Abdominal Fat Distribution in Pre- and Postmenopausal Women: The Impact of Physical Activity, Age, and Menopausal Status

J.A. Kanaley, C. Sames, L. Swisher, A.G. Swick, L.L. Ploutz-Snyder, C.M. Steppan, K.S. Sagendorf, D. Feiglin, E.B. Jaynes, R.A. Meyer, and R.S. Weinstock

Age-related increases in total body fat have been reported, but the impact of menopause on abdominal fat distribution is still unclear. The purpose of this study was to determine the impact of menopausal status on abdominal fat distribution using magnetic resonance imaging (MRI). In addition, we investigated the influence of abdominal fat distribution on blood lipid profiles and leptin concentrations. Twenty-three premenopausal (PRE), 27 postmenopausal (POST), and 28 postmenopausal women on estrogen replacement therapy (ERT) had measurements of regional abdominal fat, blood lipids, and serum leptin concentrations. The women were matched for body mass index (BMI) and total body fat mass. Age and menopausal status were not found to be significant predictors of total abdominal fat, visceral fat, or subcutaneous fat, while physical activity was a significant predictor ($P < .01$) for total abdominal fat ($R^2 = .16$), visceral fat ($R^2 = .32$) and percent visceral fat ($R^2 = .25$). There was a trend for a greater visceral fat content in the POST women compared with the PRE women (2,495.0 ± 228.4 *v* 1,770.4 ± 240.8 cm^2, respectively, $P = .06$). The percent visceral abdominal fat was significantly lower ($P < .05$) in the premenopausal women than in either postmenopausal group (PRE, 23.2% ± 1.7%; POST, 28.9% ± 1.8%; ERT, 28.9% ± 1.6%). Menopausal status and age did not influence any of the blood lipid values. Abdominal fat distribution was a significant predictor of cholesterol concentrations and the cholesterol/high-density lipoprotein-cholesterol (HDL-C) ratio, but only accounted for approximately 15% of the variability in these levels. Total body fat and physical activity accounted for 47% of the variability in leptin concentrations, while abdominal fat distribution, age, and menopausal status were not significant predictors. In conclusion, in early postmenopausal women, the level of physical activity accounts for the variability in abdominal fat distribution observed, while menopausal status and age do not play a significant role. ERT was not associated with additional benefits in abdominal fat distribution compared with postmenopausal women not on ERT or in the blood lipid profile in these women.

AFTER WOMEN ENTER menopause, their risk of cardiovascular and metabolic disease increases.[1-4] The etiology of the effect of the menopause-transition on cardiovascular and metabolic diseases is unclear, but may be linked with changes in body fat distribution. Increased abdominal visceral adipose tissue is associated with the metabolic syndrome, including type 2 diabetes, hypertension, and dyslipidemia.[2,3,5,6]

Aging alone has been associated with a decrease in lean body mass,[7] a concomitant increase in body fat, and a shifting of fat from peripheral subcutaneous depots to intra-abdominal depots.[8] It is possible that the decrease in sex steroid hormones associated with menopause may facilitate age-related increases in body fat, specifically abdominal fat.[9-11] Establishing whether this shift results in accumulation of visceral or subcutaneous abdominal fat is important, considering the known health consequences of visceral fat stores.[2] Moreover, estrogen replacement therapy (ERT) may impact the redistribution of body fat, thus altering the health risks.

Much of the previous research has used anthropometic measures to establish if changes in abdominal fat occur with the transition through menopause. Studies using circumference measures usually do not demonstrate changes in body fat distribution with menopause,[12] while significantly greater central adipose deposition is observed in postmenopausal women compared with premenopausal women when dual-energy x-ray absorptiometry (DEXA) is used.[13-15] More recently, using single slice computerized tomography (CT) imaging, Toth et al[12] reported that postmenopausal women had more visceral fat than premenopausal women, and this was independent of age and total body fat mass. However, few studies have used magnetic resonance imaging (MRI) techniques to study the shifts in abdominal fat with menopause. MRI, unlike single slice analysis of abdominal fat by CT, allows quantification of total abdominal fat, instead of estimating total abdominal fat content by extrapolating from a single slice.

Thus, the primary purpose of this study was to examine the influence of menopausal status on total abdominal fat distribution using MRI and to determine if fat distribution differs between women on estrogen replacement therapy (ERT) and those who are not on ERT. Previous studies[12-15] have not compared postmenopausal women using or not using ERT. It was hypothesized that total abdominal and visceral abdominal fat would be greatest in postmenopausal women not on estrogen compared with those women on ERT and premenopausal women.

In addition, because in a younger population, increased visceral fat is associated with increased health risks,[3] we examined whether body fat distribution may partially explain the increased risk of cardiovascular disease in postmenopausal women. Leptin concentrations were also measured in this population to establish if a relationship exists with menstrual status and abdominal fat distribution. We hypothesized that visceral

From the Department of Exercise Science, Syracuse University, Syracuse; Departments of Medicine and Radiology, Upstate Medical University, Syracuse; VA Medical Center, Syracuse, NY; Pfizer, Inc, Groton, CT; and the Department of Physiology and Radiology, Michigan State University, East Lansing, MI.

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

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fat content would be related to the blood lipid and leptin concentrations and would account for the less favorable lipid profile after menopause that is frequently reported.[5] This study differs from many earlier studies in that comparisons were made between pre and postmenopausal women (± ERT) who were matched for total body fat mass, fat free mass, and body mass index (BMI), and MRI was used to specifically evaluate and quantify visceral and subcutaneous abdominal fat.

MATERIALS AND METHODS

Subjects

Seventy-eight Caucasian pre and postmenopausal women between the ages of 45 to 60 years were recruited for this study and signed a consent form that was approved by the Syracuse University Institutional Review Board. When being recruited for the study, all potential subjects first went through a screening telephone interview to see if they qualified for the study (appropriate medical history) and were questioned for age, height, and weight to see if they matched our earlier enrolled volunteers. Before entry into the study, volunteers completed medical and physical activity questionnaires. These questionnaires were returned, and we would followed up with more questions if any questions were incomplete or vague answers were given. If appropriate, the subject was then enrolled in the study. The women were categorized on their physical activity level in 1 of the following groups: inactive, active daily life (no outside exercise), moderately active daily life (formal exercise less than 3 times//week), and active daily life (formal exercise more than 3 times/week. The physical activity questionnaires were a composite of a number of questions from other questionnaires. These questionnaires targeted leisure activities, home activities, and work demands. Two investigators reviewed these questionnaires and provided a score. If there were discrepancies, this was discussed. Most women participated in some regular exercise (eg, walking for 20 minutes 1 to 2 times a week), but only a few women had more active exercise programs. Once testing was initiated, 2 subjects were omitted because of information that we learned; testing was not completed in these women, and their data is not included.

The premenopausal women (n = 23) were not on oral contraceptives (PRE), while 27 postmenopausal women were not on ERT (POST), and 28 postmenopausal women had been taking ERT for at least 1 year (4.0 ± 0.6 years; mean ± SE). All women had a BMI less than 30 kg/m^2 and were matched for BMI and level of physical activity. Overall, the women were healthy and were free of known cardiovascular disease and other metabolic diseases. None of the women were taking medications affecting blood pressure, blood lipid, glucose, or thyroid metabolism. All of the postmenopausal women had been postmenopausal for more than 1 year, but less than 7 years (POST, 4.09 ± 0.5 years; ERT, 4.61 ± 0.5 years; mean ± SE) and had been determined to be postmenopausal by their physicians by hormonal evaluation (follicle-stimulating hormone [FSH] > 50 mIU/mL). All the postmenopausal women had gone through menopause naturally, and the women on ERT were taking either Premarin (0.625 mg; Wyeth-Ayerst, Philadelphia, PA) (n = 7) or Premarin plus medroxyprogesterone acetate (2.5 mg) (n = 18, n = 3 unknown dose). All subjects were non-mokers 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, subjects had their fasting blood lipids, leptin concentrations, and body composition assessed and were required to fill out questionnaires. On the second visit, the subjects were required to have a MRI scan, which scanned from the midchest to 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 by 1 to 2 weeks, which was dependent on the subject's schedule and MRI availability.

Methodology

Body composition. Total body fat was determined using underwater weighing. Body density was measured with the underwater weight taken simultaneously with measurement of residual lung volume.[16] Percent body fat was calculated according to the equations of Brozek et al.[17]

Regional fat distribution was quantified using MRI (GE Signa 1.5 T; General Electric, Waukesha, WI) using standard spin echo imaging with respiratory compensation time recovery (TR) = 400, (TE) = 20, field of view (FOV) 40 to 45, 256 × 256, and 1 nex. Slice thickness was 1 cm, with approximately 10 consecutive slices/scan. Total abdominal fat was calculated in the region beginning at the superior portion of the head of the femur and continued up to the most superior portion of the kidneys. This represented about 25 cm in most subjects depending on their height. For each slice area, visceral fat was determined, and this was subtracted from the total fat in that slice to calculate the subcutaneous fat. Total abdominal fat was determined by adding together all fat components of all slices.

The images were analyzed using an automated fat segmentation program running on a SUN workstation (SUN Microsystems, CA) to avoid investigator bias. The program used a fully automated global thresholding technique for fat segmentation. For each individual slice, a gradient image was constructed using Sobel operators. The optimal segmentation threshold was then computed by maximizing (simplex algorithm) the correlation between the original image versus the gradient images computed from the segmented image. Images were analyzed twice with the program operator blinded to subject name and menopausal status.

Test-retest correlations of the image analysis procedure to determine total, visceral, and subcutaneous fat were greater than 0.9999 for all analyses ($P < .0001$). Coefficients of variation were all less than 1%. This demonstrated that the automated image analysis program allowed for rapid analysis of multislice acquisitions and was a highly reliable method for analysis of body fat content.

Blood sampling. Following a 12-hour overnight fast and a 24-hour period of no exercise, at 7:00 AM, a blood sample was taken for measurement of blood lipids and leptin concentrations.

Analytical methods. Serum leptin concentrations were determined using a quantitative sandwich enzyme immunoassay (R&D Systems, Minneapolis, MN). Briefly, serum was diluted 1,000-fold and added to a polystyrene microplate coated with a murine monoclonal antibody against leptin. A second monoclonal antibody conjugated to horseradish peroxidase was then added. Optical density was determined at 450 nm with a wavelength correction at 540 nm. Values for serum leptin levels are quantified by comparison against a standard curve generated using recombinant human leptin. Intra- and interassay coefficients of variation are 3.2% and 4.4%, respectively. Blood lipids were measured by SmithKline Industries (Syracuse, NY).

Statistical analysis. The data were analyzed using Statistical Packages for the Social Sciences (SPSS, Chicago, IL, version 8.0) and are presented as means ± SE. A 1-way analysis of variance was used to determine if group differences existed between the descriptive variables. A Tukey test was used for post hoc comparison if significant differences in mean group comparisons were found. To determine the effects of age, menopausal status, and physical activity on abdominal fat distribution and blood lipid and leptin concentrations, we applied a multiple linear regression analysis technique. Three dummy variables indicating menopausal status were created, 1/condition (pre, post, ERT), each having 1 or 0. The dummy variables were applied to all subjects, and we entered 2 of the 3 dummy-variables in the regression

equation, (eg, omitting ERT) so that the linear combination accounts for all possible conditions. This technique allows us to determine statistical differences of abdominal fat distribution between ERT and PRE and between ERT and POST, and it is the appropriate way to account for the 3 conditions that are not proven to be of ratio scale (equal distances between groups) in regression analysis. The variables were entered into the regression simultaneously so that the relative contribution of each predictor variable on the outcome is unbiased. Because leptin concentrations do not increase linearly with increases in fat mass, leptin values were log transformed before being entered into the regression analysis. Total abdominal fat, visceral fat, subcutaneous fat, age, physical activity, menopausal status, and total body fat mass were entered into the regression analysis.

RESULTS

Seventy-eight women participated in this study, but blood lipid and leptin results were only available on 72 women. Table 1 shows the physical characteristics of the women who participated this study. The premenopausal women were significantly younger ($P < .01$) than either postmenopausal group, however the subjects were well matched for percent body fat, fat mass, and BMI.

Abdominal Fat Distribution

There was a trend for the postmenopausal women to have greater abdominal fat ($P = .08$, Fig 1). Visceral abdominal fat content was less in the premenopausal women than in either postmenopausal groups, and the differences in visceral fat approached significance ($P < .06$) between the PRE and POST women (PRE, 1,770.4 ± 240.8; POST, 2,495.0 ± 228.4 cm^2). The percent visceral abdominal fat (visceral abdominal fat/total abdominal fat * 100) was significantly lower ($P < .05$) in the premenopausal women (23.2% ± 1.7%) than in either of the postmenopausal groups (POST, 28.9 ± 1.8l; ERT, 28.9% ± 1.6%).

Regression analysis was used to determine the influence of age, menopausal status, and physical activity on total abdominal fat, visceral fat, subcutaneous fat, or percent visceral fat (Table 2). Physical activity was a significant predictor of total abdominal fat ($R^2 = .16$, $P < .01$) and visceral fat ($R^2 = .32$, $P < .001$), and physical activity approached significance ($P = .07$) for subcutaneous fat. Neither age nor menopausal status was found to be a significant predictor of total abdominal fat, visceral fat, or subcutaneous fat. Further physical activity was a significant ($R^2 = .25$, $P < .01$) predictor of percent visceral fat.

Blood Lipid Levels

No differences in the blood lipid profile were found between the pre and postmenopausal women. Triglycerides and low-density lipoprotein-cholesterol (LDL-C) were slightly lower in the premenopausal women than the premenopausal women (Table 3). Total abdominal fat, visceral fat, and subcutaneous fat mass were significant predictors of total cholesterol levels ($P < .01$, Table 4) and cholesterol/high-density lipoprotein-cholesterol (HDL-C) ($P < .01$). Total body fat, menopausal status, age, and physical activity were not significant predictors for these dependent variables. No significant predictors were found for triglycerides, HDL-C, or LDL-C concentrations. Although these measures of regional fat were significant predictors of the blood lipid values, they accounted for less than 15% of the variability in these measures.

Serum Leptin Concentrations

There was a trend for resting plasma leptin concentrations to be lower in the PRE women compared with the POST and ERT women (P = not significant [NS], Table 3). Forty-seven percent of the variability in resting serum leptin concentrations accounted for by total body fat mass and physical activity ($P < .001$, standard error of the estimate [SEE] = 34, $\beta = 0.0293$ and -0.144, respectively). Regional fat distribution, menopausal status, and age were not significant contributors to leptin concentration.

DISCUSSION

This is one of the first studies to use MRI to carefully quantitate subcutaneous and visceral abdominal fat in pre- and postmenopausal women matched for BMI and total fat mass. Much of the previous work documenting shifts of fat from gluteal to abdominal stores with menopause used either DEXA or anthropometric measures.[10,11,14,18] However, MRI is a superior imaging technique because of its better spatial resolution, speed, and lack of ionizing radiation. These features facilitate multislice acquisition of both total and regional differences in abdominal fat. Our MRI data showed that total abdominal fat was approximately 22% higher in premenopausal women compared with postmenopausal women not on estrogen and was 13% higher than those women on ERT. In both groups of postmenopausal women, percent visceral fat was 5% higher than in the premenopausal women. Unlike previous research,[12,19] menopausal status did not influence abdominal fat

Table 1. Physical Characteristics of the Subjects

Variable	Premenopausal (n = 23)	Postmenopausal (n = 27)	Postmenopausal + ERT (n = 28)
Age (yr)	49.0 ± 0.4	53.3 ± 0.6*†	55.5 ± 0.7*
Height (cm)	162.7 ± 1.4	161.5 ± 1.0	164.1 ± 1.0
Body weight (kg)	66.1 ± 1.9	68.1 ± 2.0	65.6 ± 2.2
Percent fat	31.5 ± 1.3	34.2 ± 1.4	33.2 ± 1.4
Fast mass (kg)	21.2 ± 1.5	23.7 ± 1.5	21.8 ± 1.5
Fat-free mass (kg)	44.8 ± 1.1	44.4 ± 1.1	43.7 ± 1.0
BMI (kg/m^2)	25.1 ± 0.7	26.1 ± 0.7	24.4 ± 0.8

*$P < .05$ *v* premenopausal; †$P < .05$ *v* postmenopausal + ERT; mean ± SE.

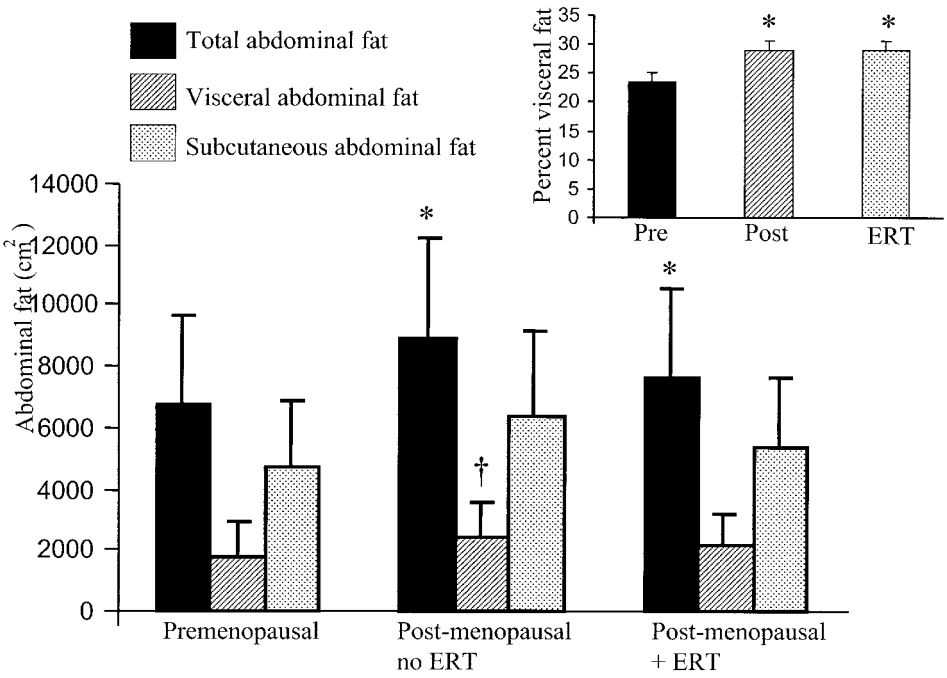

Fig 1. Regional fat distribution in the pre and postmenopausal women. The inset shows the percent visceral fat, relative to total abdominal fat. PRE, premenopausal; POST, postmenopausal not on estrogen replacement therapy; ERT, postmenopausal women not on ERT. *$P < .05$ *v* premenopausal group; n = 72; mean ± SE.

distribution, but physical activity level did account for some of the variability that occurred in these women.

Our findings demonstrate that in early menopause (≈ 4 years), dramatic increases in regional abdominal fat depots do not occur. Some changes, however, are beginning to occur. Although an absolute increase in visceral fat was not found in our study, we did observe a 5% higher percentage visceral fat in the postmenopausal women, and ERT did not alter this finding. In our findings, physical activity explained the variability in abdominal fat distribution in these women and not menstrual status. Similarly, a recent report[20] noted that weight increases in middle-aged women were not a result of menopause transition. In contrast to our findings, the only longitudinal study to date[19] found that women who had gone through natural menopause gained more fat than the women who remained premenopausal and had greater increases in their waist-to-hip ratio, indicating increases in central adiposity. Unfortunately, these investigators did not quantify abdominal fat distribution. However, supporting our findings of the importance of physical activity, Poehlman et al[19] noted that greater decreases in physical activity during leisure time were observed in the postmenopausal women.

Recently Toth et al[12] reported a preferential increase in intra-abdominal fat that is independent of age and fat mass. Using single slice CT, both visceral and subcutaneous fat were greater in their postmenopausal women. Differences in our findings and Toth's may reflect differences in our sample populations. Subjects in Toth's report were not matched for total fat mass and percent fat, and physical activity was not assessed. The higher total fat content in their postmenopausal subjects may also explain the greater abdominal fat mass.

Using DEXA analysis, Ley et al[13] showed an increase in android fat and a relative reduction in gynoid fat in postmenopausal women. Other investigators have noted a relationship between abdominal to total body fat tissue ratio and age,[14,18] but this relationship was independent of years since menopause.[14] Similarly, we noted a slight increase in total abdominal fat with menopause, but we did not demonstrate a relationship between abdominal fat or fat free mass (FFM) with years since menopause. All of our subjects were in early menopause (1 to

Table 2. Regression Coefficient for the Model to Predict Total Abdominal Fat, Visceral Fat, and Percent Visceral Fat

	Unstandardized Coefficients		Standardized Coefficients		
	β	SE	β	Significance	R^2
Total abdominal fat					
Physical activity	−1,260.7	420.5	−0.359	.004	.161
Visceral fat					
Physical activity	−684.0	138.5	−0.532	.001	.318
Percent visceral fat					
Physical activity	−4.1	1.1	−0.412	.001	.253

NOTE. Age, menopausal status, and physical activity were entered into the regression analysis, n = 72.

Table 3. Blood Lipids and Leptin Concentrations for Each of the Three Groups

Variable	Premenopausal (n = 23)	Postmenopausal (n = 27)	Postmenopausal + ERT (n = 28)
Total cholesterol (mmol/L)	194.9 ± 9.0	193.9 ± 7.8	193.0 ± 4.6
Triglycerides (mmol/L)	95.0 ± 10.2	102.1 ± 10.4	120.8 ± 15.9
LDL-C (mmol/L)	115.6 ± 5.7	120.2 ± 6.8	110.1 ± 5.3
HDL-C (mmol/L)	53.6 ± 2.5	57.4 ± 3.4	68.8 ± 7.5
Leptin (μg/L)	45.0 ± 10.6	73.3 ± 14.4	59.0 ± 9.2

NOTE. Mean ± SE.

7 years postmenopause) and thus, this may account for this lack of relationship. Greater differences in total abdominal fat and visceral fat may be found in women greater than 10 years postmenopausal.

Many investigators have speculated that estrogen/progesterone treatment in early postmenopausal women may prevent the central redistribution of fat[9]; however, our data do not support this hypothesis. Physical activity has a much more profound influence on fat distribution than ERT in this early postmenopausal population. In support of our findings, Kritz-Silverstein et al[21] reported that long-term ERT use (15 years) either continuously or intermittently was not associated with weight gain or central obesity.

Increases in central adiposity, and in particular, increased visceral fat, are related to numerous metabolic aberrations (ie, insulin resistance, hypertension, and hyperlipidemia) as shown in epidemiologic studies.[2,6] Visceral fat is more sensitive to lipolytic stimuli resulting in increased free fatty acid (FFA) levels in the portal circulation, which may stimulate hepatic very–low-density lipoprotein (VLDL) production.[22] Individuals with abdominal obesity often have reduced HDL concentrations, possibly resulting from an increased exchange of cholesterol for triglycerides in VLDL particles.[2] After menopause, increases in body fat parallel unfavorable changes in blood lipids.

If prevention of visceral fat accumulation minimizes the cardiovascular risk, ERT could exert beneficial effects on the blood lipid profile by preventing abdominal fat accumulation and altering the fat distribution.[11,13] Despite increases in the percent visceral fat in the postmenopausal women, no differences in the blood lipid profiles were seen in our women. The lack of relationship between abdominal fat and the blood lipid profile was most likely because the postmenopausal women did not have an absolute increase in the visceral fat content, which is usually associated with the poorer blood lipid profile in younger populations.

Increased risk of cardiovascular disease is associated with significant increases in visceral fat.[23] We studied healthy women in a narrow age range, who were not obese, with a mean BMI of approximately 25 kg/m^2. It is possible that the blood lipid profile does not become deleterious in postmenopausal women until a critical amount of abdominal fat has accumulated, particularly in the visceral region, and this may not take place until the women are many more years postmenopausal and more obese.

Surprisingly, the ERT group did not demonstrate lower cholesterol levels or significantly higher triglyceride levels, which is usually observed.[4,24] One longitudinal study (2 years) has shown that estrogen-progestogen therapy reduced total cholesterol and LDL-C, but did not alter HDL-C or triglyceride concentrations.[9] The Heart and Estrogen/Progestin Replacement study (HERS) found no overall effect of 4.1 years of treatment with estrogen and medroxyprogesterone acetate on the risk of nonfatal myocardial infarction.[25] Recently 3.2 years of estrogen replacement resulted in a slowing in the progression of coronary atherosclerotic lesions in women.[26] Lipoprotein a (Lp[a]), an independent risk factor for recurrent cardiovascular disease, has been reported to be lowered with estrogen and progestin treatment.[27] The estrogen/progestin replacement was more favorable in women with high initial Lp(a) levels than in women with low levels.[28]

Although we did not find any association between physical activity and the blood lipid profile, Greendale et al[29] reported that leisure physical activity was positively associated with levels of HDL-C ($P < .01$). Moderate and heavy leisure activities were associated with higher HDL-C levels, with the heavy group being significantly greater than the moderate group. Home physical activity was positively related to HDL level. The benefits of physical activity on blood lipids may indirectly affect cardiovascular disease by altering visceral fat content.[23]

Total body fat is known to influence the leptin levels, but it is still unclear whether body fat distribution or menopausal status influenced the leptin concentrations. In agreement with numerous reports,[30-33] we found that serum leptin levels were

Table 4. Regression Coefficients for Predicting Blood Lipid Values

Dependent Variable	Independent Variable	β	Intercept	*P* Value	R^2	SEE
Total cholesterol	Total abdominal fat	0.136	170.6	$P < .01$	.14	35.35
	Visceral fat	−0.127				
	Subcutaneous fat	−0.136				
Cholesterol/HDL-C	Total abdominal fat	0.131	99.9	$P < .01$	.12	28.9
	Visceral fat	−0.128				
	Subcutaneous fat	−0.139				

NOTE. Age, menopausal status, total fat mass, and physical activity were entered into the regression analysis.

positively related to the degree of adiposity in these middle-aged pre and postmenopausal women. In agreement with Considine et al,[31] we found no relationship between age and plasma leptin concentrations, while others report an inverse relationship.[33] Higher leptin concentrations in pre versus postmenopausal women have been reported,[34] while others find no differences between pre and postmenopausal women.[35,36] We did not find a relationship with menopausal status. Physical activity and total body fat accounted for 47% of the variability in leptin concentrations, while abdominal fat distribution and age did not influence leptin levels. This is consistent with one recent finding,[37] but contrasts with others who have reported that subcutaneous fat distribution is a determinant of leptin levels when comparing men and women using ultrasound.[38]

Unlike many earlier studies, this study attempted to minimize the effects of age by only selecting women in their early menopausal years and to minimize the confounding factor of total fat mass and physical activity. Further, much of the earlier work has analyzed body fat distribution with either waist-to-hip ratio or DEXA analysis, and only a few other studies have used advanced techniques of CT analysis. However, this is one of the first studies that carefully analyzed the abdominal fat distribution with MRI.

In conclusion, a greater proportion of visceral adipose tissue was observed in the postmenopausal women compared with the premenopausal women. Our data are among the first to suggest that the changes in abdominal fat distribution that are observed in the early postmenopausal years are influenced by physical activity level of these women and not menopausal status or age. Further, the blood lipid profile also was not influenced by menopausal status, but appears to be determined more so by the amount of abdominal fat. ERT does not influence either the abdominal fat accumulation or the blood lipid profile in this cohort of women. Although it is acknowledged that this study is limited by its cross-sectional design, it clearly illustrates that in the earlier postmenopausal years, if physical activity is maintained, abdominal fat distribution does not dramatically change, but the relative proportion of visceral fat is beginning to be altered. The level of physical activity in these middle-aged women appears to influence the changes in abdominal fat distribution that occur.

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