Visceral Adipose Tissue and Markers of the Insulin Resistance Syndrome in Obese Black and White Teenagers

Scott Owens, Bernard Gutin,* Paule Barbeau,* Mark Litaker,* Jerry Allison,† Matthew Humphries,* Tomoki Okuyama,* and Ngoc-Anh Le‡*

Abstract

OWENS, SCOTT, BERNARD GUTIN, PAULE BARBEAU, MARK LITAKER, JERRY ALLISON, MATTHEW HUMPHRIES, TOMOKI OKUYAMA, AND NGOC-ANH LE. Visceral adipose tissue and markers of the insulin resistance syndrome in obese black and white teenagers. *Obes Res.* 2000;8:287–293.

Objective: To determine the relationships between visceral and general adiposity, cardiovascular fitness, and markers of the insulin resistance syndrome in obese black and white teenagers.

Research Methods and Procedures: Cross-sectional survey of 81 obese 13- to 16-year-old youths. Visceral adipose tissue was measured with magnetic resonance imaging, and percentage body fat was measured with dual-energy X-ray absorptiometry. Cardiovascular fitness was assessed with a submaximal treadmill test. Fasting blood samples were analyzed for lipids/lipoproteins and insulin. Resting blood pressure was obtained using an automated cuff.

Results: Visceral adipose tissue was significantly correlated with unfavorable levels of: triacylglycerol ($r = 0.27$, $p < 0.05$), total cholesterol ($r = 0.27$, $p < 0.05$), high-density lipoprotein cholesterol ($r = -0.26$, $p < 0.05$), the ratio of total cholesterol/high-density lipoprotein cholesterol ($r = 0.42$, $p < 0.01$), low-density lipoprotein cholesterol ($r = 0.27$, $p < 0.05$), apolipoprotein B ($r = 0.38$, $p < 0.01$), and systolic blood pressure ($r = 0.30$, $p < 0.01$). Multiple regression analyses revealed that visceral adipose tissue was more powerful than percentage body fat for explaining variance in lipoproteins (e.g., for the ratio of total cholesterol/high-density lipoprotein cholesterol, $r^2 = 0.13$, $p < 0.01$, and for systolic blood pressure, $r^2 = 0.07$, $p < 0.05$). Ethnicity was the most powerful of the demographic predictors for blood lipids ($r^2 = 0.15$ for triacylglycerol with lower levels in blacks; $r^2 = 0.10$ for high-density lipoprotein cholesterol with higher levels in blacks; $r^2 = 0.06$ for the ratio of total cholesterol/high-density lipoprotein cholesterol with lower levels in blacks). Cardiovascular fitness was not retained as a significant predictor of markers of the insulin resistance syndrome.

Discussion: Some of the deleterious relationships between visceral adiposity and markers for the insulin resistance syndrome seen in adults were already present in these obese young people.

Key words: insulin resistance syndrome, teenagers, visceral adipose tissue

Introduction

In adults, obesity is associated with an insulin-resistant syndrome (IRS) that is characterized by hyperinsulinemia, dyslipidemia, hypertension, and an increased risk for cardiovascular disease (1). Relations between body fat and markers of the IRS vary with body fat distribution (2,3). Central adiposity seems to be more deleterious than peripheral fat (4,5). Within the central depot, visceral adipose tissue (VAT) tends to be more highly correlated with the IRS markers than is subcutaneous abdominal adipose tissue (SAAT) (6–9). Low levels of cardiovascular fitness have also been associated with unfavorable levels of IRS markers (10,11).

There are few studies examining VAT and markers of the IRS in adolescents, and no data have been reported for African American teenagers. In an Italian study of obese 10- to 15-year-old boys and girls, significant correlations were observed between VAT and triacylglycerol ($r = 0.46$), total

Submitted for publication August 19, 1999.
Accepted for publication in final form December 12, 1999.
*Georgia Prevention Institute, Department of Pediatrics, and †Department of Radiology, Medical College of Georgia, ‡Lipid Metabolism Laboratory, Emory University, Atlanta, Georgia.
Address correspondence to Scott Owens, PhD, Department of Health and Human Performance, Reid Gymnasium, Western Carolina University, Cullowhee, NC 28723. E-mail: owens@wpoff.wcu.edu

cholesterol ($r = 0.54$), and low-density lipoprotein cholesterol ($r = 0.60$) (12). A study of obese and non-obese adolescent girls found VAT to be significantly correlated with triacylglycerol ($r = 0.53$), high-density lipoprotein cholesterol ($r = -0.54$), and insulin ($r = 0.55$) in the obese girls only (13). Thus, some of the relationships between VAT and markers of the IRS reported for adults have been observed in obese youths.

Given that the prevalence of overweight among children and adolescents in the United States is increasing (14) and that overweight tends to track into adulthood (15,16) and has adverse health effects (17,18), understanding the nature of the relationships between the various components of body composition and disease risk becomes increasingly important. This study reports on the relationships between measures of fatness, fitness, and a number markers of the IRS in black and white obese teenagers.

Research Methods and Procedures

Subjects were 81 apparently healthy obese teenagers 13 to 16 years of age. Recruitment was accomplished with promotional flyers distributed through selected public and private schools in Augusta, GA. To be included in the study, a child needed to have a triceps skinfold greater than the 85th percentile for gender, age, and ethnicity (19), not be involved in any other weight control or exercise program, and not be restricted as to physical activity. Ethnicity was by self-designation of the parent. Interested parents and teenagers attended an orientation session and both gave informed consent in accordance with procedures of our Human Assurance Committee.

Body Fatness

VAT was selected as the component of central adiposity and was determined in the Department of Radiology at the Medical College of Georgia utilizing a 1.5-tesla magnetic resonance imaging (MRI) system (General Electric Medical Systems, Milwaukee, WI). Spin-echo techniques were used to produce T_1-weighted images demonstrating good contrast between adipose and nonadipose tissues (20). Details of the MRI acquisition were as follows: repetition time, 450 ms; echo time, 12 ms; field of view, 400–480 mm; matrix, 192×256; number of excitations, 1. Respiratory compensation was used to reduce artifacts caused by respiratory motion. With subjects in the supine position, a series of five, 1-cm thick, transverse images was acquired beginning at the inferior border of the fifth lumbar vertebra and proceeding toward the head. A 2-mm gap between images was utilized to prevent cross-talk. Respiratory compensation was used to reduce artifacts caused by respiratory motion. Tissues superior to and inferior to the five slices were saturated to reduce artifacts from flow in the aorta or inferior vena cava. VAT was segmented by thresholding and quantified as adipose tissue within a region of interest bounded by the internal-most aspect of the abdominal and oblique muscle walls and the posterior aspect of the vertebral body. Values for VAT from a single image were calculated in terms of surface area (cm^2). Volume of VAT (cm^3) from this area of the abdomen was estimated by multiplying the surface area for the individual images by the image width (1 cm) and then summing across the five images. To reduce interobserver variability, all images were analyzed by the same experienced observer. We found that the intraclass correlation coefficients for separate-day repeat analyses exceeded 0.99.

Total body percentage fat (%BF) was selected as the measure of general adiposity and was derived from dual-energy X-ray absorptiometry (DXA) (Hologic QDR-1000, Waltham, MA). Svendsen et al (21) showed DXA values agreed well with carcass analysis of pigs. We have found DXA to be reliable in children (22).

Cardiovascular Fitness

Cardiovascular fitness was determined as the oxygen consumption at a heart rate of 170 bpm (VO_{2-170}) obtained during an incremental treadmill test. The treadmill protocol involved an initial 6-minute period of walking at 2 mph, 0% grade that served as a warm-up and familiarization stage for the subjects. Subsequent stages were 2 minutes in length and involved 0.5 mph increases in treadmill speed (Stages 2 through 4) followed by 2% increases in treadmill grade (Stage 5 through test conclusion). Subjects exercised until volitional exhaustion was reached. Oxygen consumption (VO_2) was measured throughout with indirect calorimetry using a FITCO (Farmingdale, NY) metabolic measurement cart. Heart rate was obtained from a heart rate monitor (Polar Vantage XL, Port Washington, NY). Although obese children seem to be capable of maximal tests to the same degree that non-obese children are (23), a significant proportion of people fail to meet objective criteria for a true maximal oxygen consumption ($VO_{2\ max}$). If $VO_{2\ max}$ is used as the main index of cardiovascular fitness, those subjects cannot be included in analyses that include cardiovascular fitness, thereby reducing sample size. VO_{2-170} is easily measured in people varying widely in fitness, and we found it to be highly correlated with $VO_{2\ max}$ ($r = 0.88$) in 64 of the obese adolescents who did meet strict criteria for $VO_{2\ max}$ (oxygen plateau < 2 ml/kg/min increase in VO_2, a respiratory exchange ratio greater than 1.0, or a heart rate > 200 bpm).

Blood Parameters

For blood sampling, subjects reported to the laboratory between 8 and 9 AM after a 12-hour fast. A 20-mL blood sample was obtained from an antecubital vein and transferred into vacutainers containing EDTA. Tubes were inverted gently and stored in an ice bath until centrifugation. Samples were separated by low speed centrifugation (6000*g* for 10 minutes) with plasma placed in plastic vials and stored at −70 °C until analysis. Samples were analyzed at the Emory Lipid Research Laboratory, which is certified by the College of American Pathologists and participates in the National Heart, Lung, and Blood/Centers for Disease Control Lipid Standardization Program. High-density lipoprotein cholesterol (HDLC) and low-density lipoprotein cholesterol (LDLC) were determined using the homogeneous enzymatic methods (Equal Diagnostics, Exton, PA). Apolipoprotein (Apo)A-I and ApoB were measured by nephelometry using immunoturbidimetric methods (SPQ Test System; Incstar Corp., Stillwater, MN). Fasting insulin concentrations were determined using the IMx Insulin Assay (Abbot Laboratories, Abbott Park, IL), and glucose values were determined by enzymatic methods (Beckman Diagnostics, Fullerton, CA). LDL particle diameter was determined by nondenaturing polyacrylamide gradient gel electrophoresis as previously described (24).

Blood Pressure

Resting systolic and diastolic blood pressure (BP) were measured with an automated BP monitor (Dinamap; Critikon, Inc., Tampa, FL). Measurements were obtained after 10 minutes of rest in the supine position with an appropriately sized cuff placed on the left arm. Five readings were taken at 1-minute intervals, and the last three were averaged.

Data Analysis

Two-way analysis of variance was used to examine differences in anthropometric variables as related to gender, ethnicity, and their interaction. Pearson correlations were used to determine univariate relationships between measures of fatness and fitness (VAT, %BF, VO_{2-170}) and markers of the IRS. The significance level was set at $p < 0.05$. Hierarchical stepwise multiple linear regression techniques were then used to evaluate the relative contributions of the demographic (age, gender, ethnicity) and fatness/fitness (VAT, %BF, VO_{2-170}) measures to the variance in markers of the IRS. In this procedure, variables are entered in stepwise fashion within blocks (25). The procedure obtains the best model within a block before variables in subsequent blocks are considered for entry into the model. Once the best model within a block is determined, the variables selected remain in the model regardless of the variables available for selection within subsequent blocks. Thus, variables in later blocks are considered “adjusted” for those variables previously selected for inclusion. The stepwise technique circumvents the potential problem of multicollinearity, because a variable will not enter the model if it is highly correlated with a variable already in the model. For the present study, two blocks of variables were utilized. Block 1 variables included the demographic measures (age, gender, ethnicity). Block 2 included the fatness/fitness measures (VAT, %BF, VO_{2-170}) that were significantly correlated with the risk factor in question in the univariate correlational analysis. Probabilities for variables to enter or be removed during the stepwise procedure were $p \leq 0.05$ and $p \geq 0.10$, respectively. All analyses were performed using the SPSS/PC statistical program (version 8.0 for windows; SPSS, Inc., Chicago, IL).

Results

A total of 81 obese teenagers participated in this study. Table 1 displays descriptive statistics by gender and ethnic subgroups along with the ANOVA results for the subgroup comparisons (data from the lone Hispanic girl were omitted in these comparisons). Significant gender differences were observed for three of the variables with girls being older ($p = 0.002$), shorter ($p = 0.006$), and having a lower VO_{2-170} (i.e., less fit) ($p < 0.001$). Relative to ethnicity, blacks had significantly less VAT than whites ($p < 0.001$). This difference remained significant even after controlling for total adiposity ($p = 0.001$). Blacks were also significantly less fit ($p < 0.001$).

Table 2 shows the simple correlations between the fatness/fitness measures and markers of the IRS. VAT was the only fatness measure significantly associated with any of the lipid-lipoprotein variables; the strongest of these relationships being between VAT and total cholesterol/HDLC. VAT was also significantly associated with systolic BP. VO_{2-170} was positively correlated with triacylglycerol. The %BF was significantly correlated with insulin. For some markers of the IRS, no significant associations with the fatness/fitness measures were detected (LDL particle size, ApoA-I, glucose, and diastolic BP).

Table 3 summarizes results of the hierarchical stepwise multiple regression procedure in which the demographic variables (age, gender, ethnicity) were entered in Block 1 and significant fatness/fitness measures from the univariate correlational analysis were entered in Block 2. Relative to the demographic variables, age was not retained as a significant predictor for any of the eight variables shown in Table 3. On the other hand, either gender or ethnicity was a significant predictor for six of the eight IRS markers; that is, being female was predictive of lower total cholesterol, LDLC, and systolic BP. Being black was predictive of lower triacylglycerol, lower total cholesterol/HDLC, and higher HDLC. Regarding the fatness/fitness measures, VAT was a significant predictor for six of the eight IRS markers. In each case, greater VAT was predictive of a less favorable level of the IRS marker. The %BF was retained as a significant predictor only in the case of insulin, with greater %BF associated with a higher insulin concentration. The measure of fitness, VO_{2-170}, did not emerge as an independent predictor for any of the markers of the IRS.

Table 1. Descriptive statistics (mean ± SD)

		White		African American		Significant effects by two-way ANOVA*
	Combined	**Boys**	**Girls**	**Boys**	**Girls**	
Age (years)	14.9 ± 1.3 $n = 80$	14.4 ± 1.2 $n = 10$	15.3 ± 1.3 $n = 15$	14.9 ± 1.3 $n = 16$	15.2 ± 1.1 $n = 39$	Gender
Height (cm)	165 ± 7 $n = 80$	168 ± 9 $n = 10$	162 ± 6 $n = 15$	169 ± 7 $n = 16$	164 ± 8 $n = 39$	Gender
Weight (kg)	94.9 ± 19.4 $n = 80$	95.1 ± 27.7 $n = 10$	88.2 ± 17.9 $n = 15$	100.3 ± 17.8 $n = 16$	95.2 ± 18.6 $n = 39$	NS
VAT (cm^3)	306 ± 130 $n = 79$	433 ± 105 $n = 10$	355 ± 151 $n = 15$	279 ± 107 $n = 16$	263 ± 109 $n = 38$	Ethnicity
%BF	44.5 ± 6.7 $n = 78$	40.7 ± 6.6 $n = 9$	45.8 ± 5.9 $n = 15$	43.9 ± 9.3 $n = 16$	45.2 ± 5.6 $n = 38$	NS
$VO_{2\text{-}170}$ (mL/kg/minute)	19.7 ± 4.4 $n = 79$	25.0 ± 5.7 $n = 10$	20.8 ± 3.2 $n = 15$	20.5 ± 3.3 $n = 16$	17.5 ± 3.3 $n = 38$	Gender, ethnicity

* $p < 0.05$.

Discussion

This study examined relationships between two aspects of adiposity, one measure of fitness, and several markers of the IRS in obese teenaged boys and girls. It seems to be the first report to include data on VAT and IRS markers in African American teenagers. We found VAT to be significantly lower in black than in white teenagers, even after controlling for total fat mass. Less VAT in blacks compared to whites has been reported in other age groups, including 4- to 10-year-old boys and girls (26), 7- to 10-year-old girls (27), obese women (28), and young adult men (29).

VAT was more frequently associated with markers of the IRS than were %BF or $VO_{2\text{–}170}$ and was more frequently retained as a predictor variable in the multiple regression analyses. We note in passing that MRI-derived SAAT was also examined for its association with the IRS markers. As anticipated, based on results from our previous study with younger subjects (30), SAAT was not as powerful a predictor for the IRS markers as was VAT (data not shown). As for the demographic variables, gender and ethnicity were each retained as significant predictors for three of the IRS markers. The retention of gender and ethnicity, as significant predictors for the variations in some of the lipid/lipoproteins, differs from our observations in 7- to 11-year-old black and white children (30) in which VAT, but not ethnicity or gender, provided the explanatory power. It may be that the relative predictive powers of gender, ethnicity, and VAT for these variables differs in pre- vs. postpubertal subjects, although the relationships seem to be complex. In 4- to 10-year-old black and white children, Ku et al. (26) found that VAT, but not ethnicity, was a significant predictor for triacylglycerol but not for HDLC. In premenopausal women, Albu et al. (31) reported that the regression equations predicting triacylglycerol and HDLC from adiposity measures (VAT, SAAT, total body fat mass) had slopes that were not significantly different for blacks and whites. They also reported that LDLC was independently related to VAT in black but not white women. In our teenagers we found VAT and gender, but not ethnicity, to be significant predictors of LDLC. In our study of 7 to 11 year olds, VAT was a significant predictor of LDLC, but gender was not (30). The finding for ApoB in the present study mirrored that of our previous study with 7 to 11 year olds in that VAT was the only significant predictor (30). Thus, these data suggest that, in obese teenagers, VAT is the adiposity measure that carries the most explanatory weight for the common lipid-lipoprotein risk factors but that gender and ethnicity also contribute significant information about some of these variables.

A somewhat different set of relationships emerged for the nonlipid markers. For these variables, VAT was predictive only for systolic BP. We saw a similar pattern in our study of the 7 to 11 year olds, that is, VAT was the strongest predictor of the blood lipids but was not predictive of the nonlipid markers (30).

The %BF emerged as the best predictor of fasting insulin in our group of teenagers, which contrasts somewhat with the findings of Gower et al. (32) who reported that VAT, total body fat mass, and ethnicity were significant predictors for fasting insulin in normal-weight 5- to 10-year-old black and white children. Differences in pubertal and obesity status between the two studies may have contributed to the divergent findings.

Both gender and VAT displayed significant predictive power for systolic BP. Other studies of youth typically have not found VAT and systolic BP to be highly correlated

Table 2. Pearson correlations among fatness and fitness measures and markers of the IRS

Variable	VAT	%BF	$VO_{2\text{-}170}$
Triacylglycerol	0.27*	−0.14	0.26*
	$n = 79$	$n = 78$	$n = 79$
Total cholesterol	0.27*	0.04	−0.09
	$n = 79$	$n = 78$	$n = 79$
HDLC	−0.26*	0.05	−0.12
	$n = 79$	$n = 78$	$n = 79$
TC/HDLC	0.42†	0.01	−0.02
	$n = 79$	$n = 78$	$n = 79$
LDLC	0.27*	0.10	−0.16
	$n = 79$	$n = 78$	$n = 79$
LDL size	−0.16	0.06	−0.10
	$n = 79$	$n = 78$	$n = 79$
ApoA-I	−0.11	−0.01	−0.03
	$n = 79$	$n = 78$	$n = 79$
ApoB	0.38†	0.03	−0.02
	$n = 79$	$n = 78$	$n = 79$
Insulin	0.16	0.24*	−0.20
	$n = 78$	$n = 77$	$n = 78$
Glucose	0.23	0.14	0.05
	$n = 70$	$n = 70$	$n = 70$
Systolic BP	0.30†	−0.04	−0.05
	$n = 80$	$n = 79$	$n = 80$
Diastolic BP	0.10	−0.01	−0.20
	$n = 80$	$n = 79$	$n = 80$

* $p < 0.05$; † $p < 0.01$.

Table 3. Summary table for hierarchical stepwise multiple regression analyses for markers of the IRS

Dependent variable	Predictor variables retained	Block*	R^2	R^2 increase
Triacylglycerol	Ethnicity	1	0.15	
Total cholesterol	Gender	1	0.09	
	VAT	2	0.14	0.05
HDLC	Ethnicity	1	0.10	
	VAT	2	0.12	0.02
TC/HDLC	Ethnicity	1	0.06	
	VAT	2	0.19	0.13
LDLC	Gender	1	0.05	
	VAT	2	0.11	0.06
ApoB	VAT	2	0.15	
Insulin	%BF	2	0.06	
Systolic BP	Gender	1	0.10	
	VAT	2	0.17	0.07

* Block 1 variables = demographics (age, gender, ethnicity). Potential Block 2 variables = fatness/fitness variables (VAT, %BF, $VO_{2\text{-}170}$).

(12,13,27), although several adult studies have reported significant VAT-systolic BP relationships (9,33,34).

Cardiovascular fitness (VO_{2-170}) did not prove to be an independent predictor of the IRS factors, and the only significant relationship among the simple correlations was the unexpected positive association between fitness and triacylglycerol. Comparative data from similar subjects are lacking (i.e., black and white obese adolescents). In our study of obese 7- to 11-year-old black and white youths in which we measured cardiovascular fitness during supine cycling, we did not find fitness to be associated with IRS risk (35). Some adult studies have reported a fitness-IRS risk factor relationship (10,11). However, in obese men and women, Goodpaster et al. (36) did not find fitness to be related to insulin sensitivity, although insulin sensitivity was associated with fatness measures such as VAT, total body fat mass, and subcutaneous abdominal fat. One of our previous studies used a wider distribution of adiposity, and presumably fitness, to study relations of fatness and fitness to IRS factors (37). In that study, both elevated %BF and lower cardiovascular fitness were significantly associated with unfavorable levels of lipids-lipoproteins and insulin; for example, the correlation coefficients with insulin were $r = 0.78$ and $r = 0.72$ for %BF and cardiovascular fitness, respectively. Thus, it seems that, within the somewhat restricted range of adiposity we had in the subjects of the current study, %BF and cardiovascular fitness lose some of their explanatory power, whereas VAT still is predictive of unfavorable levels of several variables. Thus, these and other studies of ours (35) support the notion that fatness better predicts markers of the IRS than does fitness.

For several of the IRS markers we saw no significant associations with the adiposity measures. We had anticipated a significant (inverse) correlation between VAT and LDL particle size based on observations in a subsample of 41 subjects from our study of 7- to 11-year-old obese children in whom these two variables were significantly related ($r = -0.32$, $p < 0.05$) (30). Likewise, a significant VAT-LDL particle size relationship has been observed in some adults (38,39). We pursued this matter further by combining the data from our study of 7- to 11-year-old obese children with the data of the obese adolescents in this study, to increase the sample size and statistical power. After controlling for age, we found that the correlation between LDL size and VAT for the combined samples resulted in a nonsignificant partial r of -0.08 ($p = 0.40$).

Further studies will be needed to clarify if LDL size is significantly associated with adiposity in young people.

Clearly, some of the deleterious relationships between VAT and markers of the IRS as seen in adults were already present in this group of obese young people. Information regarding interventions that target VAT in children and adolescents are just beginning to emerge. In our recent study of 7- to 11-year-old obese black and white children, we found that children who engaged in 4 months of controlled aerobic training accumulated significantly less VAT than did the control group children who did not participate in the training (40). We also found that levels of some IRS markers (e.g., triacylglycerol, insulin) declined more during periods of physical training than during periods of no physical training (41). No evidence is available in obese teenagers concerning the influence of exercise and/or dietary interventions on VAT.

In conclusion, in this sample of obese black and white teenagers, we found VAT to be the most powerful of the adiposity measures for explaining the variance in blood lipid-lipoprotein levels; ethnicity was the most powerful of the demographic predictors for these measures with whites having less favorable levels of some of the risk factors. VAT was less informative for explaining the variance among the nonlipid IRS markers. Given the increased prevalence of obesity in this age group, the pursuit of effective obesity treatment and prevention strategies should be given a high public health priority.

Acknowledgments

Support for this work was provided by Grant HL55564 from the National Heart, Lung, and Blood Institute.

References

1. **Kissebah AH, Freedman DS, Peiris AN.** Health risks of obesity. *Med Clin N Am.* 1989;73:111–38.
2. **Gillum RF.** The association of body fat distribution with hypertension, hypertensive heart disease, coronary heart disease, diabetes and cardiovascular risk factors in men and women aged 18–79 years. *J Chronic Dis.* 1987;40:421–8.
3. **Haffner SM, Fong D, Hazuda HP, Pugh JA, Patterson JK.** Hyperinsulinemia, upper body adiposity, and cardiovascular risk factors in non-diabetics. *Metabolism.* 1988;37:338–45.
4. **Thompson CJ, Ryu JE, Craven TE, Kahl FR, Crouse JR III.** Central adipose distribution is related to coronary atherosclerosis. *Arterioscler Thromb.* 1991;11:327–33.
5. **Després JP, Allard C, Tremblay A, Talbot J, Bouchard C.** Evidence for a regional component of body fatness in the association with serum lipids in men and women. *Metabolism.* 1985;34:967–73.
6. **Després JP, Moorjani S, Ferland M, et al.** Adipose tissue distribution and plasma lipoprotein levels in obese women. Importance of intra-abdominal fat. *Arteriosclerosis.* 1989;9:203–10.
7. **Pouliot MC, Després JP, Nadeau A, et al.** Visceral obesity in men. Associations with glucose tolerance, plasma insulin, and lipoprotein levels. *Diabetes.* 1992;41:826–34.
8. **Rissanen J, Hudson R, Ross R.** Visceral adiposity, androgens, and plasma lipids in obese men. *Metabolism.* 1994;43: 1318–23.
9. **Hunter GR, Kekes-Szabo T, Snyder SW, Nicholson C, Nyikos I, Berland L.** Fat distribution, physical activity, and cardiovascular risk factors. *Med Sci Sports Exerc.* 1997;29:362–9.
10. **Nyholm B, Mengel A, Nielsen S, et al.** Insulin resistance in relatives of NIDDM patients: the role of physical fitness and muscle metabolism. *Diabetologia.* 1996;39:813–22.
11. **Endre T, Mattiasson I, Hulthen UL, Lindgarde F, Berglund G.** Insulin resistance is coupled to low physical fitness in normotensive men with a family history of hypertension. *J Hypertens.* 1994;12:81–8.
12. **Brambilla P, Manzoni P, Sironi, et al.** Peripheral and abdominal adiposity in childhood obesity. *Int J Obes.* 1994;18: 795–800.
13. **Caprio S, Hyman LD, McCarthy S, Lange R, Bronson M, Tamborlane WV.** Fat distribution and cardiovascular risk factors in obese adolescent girls: importance of the intraabdominal fat depot. *Am J Clin Nutr.* 1996;64:12–7.
14. **Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL.** Overweight prevalence and trends for children and adolescents. The National Health and Nutrition Examination Surveys, 1963 to 1991. *Arch Pediatr Adolesc Med.* 1995; 149:1085–91.
15. **Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T.** Do obese children become obese adults? A review of the literature. *Prev Med.* 1993;22:167–77.
16. **Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM.** The predictive value of childhood body mass index values for overweight at age 35 y. *Am J Clin Nutr.* 1994;59:810–19.
17. **Nieto FJ, Szklo M, Comstock GW.** Childhood weight and growth rate as predictors of adult mortality. *Am J Epidemiol.* 1992;136:201–13.
18. **Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH.** Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med.* 1992;327:1350–5.
19. **Must A, Dallal GE, Dietz WH.** Reference data for obesity: 85th and 95th percentiles of body mass index (wt/ht^2) and triceps skinfold thickness [published erratum appears in *Am J Clin Nutr.*] *Am J Clin Nutr.* 1991;53:839–46.
20. **Ross R, Leger L, Morris D, de Guise J, Guardo R.** Quantification of adipose tissue by MRI: relationship with anthropometric variables. *J Appl Physiol.* 1992;72:787–95.
21. **Svendsen OL, Harbo J, Hassager C, Christiansen C.** Accuracy of measurements by dual-energy x-ray absorptiometry in vivo. *Am J Clin Nutr.* 1993;57:605–8.
22. **Gutin B, Litaker M, Islam S, Manos T, Smith C, Treiber F.** Body-composition measurement in 9–11-y-old children by dual-energy X-ray absorptiometry, skinfold-thickness measurements, and bioimpedance analysis. *Am J Clin Nutr.* 1996; 63:287–92.
23. **Owens S, Gutin B.** Exercise testing of the child with obesity. *Pediatr Cardiol.* 1999;20:79–83.
24. **Li X, Innis-Whitehouse W, Brown W, Le N.** Protocol for the preparation of a segmental linear polyacrylamide gradient gel: simultaneous determination of Lp(a), LDL, and HDL particle sizes. *J Lipid Res.* 1997;38:2603–14.

25. **Trieber FA, McCaffrey F, Pflieger K, Raunikar RA, Strong WB, Davis H.** Determinants of left ventricular mass in normotensive children. *Am J Hypertens.* 1993;6:505–13.
26. **Ku C-Y, Gower BA, Nagy TR, Goran MI.** Relationships between dietary fat, body fat, and serum lipid profile in prepubertal children. *Obes Res.* 1998;6:400–7.
27. **Yanovski JA, Yanovski SZ, Filmer KM, et al.** Differences in body-composition of black and white girls. *Am J Clin Nutr.* 1996;64:833–9.
28. **Conway JM, Yanovski SZ, Avila NA, Hubbard VS.** Visceral adipose tissue differences in black and white women. *Am J Clin Nutr.* 1995;61:765–71.
29. **Hill JO, Sidney S, Lewis CE, Tolan K, Scherzinger AL, Stamm ER.** Racial differences in amounts of visceral adipose tissue in young adults: the CARDIA (Coronary Artery Risk Development in Young Adults) study. *Am J Clin Nutr.* 1999; 69:381–7.
30. **Owens S, Gutin B, Ferguson M, Allison J, Karp W, Le N-A.** Visceral adipose tissue and cardiovascular risk factors in obese children. *J Pediatr.* 1998;133:41–5.
31. **Albu JB, Murphy L, Frager DH, Johnson JA, Pi-Sunyer FX.** Visceral fat and race-dependent health risks in obese nondiabetic premenopausal women. *Diabetes.* 1997;46: 456–62.
32. **Gower BA, Nagy TR, Trowbridge CA, Dezenberg C, Goran MI.** Fat distribution and insulin response in prepubertal African American and white children. *Am J Clin Nutr.* 1998;67:821–7.
33. **Cigolini M, Targher G, Bergamo Andreis IA, Tonoli M, Agostino G, De Sandre G.** Visceral fat accumulation and its relation to plasma hemostatic factors in healthy men. *Arterioscler Thromb Vasc Biol.* 1996;16:368–74.
34. **Peiris AN, Sothmann MS, Hoffmann RG, et al.** Adiposity, fat distribution, and cardiovascular risk. *Ann Intern Med.* 1989;110:867–72.
35. **Gutin B, Owens S, Treiber F, Islam S, Karp W, Slavens G.** Weight-independent cardiovascular fitness and coronary risk factors. *Arch Pediatr Adolesc Med.* 1997;151:462–5.
36. **Goodpaster BH, Thaete FL, Simoneau JA, Kelley DE.** Subcutaneous abdominal fat and thigh muscle composition predict insulin sensitivity independently of visceral fat. *Diabetes.* 1997;46:1579–85.
37. **Gutin B, Islam S, Manos T, Cucuzzo N, Smith C, Stachura ME.** Relation of percentage of body fat and maximal aerobic capacity to risk factors for atherosclerosis and diabetes in black and white seven- to eleven-old children. *J Pediatr.* 1994;125:847–52.
38. **Kissebah AH, Krakower GR.** Regional adiposity and morbidity. *Physiol Rev.* 1994;74:761–811.
39. **Despres JP, Moorjani S, Lupien PJ, Tremblay A, Nadeau A, Bouchard C.** Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. *Arteriosclerosis.* 1990;10:497–511.
40. **Owens S, Gutin B, Allison J, et al.** Effect of physical training on total and visceral fat in obese children. *Med Sci Sports Exerc.* 1999;31:143–8.
41. **Gutin B, Owens S.** Role of exercise intervention in improving body fat distribution and risk profile in children. *Am J Hum Biol.* 1999;11:237–47.