Impact of Abdominal Visceral and Subcutaneous Adipose Tissue on Cardiometabolic Risk Factors: The Jackson Heart Study

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Objective: Obesity is a major driver of cardiometabolic risk. Abdominal visceral adipose tissue (VAT) and sc adipose tissue (SAT) may confer differential metabolic risk profiles. We investigated the relations of VAT and SAT with cardiometabolic risk factors in the Jackson Heart Study cohort.

Methods: Participants from the Jackson Heart Study (n = 2477; 64% women; mean age, 58 yr) underwent multidetector computed tomography, and the volumetric amounts of VAT and SAT were assessed between 2007 and 2009. Cardiometabolic risk factors were examined by sex in relation to VAT and SAT.

Results: Men had a higher mean volume of VAT (873 vs. 793 cm³) and a lower mean volume of SAT (1730 vs. 2659 cm³) than women (P < 0.0001). Per 1-SD increment in either VAT or SAT, we observed elevated levels of fasting plasma glucose and triglyceride, lower levels of high-density lipoprotein-cholesterol, and increased odds ratios for hypertension, diabetes, and metabolic syndrome. The effect size of VAT in women was larger than that of SAT [fasting plasma glucose, 5.51 ± 1.0 vs. 3.36 ± 0.9; triglyceride, 0.17 ± 0.01 vs. 0.05 ± 0.01; high-density lipoprotein-cholesterol, −5.36 ± 0.4 vs. −2.85 ± 0.4; and odds ratio for hypertension, 1.62 (1.4–1.9) vs. 1.40 (1.2–1.6); diabetes, 1.82 (1.6–2.1) vs. 1.58 (1.4–1.8); and metabolic syndrome, 3.34 (2.8–4.0) vs. 2.06 (1.8–2.4), respectively; P < 0.0001 for difference between VAT and SAT]. Similar patterns were also observed in men. Furthermore, VAT remained associated with most risk factors even after accounting for body mass index (P ranging from 0.006–0.0001). The relationship of VAT to most risk factors was significantly different between women and men.

Conclusions: Abdominal VAT and SAT are both associated with adverse cardiometabolic risk factors, but VAT remains more strongly associated with these risk factors. The results from this study suggest that relations with cardiometabolic risk factors are consistent with a pathogenic role of abdominal adiposity in participants of African ancestry. (J Clin Endocrinol Metab 95: 5419–5426, 2010)

Obesity, defined by waist circumference (WC) or body mass index (BMI), is a risk factor for insulin resistance, dyslipidemia, diabetes, and coronary heart disease in both men and women across different ethnic groups (1–12). BMI and WC are both useful indicators of obesity, but they cannot distinguish different fat compartments. Recent studies indicate that abdominal visceral adipose tissue (VAT) is a metabolically active fat depot that may differentially contribute to metabolic consequences of obesity (2–4, 6, 9, 10). Increasing abdominal sc adipose tissue (SAT) may...
also be a health risk because it has been associated with cardiometabolic risk factors and insulin resistance (3, 4).

African-Americans are disproportionately affected by obesity, but the concomitant role of visceral compared with subcutaneous adiposity is uncertain (4, 6, 13, 14). Studies that have compared the amount of VAT between African-Americans and European-Americans have consistently shown that, given similar degrees of obesity defined by BMI, African-Americans have a lower quantity of VAT despite higher rates of insulin resistance, hypertension, and diabetes (6, 8, 9, 12). This paradox suggests that either the associations of abdominal VAT and SAT vary across different ethnic groups or the increased risk for hypertension or type II diabetes in African-Americans is due to factors above and beyond abdominal adiposity. Therefore, it remains uncertain whether VAT is an important correlate of cardiometabolic risk after accounting for BMI in African-American populations (2, 4, 6, 10).

Thus, the purpose of the present analysis was to examine the association of abdominal VAT with multiple cardiometabolic risk factors. We sought to compare the strength of the association with VAT compared with SAT, as well as to test whether VAT is associated with cardiometabolic risk factors independent of BMI in a large sample of African-Americans from the Jackson Heart Study (JHS).

**Subjects and Methods**

**Study sample**

The original JHS cohort comprises 5301 African-American participants between the ages of 21 and 94 yr enrolled between September 2000 and March 2004 from the Jackson, Mississippi, metropolitan area (15, 16). The cohort was composed of four components: 1) approximately 31% of the cohort members were participants from the Atherosclerosis Risk in Communities (ARIC) study recruited to the JHS; 2) 30% were representative community volunteers who met census-derived age, sex, and socioeconomic status eligibility criteria from the Jackson, Mississippi, metropolitan area; 3) 17% were randomly ascertained from the Jackson, Mississippi, area through methods described previously (15); and 4) 22% were in the JHS family study. The sampling frame for the family study consisted of participants in any one of the ARIC, random, or volunteer samples whose family size met eligibility requirements as detailed previously (16). The cohort consisted of 5035 adults aged 35–84 yr and an additional 266 participants (251 participants aged 21–34 and 15 participants aged >85) who were added as a part of the JHS Family Study. The present study included participants who underwent multidetector computed tomography (CT) scanning from 2007 to 2009 as part of the second JHS Examination (JHS Exam 2).

Overall, 4203 participants attended the JHS Exam 2. Of these, 2478 underwent multidetector CT assessment for abdominal VAT and SAT by the end of June 2009. Of these 2478 participants imaged, 2477 had a complete covariate profile, with one missing SAT measure, and 2278 were free of cardiovascular disease, resulting in a final sample size of 2477. The study protocol was approved by the institutional review boards of the participating institutions: the University of Mississippi Medical Center, Jackson State University, and Tougaloo College. All of the participants provided informed consent.

**Multidetector CT scan protocol for measuring adiposity**

The research CT protocol included the heart and lower abdomen using a 16-channel multidetector CT system equipped with cardiac gating (Lightspeed 16 Pro; GE Healthcare, Milwaukee, WI). Quality control and image analysis were performed at a core reading center (Wake Forest University School of Medicine, Winston-Salem, NC). The protocol included scout images, one electrocardiogram-gated series of the entire heart, and a series through the lower abdomen.

The acquired abdominal imaging slices covering the lower abdomen from L3 to S1 were used for assessing VAT and SAT. Briefly, 24 contiguous 2-mm-thick slices centered on the lumbar disk space at L4–L5 were used for this analysis; 12 images before the center of the L4–L5 disk space and 12 images after the disk space were used for quantification of VAT and SAT. The abdominal muscular wall was first manually traced, and the fat volumes in different compartments were measured by semi-automated segmentation technique. Volume analysis software (Advantage Windows; GE Healthcare, Waukesha, WI) was used to segment and characterize each individual voxel as a tissue attenuation of fat using a threshold range of −190 to −30 Hounsfield units. The VAT and SAT volumes were the sum of VAT and SAT voxels over 24 slices. In this study of a randomly selected sample of 60 participants, the interclass correlation coefficient for interreader comparisons was 0.95 for both VAT and SAT.

**Risk factors and covariate assessment**

Risk factors and covariates were measured at JHS Exam 2 (2007–2009). BMI was defined as weight (in kilograms) divided by the square of height (in meters). Two measures of the waist (at the level of the umbilicus, in the upright position) were averaged to determine baseline WC for each participant. Fasting blood samples were collected according to standardized procedures, and the assessments of plasma glucose and lipids were processed at the Central Laboratory (University of Minnesota) as previously described (15, 16). Sitting blood pressure (BP) was measured twice at 5-min intervals, and the average of two measurements was used for analysis.

Participants were considered to have hypertension if they were taking antihypertensive medications, if they self-reported a diagnosis of hypertension, and/or if their systolic pressure was at least 140 mm Hg or diastolic pressure was at least 90 mm Hg. Diabetes was defined as a fasting plasma glucose level of at least 7.0 mmol/liter or if the subject was being treated with insulin or a hypoglycemic agent. Obesity was defined by a BMI of at least 30 kg/m², and modified National Cholesterol Education Program Adult Treatment Panel III criteria were used to define the metabolic syndrome (17).

**Statistical analysis**

VAT and SAT were normally distributed, and triglycerides were normalized by logarithmic transformation and the central tendency and spread represented by the median and interquartile ranges. Age-adjusted Pearson correlations of VAT and SAT were performed with each of metabolic risk factors, including systolic and diastolic BP, fasting plasma glucose, triglycerides, and high-density lipoprotein-cholesterol (HDL-C). To estimate and to
TABLE 1. Clinical characteristics of study participants who underwent CT assessment of VAT and SAT

<table>
<thead>
<tr>
<th></th>
<th>Women (n = 1596)</th>
<th>Men (n = 881)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>59 ± 11</td>
<td>58 ± 11</td>
</tr>
<tr>
<td>Abdominal VAT (cm³)</td>
<td>793.7 ± 364.8</td>
<td>873.0 ± 412.2</td>
</tr>
<tr>
<td>Abdominal SAT (cm³)</td>
<td>2659.3 ± 966.8</td>
<td>1729.5 ± 815.3</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>32.7 ± 6.9</td>
<td>29.9 ± 5.2</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>103.1 ± 14.2</td>
<td>102.4 ± 13.5</td>
</tr>
<tr>
<td>Triglyceride (mmol/liter)</td>
<td>0.97 (0.70, 1.33)</td>
<td>1.05 (0.73, 1.57)</td>
</tr>
<tr>
<td>HDL-C (mmol/liter)</td>
<td>1.50 ± 0.4</td>
<td>1.25 ± 0.3</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>5.17 ± 1.0</td>
<td>4.94 ± 1.0</td>
</tr>
</tbody>
</table>

All values are number (percentage) or mean ± SD.

* Median (25th, 75th percentile).

Results

Overall, 1596 women and 881 men were available for analysis. The mean age of the study sample was 59 yr for women and 58 yr for men. Men had higher mean volume of VAT compared with women (873 vs. 793 cm³; *P* = 0.0001), whereas women had higher SAT volumes compared with men (2659 vs. 1730 cm³; *P* = 0.0001 for difference; Table 1). Approximately 55% of participants were obese, 72% had hypertension, 55% had metabolic syndrome, and 23% had diabetes mellitus.

Correlations with VAT and SAT

Age-adjusted correlates of VAT or SAT with metabolic risk factors are displayed in Table 2. Among adiposity measures, BMI was highly correlated with VAT and SAT. In both men and women, VAT and SAT were significantly correlated with all of the tested cardiometabolic risk factors including systolic BP, triglyceride, and fasting plasma glucose, and inversely with HDL-C and physical activity score.

A multivariable-adjusted regression model with VAT, SAT, and metabolic risk factors

The results of the multivariable-adjusted linear regression analyses for the sex-specific association of VAT or SAT with both continuous and dichotomous metabolic risk factors are summarized in Tables 3 and 4. In men, per 1-SD increment in VAT, systolic BP was 1.30 mm Hg higher (*P* = 0.02) and diastolic BP was 1.36 mm Hg higher (*P* = 0.07). In contrast, we observed no association between VAT and systolic or diastolic BP, nor did we observe the association between SAT and systolic or diastolic BP in women.

The association of both VAT and SAT with the continuous measures of metabolic risk factors was highly significant in men and women. For example, the effect of a 1-SD increase in VAT for fasting plasma glucose (5.51 ± 1.0 in women, and 3.58 ± 1.0 in men) was stronger than that in SAT (3.36 ± 0.9 in women, and 2.39 ± 1.3 in men) (*P* < 0.01 in men, and 0.0001 in women for difference

TABLE 2. Age-adjusted Pearson correlation coefficients between metabolic risk factors and VAT and SAT

<table>
<thead>
<tr>
<th></th>
<th>Women</th>
<th>Men</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>VAT</td>
<td>SAT</td>
</tr>
<tr>
<td>BMI</td>
<td>0.61</td>
<td>0.83</td>
</tr>
<tr>
<td>WC</td>
<td>0.67</td>
<td>0.76</td>
</tr>
<tr>
<td>SAT</td>
<td>0.45</td>
<td>0.45</td>
</tr>
<tr>
<td>Log triglyceride</td>
<td>0.32</td>
<td>0.10</td>
</tr>
<tr>
<td>HDL-C</td>
<td>−0.32</td>
<td>−0.17</td>
</tr>
<tr>
<td>Glucose</td>
<td>0.21</td>
<td>0.14</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>0.02</td>
<td>0.00</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>0.01</td>
<td>0.00</td>
</tr>
</tbody>
</table>

All values are number (percentage) or mean ± SD.

* Median (25th, 75th percentile).
The significant association with VAT persisted after additionally being adjusted for BMI (P < 0.0001 for difference in women, and P < 0.006 in men). Strong and significant association for triglycerides and HDL-C followed similar patterns (Table 3).

For dichotomous variables, significant associations with VAT and SAT were observed for hypertension, diabetes, and metabolic syndrome among women and men (Table 4). The odds ratios in women were significantly larger in VAT than those in SAT for hypertension [1.62 (1.4–1.9) vs. 1.58 (1.4–1.8)], diabetes [1.82 (1.6–2.1) vs. 1.58 (1.4–1.8)], and metabolic syndrome [3.34 (2.8–4.0) vs. 2.06 (1.8–2.4)] (P < 0.0001 for difference between VAT and SAT). Similar patterns were also observed in men, except for metabolic syndrome [3.46 (2.8–4.3) vs. 3.57 (2.8–4.6); P < 0.0001]. The significant associations between VAT and the above metabolic risk factors persisted after additionally being adjusted for BMI. The associations of VAT and SAT with all risk factors examined were significantly different between women and men (P values range from 0.03 to 0.0001).

Because VAT and SAT are highly correlated with each other, we performed analyses to yield R² and c statistics in each of the regression models to determine the total variance explained by VAT and SAT. The results are presented in Table 5. In women, for example, the total variance for

### Table 3. Multivariablea adjusted regression coefficients of abdominal adiposity (per 1-SD increase) with metabolic risk factors

<table>
<thead>
<tr>
<th></th>
<th>Women</th>
<th></th>
<th></th>
<th></th>
<th>Men</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MV</td>
<td>p</td>
<td>p</td>
<td>MV + BMI</td>
<td>P</td>
<td>MV</td>
<td>p</td>
<td>p</td>
</tr>
<tr>
<td>SBP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VAT</td>
<td>0.70</td>
<td>0.15</td>
<td>0.47</td>
<td>0.42</td>
<td>1.30</td>
<td>0.12</td>
<td>0.0005</td>
<td></td>
</tr>
<tr>
<td>SAT</td>
<td>0.44</td>
<td>0.35</td>
<td>0.63</td>
<td>0.06</td>
<td>0.22</td>
<td>0.07</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>0.42</td>
<td>0.13</td>
<td>0.44</td>
<td>0.32</td>
<td>0.75</td>
<td>0.07</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>FPG</td>
<td>5.51</td>
<td>0.0001</td>
<td>5.88</td>
<td>0.0001</td>
<td>3.52</td>
<td>0.10</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>Log TG</td>
<td>0.17</td>
<td>0.0001</td>
<td>0.19</td>
<td>0.0001</td>
<td>0.20</td>
<td>0.0001</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>HDL-C</td>
<td>5.36</td>
<td>0.0001</td>
<td>4.21</td>
<td>0.0001</td>
<td>0.53</td>
<td>0.0001</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>SAT</td>
<td>2.85</td>
<td>0.0001</td>
<td>3.63</td>
<td>0.0001</td>
<td>0.53</td>
<td>0.0001</td>
<td>0.0001</td>
<td></td>
</tr>
</tbody>
</table>

a Adjusted for age, smoking, and alcohol use.
b P for VAT or SAT in the model.
c P for difference in effect size between VAT and SAT.

### Table 4. Multivariablea adjusted odds ratio for hypertension, diabetes, and metabolic syndrome with per 1-SD increase in abdominal VAT or SAT

<table>
<thead>
<tr>
<th></th>
<th>Women</th>
<th></th>
<th></th>
<th></th>
<th>Men</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MV</td>
<td>p</td>
<td>p</td>
<td>MV + BMI</td>
<td>P</td>
<td>MV</td>
<td>p</td>
<td>p</td>
</tr>
<tr>
<td>HTN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VAT</td>
<td>1.62</td>
<td>0.0001</td>
<td>1.29</td>
<td>0.006</td>
<td>1.55</td>
<td>0.0001</td>
<td>1.34</td>
<td>0.003</td>
</tr>
<tr>
<td>SAT</td>
<td>1.40</td>
<td>0.0001</td>
<td>1.40</td>
<td>0.0001</td>
<td>1.45</td>
<td>0.0002</td>
<td>1.34</td>
<td>0.005</td>
</tr>
<tr>
<td>DM</td>
<td>1.82</td>
<td>0.0001</td>
<td>1.40</td>
<td>0.0001</td>
<td>1.69</td>
<td>0.0002</td>
<td>1.41</td>
<td>0.008</td>
</tr>
<tr>
<td>SAT</td>
<td>1.58</td>
<td>0.0001</td>
<td>1.40</td>
<td>0.0001</td>
<td>1.78</td>
<td>0.0001</td>
<td>1.41</td>
<td>0.059</td>
</tr>
<tr>
<td>MetS</td>
<td>3.34</td>
<td>0.0001</td>
<td>2.41</td>
<td>0.0001</td>
<td>3.46</td>
<td>0.0001</td>
<td>2.53</td>
<td>0.0001</td>
</tr>
<tr>
<td>SAT</td>
<td>2.06</td>
<td>0.0001</td>
<td>2.57</td>
<td>0.0001</td>
<td>3.57</td>
<td>0.0001</td>
<td>2.53</td>
<td>0.0004</td>
</tr>
</tbody>
</table>

a Adjusted for age, smoking, and alcohol use.
b P for VAT or SAT in the model.
c P for difference in effect size between VAT and SAT.
HDL-C was 12%, which can be explained by VAT plus covariates, and slightly increased to 13% when BMI had been taken into account additionally. SAT only contributed to 5% of total variance for HDL-C. Similar results were also observed in other risk factors, and similar patterns were followed in men (Table 5).

**Discussion**

**Principal findings**

Volumetric CT measures of VAT and SAT were cross-sectionally associated with cardiometabolic risk factors, including fasting plasma glucose, triglycerides, HDL-C, hypertension, diabetes, and metabolic syndrome. These significant associations with VAT persisted after additionally adjusting for BMI, suggesting that VAT provides significant information above and beyond BMI. In addition, the magnitude of the effect of VAT and a total variance explained by adding VAT into each on regression models were consistently stronger than those of SAT in the association with cardiometabolic risk factors, indicating that VAT is more metabolically deleterious than SAT. A stronger association of VAT and SAT with most of the risk factors examined was observed for women than for men.
In the context of current literature

It has been well documented that VAT is more strongly associated with cardiometabolic risk compared with SAT (3, 4, 6, 10), that VAT remains an important correlate of metabolic risk factors after accounting for BMI (3, 18), and that sex differences exist in the relationship of VAT and SAT with cardiometabolic risk (3, 14, 19, 20). However, these prior studies have been largely restricted to populations of European ancestry. Thus, the findings from the present study expand the literature in several important ways. First, our results are consistent with prior studies that abdominal VAT is a stronger correlate of most cardiometabolic risk factors compared with abdominal SAT (3, 4, 6, 9, 11, 21–23). Second, although the BMI to VAT ratio is much larger in African compared with European ancestry populations, VAT remains an important correlate of cardiometabolic risk after accounting for BMI. Finally, we observed similar sex interactions that have previously been observed primarily in populations of European ancestry. Thus, despite different relationships between overall BMI and body fat distribution in African-Americans, our findings support a consistent and particular role for abdominal VAT with cardiometabolic risk factors in African-American populations.

The role of abdominal SAT has been debated because not all studies have observed a pathogenic role for SAT (18). The essential role for sc fat depots can best be observed in lipodystrophic syndromes, where the absence of sc fat leads to ectopic fat accumulation in muscle and liver with concomitant insulin resistance (24). This has led some investigators to hypothesize a potential beneficial role for SAT (10, 14). Recent animal studies demonstrate that transplantation of SAT into abdominal cavities of rats is associated with an improvement in insulin sensitivity (25). In humans, it is difficult to disentangle the associations of SAT compared with VAT given the higher correlations of these two fat depots. In the Framingham Heart Study, larger volumes of SAT were associated with more adverse risk factor profiles (3), although increasing SAT was associated with lower triglyceride levels only when examined within narrow ranges of VAT (18). Thus, the data from the present study have confirmed these observations that higher levels of abdominal SAT are also associated with more adverse levels of cardiometabolic risk and extend these findings to participants of African ancestry.

Effect of sex

Sex differences in body fatness or in regional adipose tissue distribution are well documented. Women are generally characterized by a greater body fat content and preferential accumulation of adipose tissue in the gluteofemoral region, whereas men are prone to abdominal fat deposition, particularly in the abdominal cavity, a condition that has been described as visceral obesity (3, 6, 26). Consistent with these observations, men in our study had higher mean volume of VAT than women for a given lower level of BMI, whereas women had more SAT. Importantly, despite known differences in body fat distribution between individuals of African compared with European ancestry, we observed similar sex interactions with VAT and cardiometabolic risk.

Potential mechanisms

VAT and SAT differ in the magnitude of the associations despite higher volumes of SAT than VAT in our study. The possible explanation for differences between VAT and SAT may be related to differences in their anatomic location, lipoprotein lipase activities, and cytokine secretion profiles. For example, lipoprotein lipase activity, expressed as a function of cell number, was significantly higher in omental than in sc adipose tissue (26, 27). SAT may preferentially release more leptin and IL-6, whereas VAT may mainly release TNF-α (28). Although relationships of VAT or SAT lipoprotein lipase activities and cytokine secretion profiles with cardiometabolic pathogenesis are not clear, it may be that lipoprotein lipase activities or endocrine factors contribute to the differential effects of VAT and SAT.

Implications

The results from this study have important implications in our understanding of the relationship between body fat distribution and cardiometabolic risk in African-Americans. First, VAT remains an important correlate of cardiometabolic risk in addition to high rates of obesity in African-Americans. More importantly, given the lower amount of VAT in African-Americans (2, 4, 6, 9, 10), the associations of abdominal obesity with cardiometabolic risk factors are consistent with the findings from studies among participants of European ancestry (3, 14, 18–20, 24).

Strengths and limitations

The precise measurement of abdominal VAT and SAT volumes using advanced imaging techniques is a marked strength of this study. A relatively large sample size from a large population-based cohort of African-Americans provides the adequate power to detect potential small but significant relations with abdominal VAT and SAT and the originality to this research field. However, the results must be reviewed with caution because the findings are derived from the cross-sectional analysis. Generalizability cannot be applied to other ethnic groups because of a primary African-American sample.
Conclusion

Abdominal VAT and SAT are both associated with adverse cardiometabolic risk factors. Despite lower levels of VAT in African-Americans, relations with cardiometabolic risk factors are consistent with a pathogenic role of visceral adiposity.

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