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Intrabdominal fat is related to metabolic risk factors in Hispanic Americans, African Americans, and in girls

Krista Casazza, PhD, RD, **Akilah Dulin-Keita, PhD**, **Barbara A. Gower, PhD**, and **Jose R. Fernandez, PhD**
Department of Nutrition Sciences and Clinical Nutrition Research Center, University of Alabama at Birmingham, Birmingham, AL

Abstract

Aim—This study aimed to test the association of individual adipose depots on cardiometabolic outcomes; whether the association varied by depot; and if the associations differed by race/ethnicity or sex in early pubertal children.

Methods—320 children (53% male) aged 7–12y self-identified as African- (AA; n=114), European- (EA; n=120), or Hispanic American (HA; n=86) participated. Insulin dynamics were assessed by intravenous glucose tolerance test; body composition with DXA; fat distribution with CT.

Results—AA had the least fat in each depot and HA had the most. Fat accumulation negatively impacted cardiometabolic outcomes independent of race/ethnicity or sex. AA and females were reproductively more mature. In AA and HA each measure of adiposity influenced the insulin sensitivity index (S_I), whereas intra-abdominal adipose tissue (IAAT) did not contribute to S_I in EA. IAAT was positively associated with blood pressure in AA, only. In females, adiposity adversely influenced cardiometabolic outcomes, such that total fat mass, IAAT, and/or SAAT was inversely associated with S_I, and positively associated with blood pressure and fasting insulin.

Conclusions—IAAT is uniquely related to metabolic risk factors in Hispanic Americans, African Americans, and girls, suggesting that either the threshold for adverse effects of IAAT is lower, or that IAAT metabolism differs in these groups.

Keywords

Intra-abdominal adipose tissue; puberty; race/ethnicity; sex differences; pediatric obesity

The alarming rise in the prevalence of obesity among the pediatric population has become one of the most serious and urgent public health problems. Many of the metabolic complications associated with obesity are already present during childhood and are closely linked to risk for the development of type 2 diabetes and cardiovascular disease. Excess adiposity is strongly associated with reduced insulin sensitivity, impaired glucose tolerance, adverse lipid profiles and elevated blood pressure in both adult (1–3) and pediatric (4–8) populations. However, the location of the excess body fat may differentially influence the severity of metabolic complications.

Corresponding Author: Krista Casazza, PhD, RD, Department of Nutrition Sciences, Webb 415, 1530 3rd Ave S, University of Alabama at Birmingham, Birmingham, AL 35294-3360. kristac@uab.edu, Telephone: (205) 975-6226, Fax: (205) 934-7050.

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Intra-abdominal adipose tissue (IAAT), in particular, has been shown to be strongly correlated with insulin dynamics and lipid metabolism in adults (1;9;10). Studies in adult humans as well as in animal models indicate that compared to the subcutaneous fat depot, IAAT is a greater contributor to adverse cardiometabolic outcomes (1;9;10). Information on the adverse effects of IAAT in children, especially early pubertal children, is limited. Adipose tissue is preferentially deposited subcutaneously in early pubertal children and IAAT is relatively low among this group (6;11). However, it has been hypothesized that beginning in childhood, as excess fat begins to accumulate, lipid overflow leads to increasing fat deposition into the IAAT compartment that may initiate cardiometabolic complications (5;12). The regional distribution of adipose tissue appears to influence cardiometabolic outcomes, specifically glucose and lipid metabolism and blood pressure (1). It is not clear if specific fat depots contribute to cardiometabolic outcomes differently in children, particularly children of diverse racial/ethnic backgrounds.

It has been clearly demonstrated that Hispanic and African Americans of every age group are disproportionately affected by cardiovascular disease and type 2 diabetes relative to their European American counterparts (13–15). Part of this ethnic discrepancy in disease risk may derive from ethnic differences in fat distribution. Racial/ethnic differences in body fat distribution as well as in metabolic risk factors have been identified in both adults and children. Hispanic American adults (5;16) and children (5) reportedly have greater IAAT than European Americans even when matched for total adiposity. More adverse cardiometabolic outcomes observed in Hispanic Americans relative to European Americans appears to be reflective of greater fat accumulation, particularly in the intra-abdominal compartment.

Unlike Hispanics, African American adults and children have been consistently shown to have less IAAT than European Americans for a given total body fat (1;3;11;17;18). Further, greater risk for type 2 diabetes among African Americans vs. European Americans is independent of BMI (14). Taken together, these observations suggest that either there are factors unrelated to adiposity underlying metabolic risk, or that the threshold for an effect of a given adipose tissue depot on metabolic risk may be lower among African Americans.

Few studies (4;6;11;19) have investigated the associations among fat depots and metabolic outcomes in children. To our knowledge no study has investigated these variables using robust measures of metabolism and body composition in a multi-ethnic cohort of early pubertal children. The aim of this study was to examine the associations of individual adipose depots with metabolic outcomes, and whether these associations vary by race/ethnicity or sex.

Methods

Participants

Participants were 320 children aged 7–12 years recruited as a part of a cross-sectional study which aims to identify racial and ethnic differences in insulin related outcomes among healthy children. Children were categorized according to parental self-report as African American (n=114), European American (n=120), or Hispanic American (n=86). The children were pubertal stage ≤3 as assessed by a pediatrician according to the criteria of Marshall and Tanner (20), had no major illnesses or medical diagnoses (e.g. asthma, diabetes) and were not taking any medications known to affect body composition or metabolism. The children and parents provided informed assent and consent, respectively, to the protocol, which was approved by the Institutional Review Board for human subjects at the University of Alabama at Birmingham (UAB). All measurements were performed at the

General Clinical Research Center (GCRC) and the Department of Nutrition Sciences at UAB between 2005 and 2008.

Protocol

Participants completed two testing sessions approximately within two weeks. In the first session, pubertal status, anthropometric measurements, and body composition by dual-energy X-ray absorptiometry (DXA) were assessed. In the second session, participants were admitted to the GCRC in the late afternoon for an overnight visit. Scans via computed tomography (CT) and two blood pressure measurements (evening and morning) were obtained. All participants were given the same meal and snack foods. After 2000h, only water and/or non-caloric decaffeinated beverages were permitted until after the morning testing session. Upon completion of the overnight fast, an intravenous glucose tolerance test (IVGTT) was performed.

Anthropometric measures

For all participants, anthropometric measurements were obtained by the same registered dietitian. Participants were weighed (Scale-tronix 6702W; Scale-tronix, Carol Stream, IL) to the nearest 0.1 kg in minimal clothing without shoes. Height was recorded without shoes using a digital stadiometer (Heightronic 235; Measurement Concepts, Snoqualmie, WA) to the nearest 0.1cm. BMI percentile was calculated from measured height and weight according to Centers for Disease Control and Prevention age- and sex- specific growth curves.

Waist circumference was measured at the “narrowest part of the torso,” the area between the ribs and iliac crest as described by Lohman et al (21). Waist circumference measures were obtained using a flexible tape measure (Gulick II; Country Technology, Inc., Gays Mills, WI) and were recorded to the nearest 0.1 cm.

Assessment of body composition

Body composition (total body fat mass, bone mass and non-bone lean tissuemass) was measured by DXA using a GE Lunar Prodigy densitometer (GE LUNAR Radiation Corp., Madison, WI). Participants were scanned in light clothing, while lying flat on their backs with arms at their sides. DXA scans were performed and analyzed using pediatric software (enCORE 2002 Version 6.10.029). In our laboratory, the coefficient of variation (CV)for repeated measures of total body fat mass is 6.55%. Intra-abdominal adipose tissue (IAAT) and subcutaneous abdominal adipose tissue (SAAT) were measured by CT scanning with a HiLight/Advantage Scanner (General Electric, Milwaukee) as previously described (22). A 5mm abdominal scan was taken at the level of the umbilicus. Scans were analyzed for cross-sectional area (cm^2) of adipose tissue using the density contour program with Hounsfield units for adipose tissue set at −190 to −30.

Intravenous Glucose Tolerance Test (IVGTT)

Following the overnight fast, a topical anesthetic was applied to the antecubital space of both arms, and flexible intravenous catheters were placed in both arms for an insulin-modified IVGTT, as described elsewhere (23). Acute insulin response to glucose (AIRg), an approximation of first phase insulin secretion, was calculated as the incremental area under the curve for insulin during the first 10 minutes after glucose injection using trapezoidal methodology (24). Glucose and insulin values were entered into the MINMOD Millenium version computer program for determination of the insulin sensitivity index (S_I).

Assay of glucose and lipids

Glucose and lipids were analyzed using a SIRRUS analyzer. Glucose was assayed by the glucose oxidase method (interassay CV 2.56%). Triglycerides were assessed with the glycerylphosphate (GPO) method. HDL-cholesterol was analyzed using a two-reagent system involving stabilization of LDL, VLDL, and chylomicrons using cyclodextrin and dextrin sulfate, and subsequent enzymatic-colorometric detection of HDL-C.

Blood Pressure

Evening and morning blood pressure (Dinamap Pro 200 automated pediatric cuff, GE Medical Systems) were measured during the overnight inpatient stay. On each occasion, blood pressure was taken twice in the seated position, 5 minutes apart, after at least 10 minutes of seated rest. The evening measurements were taken at approximately 1800 h on the evening of the overnight stay. The morning measurements were taken shortly after awakening at approximately 0700 h of the overnight stay. The evening and morning measurements did not significantly differ from one another and therefore, were averaged to yield final blood pressure values.

Socioeconomic Status (SES)

Geographic differences in health status, based on socioeconomic status are well documented. In an effort to control for the effect of differences in socioeconomic status (SES) on cardiometabolic outcomes, SES was measured with the Hollingshead 4-factor index of social class (25). This measure combines the educational attainment and occupational prestige for the number of working parents in the child's family. Scores range from 8 to 66, with higher scores indicating higher theoretical social status.

Statistical Analyses

Differences in descriptive statistics by sex and race/ethnicity were examined using ANOVA with Duncan's post-hoc analysis. Multiple linear regression was used to test contributions of fat depots to various cardiometabolic outcomes. Overall multiple regression models (testing associations in the entire sample (n=320)) were adjusted for age, pubertal stage, sex, race/ethnicity (using orthogonal coding) and SES. Models including IAAT were adjusted for total fat mass. Total fat mass and SAAT were highly correlated (r=0.89) and therefore were not included together in models. For those models evaluating the dependent variable blood pressure, height was added as a covariate; for models evaluating HDL-C, triglycerides was added as a covariate; and for models evaluating AIRg, S_I was added as a covariate. Interaction terms tested moderation by sex and ethnicity of relationships between fat depots and cardiovascular outcomes. Subsequently, models were evaluated by race/ethnicity or sex where appropriate (based on significance of interaction term, $p<0.05$). Models stratified by sex included race/ethnicity as a covariate and models stratified by race/ethnicity included sex as a covariate. It was determined that a minimum of 57 participants per group was necessary for an analysis providing 80% power with a corresponding effect size = 0.25 at $p = 0.05$.

To conform to the assumptions of linear regression, all statistical models were evaluated for residual normality and logarithmic transformations were performed when appropriate. All data were analyzed using SAS 9.1 software.

Results

Participant characteristics (n=320; 53% male) are presented in Table 1. There were no differences among racial/ethnic groups in age or weight of the children. European Americans reported a higher SES than African Americans, who, in turn reported a higher

SES than Hispanic Americans. Hispanic American children had greater adiposity as measured by BMI percentile, waist circumference, total fat mass, IAAT and SAAT than European- and African Americans. African Americans had greater lean mass and were reproductively more mature than both European- and Hispanic Americans. European Americans had lower fasting insulin, a lower AIRg and a higher S_I than both African- and Hispanic Americans. African Americans had higher systolic blood pressure, lower fasting glucose, and higher HDL-C than both European and Hispanic American children. Hispanic Americans had higher triglycerides than both European- and African Americans. Females had a lower S_I than males, but otherwise did not metabolically differ (data not shown).Females were reproductively more mature and had more total fat mass and SAAT, while males had more lean mass (data not shown).

Table 2 illustrates the contribution of each of the fat depots to metabolic outcomes in the total sample. In the total sample, each fat depot was inversely associated with S_I, and positively associated with AIRg, fasting insulin, blood pressure and the lipid profile (except IAAT and AIRg).

Table 3 presents the p-values representing the significance of the interactions between the fat depot and race/ethnicity or sex. The interaction term including total fat was significant for S_I and fasting insulin; the term including IAAT was significant for S_I only; and the interaction term including SAAT was significant for S_I and blood pressure. Subsequent analysis by race/ethnicity revealed differences in the contribution of fat depots to cardiometabolic outcomes (Table 4). In European Americans, total fat mass and SAAT (but not IAAT) were inversely associated with S_I, whereas in African Americans and Hispanic Americans each measure of adiposity influenced S_I. IAAT was positively associated with blood pressure in African Americans, but did not contribute to blood pressure in European or Hispanic Americans. Though the interaction term was significant, stratified analysis did not indicate a racial/ethnic difference in the contribution of total fat mass to fasting insulin.

Significant sex by fat depot interactions were also observed. The interaction term including total fat was significant for AIRg; the term including IAAT, was significant for S_I, AIRg, and blood pressure, and the interaction term including SAAT was significant for S_I and blood pressure. Subsequent analysis by sex indicated that in females, both IAAT and SAAT were inversely associated with S_I, whereas neither of those depots influenced S_I in males. Similarly, total fat mass and IAAT contributed to a higher AIRg in females with no relationship in males. Our findings for blood pressure indicated SAAT was associated with increased blood pressure in boys, whereas IAAT influenced blood pressure in girls.

Additionally, there were no significant associations between socioeconomic status and body composition. The lack of significance is supported by others (8;16) which suggests that inconsistent relationships emerge across racial/ethnic groups such that European American girls are protected, but relatively little protection is offered for Hispanic and African American children.

DISCUSSION

Numerous studies in adults have demonstrated a relationship between fat accumulation, particularly in the intra-abdominal compartment, and adverse cardiometabolic profiles. Intra-abdominal adipose tissue, relative to subcutaneous, has been described as metabolically unique and has been implicated as a greater contributor to adverse metabolic outcomes (1;9;10).

Several studies have suggested that in young children, particularly non-obese children, the contribution of IAAT to metabolic outcomes is limited(7;18).Further, although European-

American prepubertal children had greater IAAT than African American prepubertal children (Tanner stage 1), this difference in IAAT did not account for ethnic/racial differences in cardiometabolic outcomes.

In contrast, our findings here, with a somewhat wider range of reproductive maturity (Tanner stage 1–3) demonstrate adverse associations between IAAT and risk factors that differed with both race/ethnicity and sex. We suggest two plausible hypotheses to help explain these findings. First, the threshold for IAAT may be different between populations or sex groups. The contribution of IAAT to S_I noted among African Americans (who had the least amount of IAAT) and Hispanic Americans (who had the most IAAT), while no association noted in European Americans suggest the possibility of differing thresholds by race/ethnicity. Previous research in adults of European origin has indicated that the adverse cardiometabolic outcomes accompanying IAAT accrual are manifest above a certain threshold volume (26;27). The clinical significance of this value when used in populations of ancestral backgrounds other than European is not clearly established. For example, it has been suggested that among persons of Japanese ancestry the adverse effects of IAAT may occur at as low as $60cm^2$ (28). The same may be true for AA, who are disproportionately affected by CVD and type 2 diabetes, yet have a lower accumulation of IAAT than EA(5;7). A threshold for adverse metabolic effects of IAAT has not been established for AA, nor has any established values been identified in children.

We also found that IAAT contributed to risk in females, but not males. Although females had greater overall adiposity, the volume of IAAT did not differ by sex. The girls were on average reproductively more mature and although not yet menstruating, likely to have at least some changes in the hormonal environment. Shen et al (29) recently reported sex was not a predictor of IAAT accumulation before puberty and the sexual dimorphism illustrated between males and females only exist in pubertal children. As such, our second plausible hypothesis is that that reproductive maturity may lead to an “activation” of the IAAT compartment. Cruz and colleagues identified a significant contribution of IAAT to S_I in their Hispanic American cohort, who was on average a year older than our children, and was reproductively more mature (Tanner stage 2) (5). In our sample, there were more Hispanic children categorized as obese based on total adiposity relative to European and African Americans (who did not differ in exceeding obesity cut-point). However, we did not observe an association between IAAT and risk factors in our Hispanic subjects, who were younger than those of Cruz et al. Other researchers have likewise demonstrated the detrimental effects of IAAT in European-American (4) adolescents in the later stages of puberty as well as in reproductively mature obese adolescents (4;30). Taken together, present and previous observations suggest the possibility that reproductive maturity may play a role in the contribution of IAAT to metabolic risk in children.

A possibility that may relate reproductive maturity to activation of IAAT is inflammation. IAAT is strongly associated with the production of pro-inflammatory cytokines, which may be regulated in part by the reproductive hormonal milieu. Estrogen has recently been recognized as exerting pro-inflammatory effects under certain conditions (e.g. oxidative stress, increased catecholamine release), which may accompany puberty. Thus, endocrine changes during puberty may alter the endocrine and paracrine secretions of adipose tissue. These changes combined with other unique aspects of pubertal metabolism, such as increased free fatty acid flux and insulin resistance, and may act synergistically to initiate an adverse cardiometabolic profile.

The strengths of this study were the robust measures of insulin sensitivity and body composition in a multi-ethnic population with wide range of body habitus. A limitation of this study was its cross-sectional nature preventing the establishment of cause and effect

relationships; longitudinal data will be required to determine the long-term contribution of individual adipose depots to cardiometabolic outcomes in early pubertal children. In addition, the sample included only participants from a discrete geographic area, limiting the generalizablity. Because hormones levels were not analyzed, we cannot say with certainty that peripubertal changes in reproductive hormones are responsible for the observed associations between IAAT and metabolic risk.

In conclusion, excess fat accumulation negatively impacts cardiometabolic outcomes in early pubertal children independent of race/ethnicity or sex. IAAT is uniquely related to metabolic risk factors in Hispanic Americans, African Americans, and girls, suggesting that either the threshold for adverse effects of IAAT is lower, or that IAAT metabolism differs in these groups. Further research is needed to identify ethnic-specific cut-points for IAAT, and to determine whether pubertal or endocrine status (e.g. via reproductive hormone concentration) affects these cut-points.

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Table 1

Population characteristics. (mean ± SE unless otherwise indicated)

	Total (n=320)	EA (n=120)	AA (n=114)	HA (n=86)
Age (yrs)	9.6±0.1	9.6±0.1	9.6±0.1	9.4±0.2
Male n (%)	170 (53)	62 (52)	63 (55)	45 (52)
Tanner	1.5±0.1	1.3±0.1^a	1.7±0.1^b	1.4±0.1^a
I				
Male	120	48	36	36
Female	84	40	19	25
II				
Male	38	11	19	8
Female	40	13	16	11
III				
Male	12	2	8	2
Female	26	6	16	4
SES	38.5±0.8	49.3±0.9^a	37.0±1.1^b	25.6±1.3^c
Height (cm)	139.5±0.6	140.1±9.7^{ab}	140.7±1.0^a	137.0±1.2^b
Weight (kg)	36.5±0.5	35.5±0.8	37.0±0.9	37.7±1.1
BMI Percentile	66.5±1.4	60.2±2.4 ^{a}	63.8±2.5 ^{a}	79.3±1.9 ^{b}
WC (cm)	64.3±0.5	63.0±0.7^a	62.7±0.8^a	68.3±1.2^b
Total Fat Mass (kg)	8.7±0.3	8.2±0.5^a	8.0±0.6^a	10.3±0.6^b
Total Lean Mass (kg)	25.6±0.3	25.4±0.5^a	26.9±0.5^b	24.1±0.6^a
IAAT (cm^2)	33.0±1.5	34.1±2.7^a	27.0±2.0^b	41.2±3.3^a
SAAT (cm^2)	93.7±5.2	86.4±8.9^a	81.9±8.6^a	121.8±8.4^b
Fasting Insulin	12.6±0.4	10.9±0.4^a	13.0±0.6^b	14.3±0.9^b
Fasting Glucose (mg/dL)	97.4±0.4	97.6±0.6^a	94.8±0.6^b	100.1±0.7^c
S_I	5.8±0.2	7.1±0.4^a	4.5±0.3^b	5.4±0.4^b
AIRg	918.9±46.2	621.6±41.5^a	1238.4±89.6^b	989.0±105.7^c
TG (mg/dL)	66.5±2.1	66.2±2.9^a	54.6±2.8^a	82.4±5.2^b
HDL-C (mg/dL)	50.3±0.7	48.3±1.1^a	55.3±1.3^b	46.5±1.4^a
Systolic BP (mm Hg)	103.4±0.6	102.0±1.0^a	106.6±1.0^b	101.4±1.0^a

a–c superscripts represent differences between racial/ethnic groups.

SES = socioeconomic status; BMI= body mass index; WC= waist circumference; IAAT = intra-abdominal adipose tissue; SAAT = subcutaneous abdominal adipose tissue; BP = Blood pressure; S_I= insulin sensitivity Index; AIRg= acute insulin response to glucose; TG= triglyceride concentration; HDL-C = high density lipoprotein cholesterol.

Table 2

Multiple regression analysis illustrating the contribution of IAAT, subcutaneous adipose tissue, and total adiposity on cardiometabolic outcomes.

	S_I		7AIRg		Fasting Insulin		BP		TG		HDL-C	
	β	**p-value**	**β**	**p-value**	**β**	**p-value**	**β**	**p-value**	**β**	**p-value**	**β**	**p-value**
Total Sample												
Total Fat Mass	**−0.40**	**<0.001**	**0.16**	**0.006**	**0.53**	**<0.001**	**0.24**	**0.001**	**0.27**	**0.009**	**−0.31**	**0.001**
IAAT	**−0.24**	**0.006**	0.11	0.184	**0.32**	**<0.001**	**0.25**	**0.015**	**0.37**	**<0.001**	**−0.29**	**0.002**
SAAT	**−0.41**	**<0.001**	**0.16**	**0.030**	**0.49**	**<0.001**	**0.21**	**0.011**	**0.34**	**<0.001**	**−0.20**	**0.005**

Note: Overall models adjusted for age, pubertal stage, sex, race/ethnicity and socioeconomic status. Models including IAAT were adjusted for total fat. AIRg models included S_I as a covariate, blood pressure models included height as a covariate, and HDL-C models included triglyceride concentration as a covariate.

IAAT = intra-abdominal adipose tissue; SAAT = subcutaneous abdominal adipose tissue; BP = Blood pressure; S_I= insulin sensitivity Index; AIRg= acute insulin response to glucose; insulin= fasting insulin; TG= triglyceride concentration; HDL-C = high density lipoprotein cholesterol.

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Table 3

P-values testing the interaction between fat depot and race/ethnicity or sex . Models which included a significant interaction term were stratified and analyzed accordingly.

	Interaction Terms					
	Total Fat* Ethnic	**Total Fat*Sex**	**IAAT* Ethnic**	**IAAT*Sex**	**SAAT***	**SAAT* Sex**
S_I	**<0.001**	0.233	**0.005**	**0.002**	**0.036**	**0.009**
AIRg	0.613	**0.022**	0.661	**0.015**	0.704	0.342
Insulin	**0.043**	0.323	0.592	0.127	0.117	0.394
SysBP	0.270	0.468	0.166	**0.025**	**0.010**	**0.033**
TG	0.44	0.19	0.4764	0.094	0.985	0.260
HDL-C	0.38	0.32	0.530	0.958	0.392	0.635

IAAT = intra-abdominal adipose tissue; SAAT = subcutaneous abdominal adipose tissue; BP = Blood pressure; S_I= insulin sensitivity Index; AIRg= acute insulin response to glucose; insulin= fasting insulin; TG= triglyceride concentration; HDL-C = high density lipoprotein cholesterol.

Table 4

Multiple regression analysis illustrating the contribution of IAAT, subcutaneous adipose tissue, and total adiposity on cardiometabolic outcomes stratified by race/ethnicity or sex.

	S_I		AIRg		Fasting Insulin		BP	
	β	**p-value**	**β**	**p-value**	**β**	**p-value**	**β**	**p-value**
European Americans								
Total Fat Mass	**−0.33**	**0.001**			**0.51**	**<0.001**		
IAAT	−0.18	0.096						
SAAT	**−0.47**	**<0.001**					0.24	0.107
African American								
Total Fat Mass	**−0.26**	**0.013**			**0.52**	**<0.001**		
IAAT	**−0.32**	**0.020**						
SAAT	**−0.32**	**0.005**					**0.31**	**0.043**
Hispanic Americans								
Total Fat Mass	**−0.61**	**<0.001**			**0.57**	**<0.001**		
IAAT	**−0.39**	**0.044**						
SAAT	**−0.53**	**<0.001**					0.05	0.736
Male								
Total Fat Mass			0.11	0.183				
IAAT	0.10	0.501	0.07	0.661			0.04	0.978
SAAT	**−0.37**	**<0.001**					**0.36**	**0.002**
Female								
Total Fat Mass			**0.23**	**<0.001**				
IAAT	**−0.35**	**0.001**	**0.19**	**0.037**			**0.41**	**0.002**
SAAT	**−0.45**	**<0.001**					0.02	0.839

Models including IAAT were adjusted for total fat. AIRg models included S_I as a covariate, blood pressure models included height as a covariate, and HDL-C models included triglyceride concentration as a covariate. IAAT = intra-abdominal adipose tissue; SAAT = subcutaneous abdominal adipose tissue; BP = Blood pressure; S_I= insulin sensitivity Index; AIRg= acute insulin response to glucose; insulin= fasting insulin; TG= triglyceride concentration; HDL-C = high density lipoprotein cholesterol.