

Fat distribution and insulin response in prepubertal African American and white children¹⁻³

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ABSTRACT Ethnic differences in obesity-related disease prevalence may relate to differences in fat distribution or metabolism. We conducted a study in 73 African American and white children to examine the relation between fat distribution and insulin and to determine whether ethnic differences in fat distribution or in adiposity-insulin relations contribute to differences in insulin concentrations. Fasting and postchallenge insulin concentrations were determined by oral-glucose-tolerance test, total body fat by dual-energy X-ray absorptiometry, and subcutaneous abdominal (SAAT) and intraabdominal (IAAT) adipose tissue by computerized tomography. African Americans had greater fasting insulin ($\bar{x} \pm SD$: 79 ± 37 compared with 55 ± 23 pmol/L, $P < 0.01$), incremental 30-min insulin (567 ± 438 compared with 300 ± 304 pmol/L, $P < 0.001$), and incremental area under the insulin curve (AUC; 262 ± 209 compared with 164 ± 156 pmol/L, $P < 0.01$). In multiple linear regression, fasting insulin was independently related to total fat within both ethnic groups (model $R^2 = 0.42$ and 0.52 for African Americans and whites, respectively), incremental 30-min insulin to total fat and IAAT in whites only (model $R^2 = 0.71$), and AUC to SAAT in African Americans only (model $R^2 = 0.49$). Adjusting insulin indexes for adiposity did not eliminate the significant effect of ethnicity. In general, relations between adiposity and insulin were stronger in whites than in African Americans. African American children had higher insulin concentrations than white children after total body fat, IAAT, and SAAT were controlled for. However, strong relations between adiposity (total and abdominal) and insulin in both groups suggest that obesity may contribute to disease risk regardless of ethnicity. *Am J Clin Nutr* 1998;67:821-7.

KEY WORDS Insulin, fat distribution, African Americans, obesity, diabetes, whites, children, ethnicity, abdominal adipose tissue, dual-energy X-ray absorptiometry, computerized tomography, type 2 diabetes

INTRODUCTION

The incidence of type 2 diabetes in children and adolescents is increasing, particularly in African Americans (1). In adults, type 2 diabetes is associated not only with obesity, but with abdominal and intraabdominal (visceral) adiposity (2-6).

Few studies have examined the relation between fat distribution and indexes of insulin secretion and action in African American children and adolescents. Percentage body fat, but not

waist-hip ratio, was correlated with fasting insulin concentrations in both African American and white children aged 7-11 y (7). African American children had higher fasting insulin concentrations than white children, even after adiposity was adjusted for. Likewise, African Americans (mean age ≈ 12 y) were found to have higher 30-min insulin concentrations during an oral-glucose-tolerance test (OGTT) than whites (8). In this latter study, abdominal skinfold-thickness measurements, independent of peripheral skinfold-thickness measurements, were related to postchallenge insulin concentrations in the combined group. Only one study examined the contribution of subcutaneous abdominal adipose tissue (SAAT) and intraabdominal adipose tissue (IAAT) to insulin in a group of predominantly prepubertal, obese African American girls (9). In this study, fasting and 2-h insulin concentrations were related to SAAT in African Americans but not whites and were not related to IAAT in either group. The present study was conducted to examine the relation between fat distribution and insulin concentration in African American and white children, and to determine whether ethnic differences in fat distribution or in adiposity-insulin relations contribute to differences in insulin concentrations.

SUBJECTS AND METHODS

Subjects

A heterogeneous group of children was recruited by newspaper and radio advertisements and by word of mouth. Subjects were selected on the basis of age (5-10 y old), Tanner stage [determined

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by physician evaluation of both breast and pubic hair in girls (10) and genitalia in males (11); only children in Tanner stage I were included in the present study], and medical history [children were ineligible if they were taking medications known to affect body composition (eg, methylphenidate or growth hormone), had a diagnosis of syndromes or diseases known to affect body composition or fat distribution (eg, Cushing syndrome, Down syndrome, and type 1 diabetes), or a diagnosis with any major illness since birth]. Ethnicity was determined by self-report. This study was approved by the Institutional Review Board at the University of Alabama at Birmingham (UAB) and parents provided informed consent before testing commenced.

Protocol

Children were admitted to the General Clinical Research Center (GCRC) in the late afternoon for an overnight visit. On arrival, anthropometric measurements were obtained. A computerized tomography (CT) scan was conducted in the Department of Radiology at UAB at ≈ 1700 . The children were served dinner and an evening snack, consuming all food before 2000. All children were fed a fixed meal with 55% of energy as carbohydrate, 15% as protein, and 30% as fat. Consumption of only water and nonenergetic, noncaffeinated beverages was permitted between 2000 and testing the following morning. Two weeks after testing at the GCRC, children came to the Energy Metabolism Research Unit at UAB for body-composition analysis by dual-energy X-ray absorptiometry (DXA).

Oral-glucose-tolerance test

At 0600 a topical anesthetic (Emla cream; Astra Pharmaceutical Products, Inc, Westborough, MA) was applied to the antecubital space of one arm, and at ≈ 0700 a flexible catheter was inserted into a vein. Blood samples were drawn at -20 and -10 min relative to glucose ingestion. At time zero, children were instructed to drink a dextrose solution (1.75 g dextrose/kg body mass; 75 g maximum) within 5 min. Additional blood samples were collected at 30-min intervals from 30 to 180 min post-ingestion. Blood was centrifuged at $1800 \times g$ for 10 min at 4°C within 1 h of collection, sera were separated and placed in cryovials, and all samples were stored at -85°C until assayed for glucose and insulin (within 5 mo of collection).

Assay of glucose and insulin

Glucose was measured in 10 μL serum by using an Ektachem DT II System (Johnson and Johnson Clinical Diagnostics, Rochester, NY). In our laboratory this analysis has a mean intraassay CV of 0.61% and a mean interassay CV of 1.45%. Insulin was assayed in duplicate 200- μL aliquots with Diagnostic Products Corporation Coat-A-Count kits (Los Angeles). According to the supplier, cross-reactivity of this assay with proinsulin is $\approx 40\%$ at midcurve; C-peptide is not detected. In our laboratory this assay has a sensitivity of 11.4 pmol/L (1.9 $\mu\text{U}/\text{mL}$), a mean intraassay CV of 5%, and a mean interassay CV of 6%. Commercial quality control sera with low, medium, and high insulin concentrations (Lyphochek; Bio-Rad Corp, Anaheim, CA) were included in every assay to monitor variation over time.

Oral-glucose-tolerance test measures

Three main outcome measures were selected from the OGTT data: fasting insulin and incremental 30-min insulin concentra-

tions and incremental area under the insulin curve (AUC). Incremental 30-min insulin concentrations were used as an index of insulin secretion (12, 13) and incremental insulin AUC was used as an index of insulin resistance (13). Fasting insulin was the mean of two baseline samples drawn 10 min apart. Incremental 30-min insulin was calculated as the difference between 30-min and fasting insulin values. Incremental insulin AUC was calculated by dividing the AUC, as determined with the trapezoidal method (14), by 180 min and subtracting the fasting insulin value.

Measurement of body composition and fat distribution

SAAT and IAAT were measured by CT scanning with a HiLight/Advantage Scanner (General Electric, Milwaukee) as described previously (15). A single-slice scan (5 mm) of the abdomen was performed at the level of the umbilicus and analyzed for cross-sectional area of adipose tissue by using the density contour program. CT data are presented as cross-sectional area of tissue (cm^2) with Hounsfield units for adipose tissue as -190 to -30 . We have shown the test-retest reliability for IAAT to be 1.7% (16). All scans were analyzed by the same investigator (TRN). The total-body radiation dose to each subject was ≈ 0.0026 Gy (0.26 rad). This total-body radiation dose is less than that received from a standard chest X-ray.

Body composition (fat mass and fat-free mass) was measured by DXA using a Lunar DPX-L densitometer (Lunar Radiation Corp, Madison, WI). We previously validated the use of DXA against carcass analysis in a pig model that encompassed the pediatric weight range (17). Subjects were scanned in light clothing while lying flat on their backs with their arms at their sides. DXA scans were performed and analyzed with pediatric software version 1.5e. Weight-for-height percentile was calculated relative to the growth standards of the National Center for Health Statistics (18) by using EpiInfo, version 6 (Centers for Disease Control and Prevention, Atlanta).

Statistics

All statistics were performed with SAS version 6.11 for Windows (SAS Institute, Inc, Cary, NC). The following variables were not normally distributed and were therefore log-transformed before analysis: total fat, IAAT, SAAT, fasting insulin concentration, incremental 30-min insulin concentration, and incremental insulin AUC. After log transformation, incremental insulin AUC, SAAT, and total fat remained skewed in distribution but were closer to normality than before transformation [$P < W$ (Shapiro-Wilk statistic) = 0.01, 0.002, and 0.005, respectively, compared with an untransformed value of $P < W = 0.0000$ for all]. Preliminary analyses indicated that sex had no independent effects on main outcome variables; thus, all between-group comparisons are shown with the sexes combined and sex was omitted from the regression models. Analysis of variance (ANOVA) was used to compare body composition and OGTT measures between African American and white children. This was done both with sexes combined and only in boys; the latter was to show that ethnic differences were not due to the greater number of girls in the African American group. Pearson correlation analysis was used to examine correlations between adipose tissue variables and main outcome measures from the OGTT (fasting insulin, incremental 30-min insulin, and incremental insulin AUC) within each ethnic subgroup of children. Multiple linear regression was used to identify variables with independent effects on fasting insulin, incremental 30-min insulin, and incremental insulin AUC. The models



included IAAT, SAAT, total fat, and ethnicity. Because ethnicity emerged as significant in all models, subsequent analyses also were conducted within each ethnic group. Adipose depots that were independently related to main outcome measures were used in subsequent analysis of covariance (ANCOVA), with ethnicity as the class variable. For all tests, differences or effects with $P < 0.05$ were considered significant.

RESULTS

African American and white children did not differ with respect to age or body composition, either with sexes combined (Table 1) or within only boys (Table 2). The mean weight-for-height percentile for African American children was 67% and that for white children was 71%; 46% of the African American children and 35% of the white children were above the 85th percentile for National Center for Health Statistics growth standards. Normal glucose tolerance, defined as 2-h glucose concentration < 7.77 mmol/L (140 mg/dL) (19), was measured in all but four children (three African American girls and one white boy) who had 2-h glucose values between 7.77 and 8.33 mmol/L (140 and 150 mg/dL). Because impaired glucose tolerance was not an exclusion criterion, these children were not removed from the data set. However, results did not differ if analyses were conducted without data from these individuals. Fasting insulin concentration was higher and fasting glucose lower in African Americans than in whites (Table 1, Figure 1). Thirty-minute insulin (unadjusted and incremental), 90-min insulin, and incremental insulin AUC were higher in African American than in white children. Correlation analysis indicated that total fat, IAAT, and SAAT were related to fasting insulin, incremental 30-min insulin, and incremental insulin AUC in both white and African American children (Table 3).

In multiple linear regression for the dependent variable fasting insulin, total fat ($P < 0.01$) and ethnicity ($P < 0.01$) were positively related, and IAAT ($P < 0.05$) was negatively related, after SAAT was adjusted for. The only variable independently related to incremental 30-min insulin concentration was ethnicity ($P < 0.001$, positive). Both SAAT ($P < 0.05$) and ethnicity ($P < 0.001$) had significant, positive, independent effects on incremental

insulin AUC (Figure 2). When analyses were conducted within each ethnic group, both total fat and IAAT were independently related to fasting insulin in African Americans, whereas only total fat was significant in whites (Table 4). No measure of adiposity was independently related to incremental 30-min insulin concentration in African American children, whereas total fat and IAAT were related in white children. SAAT emerged as independently affecting insulin AUC in African American children, whereas no variable emerged as independent in white children.

Adjusting fasting insulin for total fat with ANCOVA did not eliminate the significant effect of ethnicity observed with the ANOVA (70 ± 4 and 52 ± 3 pmol/L, adjusted geometric mean \pm SEM for African American and white children, respectively, $P < 0.001$). Likewise, adjusting incremental insulin AUC for SAAT did not alter the effect of ethnicity (218 ± 16 and 122 ± 11 pmol/L, adjusted geometric mean \pm SEM for African American and white children, respectively, $P < 0.001$; Figure 2). The slopes of the relations between incremental 30-min insulin and both total fat and IAAT differed with ethnicity; thus, these data could not be subjected to ANCOVA.

DISCUSSION

The main findings of this study are 1) African American children had higher fasting and postchallenge insulin concentrations than white children, 2) the effect of ethnicity was not eliminated by adjusting for total or regional adiposity, and 3) adiposity explained more of the variance in fasting and postchallenge insulin in white than in African American children. Additionally, ethnicity affected the relation between fat distribution and insulin, with SAAT being independently related to insulin AUC only in African American children, and IAAT being independently related to 30-min insulin concentration only in white children. The greater fasting and postchallenge insulin concentrations in the African American children were not explained by total or regional adiposity. The African American children were neither more obese than nor had more SAAT or IAAT than the white children. Adjusting the various insulin measures for total and regional body fat, either by multiple linear regression or by ANCOVA, did not eliminate the significant effect of ethnicity.

TABLE 1

Descriptive statistics for 73 African American and white prepubertal children¹

	African American (n = 20 boys and 23 girls)	White (n = 21 boys and 9 girls)
Age (y)	7.6 \pm 1.7 (4.6–10.4)	8.1 \pm 1.3 (5.6–10.4)
Weight (kg)	32.0 \pm 12.9 (14.0–69.0)	30.0 \pm 6.4 (20.4–45.0)
Total fat mass (kg)	10.1 \pm 7.7 (2.7–31.7)	8.1 \pm 4.4 (2.6–19.7)
Percentage fat (%)	28.6 \pm 9.8 (14.4–47.4)	25.9 \pm 9.0 (11.4–43.9)
IAAT (cm ²)	26.7 \pm 22.5 (7.1–114.4)	29.0 \pm 16.5 (7.0–77.4)
SAAT (cm ²)	84.0 \pm 100.5 (8.3–462.7)	80.8 \pm 65.2 (13.6–230.0)
Total lean mass (kg)	20.5 \pm 5.5 (10.6–34.5)	20.5 \pm 3.1 (14.5–25.9)
Fasting glucose (mmol/L)	5.0 \pm 0.29 ² (4.3–5.7)	5.1 \pm 0.33 (4.6–6.1)
Fasting insulin (pmol/L)	79 \pm 37 ³ (30–192)	55 \pm 23 (24–114)
30-min insulin (pmol/L) ⁴	567 \pm 438 ⁵ (120–2364)	300 \pm 304 (78–1560)
Insulin AUC (pmol/L) ⁴	262 \pm 209 ³ (75–968)	164 \pm 156 (46–797)

¹ $\bar{x} \pm$ SD; range in parentheses. IAAT, intraabdominal adipose tissue; SAAT, subcutaneous abdominal adipose tissue; AUC, area under the curve. To convert values for insulin to μ U/mL, divide by 6; to convert values for glucose to mg/dL, divide by 0.05551.

^{2,3,5} Significantly different from white children: ² $P < 0.05$, ³ $P < 0.01$, ⁵ $P < 0.001$.

⁴ Incremental.

TABLE 2Descriptive statistics for 41 African American and white prepubertal boys¹

	African American (n = 20)	White (n = 21)
Age (y)	7.7 ± 1.7 (5.1–10.4)	8.2 ± 1.4 (5.6–10.4)
Weight (kg)	33.7 ± 14.3 (19.9–69.0)	30.2 ± 6.8 (20.4–45.0)
Total fat mass (kg)	9.7 ± 8.6 (3.7–31.7)	7.8 ± 4.8 (2.6–19.7)
Percentage fat (%)	25.5 ± 9.9 (14.4–47.4)	23.4 ± 9.7 (11.4–43.9)
IAAT (cm ²)	29.8 ± 29.4 (7.9–114.4)	26.5 ± 15.0 (7.0–61.2)
SAAT (cm ²)	84.9 ± 126.6 (8.3–462.7)	75.8 ± 68.8 (13.6–230.0)
Total lean mass (kg)	22.5 ± 5.8 (14.6–34.5)	21.0 ± 3.1 (15.4–25.9)
Fasting glucose (mmol/L)	4.9 ± 0.34 (4.4–5.7)	5.1 ± 0.28 (4.6–5.8)
Fasting insulin (pmol/L)	70 ± 31 ² (30–168)	52 ± 21 (24–114)
30-min insulin (pmol/L) ³	517 ± 410 ⁴ (156–1728)	263 ± 220 (78–792)
Insulin AUC (pmol/L) ³	236 ± 203 ² (75–968)	141 ± 116 (46–559)

¹ $\bar{x} \pm SD$; range in parentheses. IAAT, intraabdominal adipose tissue; SAAT, subcutaneous abdominal adipose tissue; AUC, area under the curve. To convert values for insulin to $\mu\text{U/mL}$, divide by 6; to convert values for glucose to mg/dL , divide by 0.05551.

³ Incremental.

^{2,4} Significantly different from white children: ² $P < 0.05$, ⁴ $P < 0.01$.

These results agree with those of others who found ethnic differences in insulin concentration in children and adolescents independent of adiposity (7, 20), but differ from a report of no ethnic difference (9). In the latter study, the African American group had less upper-body fat than the white group, a difference that might have acted to minimize potential ethnic differences in insulin concentration. Thus, available data suggest that greater total or regional adiposity is unlikely to be the major cause of

the higher insulin concentrations reported in African American children.

The higher postchallenge insulin concentrations observed in the African American children were apparent at both 30 min and in the incremental AUC. These measures were selected as approximations of insulin secretion (12) and resistance (13), respectively, two possible sources of hyperinsulinemia. Choice of these approximations was based on the relation in adults between OGTT-derived insulin and glucose concentrations and actual measurements of first-phase insulin secretion after intravenous glucose administration (12), and insulin resistance during a pancreatic suppression test (13). The greater incremental insulin AUC in the African American children suggested that this group was more insulin resistant than the white children. However, adding incremental 30-min insulin to the multiple regression model for incremental insulin AUC eliminated the independent effect of ethnicity ($P = 0.441$), indicating that the greater AUC was due solely to the greater insulin secretion early in the test. Therefore, we suggest that the greater postchallenge insulin concentration observed in the African American children was due to greater pancreatic responsiveness and not to greater insulin resistance. This hypothesis is supported by recent data in prepubertal children showing no ethnic difference in insulin sensitivity assessed with a hyperglycemic clamp (20).

Despite having higher insulin concentrations, African American children had significantly lower fasting glucose and slightly (but not significantly) lower postchallenge glucose concentrations throughout the OGTT. Lower fasting glucose (21) and glucose AUC (22) in African Americans have been reported in other studies as well. Although these observations may reflect the greater insulin-independent glucose disposal observed in African Americans (23), the cause of lower glucose concentrations in this group is yet to be determined.

We examined the possibility that ethnic differences exist in the relation between total or regional adiposity and insulin in children. Such differences may appear either in the amount of variance explained by adiposity (R^2), in the slope of the line of the relation between adiposity and insulin, or in independent effects of individual components of adiposity after all other components are adjusted for. In our subject population, the regression-model R^2 values for insulin were in general higher in whites than in African Americans, despite the greater range of both dependent and inde-

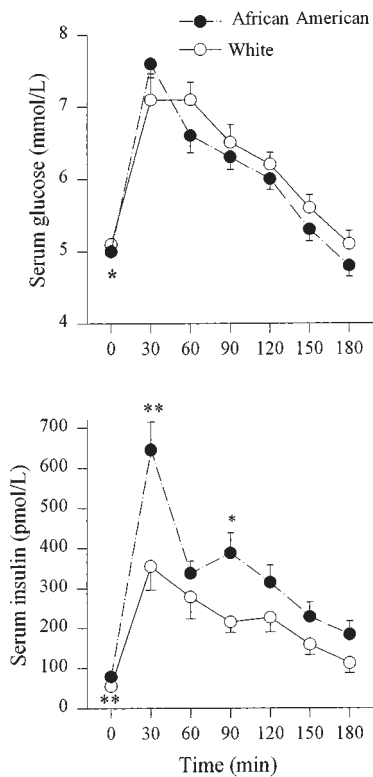


FIGURE 1. Serum glucose and insulin response to oral glucose administration in African American and white children. $\bar{x} \pm \text{SEM}$. *, ** Significant differences between groups at a given time: * $P < 0.05$, ** $P < 0.01$. To convert values for insulin to $\mu\text{U/mL}$, divide by 6; to convert values for glucose to mg/dL , divide by 0.05551.

TABLE 3

Pearson correlation coefficients for oral-glucose-tolerance-test measures and adiposity indexes for 73 prepubertal African American and white children¹

	Log total fat mass		Log IAAT		Log SAAT	
	AA	W	AA	W	AA	W
Log fasting insulin	0.57 ²	0.71 ²	0.40 ³	0.50 ³	0.55 ²	0.61 ²
Log 30-min insulin ⁴	0.58 ²	0.80 ²	0.52 ²	0.79 ²	0.60 ²	0.77 ²
Log insulin AUC ⁴	0.59 ²	0.73 ²	0.59 ²	0.64 ²	0.69 ²	0.68 ²

¹ AA, African American; W, white; IAAT, intraabdominal adipose tissue; SAAT, subcutaneous abdominal adipose tissue; AUC, area under the curve.² $P < 0.001$.³ $P < 0.01$.⁴ Incremental.

pendent variables in the latter. Additionally, the slope of the regression line between total fat and incremental 30-min insulin concentration was greater in whites. A positive and independent contribution of IAAT to 30-min insulin concentration was observed only in whites (Table 4); however, SAAT was independently related to insulin AUC only in African Americans. A negative contribution of IAAT to fasting insulin was observed in African Americans; the potential physiologic implications of this finding are not clear. A unique role for SAAT in African Americans was likewise reported by Yanovski et al (9), who found that

SAAT, assessed by multiple magnetic resonance imaging scans, was associated with fasting and postchallenge insulin concentrations in African American but not white girls matched for body mass index, age, Tanner stage, and socioeconomic status. Taken together, these two studies suggest that although adiposity in general (all components) may explain more of the variance in insulin concentration in white children, SAAT is uniquely related to insulin concentration in African American children.

Several studies have suggested that sex may influence analysis of ethnic differences in insulin concentration and adiposity-insulin relations. Insulin concentrations are higher in African American females (relative to males or whites), independent of body mass index (24), and are significantly related to the ratio of central to peripheral skinfold thicknesses in all race-sex groups except African American males (8). The present study did not include enough white girls ($n = 9$) to make firm conclusions about the influence of sex in this regard, that of Yanovski et al (9) included only females, and differences between studies in the methods used (CT, magnetic resonance imaging, skinfold thicknesses, body mass index, and DXA) make it difficult to compare results. Thus, further study is needed to clarify the importance of sex in explaining insulin concentrations both within and between ethnic groups.

Similar to the present results, studies in adults have indicated that African Americans have greater fasting insulin concentrations and insulin secretion than whites, independent of obesity (25–29). The basis for these differences is not clear. A recent report indicated that physical fitness, as reflected in maximal oxygen consumption, may play a role in the ethnic difference in insulin secretion (20). [In our cohort, adjustment for maximal oxygen consumption did not eliminate the ethnic difference in postchallenge insulin concentration (BA Gower, TR Nagy, CA Trowbridge, C Dezenberg, and MI Goran, unpublished observations, 1997).] Additionally, ethnic differences in diet, socioeconomic status, genetics, or other factors may contribute to the higher fasting and postchallenge insulin concentrations observed in African Americans.

A limitation of the present study is the cross-reactivity with proinsulin of the insulin antiserum used. Ethnic differences in proinsulin secretion (none have been reported) could result in erroneous conclusions regarding the concentration of bioactive insulin and thereby insulin sensitivity. Additionally, reduced hepatic insulin extraction among African Americans relative to whites (24, 25) could result in greater serum insulin concentrations independent of pancreatic responsiveness and lead to misleading conclusions regarding insulin secretion.

In summary, the present study found that African American children had significantly greater fasting and postchallenge insulin concentrations than white children that were not explained by differences in body fat or fat distribution. However,

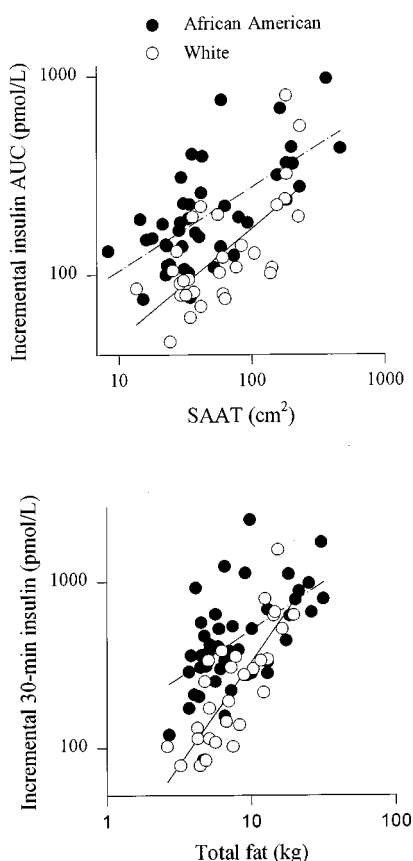


FIGURE 2. Regression of incremental insulin area under the curve (AUC) on subcutaneous abdominal adipose tissue (SAAT) ($R^2 = 0.48$ and slope = 0.45 for African Americans; $R^2 = 0.46$ and slope = 0.55 for whites; $P > 0.05$ for difference in slope) and regression of incremental 30-min insulin concentration on total fat in children ($R^2 = 0.33$ and slope = 0.57 for African Americans; $R^2 = 0.64$ and slope = 1.22 for whites; $P < 0.01$ for difference in slope). To convert values for insulin to $\mu\text{U/mL}$, divide by 6.

TABLE 4
Multiple linear regression models by ethnicity¹

Independent variable	African American		White	
	Parameter estimate ± SEE	P	Parameter estimate ± SEE	P
Model A, dependent variable:				
log fasting insulin				
Log total fat (kg)	0.46 ± 0.20	0.028	0.71 ± 0.25	0.009
Log IAAT (cm ²)	-0.47 ± 0.20	0.023	-0.08 ± 0.18	0.662
Log SAAT (cm ²)	0.25 ± 0.14	0.080	-0.07 ± 0.20	0.726
Model B, dependent variable: log incremental 30-min insulin				
Log total fat (kg)	0.26 ± 0.30	0.404	0.87 ± 0.39	0.035
Log IAAT (cm ²)	-0.19 ± 0.30	0.539	0.65 ± 0.28	0.026
Log SAAT (cm ²)	0.36 ± 0.21	0.101	-0.16 ± 0.30	0.614
Model C, dependent variable				
log incremental insulin AUC				
Log total fat (kg)	-0.12 ± 0.27	0.660	0.78 ± 0.39	0.058
Log IAAT (cm ²)	-0.06 ± 0.27	0.807	0.23 ± 0.28	0.423
Log SAAT (cm ²)	0.56 ± 0.19	0.005	-0.06 ± 0.31	0.847

¹ Model A: $R^2 = 0.42$ for African Americans, 0.52 for whites. Model B: $R^2 = 0.38$ for African Americans, 0.71 for whites. Model C: $R^2 = 0.49$ for African Americans, 0.54 for whites. IAAT, intraabdominal adipose tissue; SAAT, subcutaneous abdominal adipose tissue; AUC, area under the curve.

insulin measures were related to adiposity within both ethnic groups. Thus, for both white and African American individuals, obesity is likely to increase disease risk. For African Americans it will be important to identify the source and disease implications of greater adiposity-independent fasting and postchallenge insulin concentrations.

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