Racial Differences in Subcutaneous and Visceral Fat Distribution in Postmenopausal Black and White Women

J.A. Kanaley, I. Giannopoulou, G. Tillapaugh-Fay, J.S. Nappi, and L.L. Ploutz-Snyder

Most studies examining racial disparities in abdominal fat distribution have focused on premenopausal women. The purpose of this report was to determine if racial differences exist in the abdominal fat distribution in postmenopausal white and black women. Fifty-four women (33 white and 21 black) were scanned by magnetic resonance imaging (MRI) to determine abdominal fat distribution, were measured by hydrostatic weighing for percent body fat, and had their fasting blood lipids, glucose, and insulin levels measured. These women were matched for age (mean age, 53.5 ± 0.9 years) and percent body fat (black: 39.6% ± 2.3%, white: 37.3% ± 1.2%). When adjusted for total body fat mass and hormone replacement therapy (HRT), total abdominal fat (white: 10,352.1 ± 535.2, black: 11,220.4 ± 670.1 cm^3) was not statistically different between groups, but the visceral fat content was significantly higher in the white women (white: 2,943.5 ± 220.4, black: 2,332.6 ± 176.1 cm^3). The percent visceral fat was also higher in these women (white: 30.5% ± 1.3%, black: 22.1% ± 1.6%, $P < .01$). Subcutaneous adipose tissue (SAT) was significantly higher in the black women (white: 7,408.6 ± 450.2, black: 8,887 ± 563.1 cm^3, $P < .05$). No significant differences were found in the insulin concentrations or the blood lipid profile of these women. Regardless of race, visceral fat was a significant predictor of log triglyceride, low-density lipoprotein-cholesterol (LDL-C), cholesterol/LDL-C, insulin levels, and insulin resistance. Race was only found to contribute to 8% of the variability of LDL-C. HRT use had no effect on abdominal fat distribution or the blood lipid profile in this cohort of women. In conclusion, disparities in abdominal fat distribution between black and white women continue to exist in the early postmenopausal years, and the regression results indicate that the absolute amount of visceral fat, and not the relative amounts of visceral fat, is the best predictor of the blood lipid profile and insulin sensitivity. HRT use did not result in differences in abdominal fat distribution in these women. Factors, such as genetics and lifestyle, must play a larger role in explaining the increased health risk in black women.

EXCESSIVE VISCERAL adipose tissue (VAT) has been associated with insulin resistance, coronary artery disease, diabetes, and hypertension by numerous investigators,[1,2] yet the majority of the research studying this association has focused on white men and women. Epidemiologic studies indicate that ~30% of the black population are overweight by the end of the third decade of life, that these rates increase to 60% by 50 years of age, and that this population has a higher risk of hyperlipidemia, diabetes, and the metabolic syndrome compared with whites.[3,4] Research comparing black and white women has primarily focused on younger, premenopausal women (age ~35 years).[5-9] Premenopausal black women tend to accumulate abdominal fat in the subcutaneous region compared with the white population who accumulate abdominal fat in the visceral region.[6] Only one investigation has examined postmenopausal obese women and has reported similar findings.[10] Considering that obesity is more prevalent in black women, little research has been conducted in this older population to establish the racial differences in abdominal fat distribution and the relationship with the blood lipid profile and insulin concentrations.

In older women, confounding the problem of obesity is menopause. Menopause is associated with an increase in waist measurements and is suspected to cause a shifting of fat deposition.[11] Controversy exists whether changes in abdominal fat distribution occurs with menopause, with some cross-sectional studies indicating an increase in visceral fat content with menopause,[12] while others find no change in visceral fat content.[13] With the potential changes in abdominal fat distribution with menopause and/or aging, it is possible that the racial differences that exist in abdominal fat distribution in the premenopausal years may or may not exist when women are studied in the postmenopausal years. In addition, there is controversy whether hormone replacement therapy (HRT) prevents this shifting of body fat with menopause. Some studies using dual energy x-ray absorptiometry (DXA) or waist circumferences note an increase in central adiposity with menopause,[11,14-16] but there are a few recent studies using computed tomography, which indicate that women on HRT do not have lower levels of visceral or subcutaneous fat.[17,18]

The purpose of this study was to determine whether differences exist in VAT and subcutaneous adipose tissue (SAT) between postmenopausal black and white women. This study also examined whether total abdominal fat, VAT, and/or SAT predicted serum lipoproteins and calculated insulin resistance in older healthy postmenopausal black and white women. We hypothesized that disparities in abdominal fat distribution would be observed with white women having more visceral fat than black women. Further, we hypothesized that race would be a significant predictor of blood lipid levels and that blood lipids would be related to these differences in abdominal fat distribution.

MATERIALS AND METHODS

Subjects

Thirty-three white women and 21 black postmenopausal women, ranging in age from 50 to 65 years were recruited for this study. All

From the Department of Exercise Science, Syracuse University, Syracuse; and the Department of Radiology, Upstate Medical University, Syracuse, NY.

Submitted May 1, 2002; accepted August 15, 2002.

Supported in part by Grant No. RO3AG16370-01 from the National Institute on Aging.

Address reprint requests to J.A. Kanaley, PhD, 820 Comstock Ave, Rm 201, Syracuse, NY 13244.

0026-0495/03/5202-0004$35.00/0

doi:10.1053/meta.2003.50024

subjects signed a consent form that was approved by the Syracuse University and SUNY Upstate Medical University Institutional Review Board. The women had been postmenopausal for more than 1 year, but less than 7 years (mean, 4.61 ± 0.5 years; mean ± SE) and had been determined to be postmenopausal by their physicians by hormonal evaluation (follicle-stimulating hormone [FSH] > 50mIU/mL). Seventeen white and 8 black women were on HRT for at least 1 year (Premarin, 0.625 mg [Wyeth-Ayerst, Philadelphia, PA] or Premarin plus medroxyprogesterone acetate, 2.5 mg). Women were selected over a range of body mass index (BMI) (22 to 40 kg/m^2), were healthy, and free of known cardiovascular disease and other metabolic diseases. None of the women were taking medications affecting blood pressure, blood lipids, or glucose metabolism. All subjects were non-smokers, euthyroid, and were weight stable for at least 2 months before the study.

Experimental Design

All women were required to come for 2 visits to complete the testing. On the first visit, after a 12-hour overnight fast and a 24-hour period of no exercise, subjects had their fasting blood lipids, glucose and insulin concentrations, and body composition assessed. Health and physical activity questionnaires were completed on this visit. Three independent investigators evaluated the physical activity questionnaires following a procedure previously described.[13] On the second visit, the subjects were required to have an abdominal magnetic resonance imaging (MRI) scan; contiguous transaxial image slices covering from the midchest to the midthigh region. Body weight was taken at all visits to ensure that weight had not changed. The first and second visits were separated by approximately 1 to 2 weeks, which was dependent on the subject's schedule and MRI availability.

Methodology

Body composition and body fat distribution. Total body fat was determined using underwater weighing. Body density was measured with the underwater weight taken simultaneously with measurement of residual lung volume.[19] Percent body fat was calculated using age-specific equations.[20]

Regional fat distribution was quantified from the MRIs with techniques previously reported.[13] The MRIs were obtained (GE Signa 1.5 T MRI scanner; General Electric, Milwaukee, WI) using standard T1 weighted spin echo imaging with respiratory compensation. The MR spin echo pulse sequence parameters used time relaxation (TR) = 400 ms, time echo (TE) = 20 ms, field of view (FOV) = 40 to 48, acquisition matrix = 256 × 256, with nex = 2. Slice thickness was 1 cm, with approximately 40 consecutive slices/total scan acquired. Total abdominal fat was determined from the MRIs of the region beginning at the superior portion of the head of the femur and inclusive to the most superior portion of the kidneys. The area included for analysis represented about 25 cm in most subjects depending on their height. Visceral fat pixel intensity was segmented out at each MRI slice level and used to determine the total volume of fat for the slice. The total visceral fat volume subtracted from the total fat (VAT plus SAT) of that slice determined the total subcutaneous fat of that slice level. Total volume of abdominal fat was determined by adding together the1-cm slice volume measurements of all consecutive slices. The digital image data were analyzed using an automated pixels representing fat segmentation program running on a SUN workstation (SUN Microsystems, Santa Clara, CA) (note that there is still user 'bias' in determining the maximum thresholding to be accepted). Test-retest for this method was r = .9999 (P < .0001).[13]).

Analytical methods. The fasting blood sample was analyzed for triglycerides, total cholesterol, high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), insulin, and glucose levels. The blood lipids were analyzed by Quest Diagnostic (Syracuse, NY). Glucose concentrations were determined using a Yellow Springs Instrument (Yellow Springs, OH). Insulin levels were determined by enzyme-linked immunosorbent assay (ELISA) from Diagnostic Laboratories (Webster, TX).

Table 1. Subject Characteristics of All of the White and Black Women

	White (n = 33)	Black (n = 21)
Age (yr)	54.2 ± 1.6	52.3 ± 1.0
Height (m)	1.62 ± 0.01	1.63 ± 0.01
Weight (kg)	72.2 ± 3.6	83.1 ± 3.7*
BMI (kg/m^2)	28.0 ± 1.9	31.2 ± 1.3*
Percent fat	37.3 ± 1.2	39.6 ± 2.3
Fat mass (kg)	26.6 ± 1.5	33.7 ± 2.8*
Waist (cm)	95.9 ± 4.6	113.4 ± 2.5*
Years since menopause	4.2 ± 0.5	6.6 ± 1.2
Years of HRT use	4.3 ± 0.7	6.5 ± 1.9
Glucose (mg/dL)	82.3 ± 2.3	82.8 ± 1.7
Insulin (μU/mL)	10.5 ± 1.5	14.0 ± 2.9
HOMA-IR	2.1 ± 0.3	3.0 ± 0.7

NOTE. Data are mean ± SE.

Abbreviation: HOMA-IR, homeostasis model assessment of insulin resistance.

*P < .05 between groups.

Statistical analysis. The data were analyzed using Statistical Packages for the Social Sciences (SPSS, Chicago, IL, version 10.0) and are presented as means ± SE. The homeostasis model assessment of insulin resistance (HOMA-IR) was used to estimate insulin resistance from fasting glucose and insulin levels.[21] An analysis of covariance (ANCOVA) was used to determine if racial differences existed between the descriptive and abdominal fat variables, with HRT status and total body fatness as a covariate. Serum triglyceride levels were logarithmically transformed to yield a normal distribution before parametric analysis. Multiple regression analysis was used to predict blood lipids or insulin resistance from the following predictors: total abdominal fat, visceral fat, subcutaneous fat, percent visceral fat, total body fat, HRT status, physical activity, and race. All variables were entered into the equation simultaneously, and squared semipartial correlation coefficient (sr^2) values were calculated for each predictor to determine both the combined and independent contributions of each variable to blood lipids or insulin resistance.

RESULTS

The descriptive variables of these 2 groups of subjects are shown in Table 1. The subjects were similar in height and years since menopause and were well matched on percent body fat (black: 39.6% ± 2.3% *v* whites: 37.3% ± 1.2%, P < .05). The black women were slightly younger and heavier, had a higher BMI, and had a greater total body fat mass (P < .01) than the white women. The white women had a higher overall score on physical activity than the black women (3.0 ± 0.1 and 2.4 ± 0.2, respectively, P < .01), and higher physical activity levels were negatively correlated with lower levels of fat mass and total abdominal fat (r = -.42 and -.42, respectively, P < .01).

The black women had significantly greater total abdominal fat and subcutaneous fat than the white women (12,923.5 ± 1,029.9 and 10,280.0 ± 851.3 cm^3 *v* 9,234.4 ± 834.3 and 6,494.9 ± 689.6 cm^3; P < .01, respectively), while the size of the visceral fat depot was similar between groups (2,739.5 ± 207.6 *v* 2,643.5 ± 256.2 cm^3, respectively). Percent visceral fat

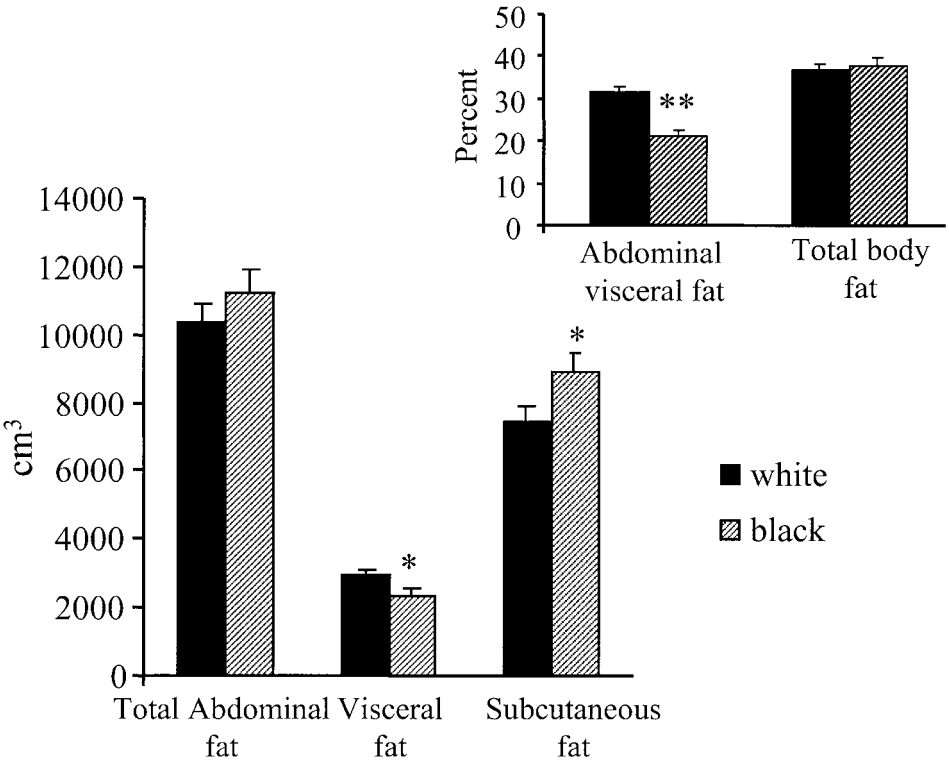

Fig 1. Comparison of abdominal fat measures adjusted for total body fat mass in black and white postmenopausal women. Inset: total body fat and percent visceral fat for each respective group. *$P < .05$, between groups, **$P < .01$ between groups.

(visceral fat/total abdominal fat) was significantly higher in the white women than in the black women (31.2% ± 1.4% and 21.0% ± 1.7%, respectively, $P < .01$). When adjusted for total body fat mass and HRT, total abdominal fat was not statistically different between groups, but the absolute amount of visceral fat content was now significantly higher in the white women. The SAT remained significantly higher in the black women ($P < .01$; Fig 1). When visceral fat was expressed relative to total abdominal fat mass, the white women had a higher percent visceral fat than black women ($P < .01$; inset Fig 1). The ratio of VAT to SAT was not different between groups. Women on HRT were not found to have higher total abdominal, visceral, or subcutaneous fat.

There were no significant differences in serum lipids between groups. Using multiple regression analysis, we determined whether abdominal fat distribution could be used to predict blood lipids, as well as glucose and insulin levels and insulin resistance (Table 2). VAT was the strongest predictor of log triglyceride, LDL-C levels, and the cholesterol/HDL-C ratio and accounted for 30%, 28%, and 34%, respectively, of the variability ($P < .001$). Although representing only a small percentage of the variability, race was found to be a significant predictor (8.7%, $P < .01$) only for LDL-C concentrations. Total body fat mass accounted for 18% of the variability of HDL-C concentrations, but none of the abdominal fat distribution measures were significant predictors of this variable. Physical activity and HRT were not found to be significant predictors of any of the blood lipid variables. Physical activity

Table 2. Regression Results for the Prediction of Blood Lipid and Insulin Measures Using Abdominal Fat Distribution, Race, and Total Body Fat Mass

Dependent Variable	Independent Variable	β	Beta	P	Multiple R	R^2	sr^2	Overall P
Log triglyceride	Visceral fat	0.047	0.747	.009	.551	.304	0.304	.001
HDL-C	Fat mass	−0.636	−0.428	.001	.428	.183	0.183	.001
LDL-C	Visceral fat	0.011	0.454	.000	.530	.281	0.205	.000
	Race	16.196	0.295	.019			0.087	
Cholesterol/HDL-C	Visceral fat	0.0004	0.571	.000	.580	.336	0.336	.001
Insulin	Visceral fat	0.0004	0.438	.002	.473	.224	0.224	.004
HOMA-IR	Visceral fat	0.0013	0.615	.000	.542	.294	0.250	.002

NOTE. Multiple linear regression was used for the above variables except the HOMA-IR, which was determined using block regression analysis.

Abbreviations: HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance.

probably played little role in predicting abdominal fat because all the women had a similar amount of physical activity.

Glucose concentrations were similar between the white and black women, while the insulin levels were slightly, but not significantly, higher in the black women than in the white women (Table 1). Regression analysis did not find any significant predictors for the glucose concentrations in this cohort of postmenopausal women. Race was not a significant predictor of insulin concentrations, while visceral fat accounted for 22% of the variability in the insulin levels ($P < .01$, Table 2). The initial regression analysis found visceral fat, race, and subcutaneous fat to be significant predictors of HOMA-IR; however, subcutaneous fat was found to be a suppressor variable.[22] To determine if subcutaneous fat artificially inflated the contribution of visceral fat and/or race in predicting HOMA-IR, we subsequently ran a block regression analysis with these variables to determine the independent contributions. The results of the block regression analysis showed that only visceral fat was a significant predictor of insulin resistance ($R^2 = .25$, $P < .01$). These results are found in Table 2.

DISCUSSION

Considerable research has been conducted establishing the relationship between abdominal fat distribution and health risks in white women. Overall, these findings have been generalized to all populations and have indicated that increases in abdominal fat, particularly visceral fat, results in increased health risks for a number of chronic diseases.[23,24] In recent years, studies have demonstrated differences in intrabdominal fat distribution between white and black premenopausal women, such that black women have less abdominal VAT and more abdominal SAT.[2,4-9,25] Although some earlier studies suggest visceral fat accumulates with menopause,[12] based on our previous work,[13] we hypothesized that racial differences in abdominal fat distribution would exist in postmenopausal women. When adjusted for total fat mass, we observed that black women had more subcutaneous fat, less visceral fat, and that the percent visceral fat was ~8% lower in the black women compared with the white women when adjusted for total fat mass. Although menopause has been associated with shifts in abdominal fat by some investigators,[11,12] which could potentially remove the racial disparities seen in abdominal fat distribution in premenopausal women, our study demonstrates that the disparities in abdominal fat distribution between black and white women still exist at least 5 years postmenopause. In addition, HRT use did not result in differences in abdominal fat distribution in either group.

Conway et al[6] demonstrated less VAT in premenopausal, black women compared with premenopausal, white women of comparable BMI and % body fat. In their younger population, they also noted that VAT expressed as a percentage of total abdominal adipose tissue was approximately 5% less in black women than in white women, but that SAT was 4% to 6% greater in black women. Other studies have had similar findings,[2,7] except for 1 study,[25] which reported no differences in VAT and SAT in young, premenopausal, obese black and white women. In our postmenopausal women when adjusted for total body fat mass and HRT, the SAT was ~20% greater in the black women, while the percent visceral fat was ~8% greater in white women than in black women.

Studies on black women have reported the incidence of overweight to be as high as 49%, and that the risk of diabetes, coronary artery disease, and insulin resistance is greater in this population.[26] Yet paradoxically, many studies report that premenopausal blacks have lower absolute and relative VAT than age-matched white women. In our study despite no racial differences in blood lipid levels, VAT accounted for ~19% to 30% of the variability in the blood lipid profile, which is in agreement with earlier work on pre and postmenopausal women.[2,10] Race was only a significant contributor (8%) of LDL-C levels, which contrasts with earlier work.[2] Total body fat was the only significant predictor of HDL-C in this cohort of postmenopausal women, which contrasts with the earlier studies on postmenopausal women.[10] With the known changes in cardiovascular risk associated with menopause, it is possible that race is not a significant predictor of blood lipids postmenopause, because the menopause-evoked changes in cardiovascular risk may outweigh the racial differences. More importantly, the results of the regression analysis highlight that it is the absolute amount of VAT, and not the relative amount of VAT, which is more important when predicting the lipid profile. Although the black women have a greater total body fat mass, this only seems to play a role in modifying the HDL levels. Regardless of race and total fat mass, visceral fat content is the only significant variable impacting the blood lipid profile.

From pre to postmenopause there is some evidence to suggest that there may be an increase in central obesity. Some studies using waist circumference[11] and studies using DXA[15] have suggested increases in abdominal fat with menopause, while we recently reported that increases in abdominal fat around the menopausal years were associated with differences in physical activity and were not due to menopausal status.[13] The use of HRT has been suggested to prevent increases in central adiposity, and 1 study demonstrated that 2 years of HRT attenuated the increase in abdominal fat in postmenopausal women.[27] An additional study reported a reduction in waist circumference and central abdominal fat measured by DXA with 6 months of HRT use.[28] In contrast in the present study, women using HRT (~ 4.6 years) did not display differences in abdominal fat distribution compared with the non-HRT users. This finding is in agreement with the recent reports of Ryan et al[17] and Sites et al.[18]

VAT has also been linked with impaired insulin function leading to hyperinsulinemia and alterations in lipid metabolism,[29] but the mechanism of action is unknown. In postmenopausal women, intrabdominal fat has been reported to explain 24% of the variability in insulin sensitivity,[30] which agrees with other findings.[25,31] Similarly, in our cohort of white and black postmenopausal women, visceral fat explained 25% of the variability in calculated insulin resistance. This is an interesting finding because the black women had greater total abdominal fat, yet similar levels of visceral fat compared with the white women, and the black women still exhibited poorer insulin resistance. More research is needed to identify the underlying physiologic factors that can explain this discrepancy.

These differences of abdominal fat patterning among different races suggest caution must be heeded in assuming only

VAT is associated with increased health risks.[32] Black women have a lower visceral fat area,[4] but also have a lower insulin sensitivity index than white women. Goodpaster et al[33] reported that subcutaneous abdominal fat had as strong an association with insulin resistance as visceral fat, suggesting increased fat content was an important independent marker of insulin resistance in obesity in younger men and women. However, in the present study in older women, percent visceral fat, SAT, or total body fat was not related to insulin resistance. Subcutaneous abdominal fat may potentially be more of an important predictor in a younger white population. Brochu et al[34] recently reported that there may be a subgroup of obese postmenopausal women who have a high accumulation of body fat, yet are metabolically normal and have high insulin sensitivity. This may explain why only a small portion of the variability of the blood lipids and insulin resistance are explained by VAT and race.

Investigators[2,5] have looked for surrogates for the measurement of VAT because of expense and convenience. When entered into the regression equation separately, waist circumference and sagittal diameter were found to be significant predictors ($R^2 = .20$, $R^2 = .17$; $P < .01$) of visceral fat. Although these measures are significant predictors of VAT they were not significant variables in predicting the blood lipids or insulin resistance, and thus would not be a good substitute for the visceral fat measure.

In conclusion, this study clearly demonstrates that postmenopausal black women have more SAT, but less VAT or percent visceral fat than white women. The absolute amount of VAT and not the relative amounts of visceral fat or total fat mass is the best predictor of the blood lipid profile and insulin sensitivity. Race was a minor contributor. The use of HRT did not result in differences in abdominal fat distribution in this cohort of women. Most likely other factors, such as genetics and lifestyle, play a larger role in explaining the increased health risk in the black women.

ACKNOWLEDGMENT

The authors thank R. Ploutz-Snyder, PhD for statistical consulting assistance.

REFERENCES

1. Matsuzawa Y, Shimomura I, Nakamura T, et al : Pathophysiology and pathogenesis of visceral fat obesity. Obes Res 3:187S-194S, 1995 (suppl 2)

2. Perry A, Applegate E, Jackson M, et al: Racial differences in visceral adipose tissue but not anthropometric markers of health-related variables. J Appl Physiol 89:636-643, 2000

3. Centers for Disease Control: Chronic Disease in Minority Populations. Atlanta, GA: Public Health Services, CDC, 1994

4. Lovejoy J, de la Bretonne J, Klemperer M, et al: Abdominal fat distribution and metabolic risk factors: Effects of race. Metabolism 45:1119-1124, 1996

5. Albu J, Murphy L, Frager D, et al: Visceral fat and race-dependent health risks in obese nondiabetic premenopausal women. Diabetes 46:456-462, 1997

6. Conway J, Yanovski S, Avila N, et al: Visceral adipose tissue differences in black and white women. Am J Clin Nutr 61:765-771, 1995

7. Hill J, Sidney S, Lewis C, et al: 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 69:381-387, 1999

8. Lovejoy J, Smith S, Rood J: Comparison of regional fat distribution and health risk factors in middle-aged white and African American women: The Healthy Transitions Study. Obes Res 9:10-16, 2001

9. Weinsier R, Hunter G, Gower B, et al: Body fat distribution in white and black women: Different patterns of intraabdominal and subcutaneous abdominal adipose tissue utilization with weight loss. AmJ Clin Nutr 74:631-636, 2001

10. Berman D, Rodrigues L, Nicklas B, et al: Racial disparities in metabolism, central obesity, and sex hormone-binding globulin in postmenopausal women. J Clin Endocrinol Metab 86:97-1003 , 2001

11. Poehlman ET, Toth MJ, Gardner AW: Changes in energy balance and body composition at menopause: A controlled longitudinal study. Ann Intern Med 123:673-675, 1995

12. Toth MJ, Tchernor A, Sites CK, et al: Effect of menopausal status on body fat composition and abdominal fat distribution. Int J Obes 24:226-231, 2000

13. Kanaley J, Sames C, Swisher L, et al: Abdominal fat distribution in pre- and postmenopausal women: The impact of physical activity, age and menopausal status. Metabolism 50:976-982, 2001

14. Panotopoulos G, Ruiz JC, Raison J, et al: Menopause, fat and lean distribution in obese women. Maturitas 25:11-19, 1996

15. Svendsen OL, Hassager C, Christiansen C: Age- menopause-associated variations in body composition and fat distribution in healthy women as measured by dual-energy x-ray absorptiometry. Metabolism 44:369-373, 1995

16. Trémollieres FA, Poulilles J-M, Ribot CA: Relative influence of age and menopause on total and regional body composition changes in postmenopausal women. Am J Obstet Gynecol 175:1594-1600, 1996

17. Ryan AS, Nicklas BJ, Berman DM: Hormone replacement therapy, insulin sensitivity, and abdominal obesity in postmenopausal women. Diabetes Care 25:127-133, 2002

18. Sites CK, Brochu M, Tchernof A, et al: Relationship between hormone replacement therapy use with body fat distribution and insulin sensitivity in obese postmenopausal women. Metabolism 50:835-840, 2001

19. Organ LW, Eklund AD, Ledbetter JD: An automated real time underwater weighing system. Med Sci Sports Exerc 26:383-391, 1994

20. Deurenberg P, Westrate J, van der Kooy K: Is an adaptation of Siri's formula for the calculation of body fat percentage from body density in the elderly necessary? Eur J Clin Nutr 43:559-568, 1989

21. Haffner A, Miettinen H, Stern M: The homeostasis model in the San Antonio Heart Study. Diabetes Care 20:1087-109, 1997

22. Tabachnick B, Fidell L: Using Multivariate Statistics (ed 2). New York, NY, Harper Conlins, 1989

23. Rexrode K, Carey V, Hennekens C, et al : Abdominal adiposity and coronary heart disease in women. JAMA 280:1843-1848, 1998

24. Williams M, Hunter G, Kekes-Szabo T, et al: Intra-abdominal adipose tissue cut-points related to elevated cardiovascular risk in women. Int J Obes Related Metab Disord 20:613-617, 1996

25. Marcus M, Murphy L, Pi-Sunyer F, et al: Insulin sensitivity and serum triglyceride level in obese white and black women: Relationship to visceral and truncal subcutaneous fat. Metabolism 48:194-199, 1999

26. Harris M: Epidemiological correlates of NIDDM in Hispanics, whites, and blacks in the US population. Diabetes Care 14:639-648, 1991

27. Haarbo J, Marslew U, Gotfredsen A, et al: Postmenopausal hormone replacement therapy prevents central distribution of body fat after menopause. Metabolism 40:1323-1326, 1991

28. Samaras K, Hayward CS, Sullivan D, et al: Effects of postmenopausal hormone replacement therapy on central abdominal fat, glycemic control, lipid metabolism, and vascular factors in type 2 diabetes: A prospective study. Diabetes Care 22:1401-1407, 1999

29. Frayn K: Visceral fat and insulin resistance—Causative or correlative? Br J Nutr 83:S71-S77, 2000 (suppl 1)

30. Rendell M, Hulthen U, Tornquist C, et al: Relationship between abdominal fat compartments and glucose and lipid metabolism in early postmenopausal women. J Clin Endocrinol Metab 86:744-749, 2001

31. Ross R, Fortier L, Hudson R: Separate associations between visceral and subcutaneous adipose tissue distribution, insulin and glucose levels in obese women. Diabetes Care 19:1404-1411, 1996

32. Dowling H, Pi-Sunyer F: Race-dependent health risks of upper body obesity. Diabetes 42:537-543, 1993

33. Goodpaster B, Thaete F, Simoneau J-A, et al: Subcutaneous abdominal fat and thigh muscle composition predict insulin sensitivity independently of visceral fat. Diabetes 46:1579-1585, 1997

34. Brochu M, Tchernof A, Dionne I, et al: What are the physical characteristics associated with a normal metabolic profile despite a high level of obesity in postmenopausal women. J Clin Endocrinol Metab 86:1020-1025, 2001