT to SAT. Hence, such measures cannot advance science regarding the particular roles of VAT *versus* SAT in specific genotypes or phenotypes such as the "metabolically healthy" obese or "metabolically unhealthy" lean (26-28). Moreover, manual measures are subject to high inter-rater variability (29). Considering these limitations, computed tomography scan (CT) and magnetic resonance imaging (MRI) are the recognized gold standards for quantifying and comparing regional fat amount, type and distribution in research settings. As such, these imaging techniques have contributed greatly to understanding metabolic phenotypes as well as disparities in vulnerability for cardiometabolic disease (30, 31). However, the complex and costly technology of CT and MRI limits their general clinical utility (32). Thus, there remains need for more practical, accessible and economical methods to evaluate adiposity and determine its role in cardiometabolic risk, disease and treatment.

In contrast to CT and MRI, dual energy X-ray absorptiometry (DEXA), originally designed to assess bone mineral density, allows estimation of whole body composition from a two-dimensional X-ray with low radiation exposure, short-scanning time, high precision and low cost (33). Most often, DEXA estimated intra-abdominal adipose tissue has derived from formulas based on manual manipulation of the abdominal region of interest using anatomical landmarks at the level of the 2nd to 5th lumbar vertebrae (34-36). However, such estimation has not distinguished VAT from SAT and the most reliable estimates have come from non-obese persons (35, 37, 38). Nevertheless, we previously showed strong correlations between DEXA and water-suppressed T1 weighted MRI measures of total and regional body composition, with coefficients of variation less than 2% for DEXA-derived adiposity measures (39). More recently, algorithms in updated versions of the software used on DEXA scanners have been developed to segment fat within the android region into VAT and SAT (40). This DEXA-VAT method has been validated against CT in subjects with BMI ranging from 18.5 to 40 kg/m^2 and had a coefficient of determination (r^2) of 0.959 for women and 0.949 for men (40). Yet, the relationship between DEXA-VAT and cardiometabolic risk factors has not been well established.

The purpose of this study was to test the hypothesis that DEXA-VAT is more robustly associated with cardiometabolic risk in obese women of European American and African American descent compared to other commonly acquired anthropometric and body composition indicators. Secondly, to test the hypothesis that relationships between DEXA-derived VAT and established cardiometabolic risk factors differ in obese women by race, and if so, determine thresholds by race that identify the amount of DEXA-VAT that elevates risk. Finally, to determine which anthropometric, body composition and clinical variables most robustly predicts impaired glucose tolerance and the metabolic syndrome in obese women.

METHODS

Subjects

This study is a cross-sectional analysis of DEXA acquired whole body scans from obese women enrolled in clinical trials conducted at the Vanderbilt Clinical Research Center (VCRC) between 2008 and 2013. Subjects were recruited from local media and electronic advertisements. Scans were included if female subjects were age ≥ 21 years, BMI was ≥ 30 kg/m^2, and they were non-smokers. Race was self-identified. To be included in the analysis, subjects had anthropometric (height, weight, waist and hip circumferences), biochemical and clinical data obtained simultaneous with the DEXA scan using standardized protocols. Each subject provided written informed consent and study procedures were approved by the Vanderbilt University Institutional Review Board. Prior to analysis, subject records were de-identified and stored in a Vanderbilt REDCap database (41).

Anthropometry

Physical measures were obtained using standard methods to determine clinical trial eligibility and acquire baseline data. Height was measured to the nearest 0.1cm on a wall-mounted stadiometer. Weight was measured to the nearest 0.1kg on a calibrated digital platform scale without shoes, hats, outer clothing or pocket items. Waist and hip circumferences were measured via flexible measuring tape to the nearest 0.1cm above the right iliac crest and at the fullest extension of the buttocks, respectively. BMI, waist-hip and waist-height were calculated as ratios.

Dual Energy X-Ray Absorptiometry

Total and regional body composition was acquired by a certified densitometrist using a Lunar iDXA whole body scanner (GE Healthcare, Madison, WI) with enCore 2007 software (version 11.4). Before each acquisition, the scanner was phantom calibrated according to manufacturer instructions. Duplicate scans after repositioning 12 subjects showed coefficients of variation <2% for fat and lean total and trunk masses (39). Scans were imported into an updated version of the software (version 13.6) and reanalyzed using algorithms that provided automatic segmentation of VAT from total abdominal fat within the android region. VAT mass (g) was automatically transposed into area (cm^2) using a constant correction factor (0.94 g/ml) that is consistent with the density of adipose tissue (40, 42).

Clinical and Biochemical Cardiometabolic Risk Factors

Systolic and diastolic blood pressure (SBP, DBP) and heart rate were obtained by VCRC research nurses with subjects in a supine resting state using a calibrated sphygmomanometer with a large size cuff. Fasting glucose, insulin, lipid profile (serum total cholesterol, LDL-cholesterol [LDL-C], HDL-cholesterol [HDL-C] and triglycerides [TG]) and high sensitivity C-reactive protein (hsCRP) were processed at the Vanderbilt Pathology Laboratory using standard procedures. Serum leptin was processed at the Vanderbilt Diabetes Hormone Core Laboratory. Insulin resistance (HOMA-IR) was scored using the HOMA2-IR model (43). Glucose tolerance was measured using a standard 75g oral glucose tolerance test or a frequently sampled intravenous glucose tolerance test (44, 45).

Statistical Analysis

Data were analyzed using R Statistical Software version 3.0.1 (http://www.r-project.org/). The level of significance was set at $\alpha = 0.05$. Categorical variables are presented as frequency and percentages while continuous variables as mean ± standard deviation (SD).

Data were checked for normality by visual inspection of histograms and stem and leaf plots. We used Wilcoxon signed-rank tests to assess the significance of inter-group differences. In order to determine whether DEXA-derived VAT is more robustly associated with cardiometabolic risk factors than anthropometric and other body composition variables, we fitted multiple linear regression models with VAT, anthropometric and body composition variables as outcome variables and cardiometabolic risk factors as independent variables. We then compared the coefficients of determination across the outcome variables while adding race and the interaction between race and the independent variable in the models to identify potential effects of race on these relationships. Next, we calculated areas under the curve (AUC) using binary logistic regression. Regression modeling was performed with VAT unadjusted and adjusted for body surface area. As no significant differences were detected between models, we present results only using unadjusted VAT. To identify variables from the binary regression models that most minimized sum of the squares of the errors we performed bootstrapping to resample the individual observations with replacement (N = 200) and least absolute shrinkage and selection operator (LASSO) regression modeling. This process allowed identification of significant predictors for two separate cardiometabolic outcomes: 1) impaired glucose tolerance (IGT) defined as having fasting glucose ≥ 100 mg/dl, 2-hour glucose between 140 and 199 mg/dl (46), and/or glucose disappearance constant (K_G) < 1.5 (44, 47); and 2) the metabolic syndrome (MetSx) defined as having ≥ 3 of the 5 National Cholesterol Education Program's Adult Treatment Panel III criteria as modified by the American Heart Association (48). With anthropometric, body composition and cardiometabolic risk factors as independent variables, the residual deviance was treated as a chi square value to test the overall fit of each model. We also calculated the odds ratio for each independent variable in our final logistic regression models. For each race, we established a VAT threshold to determine at what point the probability of IGT and MetSx was at least 50% while other independent variables were fixed at their mean values.

RESULTS

Demographics and Baseline Characteristics

DEXA scans were acquired from 229 women who ranged in age from 21 to 69 years old and in BMI from 30.0 to 49.5 kg/m^2. Of these 229, 123 (53.7%) reported being of European American (EA) descent and 106 (46.3%) reported being of African American (AA) descent. Total body fat ranged from 32.0 to 56.0% and VAT ranged from 173 to 5655cm^2. Despite having similar mean age, height and % body fat (Table 1), the EA women had lower lean body mass, BMI, HC, WHtR, SBP, DBP, fasting insulin and HOMA-IR. Simultaneously, mean DEXA-VAT and serum TG levels were lower in the AA women.

DEXA-VAT and Cardiometabolic Risk Factors

In both groups, DEXA-VAT was positively associated with TG, fasting glucose, fasting insulin and HOMA-IR, and negatively associated with HDL-C. DEXA-VAT was also positively associated with SBP and DBP in EA women, but not AA women. DEXA-VAT was positively associated with hs-CRP in AA women, but not EA women. We next assessed whether DEXA-VAT was more strongly associated with cardiometabolic risk factors compared to the other anthropometric and body composition variables (Table 2).

Incorporating the interaction between DEXA-VAT and race, the strength of the relationships between SBP and DBP to DEXA-VAT, BMI, WC, HC, WHR and WHtR were similar. However, DEXA-VAT explained 10.2% more of the total variance in SBP and 11.7% more of the total variance in DBP than the other body composition variables (%body fat, %trunk fat, %android fat and android/gynoid ratio). WHR and android/gynoid ratio explained more of the total variance in HDL-C than DEXA-VAT or WC (+13.8%, +12.2%, +7.5% and +6.9%, respectively). Yet, DEXA-VAT explained 24.6% more of the total variance in TG than all other variables. Although WC explained as much of the total variance in fasting insulin as DEXA-VAT ($r^2 = 21.5\%$ *vs* 21.7%, respectively), DEXA-VAT explained 31.8% more of the total variance in fasting glucose and 25.9% more of the total variance in HOMA-IR than all other variables.

DEXA-VAT and Impaired Glucose Tolerance

The proportion of EA and AA women with IGT did not differ (29.3 *vs* 29.2%, X_2 (1) = 0.00, $P = 0.100$). To determine whether DEXA-VAT was a more robust predictor of IGT than the other anthropometric and body composition variables, we performed binary logistic regression. DEXA-VAT had the highest area under the curve (AUC = 0.766), with AUC values for anthropometric and other body composition variables ranging from 0.534 to 0.703 (Table 3). Adjusting the AUC for race (0.766) or incorporating the interaction between DEXA-VAT and race (0.759) did not change the significance of the relationship between DEXA-VAT and IGT. However, there was a difference by race in the amount of DEXA-VAT with regard to the probability of developing IGT; in EA women having DEXA-VAT of ≥ 2550cm^2 increased the likelihood of developing IGT by ≥ 50% whereas in AA women having DEXA-VAT of ≥ 2120cm^2 increased the likelihood of developing IGT by ≥ 50% (Figure 1).

DEXA-VAT and Metabolic Syndrome

The proportion of EA and AA women with metabolic syndrome also did not differ significantly (39.0 *vs* 48.1%, X_2 (1) = 1.92, $P = 0.17$). Again, we used binary logistic regression to determine whether DEXA-VAT was a more robust predictor of MetSx in comparison to the other anthropometric and body composition variables in these obese women. As with IGT, DEXA-VAT had the highest area under the curve (AUC = 0.749) for MetSx, with AUC values for anthropometric and body composition variables ranging from 0.506 to 0.712. Neither adjusting the AUC for race (0.755) nor incorporating the interaction between DEXA-VAT and race (0.749) altered the significance of the relationship between DEXA-VAT and MetSx. Also similar to IGT, there was a difference by race in the amount of DEXA-VAT with regard to the probability of developing MetSx; in EA women having DEXA-VAT of ≥ 1713cm^2 increased the likelihood of developing MetSx by ≥ 50% whereas in AA women having DEXA-VAT of ≥ 1320cm^2 increased the likelihood of developing MetSx by ≥ 50% (Figure 2).

Multivariate Models for Impaired Glucose Tolerance and Metabolic Syndrome

The final analyses were performed to determine the best fitting multivariate regression models to predict IGT and MetSx in these obese women, using the anthropometric, body composition and cardiometabolic variables that significantly improved the binary regression models. Bootstrapping with LASSO yielded DEXA-VAT, SBP, fasting insulin and hsCRP as the independent variables that accounted for most of the variability in the outcome of IGT (Table 4). DEXA-VAT most significantly predicted IGT ($P < 0.001$). Accounting for SBP, fasting insulin and hsCRP in the model, each standard deviation increase in VAT (SD = 880 cm^2) increases the odds of having IGT by 3.04-fold. Comparing the residual deviance for the multivariate model with and without race, as well as the interaction between DEXA-VAT and race, did not alter the significance of the model ($P = 0.49$).

With MetSx as the outcome variable (Table 5), bootstrapping with LASSO yielded DEXA-VAT, HOMA-IR and LDL-C as the independent variables most accounting for the variability in MetSx. Like IGT, DEXA-VAT most significantly predicted MetSx ($P < 0.001$). Accounting for HOMA-IR and LDL-C in the model, each standard deviation increase in VAT increases the odds of having MetSx by 3.28-fold. Again, comparing the residual deviance for the multivariate model with and without race, as well as the interaction between DEXA-VAT and race, did not alter the significance of the model ($P = 0.31$).

DISCUSSION

We previously demonstrated that DEXA whole body scans are as reliable as whole body continuous MRI for measurement of total fat, total lean, trunk fat and trunk lean masses (39). The present study extends our prior findings by showing that estimating VAT directly from DEXA whole body scans is a more robust indicator of cardiometabolic risk in women with Class I and II obesity of European American and African American descent than other more commonly acquired anthropometric (weight, BMI, waist circumference, waist-hip ratio, waist-height ratio) and DEXA-derived surrogate measures of intra-abdominal adipose tissue (percent body fat, percent trunk fat, percent android fat, android/gynoid ratio). Simultaneous assessment of bivariate relationships with risk factors and comparison of the areas under the curve between VAT and each anthropometric and body compositon variable using binary logistic regression modeling confirmed the strength of the relationships between DEXA-VAT and risk factors in both groups of obese women.

Upon evaluating each anthropometric and body compositon biomarker individually, one detectable difference was that DEXA-VAT correlated with hsCRP levels only in AA women even though the high body mass and elevated serum concentrations of C-reactive protein suggest presence of systemic inflammation in all these women. Importantly, animal and humans studies confirm that systemic inflammation in obesity is mechanistically linked to insulin resistance and future development of type 2 diabetes and cardiovascular disease (49-51) as VAT secretes pro-inflammatory cytokines such as TNF-α and IL-6 along with chemokines such as macrophage migration inhibitory factor and the CC chemokine receptor 2 (52, 53). While one study showed higher TNF-α and soluble TNF receptors in EA women (54), others have reported greater levels of of inflammatory biomarkers such as serum CRP concentration in African Americans. The higher rates of cardiometabolic disease in AA women suggest greater sensitivity and/or consequences from existing in this state of chronic inflammation (55). While overall body fat or total trunk fat might explain this disparity, the present cohort were well matched in both total fat mass, percent total body fat, percent trunk fat and percent android fat. Future investigation of other physiological stress factors that may predispose differential response to inflammatory signals may help explain this phenomenon. It is also possible that differences in dietary intakes or physical activities play a role in the relationship between VAT and inflammaton. For example, some long chain fatty acids (i.e., palmitate (56, 57)), and dietary glycemic load (58, 59) have been shown to induce inflammation and peripheral insulin resistance.

Another detectable difference was that DEXA-VAT correlated with blood pressure (SBP and DBP) only in EA women. Neural mechanisms have been suggested as a possible link between excess adiposity and high blood pressure. In prior work, we found that increased sympathetic nerve activity as measured by direct recording of sympathetic nerves in skeletal muscle contributed to obesity related hypertension (60). Notably, VAT (measured by CT) correlates better with muscle sympathetic nerve activity than other body composition measures. Overall, little investigation has been conducted directly assessing racial differences in the association between VAT and blood pressure. One study has reported that the association between the sympathetic nervous system and obesity is less strong in AA women (61). Yet, findings based on the relationship between WC and SBP in women show conflicting results with regard to race differences (62, 63). With the high prevalence of hypertension in the African American population, 42% *vs* 28% in white adults (64), it may be that mechanisms unrelated to VAT byproducts better explain this profound disparity.

Importantly, we further extend current findings by showing that DEXA-derived VAT was the strongest predictor of having impaired glucose tolerance in both EA and AA women. Now recognized as a condition of prediabetes, IGT currently affects > 40 million U.S. adults, elevating risk for progression to full-blown type 2 diabetes (65). In fact, the odds of having IGT was 3-fold greater for each standard deviation increase in DEXA-VAT, underscoring the physiological role of VAT in the development of impaired insulin action. Having thresholds of DEXA-VAT for obese EA and AA women that elucidate at what point the probability of having impaired glucose tolerance is at least 50% provides clinically meaningful information as one-third of Americans who do develop type 2 diabetes remain undiagnosed (65, 66).

While other studies have reported VAT thresholds, most of the work has been performed in white adults and has focused solely on the construct of the metabolic syndrome. Two studies that reported VAT thresholds in African Americans used cross-sectional CT scans to identify VAT (67, 68). Shedding additional light on the importance of VAT in the ethnic disparities of cardiometabolic risk, the thresholds determined in the present study from DEXA-VAT were lower in AA women for both IGT and MetSx. While our finding for MetSx is in agreement with recent findings from a larger biracial sample where DEXA-VAT was assessed similarly using a Hologic brand scanner (69), our results suggest that in obese women VAT is highly pathogenic in both EA and AA women.

A considerable strength of this cohort of obese women was that the EA and AA women were not only similar in age but unusually homogeneous in height, waist circumference, total fat mass as well as percentage total body fat, trunk fat, android fat and android/gynoid ratio. Moreover, the proportion of EA and AA women in the cohort was similar which provided the power to make comparisons and detect inter-group differences. A relative limitation of this study was the cross-sectional design of our analyses which restricts inference with regard to causality and we included obese women only which restricts the generalizability of the findings. Another limitation was the inability to screen subjects for use of antihypertensive or lipid-lowering medications which might influence the biochemical results. Future work is needed with the newer DEXA algorithms using prospective and more representative population samples.

In conclusion, the present findings contribute to the accumulating body of evidence that show DEXA-VAT is a robust indicator of cardiometabolic risk. Specifically, this study shows that DEXA-VAT predicts having IGT and MetSx in women with Class I and II obesity who are of both EA and AA descent. By being able to determine cut-points in DEXA-VAT that identify at what level risk for having IGT and MetSx elevates substantially in each group, DEXA-VAT provides robust information regarding detrimental health consequences of accumulating intra-abdominal fat. Compared to other imaging techniques, DEXA scanners are widely available, radiation exposure and patient burden is low, and cost is modest. With VAT being a fundamentally meaningful measure of cardiometabolic risk and anthropometric measures being unable to discern fat *versus* lean mass or differentiatie type of fat, recently developed algorithms for assessing VAT by DEXA provide clinically useful information to determine cardiometabolic risk and aid in the design of phenotype specific interventions to reduce risk.

Acknowledgements

This study was supported by a grant from the Dr. Robert C. and Veronica Atkins Foundation to Dr. Silver, NIH K23 HL103976 PhRMA Foundation Career Development Award to Dr. Shibao, and resources from Vanderbilt CTSA award UL1TR000445 from the NIH National Center for Advancing Translational Sciences.

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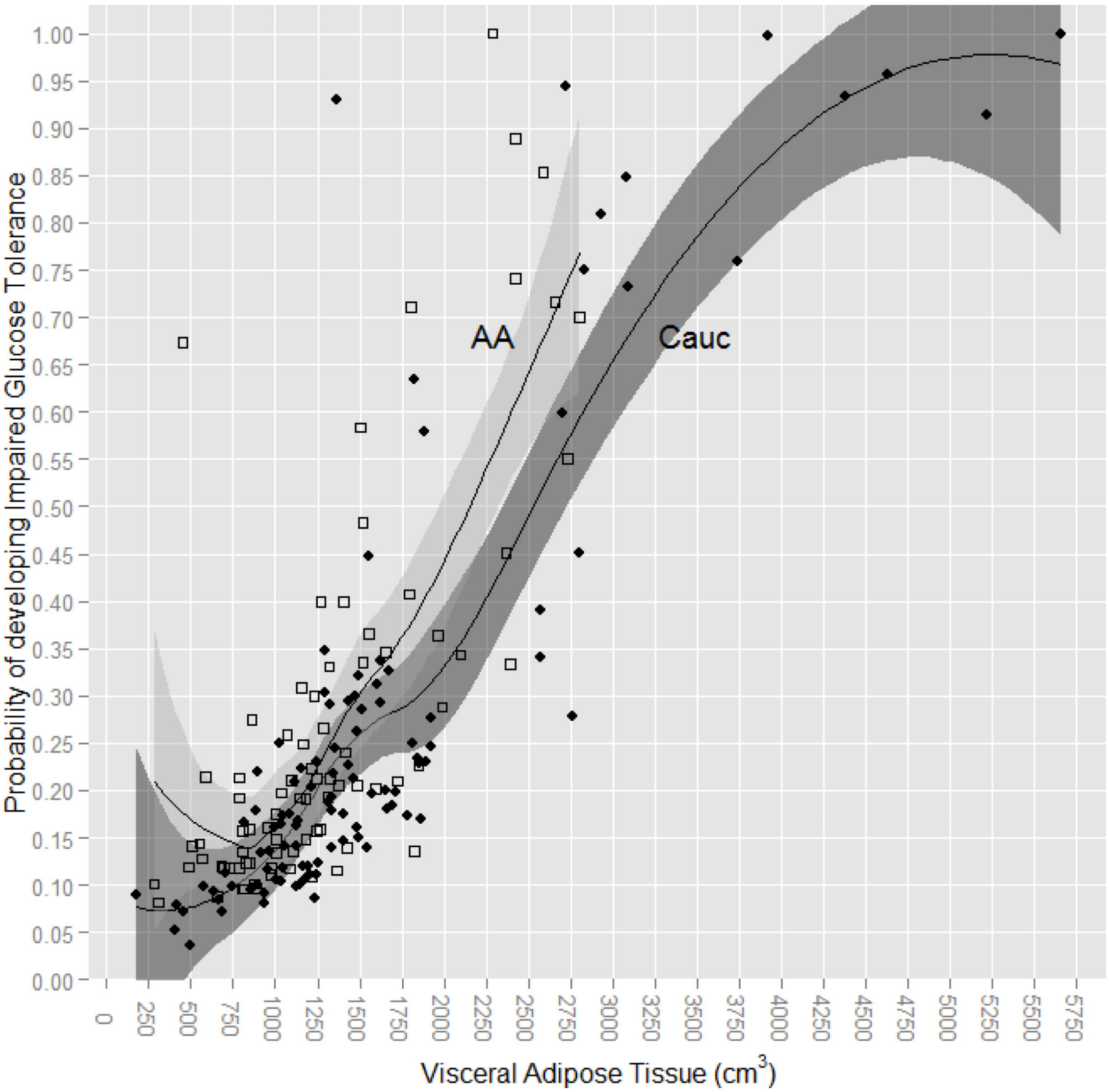

Figure 1.

Probability of Developing Impaired Glucose Tolerance by Amount of VAT in Obese European American and African American Women

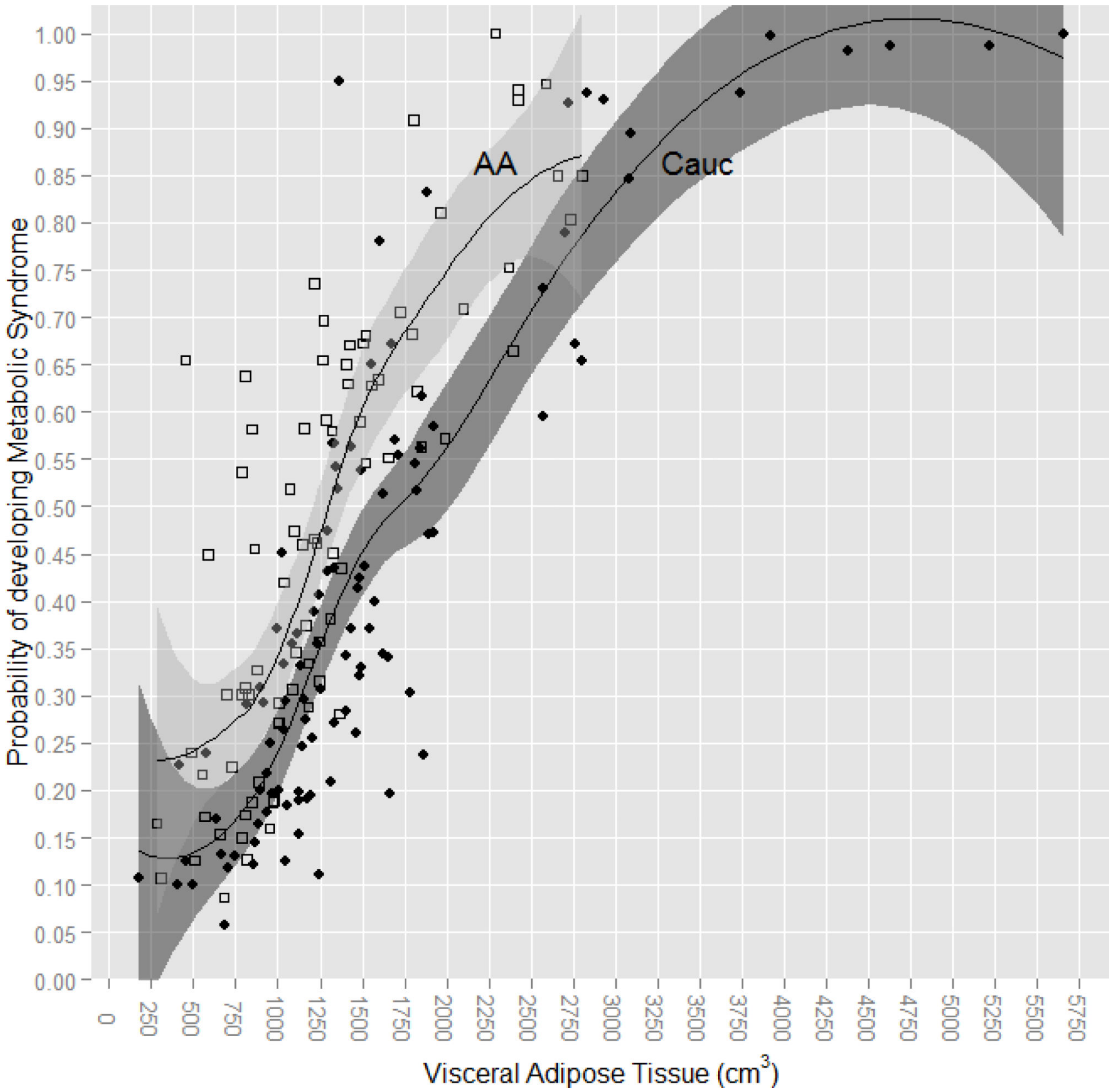

Figure 2.

Probability of Developing Metabolic Syndrome by Amount of VAT in Obese European American and African American Women

Table 1

Anthropometric, Body Composition and Clinical Cardiometabolic Risk Factors in 229 Obese Women

	Caucasian	**African American**
	n = 123	**n = 106**
Age (y)	38.6 ± 8.3	39.7 ± 9.1
Height (cm)	165.2 ± 9.2	163.6 ± 7.9
Weight (kg)	95.9 ± 13.7	100.3 ± 16.4
Body Mass Index (kg/m2)	35.1 ± 3.5	37.3 ± 4.8**
Waist Circumference (cm)	104.8 ± 10.7	107.3 ± 11.4
Hip Circumference (cm)	118.2 ± 7.5	121.3 ± 10.3*
Waist/Hip ratio	0.89 ± 0.08	0.89 ± 0.08
Waist/Height ratio	0.63 ± 0.05	0.65 ± 0.06*
Lean Mass (kg)	48.8 ± 8.4	51.5 ± 8.3**
Fat Mass (kg)	43.2 ± 7.8	44.7 ± 9.7
Body Fat (%)	46.9 ± 4.6	46.2 ± 4.5
Trunk Fat (%)	50.5 ± 4.6	49.6 ± 5.6
Android Fat (%)	53.5 ± 5.0	53.0 ± 6.3
Android/Gynoid Ratio	0.59 ± 0.17	0.57 ± 0.15
VAT area (cm2)	1646.4 ± 1007.5	1300.2 ± 661.7**
Systolic Blood Pressure (mm Hg)	122.1 ± 11.8	126.8 ± 13.1**
Diastolic Blood Pressure (mm Hg)	72.4 ± 8.1	76.6 ± 8.9**
Heart Rate (bpm)	77.9 ± 11.8	77.5 ± 11.1
Total cholesterol (mg/dL)	170.3 ± 30.4	172.5 ± 34.7
HDL-cholesterol (mg/dL)	46.7 ± 13.6	47.5 ± 13.3
LDL-cholesterol (mg/dL)	102.1 ± 26.6	109.2 ± 31.7
Triglycerides (mg/dL)	107.1 ± 61.7	82.7 ± 48.6***
hs-CRP (mg/L)	5.1 ± 5.8	6.6 ± 7.2
Leptin (ng/mL)	33.0 ± 10.4	32.3 ± 11.9
Glucose (mg/dL)	103.1 ± 42.9	97.8 ± 25.9
Insulin (mu/mL)	11.4 ± 8.6	14.3 ± 10.9**
HOMA-IR (score)	3.2 ± 3.9	3.6 ± 4.4**

Wilcoxon signed rank test

* $P < 0.05$

** $P < 0.01$

*** $P < 0.001$

Table 2

Relationships Between Anthropometric and Body Composition Independent Variables and Cardiometabolic Risk Factors[a]

	Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)	Heart Rate (bpm)	Total Cholesterol (mg/dL)	HDL Cholesterol (mg/dL)	LDL Cholesterol (mg/dL)	Triglycerides (mg/dL)	Hs-CRP (mg/L)	Glucose (mg/dL)	Insulin (mU/mL)	HOMA-IR
VAT volume (cm^2)	0.102**	0.117**	0.005	0.020	0.075***	0.041	0.246***	0.07	0.318***	0.220***	0.259***
Body Mass Index (kg/m2)	0.060**	0.073**	0.001	0.005	0.002	0.018	0.075	0.032*	0.03	0.113***	0.060***
Waist Circumference (cm)	0.071**	0.070*	0.004	0.013	0.070***	0.038	0.179***	0.068**	0.200***	0.215***	0.215***
Hip Circumference (cm)	0.056*	0.088*	0.008	0.008	0.002	0.027	0.045	0.016	0.016	0.060**	0.033*
Waist/Hip Ratio	0.045	0.087	0.006	0.005	0.138***	0.019	0.201***	0.065*	0.218***	0.136***	0.144***
Waist/Height Ratio	0.059*	0.071*	0.009	0.004	0.033**	0.021	0.141***	0.050**	0.080***	0.143***	0.102***
Bodyfat (%)	0.065	0.093	0.006	0.007	0.043*	0.026	0.063	0.052*	0.112***	0.032	0.017
Trunkfat (%)	0.052	0.07	0.007	0.007	0.022	0.028	0.056	0.065**	0.017	0.083***	0.015
Android fat (%)	0.045	0.067	0.005	0.011	0.016	0.034	0.059	0.058**	0.014	0.090***	0.02
Android/Gynoid fat Ratio	0.056	0.081	0.003	0.018	0.122***	0.039	0.212***	0.091	0.216***	0.151***	0.160***

VAT = visceral adipose tissue, HDL-Cholesterol = high-density lipoprotein cholesterol, LDL-Cholesterol = low-density lipoprotein cholesterol,

Hs-CRP = high sensitivity c-reactive protein, HOMA-IR = homeostatic model assessment-insulin resistance

[a] R^2 values from Binary Logistic Regression Modelling Accounting for the Interaction between Independent Variable and Race

$^{*}P < 0.05$

$^{**}P < 0.01$

$^{***}P < 0.001$

Table 3

Comparison of Areas Under the Curve For Relationships with Glucose Tolerance and Metabolic Syndrome

	Impaired Glucose Tolerance			**Metabolic Syndrome**		
	AUC of the IV[**]	**AUC × Race**	**AUC × Race x IV**	**AUC of the IV**	**AUC × Race**	**AUC × Race × IV**
VAT volume (cm^2)[*]	0.766	0.767	0.760	0.749	0.755	0.750
Body Mass Index (kg/m^2)	0.604	0.767	0.760	0.640	0.629	0.622
Waist Circumference (cm)	0.670	0.656	0.668	0.674	0.682	0.680
Hip Circumference (cm)	0.518	0.532	0.541	0.538	0.556	0.543
Waist/Hip Ratio	0.699	0.687	0.689	0.676	0.683	0.683
Waist/Height Ratio	0.617	0.604	0.613	0.655	0.648	0.645
Bodyfat (%)	0.566	0.556	0.540	0.505	0.529	0.598
Trunkfat (%)	0.533	0.548	0.574	0.562	0.562	0.547
Android fat (%)	0.530	0.550	0.542	0.555	0.555	0.544
Android/Gynoid fat Ratio	0.703	0.695	0.695	0.712	0.720	0.708

[*] VAT = visceral adipose tissue

[**] IV = independent variable; AUC from bias corrected bootstrapping

Table 4

Multivariate Regression for Impaired Glucose Tolerance in 229 Obese Women

Impaired Glucose Tolerance		**OR**	**SE**	**z**	***p* value**	**Lower**	**95% CI Upper**
Model 1	Fasting Insulin	1.62	0.24	2.02	0.04	1.03	2.63
	VAT	3.04	0.26	4.34	< 0.001	1.91	5.23
	SBP	1.06	0.20	0.31	0.76	0.72	1.57
	HsCRP	1.11	0.17	0.59	0.55	0.77	1.56
	Race	1.34	0.40	0.74	0.46	0.61	2.99
Model 2	Model 1 * Race	Deviance		Df	*p* value		
		0.458		1	0.49		

Table 5

Multivariate Regression for Metabolic Syndrome in 229 Obese Women

Metabolic Syndrome		**OR**	**SE**	**z**	***p* value**	**Lower**	**95% CI Upper**
Model 1	VAT	3.28	0.29	3.96	< 0.001	1.89	6.14
	HOMA-IR	2.92	0.41	2.64	0.008	1.39	6.93
	LDL	1.54	0.19	2.22	0.03	1.06	2.29
	Race	2.02	0.37	1.89	0.05	0.98	4.22
Model 2	Model 1 * Race	Deviance	Df		*p* value		
		1.044	1		0.31		