Comparison of Regional Fat Distribution and Health Risk Factors in Middle-Aged White and African American Women: The Healthy Transitions Study

Jennifer C. Lovejoy, Steven R. Smith, and Jennifer C. Rood

Abstract

LOVEJOY, JENNIFER C., STEVEN R. SMITH, AND JENNIFER C. ROOD. Comparison of regional fat distribution and health risk factors in middle-aged white and African American women: The Healthy Transitions Study. *Obes Res.* 2001;9:10–16.

Objective: Both ethnicity and menopause appear to influence intra-abdominal fat distribution. This study evaluated intra-abdominal fat distribution and obesity-related health risks in perimenopausal white and African American women.

Research Methods and Procedures: Baseline data from a longitudinal study of changes in body composition and energy balance during menopause are reported. Healthy women (55 African Americans and 103 whites) who were on no medication and had at least five menstrual cycles in the previous 6 months were recruited. Body composition was assessed by DXA, and visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) were assessed by computed tomography scan. SAT was divided into deep and superficial layers demarcated by the fascia superficialis.

Results: African American women were slightly younger (46.7 ± 0.2 vs. 47.7 ± 0.2 years, $p = 0.002$) and fatter (42.4% ± 1.0% vs. 39.4% ± 0.8% body fat, $p = 0.02$) than white women. In unadjusted data, African Americans had significantly more total abdominal fat and total, deep, and superficial SAT than whites. After adjustment for percent body fat and age, only total and superficial SAT remained significantly higher in African Americans. VAT, although slightly less in African American women, did not differ significantly by race. In multiple regression analysis, VAT was the strongest predictor of serum lipids, glucose, and insulin in women of both races, although superficial SAT was significantly associated with fasting glucose in whites.

Conclusions: Middle-aged African American women have larger SAT depots, adjusted for total body fatness, but do not differ from white women with regard to VAT. The complexity of the relationship between abdominal fat and metabolic risk is increased by ethnic differences in such associations.

Key words: menopause, ethnic differences, abdominal fat, heart disease risk, type 2 diabetes

Introduction

Menopause tends to be associated with increases in body weight (1,2) and a shift to abdominal fat distribution (3–6). Alterations in circulating hormone levels appear to regulate the shift in fat distribution because exogenous estrogen replacement therapy reduces abdominal adiposity in postmenopausal women (7). Increases in body weight at menopause appear to be less directly regulated by estrogen, although hormonally induced changes in energy expenditure and/or dietary intake patterns may play a role in weight gain at menopause (2).

Several reports indicate ethnic differences in intra-abdominal fat distribution in reproductive age women, although most of these investigations had small subject numbers (8–10). In these studies, African American women consistently had less abdominal visceral adipose tissue (VAT) and more abdominal subcutaneous adipose tissue (SAT) than did white women after adjusting for total body fatness. In a larger cohort from the Coronary Artery Risk Development in Young Adults (CARDIA) study, Hill et al.

Submitted for publication May 1, 2000.
Accepted for publication in final form September 5, 2000.
Pennington Biomedical Research Center, Louisiana State University, Baton Rouge, Louisiana.
Address correspondence to Jennifer C. Lovejoy, Women's Nutrition Research Program, Pennington Biomedical Research Center, 6400 Perkins Road, Baton Rouge, LA 70808.
E-mail: lovejoj@pbrc.edu

(11) reported less VAT in African American men but no difference in VAT between African American and white women, after adjustment for total body fat. Because of the suggested importance of VAT for determining cardiovascular and diabetes risk (12,13), understanding race differences in intra-abdominal fat distribution and its relation to health risk are critical.

Recently, evidence regarding two distinct layers of abdominal SAT has been presented, suggesting that the deep layer of SAT (DSAT), similar to VAT, is associated with adverse health consequences (14,15). Although the importance of VAT in mediating the health risks of obesity is generally well accepted, several authors have suggested that SAT may also play an important role (9,16,17). The DSAT layer may mediate the metabolic role of SAT; however, more research is needed to determine the importance of SAT and its individual anatomic layers in obesity-related pathology.

To our knowledge, there have been no studies of ethnic differences in intra-abdominal fat in women in the menopausal transition period. Studies showing increases in intra-abdominal fat with menopause have been almost exclusively conducted in white populations. Because African American women are more likely to be obese and have greater risk for development of chronic diseases (e.g., diabetes and heart disease) later in life, it is important to study the impact of menopause on fat distribution in this population. Therefore, we designed a longitudinal study, the Healthy Transitions Study, to examine changes in body composition and energy balance in healthy African American and white women. Baseline data on intra-abdominal fat distribution and health risk in this population are discussed in the present report.

Research Methods and Procedures

Subjects

Subjects were recruited by advertisement and word of mouth from the Baton Rouge, Louisiana area. To be eligible for the study, subjects had to be healthy, ≥43 years of age, and have had at least five menstrual periods in the 6 months before screening. All potential volunteers underwent a three-step screening process that included measures of blood chemistry and lipids, a physical examination, and a psychological interview to determine their ability to complete a 4-year longitudinal study. Women were excluded if they were taking regular medication (including hormones), were not having regular menstrual cycles, or had clinically abnormal results on laboratory tests or physical examination. If the volunteer was found to be eligible, she signed an informed consent form and completed baseline assessments (discussed below). A total of 55 African American women and 103 white women were included. The Louisiana State University Institutional Review Board approved the protocol and the informed consent form.

Body Composition and Fat Distribution

Height and weight were assessed in overnight fasted subjects wearing a hospital gown. Body composition (fat and lean mass) was determined by DXA (Hologic QDR2000, Waltham, MA). In addition, all subjects had an abdominal computed tomography scan at the level of the interspace between the fourth and fifth lumbar vertebrae (10-mm thick) for determination of abdominal fat distribution (GE High Speed Advantage; GE Medical Systems, Milwaukee, WI). Images were stored on digital tape for analysis at the Pennington Center using the Analyze software package (CNSoftware, Rochester, MN) run on a Sun Sparc 20 workstation (Sun Microsoft, San Jose, CA). The software allows for segmentation of sequential images into adipose and nonadipose tissue pixel values (Houndsfield units [HU]). The adipose tissue pixel values for each subject were determined using a histogram sampling technique. This results in HU values that are most appropriate for each person and decreases the error due to volume averaging and scanner drift over time. Overall, differences between individuals are ~3 to 5 HU. HUs for the upper and lower boundary were approximately −30 to −190, respectively.

Total abdominal adipose tissue (TAT) was defined as the sum of adipose tissue pixels inside a line tracing of the skin. VAT was segmented by drawing a line around the interior of the peritoneal cavity and summing all adipose tissue pixels within this area. The difference between TAT and VAT was considered to represent SAT. DSAT was measured as the area between the clearly demarcated circumferential fascia superficialis and the abdominal muscle wall. Superficial SAT (SSAT) was measured as the area between the fascia superficialis and the skin. A single reader performed all image analysis. The coefficient of variation (CV) for measures of VAT and DSAT in our laboratory was 10.5% and 9.8%, respectively (repeat measures on the same subjects made 1 to 2 weeks apart).

Health Risk Factors

Risk factors for heart disease that were measured included supine blood pressure (systolic and diastolic) and serum lipids and lipoproteins (total cholesterol, triglycerides, high-density lipoprotein–cholesterol [HDL-C], and low-density lipoprotein–cholesterol [LDL-C]). Diabetes risk factors that were measured included serum glucose and insulin. The blood pressure measurement was made in triplicate after a 10-minute rest period in the supine position. Arm circumference was measured and an appropriately

sized blood pressure cuff used. All blood samples were collected in the overnight fasted state.

Laboratory Methods

Total cholesterol, HDL-C, and triglycerides were measured on the Beckman Synchron CX5 autoanalyzer (Beckman, Brea, CA). The dextran sulfate precipitation method was used for the HDL-C measurement. LDL-C was calculated using the Friedewald equation, assuming triglycerides were within normal limits. Glucose was determined using the glucose oxidase method on a Beckman Synchron CX7 instrument. Insulin concentrations were determined using a microparticle enzyme immunoassay on an Abbott IMx analyzer (Abbott Laboratories, Abbott Park, IL). This assay has <1% cross-reactivity with proinsulin. Between-run CVs for all assays except the insulin one were <2.5%; the insulin assay had a CV of 6.6%. The Pennington clinical chemistry laboratory is lipid-certified by the Centers for Disease Control Lipid Standardization Program and is certified on all tests through the College of American Pathologists.

Data Analysis

Data were analyzed with an SAS version 6.12 (SAS Institute, Cary, NC) run on a personal computer. Descriptive statistics were calculated for each variable and normality was assessed. Variables that were not normally distributed were log-transformed before analysis. The General Linear Models procedure was used to assess differences between African American and white women, adjusted as needed for covariates. Correlation analyses were performed to determine the relationship between intra-abdominal fat depots and health risk factors. Multiple regression analysis using the R^2 selection procedure was also performed to examine the relative importance of the different abdominal fat depots on health risk factors. A p value of 0.05 was considered significant.

Results

Characteristics of the population are shown in Table 1. African American women were slightly but significantly younger than white women. The African American women had a significantly higher body mass index, body fat mass, lean mass, and percentage body fat than did the white women. There were no significant differences in serum lipids, lipoproteins, or glucose between groups. Fasting insulin and systolic and diastolic blood pressure were significantly higher in the African American women, consistent with greater insulin resistance in this population (Table 1).

Abdominal fat distribution in women of both races is shown in Table 2. When the data were not adjusted for body composition or age, African American women had significantly higher total abdominal fat by computed tomography scan and higher SAT in both deep and superficial layers than did white women. There was no race difference in VAT in the unadjusted data.

Table 1. Characteristics of 103 white and 55 African American women

	African American	White
Age (years)	46.7 ± 0.24 (44–51)	47.7 ± 0.21* (43–56)
Body mass index (kg/m^2)	28.8 ± 0.79 (18.5–47.5)	25.3 ± 0.49* (18.9–36.3)
Fat mass (kg)	33.3 ± 1.6 (13–71.9)	27.3 ± 1.0* (10.5–56.4)
Lean mass (kg)	40.8 ± 0.8 (29.7–56.7)	37.8 ± 0.4* (27.2–51.5)
Percent body fat (%)	42.4 ± 1.0 (24.2–55.9)	39.4 ± 0.8* (20.0–57.0)
Cholesterol (mg/dL)	204.4 ± 6.4 (126–369)	193.2 ± 3.2 (112–335)
LDL-C (mg/dL)	128.5 ± 5.7 (48.8–257.1)	117.5 ± 2.7 (58–209)
HDL-C (mg/dL)	57.9 ± 1.7 (31.5–101.6)	57.6 ± 1.3 (31.9–100.7)
Glucose (mg/dL)	95.6 ± 1.2 (80–125.5)	92.9 ± 0.8 (74–123)
Insulin (μU/mL)	10.4 ± 0.9 (2.2–40)	6.1 ± 0.3* (1.3–20.7)
Diastolic blood pressure (mm Hg)	79.6 ± 1.3 (65–118)	76.0 ± 0.7* (58–101)
Systolic blood pressure (mm Hg)	125.5 ± 2.4 (88–171)	119.4 ± 1.2* (96–157)

Data are mean ± SEM with ranges in parentheses.
* Significant differences between race groups by t test adjusted for equal or unequal variance as appropriate.

Data were also analyzed with adjustment for total body fatness (percent fat from DXA) and age as covariates (Table 2). The effect of this adjustment was that there was no longer a significant race difference in total abdominal fat or DSAT; however, SAT and SSAT fat remained significantly higher in African American women. There was a slight, nonsignificant trend for African American women to have less VAT after adjustment for total body fat and age.

Correlations between intra-abdominal fat and health risk (Table 3) were generally similar in women of both races. However, correlations (r value or R^2) between abdominal fat measures and total cholesterol, LDL-C, and fasting insulin tended to be larger in white women.

Table 2. Abdominal fat measures in 103 white and 55 African American women (mean ± SEM)

	African American	White
Total abdominal fat (cm^2)		
Unadjusted	466.1 ± 23.1	387.9 ± 16.4†
Adjusted*	431.7 ± 12.4	405.2 ± 8.7
Visceral fat (cm^2)		
Unadjusted	98.2 ± 11.6	96.0 ± 6.0
Adjusted	89.2 ± 7.2	100.6 ± 5.1
Subcutaneous fat (cm^2)		
Unadjusted	373.1 ± 17.7	295.6 ± 12.6†
Adjusted	347.5 ± 9.6	308.5 ± 6.7†
Deep subcutaneous fat (cm^2)		
Unadjusted	183.9 ± 9.5	156.1 ± 6.8†
Adjusted	172.0 ± 5.9	162.1 ± 4.1
Superficial subcutaneous fat (cm^2)		
Unadjusted	182.7 ± 9.5	134.4 ± 6.8†
Adjusted	167.4 ± 5.8	142.2 ± 4.1†

* Means adjusted for percent body fat and age.
† $p < 0.05$ between groups.

whereas correlations between abdominal fat measures and HDL-C and fasting glucose were greater in the African American group. Furthermore, DSAT was more strongly correlated with several risk factors, including total cholesterol and LDL-C, than was VAT in one or both ethnic groups.

Multiple regression analysis was performed to examine the relative contribution of VAT, DSAT, and SSAT to variation in health risk factors after forcing total percent body fat from DXA into the model (Table 4). In both African American and white women, VAT was the strongest independent predictor of most risk factors. The exception was fasting glucose in white women, for which SSAT was the strongest predictor, although only a small proportion of the variance in glucose was explained by any body fat variable. There was a significant interaction between race and SSAT for fasting glucose ($p = 0.05$), suggesting that the relationship between glucose and SSAT differs significantly depending on ethnicity.

When the regression model was run without forcing body fat into the model (data not shown), VAT was the strongest independent predictor of cholesterol, glucose, and insulin in African Americans; SSAT was the strongest predictor of both LDL-C and HDL-C in this group. In the white women, VAT was the strongest independent predictor of LDL-C and HDL-C and fasting insulin; DSAT was the strongest predictor of total cholesterol; and SSAT was the strongest predictor of fasting glucose.

Table 3. Correlation coefficients between intra-abdominal fat and metabolic risk factors in 55 African American and 103 white women (values are Pearson r values)

	TAT	VAT	SAT	DSAT	SSAT
Cholesterol					
African American	0.24	0.22	0.22	0.19	0.21
White	0.31*	0.32*	0.36*	0.40*	0.30*
HDL-C					
African American	−0.37*	−0.35*	−0.32*	−0.27	−0.38*
White	−0.33*	−0.33*	−0.23*	−0.20*	−0.22*
LDL-C					
African American	0.29*	0.24	0.28	0.24	0.26
White	0.36*	0.34*	0.37*	0.42*	0.32*
Log insulin					
African American	0.47*	0.41*	0.49*	0.39*	0.42*
White	0.63*	0.57*	0.56*	0.54*	0.51*
Glucose					
African American	0.38*	0.41*	0.31*	0.37*	0.23
White	0.20	0.14	0.23*	0.17	0.24*

* $p < 0.05$.

Table 4. Results of multivariate regression (R^2) assessing the relationship of abdominal fat compartments to health risk factors in 55 African American and 103 white women

	African American		White	
Total cholesterol	Model 1: VAT	$R^2 = 0.09$	Model 1: VAT	$R^2 = 0.19$
	Model 2: VAT, SSAT	$R^2 = 0.09$	Model 2: VAT, DSAT	$R^2 = 0.19$
	Model 3: VAT, SSAT, DSAT	$R^2 = 0.09$	Model 3: VAT, SSAT, DSAT	$R^2 = 0.20$
LDL-C	Model 1: VAT	$R^2 = 0.14$	Model 1: VAT	$R^2 = 0.27$
	Model 2: VAT, SSAT	$R^2 = 0.14$	Model 2: VAT, SSAT	$R^2 = 0.28$
	Model 3: VAT, SSAT, DSAT	$R^2 = 0.14$	Model 3: VAT, SSAT, DSAT	$R^2 = 0.28$
HDL-C	Model 1: VAT	$R^2 = 0.25$	Model 1: VAT	$R^2 = 0.14$
	Model 2: VAT, DSAT	$R^2 = 0.27$	Model 2: VAT, DSAT	$R^2 = 0.18$
	Model 3: VAT, DSAT, SSAT	$R^2 = 0.27$	Model 3: VAT, SSAT, DSAT	$R^2 = 0.18$
Fasting glucose	Model 1: VAT	$R^2 = 0.20$	Model 1: SSAT	$R^2 = 0.06$
	Model 2: VAT, SSAT	$R^2 = 0.23$	Model 2: SSAT, VAT	$R^2 = 0.07$
	Model 3: VAT, SSAT, DSAT	$R^2 = 0.25$	Model 3: VAT, SSAT, DSAT	$R^2 = 0.08$
Log fasting insulin	Model 1: VAT	$R^2 = 0.27$	Model 1: VAT	$R^2 = 0.40$
	Model 2: VAT, DSAT	$R^2 = 0.28$	Model 2: VAT, SSAT	$R^2 = 0.41$
	Model 3: VAT, SSAT, DSAT	$R^2 = 0.28$	Model 3: VAT, SSAT, DSAT	$R^2 = 0.41$

R^2 values are presented for each model after percent body fat from DXA is forced into the model.

Discussion

Results of the present study show that middle-aged, premenopausal African American women have greater amounts of total abdominal fat than white women, after adjustment for total body fatness, and that the difference is due to larger subcutaneous fat depots. VAT was generally the strongest predictor of cardiovascular and metabolic risk factors in both the white and African American women, after adjustment for total fatness, although SSAT was the strongest predictor of fasting glucose in the white women.

Several small studies have suggested that African American women have less VAT than age- and weight-matched white women (8–10), although a study in the larger CARDIA cohort did not find race differences in visceral fat in young women (11). Our results confirm the data from CARDIA in a population of middle-aged premenopausal women, although the present study did show a slight, nonsignificant trend toward African American women having less VAT after adjustment for total fat (Table 2). African American women had significantly greater amounts of SAT even after adjustment for total body adiposity, as we (9) and others (11) have reported previously.

In general, research has supported a strong relationship between VAT and health risk, although most studies of this question have been performed in whites (12,13). The role of VAT in metabolic and cardiovascular risk has been hypothesized to relate to the impact of metabolic products from the adipocytes draining into the portal circulation. For example, regional differences in adipose tissue lipolysis resulting in increased free fatty acid levels in the portal system have been implicated in the disordered insulin and triglyceride metabolism of abdominal obesity (18,19).

Despite the apparent importance of VAT in metabolic disorders of obesity, several groups have argued the potential importance of abdominal SAT (16,17). Recently, our group (14) and Kelley et al. (15) have recognized the importance of subdividing abdominal SAT into two layers, deep and superficial. We have observed that the DSAT layer is similar to VAT in terms of its relationship to health risk factors (20), although these previous investigations used primarily white populations. The current results suggest that VAT is generally the best correlate of health risk factors in both whites and African Americans, although SSAT was a strong independent predictor of glucose in whites. In the present study, DSAT was an element of the best predictive model for HDL-C in women of both races and of fasting insulin in African Americans but did not appear to have predictive value as a single variable once body fat was included in the model.

In our previous study of fat distribution and health risk, we found that VAT was similarly correlated with most health risk factors in African American and white women but that SAT was more strongly correlated with insulin resistance in the African American women (9). We did not separate DSAT and SSAT in our previous study, so we speculate that some of the strength of the association between total SAT and health risk factors might disappear if DSAT was taken into account. In the present study, we

observed a slightly larger correlation between SAT and fasting insulin (r = 0.49) than between VAT and fasting insulin (r = 0.41) in African Americans; however, neither DSAT nor SSAT seemed strikingly correlated with insulin (Table 3).

The possible different roles of the deep and superficial depots of the subcutaneous fat layer are not completely clear. In a porcine model of adipose tissue development, it was shown that the superficial adipose tissue has a different embryonic origin than deep adipose tissue (perifollicular stromal cells vs. mesenchyme). Mersmann and Leymaster (21) have proposed that SSAT plays a thermoinsulatory role, whereas the deeper layer may play a more metabolic role. Several other studies in the pig support differences between deep and superficial adipose tissue in lipogenic enzyme activity (22,23). Although there have been few studies in humans addressing these questions, the porcine data support the idea that the two subcutaneous adipose depots may be distinct metabolically as well as anatomically.

A variety of factors other than ethnicity are involved in regional fat distribution. Gender, total body fatness, and age are known to be determinants of VAT accumulation (24). Hormonal factors are also critical. Previous studies have shown that changes in circulating estrogen at menopause are likely to play a critical role in the redistribution of body fat, with estrogen-replacement therapy preventing or ameliorating the shift in body fat at menopause (7). Other hormones, including ovarian and adrenal androgens and cortisol, and sex hormone-binding globulin are also associated with increased abdominal obesity (24).

Poehlman et al. (2) have shown longitudinally that changes in diet and physical activity, as well as resting energy expenditure, occur during the menopause transition, although it is likely that these environmental factors influence total body weight to a greater extent than fat distribution per se. Ethnic differences in dietary intake, physical activity, basal metabolism, and certain hormones (including androgens) have been reported by a number of investigators. Our ongoing longitudinal study of this cohort as they move through the menopausal transition will address how each of these factors influences menopause-related accumulations of total and abdominal fat.

In summary, the present study shows that middle-aged, premenopausal African American women have larger SAT depots than white women after adjustment for differences in total body fatness. Abdominal fat measures in general are correlated with metabolic and cardiovascular risk factors in women of both ethnic groups, as expected, with VAT generally the strongest predictor of risk factors. However, SSAT and DSAT both were strong predictors for certain dependent variables in both ethnic groups. This confirms the importance of analyzing the two anatomical layers of abdominal SAT separately to assess the role of abdominal fat in health risk. Taken together, these results suggest that the relationships between adipose tissue compartments and metabolic risk are complex, involving depot, gender, and ethnic differences in effects. Further studies comparing men and women, as well as individuals from different ethnic groups, are needed to understand the complexity of the relationships between specific intra-abdominal fat depots and the health risks associated with obesity.

Acknowledgments

This work was supported by National Institutes of Health Grant R01-DK50736A (to J.C.L.). We thank the volunteers who participated in this study, and the staff of the outpatient clinic at Pennington Biomedical Research Center for their assistance. In particular, Mark Klemperer, the study coordinator, and Julia Wright, who performed the computed tomography scan analysis, are thanked for their contribution.

References

1. **Wing RR, Matthews KA, Kuller LH, Meilahn EN, Plantinga P.** Waist to hip ratio in middle-aged women: associations with behavioral and psychosocial factors and with changes in cardiovascular risk factors. *Arterioscler Thromb.* 1991;11:1250–7.
2. **Poehlman ET, Toth MJ, Gardner AW.** Changes in energy balance and body composition at menopause: a controlled longitudinal study. *Ann Intern Med.* 1995; 123:673–5.
3. **Toth MJ, Tchernof A, Sites CK, Poehlman ET.** Effect of menopausal status on body composition and abdominal fat distribution. *Int J Obes.* 2000;24:226–31.
4. **Svendsen OL, Hassager C, Christiansen C.** Age- and menopause-associated variations in body composition and fat distribution in healthy women as measured by dual-energy X-ray absorptiometry. *Metabolism.* 1995;44:369–73.
5. **Kotani K, Tokunaga K, Fujioka S, et al.** Sexual dimorphism of age-related changes in whole-body fat distribution in the obese. *Int J Obes.* 1994;18:207–12.
6. **Zamboni M, Armellini F, Milani MP, et al.** Body fat distribution in pre- and post-menopausal women: metabolic and anthropometric variables and their inter-relationships. *Int J Obes.* 1992;16:495–504.
7. **Haarbo J, Marslew U, Gotfredsen A, Christiansen C.** Postmenopausal hormone replacement therapy prevents central distribution of body fat after menopause. *Metabolism.* 1991; 40:1323–6.
8. **Conway JM, Yanovski SZ, Avila NA, et al.** Visceral adipose tissue differences in black and white women. *Am J Clin Nutr.* 1995;61:765–71.
9. **Lovejoy JC, de la Bretonne JA, Klemperer M, Tulley R.** Abdominal fat distribution and metabolic risk factors: effects of race. *Metabolism.* 1996;45:1119–24.

10. **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.
11. **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.
12. **Despres JP, Moorjani S, Lupien PJ, Tremblay A, Madeau A, Bouchard C.** Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. *Arteriosclerosis.* 1990;100:497–511.
13. **Kissebah AH, Krakower GR.** Regional adiposity and morbidity. *Physiol Rev.* 1994;74:761–811.
14. **Smith SR.** Regional fat distribution. In: Bray GA, Ryan DH, eds. *Nutrition Genetics and Obesity.* Vol. 9. Baton Rouge, LA: Louisiana State University Press; 1999, pp. 433–58.
15. **Kelley DE, Thaete FL, Troost F, Heuwe T, Goodpaster BH.** Subdivisions of subcutaneous abdominal adipose tissue and insulin resistance. *Am J Physiol.* 2000;278:E941–E948.
16. **Goodpaster BH, Thaete FL, Simoneau J-A, Kelley DE.** Subcutaneous abdominal fat and thigh muscle composition predict insulin sensitivity independently of visceral fat. *Diabetes.* 1997;46:1579–85.
17. **Abate N, Garg A, Peshock RM, Stray-Gunderson J, Grundy SM.** Relationships of generalized and regional adiposity to insulin sensitivity in men. *J Clin Invest.* 1995;96:88–98.
18. **Arner P.** Role of antilipolytic mechanisms in adipose tissue distribution and function in man. *Acta Med Scand Suppl.* 1988;723:147–52.
19. **Mauriege P, Despres JP, Marcotte M, et al.** Abdominal fat cell lipolysis, body fat distribution, and metabolic variables in premenopausal women. *J Clin Endocrinol Metab.* 1990;71: 1028–35.
20. **Smith SR, Lovejoy JC, Greenway F, et al.** Contributions of total body fat, abdominal subcutaneous adipose tissue compartments, and visceral adipose tissue to the metabolic complications of obesity. *Metabolism.* 2001. (in press).
21. **Mersmann HJ, Leymaster KA.** Differential deposition and utilization of backfat layers in swine. *Growth.* 1984;48: 321–30.
22. **Anderson DB, Kauffman RG.** Cellular and enzymatic changes in porcine adipose tissue during growth. *J Lipid Res.* 1973;14:160–8.
23. **Hood RL, Allen CE.** Lipogenic enzyme activity in adipose tissue during the growth of swine with different propensities to fatten. *J Nutr.* 1973;103:353–62.
24. **Bjorntorp P.** Visceral obesity: a "civilization syndrome." *Obes Res.* 1993;1:206–22.