

# Abdominal Obesity and Association With Atherosclerosis Risk Factors

## *The Uberlândia Heart Study*

Leonardo S. Roeвер, MHS, Elmiro. S. Resende, PhD, Angélica. L. D. Diniz, PhD, Nilson Penha-Silva, PhD, Fernando. C. Veloso, MHS, Antonio Casella-Filho, PhD, Paulo .M.M. Dourado, PhD, and Antonio .C.P. Chagas, PhD

**Abstract:** Ectopic visceral fat (VF) and subcutaneous fat (SCF) are associated with cardiovascular risk factors. Gender differences in the correlations of cardiovascular disease risk factors and ectopic fat in the Brazilian population still lacking.

Cross-sectional study with 101 volunteers (50.49% men; mean age  $56.5 \pm 18$ , range 19–74 years) drawn from the Uberlândia Heart Study underwent ultrasonography assessment of abdominal visceral adipose tissue with convex transducer of 3.5 MHz of frequency. The thickness of VF was ultrasonographically measured by the distance between the inner face of the abdominal muscle and the posterior face of abdominal aorta, 1 cm above the umbilicus. The SCF thickness was measured with a 7.5 MHz linear transducer transversely positioned 1 cm above the umbilical scar. The exams were always performed by the same examiner. Ectopic fat volumes were examined in relation to waist circumference, blood pressure, and metabolic risk factors.

The VF was significantly associated with the levels of triglycerides ( $P < 0.01$ ,  $r = 0.10$ ), HDL cholesterol ( $P < 0.005$ ,  $r = 0.15$ ), total cholesterol ( $P < 0.01$ ,  $r = 0.10$ ), waist circumference ( $P < 0.0001$ ,  $r = 0.43$ ), systolic blood pressure ( $P < 0.001$ ,  $r = 0.41$ ), and diastolic blood pressure ( $P < 0.001$ ,  $r = 0.32$ ) in women, and with the levels of triglycerides ( $P < 0.002$ ,  $r = 0.14$ ), HDL cholesterol ( $P < 0.032$ ,  $r = 0.07$ ), glucose ( $P < 0.001$ ,  $r = 0.15$ ), alanine aminotransferase (ALT) ( $P < 0.008$ ,  $r = 0.12$ ), gamma-GT ( $P < 0.001$ ,  $r = 0.30$ ), waist circumference ( $P < 0.001$ ,  $r = 0.52$ ), systolic blood pressure ( $P < 0.001$ ,  $r = 0.32$ ), and diastolic blood pressure ( $P < 0.001$ ,  $r = 0.26$ ) in men. SCF was significantly associated with the levels of triglycerides ( $P < 0.01$ ,  $r = 0.34$ ), LDL cholesterol ( $P < 0.001$ ,  $r = 0.36$ ), total cholesterol ( $P < 0.05$ ,  $r = 0.36$ ), waist circumference ( $P < 0.0001$ ,  $r = 0.62$ ), systolic and diastolic blood pressure ( $P < 0.05$ ,  $r = 0.34$ ) in women, and with the waist circumference ( $P < 0.001$ ,  $r = 0.065$ ), and MetS ( $P < 0.05$ ,  $r = 0.11$ ) in men.

The VF and SCF were correlated with most cardiovascular risk factors in both genders but our findings support the idea that there are gender differences in the correlations between ectopic fat deposition and the cardiovascular risk factors.

(*Medicine* 95(11):e1357)

**Abbreviations:** ALT = alanine aminotransferase, AST = aspartate transaminase, BMI = body mass index, CT = computed tomography, CVD = cardiovascular disease, GGT = gamma glutamyl transferase, HDL-C = high density lipoprotein cholesterol, HOMA-IR = Homeostatic Model Assessment for Insulin Resistance, LDL-C = low density lipoprotein cholesterol, M = men, MetS = metabolic syndrome, MRI = magnetic resonance imaging, non-HDL = nonhigh density lipoprotein cholesterol, SCF = subcutaneous fat, VF = visceral fat, W = women, WC = waist circumference.

## INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality in the world. Ectopic fat is a risk factor for multiple CVD risk factors, including hypertension, dyslipidemia, diabetes, and the metabolic syndrome (MetS).<sup>1–5</sup> In particular, the visceral fat (VF) compartment may be a pathogenic fat depot. VF has been termed an endocrine organ, in part because it secretes adipocytokines and other vasoactive substances that can influence the risk of developing metabolic traits.<sup>6–10</sup>

Waist circumference (WC) is an imprecise measure of abdominal adiposity because it is a function of both the subcutaneous fat (SCF) and VF compartments.<sup>7</sup> Available studies report relations of greater SAT and VF with a higher prevalence of impaired fasting glucose, diabetes, insulin resistance, hypertension, lipids, MetS, inflammation, and risk factor clustering.<sup>8,11–24</sup>

The aim of this study was to analyze the association among SCF and VF and other obesity-related parameters, such as waist circumference, blood pressure, and metabolic risk factors.

## METHODS

### Study Sample

This prospective cross-sectional case–control study with 101 volunteers (50.49% men; mean age  $56.5 \pm 18$ , range 19–74 years) drawn from the Uberlândia Heart Study underwent ultrasonography assessment of abdominal adipose tissue. The study was a random sample of individuals that required medical service hospital. The study was approved by the institutional review boards of the Federal University of Uberlândia. All subjects provided written informed consent. All patients received the first diagnosis of related disorders in the study, and did not use medications that affected the lipid profile, blood pressure, and blood glucose. Those with kidney liver or chronic respiratory failure, as well as subjects with severe apnea, morbid

Editor: Leonardo Gilardi.

Received: June 18, 2015; revised and accepted: July 9, 2015.

From the Federal University of Uberlândia (LR, ESR, ALDD, NP-S, FCV), Heart Institute (InCor), HCFMUSP, University of São Paulo Medical School, São Paulo (AC-F, PMMD, ACPC), and Faculty of Medicine ABC, Santo André (ACPC), Brazil.

Correspondence: Leonardo S. Roeвер-Borges, Department of Clinical Research, Av. Pará, 1720, Bairro Umuarama Uberlândia, MG, CEP 38400-902, São Paulo, Brazil (e-mail: leonardoroever@hotmail.com).

The authors have no conflicts of interest to disclose.

Copyright © 2016 Wolters Kluwer Health, Inc. All rights reserved.

This is an open access article distributed under the Creative Commons Attribution-NoDerivatives License 4.0, which allows for redistribution, commercial and non-commercial, as long as it is passed along unchanged and in whole, with credit to the author.

ISSN: 0025-7974

DOI: 10.1097/MD.0000000000001357

obesity, cancer, neurodegenerative diseases, or receiving psychiatric medications were excluded.

### Abdominal Adipose Tissue Measurements

The ultrasound examination was performed always by the same examiner with an equipment Versa-Pro (Siemens, Erlangen, Germany), using the preset for abdominal examination. The Assessment of abdominal visceral adipose tissue was done with convex transducer of 3.5 MHz of frequency. The thickness of VF was ultrasonographically measured by the distance between the inner face of the abdominal muscle and the posterior face of abdominal aorta, 1 cm above the umbilicus (Figure 1). The SCF thickness (Figure 2) was measured with a 7.5 MHz linear transducer transversely positioned 1 cm above the umbilical scar. During the ultrasound scan, the examiner took care not to press the transducer in the abdomen, in order to not underestimate the thickness of the subcutaneous.

### Risk Factor and Covariate Assessment

Risk factors and covariates were measured at the contemporaneous examination. Body mass index (BMI), defined as weight (in kilograms) divided by the square of height (in meters), was measured at each index examination. WC was measured at the level of the umbilicus. Abdominal obesity was defined as  $\geq 80$  cm in women and  $\geq 94$  cm in men. Hypertension was defined as systolic blood pressure 130 mmHg, diastolic blood pressure 85 mmHg. Total, high-density lipoprotein (HDL-C) and low density lipoprotein (LDL-C) cholesterol, and triglycerides were measured on fasting morning samples. Non-HDL-C is easily calculated from a lipid profile (non-HDL-C = total cholesterol minus HDL-C). Diabetes was defined as a fasting plasma glucose level  $\geq 126$  mg/dL. Impaired fasting glucose was defined as a fasting plasma glucose level of 100 to 125 mg/dL among those not treated for diabetes. The main outcome measures were age-standardized prevalence of the MetS per the harmonized American Heart Association/National Heart, Lung, and Blood Institute definition and its component abnormalities. The control group was considered which did not have cardiovascular risk factors. Patients after diagnosis needed medication were referred to specialized treatment.

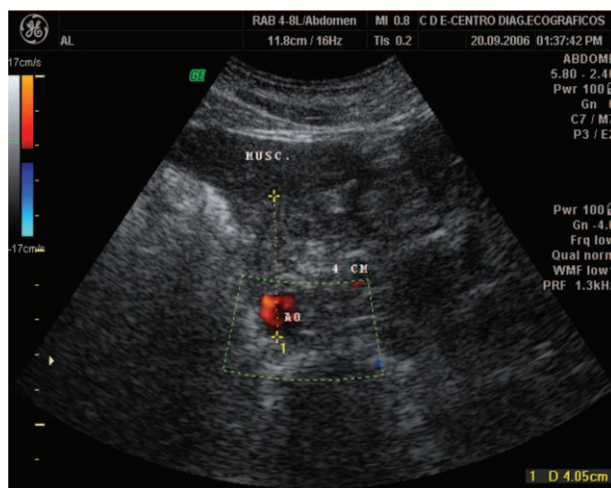


FIGURE 1. The thickness of visceral fat.

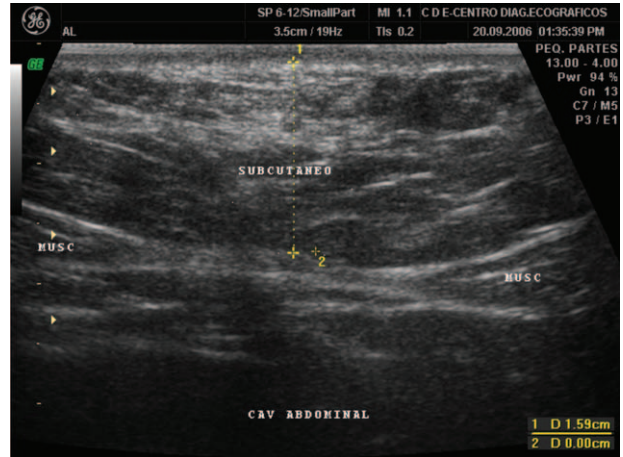


FIGURE 2. The thickness of subcutaneous fat.

### Statistical Analysis

SCF and VF were normally distributed. Sex-specific age-adjusted Pearson correlation coefficients were used to assess simple correlations between SCF and VF and metabolic risk factors. Multivariable linear and logistic regression was used to assess the significance of covariate-adjusted cross-sectional relations between continuous and dichotomous metabolic risk factors and SCF and VF. A *P*-value 0.05 was considered to indicate significance. SPSS Version 21 software (SPSS, Chicago, IL) was used.

## RESULTS

### Baseline Characteristics

Overall, 49 women (W) and 52 men (M) were available for analysis. The mean age of the study sample was 48 W and 52 M years, and 48.5% were women (Table 1); 40.2% was hypertensive, 39.3% had obese, 61.8% abdominal obesity, 32% hypertriglyceridemia, 33.2% low HDL-C and high LDL-C, 40.2% high total cholesterol, 33.2% high non-HDL-C, 22.7% mixed dyslipidemia, 20.2% impaired fasting glucose, and 41.1% had MetS. Mean visceral and SCF thickness were 4.9 and 2.7 cm in W and 6.8 and 2 cm in M, respectively.

### Correlations With VF and SCF

Correlations of VF and SCF with metabolic risk factors are shown in Table 2. VF was significantly associated with the levels of triglycerides ( $P < 0.01$ ,  $r = 0.10$ ), HDL cholesterol ( $P < 0.001$ ,  $r = 0.15$ ), total cholesterol ( $P < 0.01$ ,  $r = 0.10$ ), waist circumference ( $P < 0.0001$ ,  $r = 0.43$ ), systolic blood pressure ( $P < 0.001$ ,  $r = 0.41$ ), and diastolic blood pressure ( $P < 0.001$ ,  $r = 0.32$ ) in women, and with the levels of triglycerides ( $P < 0.002$ ,  $r = 0.14$ ), HDL cholesterol ( $P < 0.032$ ,  $r = 0.07$ ), glucose ( $P < 0.001$ ,  $r = 0.15$ ), alanine aminotransferase (ALT) ( $P < 0.008$ ,  $r = 0.12$ ), gamma-GT ( $P < 0.001$ ,  $r = 0.30$ ), waist circumference ( $P < 0.001$ ,  $r = 0.52$ ), systolic blood pressure ( $P < 0.001$ ,  $r = 0.32$ ), and diastolic blood pressure ( $P < 0.001$ ,  $r = 0.26$ ) in men.

SCF was significantly associated with the levels of triglycerides ( $P < 0.01$ ,  $r = 0.34$ ), LDL cholesterol ( $P < 0.001$ ,  $r = 0.36$ ), total cholesterol ( $P < 0.05$ ,  $r = 0.36$ ),

TABLE 1. Study Sample Characteristics

	Men (52)	Women (49)
Age, y	52 (13)	48 (6.4)
BMI, kg/m <sup>2</sup>	25.9 (4.1)	26.3 (3.4)
Overweight (BMI ≥25 and ≥30), %	26.9	26.5
Obesity Grade 1 (BMI ≥30 and ≥35), %	7.6	18.3
WC, cm	96.2 (11.9)	85.2 (10.9)
WC ≥94 M and W 80 cm, %	55.7	69.3
Triglycerides, mg/dL	167.6 (39–638)	123.8 (44–490)
Hypertriglyceridemia ≥ 150 mg/dL, %	40.3	24.4
HDL cholesterol, mg/dL	45.7 (11.7)	52.2 (11.2)
HDL ≤ 40 M and 50 W (mg/dL), %	34.6	32.6
LDL cholesterol, mg/dL	115 (28.4)	119.7 (39.4)
LDL cholesterol, mg/dL ≥ 130%	34.6	32.6
Total cholesterol, mg/dL	191.3 (38.4)	196.2 (40.3)
Total cholesterol, mg/dL ≥ 200%	36.5	44.8
Non-HDL-C, mg/dL	140.6 (35.7)	141.9 (42)
Non-HDL-C, ≥160 M and 150 W (mg/dL), %	32.6	34.6
Mixed dyslipidemia, %	13.4	32.6
AST, U/L	17.2 (7.8)	12.9 (2.9)
ALT, U/L	40.3 (23.3)	25.2 (11)
Gamma-GT, U/L	43.7 (34.4)	25.2 (17.1)
Systolic blood pressure, mmHg	126.4 (15.4)	121.3 (16.8)
Diastolic blood pressure, mmHg	84.8 (9.5)	81.5 (9.7)
Hypertension, %	48.7	32.6
Fasting plasma glucose, mg/dL	96.3 (10.8)	90.8 (10.8)
Impaired fasting glucose, %	28.8	12.2
MetS, %	38.4	44.8
Postmenopausal, %	...	36.7
Hormone replacement therapy, %	...	35.6
SCF, cm	2 (0.8)	2.7 (1)
VF, cm	6.8 (2)	4.9 (1.6)

Data are presented as mean ± SD when appropriate.

ALT = alanine aminotransferase, AST = aspartate transaminase, BMI = body mass index, GGT = gamma glutamyl transferase, HDL-C = high density lipoprotein cholesterol, LDL-C = low density lipoprotein cholesterol, M = men, MetS = metabolic syndrome, non-HDL = nonhigh density lipoprotein cholesterol, SCF = subcutaneous fat, VF = visceral fat, W = women, WC = waist circumference.

waist circumference ( $P < 0.0001$ ,  $r = 0.62$ ), systolic and diastolic blood pressure ( $P < 0.05$ ,  $r = 0.34$ ) in woman, and with the waist circumference ( $P < 0.001$ ,  $r = 0.65$ ), and MetS ( $P < 0.05$ ,  $r = 0.11$ ) in men.

### Multivariable-Adjusted Regressions With VF, SCF, and Metabolic Risk Factor Variables

Results of multivariable-adjusted general linear regression analyses for VF and SCF for both continuous and dichotomous metabolic risk factors are shown in Table 3. The VF was significantly associated with the levels of triglycerides ( $P < 0.01$ ,  $r = 0.12$ ), waist circumference ( $P < 0.0001$ ,  $r = 0.13$ ), systolic blood pressure ( $P < 0.001$ ,  $r = 0.44$ ), LDL-C ( $P < 0.01$ ,  $r = 0.17$ ), SBP ( $P < 0.001$ ,  $r = 0.42$ ), and DBP ( $P < 0.001$ ,  $r = 0.33$ ) in women, and with the levels of triglycerides ( $P < 0.001$ ,  $r = 0.16$ ), HDL cholesterol ( $P < 0.05$ ,  $r = 0.09$ ), glucose ( $P < 0.001$ ,  $r = 0.15$ ), ALT ( $P < 0.05$ ,  $r = 0.13$ ), gamma-GT ( $P < 0.0001$ ,  $r = 0.32$ ), waist circumference ( $P < 0.001$ ,  $r = 0.53$ ), SBP ( $P < 0.001$ ,  $r = 0.28$ ), DBP ( $P < 0.0001$ ,  $r = 0.34$ ), FPG ( $P < 0.001$ ,  $r = 0.02$ ), and MetS ( $P < 0.001$ ,  $r = 0.11$ ) in men.

SCF was significantly associated with waist circumference ( $P < 0.0001$ ,  $r = 0.18$ ) in women, and with waist circumference ( $P < 0.001$ ,  $r = 0.26$ ), SBP ( $P < 0.05$ ,  $r = 0.09$ ), and MetS ( $P < 0.05$ ,  $r = 0.11$ ) in men.

### Sex Interaction

We observed a significant sex interaction, which suggests that SCF are associated with more adverse risk factor profiles in women, and VF in men (Table 3).

### DISCUSSION

In the Uberlândia Heart Study, thickness US measures of both VF and SCF were correlated with multiple metabolic risk factors, although risk factor correlations with VF were consistently significantly stronger than those for SCF. VF was more strongly associated with metabolic risk factors in men than in women after multivariable-adjusted regressions. SCF was more strongly associated with metabolic risk factors in women than in men after correlation.

VF has traditionally been considered the more associated with risk factors compartment compared with SCF, but data confirming these relations in women and men have been lacking.

**TABLE 2.** Sex-Adjusted Pearson Correlation Coefficients Between Metabolic Risk Factors and VF and SCF Thickness

	Men				Women			
	VF		SCF		VF		SCF	
	C	MetS	C	MetS	C	MetS	C	MetS
BMI, kg/m <sup>2</sup>	0.18	-0.32	0.11	0.41	0.49 <sup>‡</sup>	0	0.39*	0.13
WC, cm	0.67 <sup>‡</sup>	0.51 <sup>‡</sup>	0.65 <sup>‡</sup>	0.32	0.66 <sup>‡</sup>	0.21	0.62 <sup>‡</sup>	0.68 <sup>†</sup>
Triglycerides, mg/dL	0.25	0.01	0.31	-0.20	0.25	-0.04	0.34 <sup>†</sup>	0.15
HDL cholesterol, mg/dL	0	0.7	-0.30	0.23	-0.20	0.02	-0.09	0.06
LDL cholesterol, mg/dL	0.24	-0.06	0.24	-0.013	0.47 <sup>†</sup>	0.14	0.46 <sup>†</sup>	-0.31
Total cholesterol, mg/dL	0.23	0.21	0.10	-0.43*	0.29	0.09	0.36*	-0.23
Non-HDL, mg/dL	0.18	-0.1	-0.15	0.30	0.10	0.15	0.20	-0.16
AST, U/L	0.09	0.07	0.16	0.01	-0.06	0.43	0.02	0.03
ALT, U/L	0.10	0.28	0.07	0.15	0.14	0	0.18	0.05
Gamma-GT	0.41*	0.49*	0.23	-0.07	0.11	-0.03	-0.013	0.02
Systolic blood pressure, mmHg	0.31	0.37	-0.01	0.19	0.34*	0.05*	0.34*	0.49
Diastolic blood pressure, mmHg	0.06	0.35	0.35	0.35	0.88 <sup>‡</sup>	0.19	0.34*	0.37
Fasting plasma glucose, mg/dL	0.23	0.45*	0.32	-0.23	0.14	0.05	0.14	-0.07
MetS		0.41 <sup>‡</sup>		0.11*		0.21 <sup>†</sup>		0

ALT = alanine aminotransferase, AST = aspartate transaminase, BMI = body mass index, C = control, GT = gamma glutamyl transferase, HDL-C = high density lipoprotein cholesterol, LDL-C = low density lipoprotein cholesterol, MetS = metabolic syndrome, non-HDL = nonhigh density lipoprotein cholesterol, SCF = subcutaneous fat, VF = visceral fat, WC = waist circumference.

\* *P* < 0.05.

† *P* < 0.01.

‡ *P* < 0.001.

**TABLE 3.** Sex-Specific Multivariable-Adjusted\* Regressions for VF and SCF With Continuous Metabolic Risk Factors (Top) and Dichotomous Risk Factors

	Men				Women			
	VF		SCF		VF		SCF	
	C	MetS	C	MetS	C	MetS	C	MetS
BMI, kg/m <sup>2</sup>	0.10	-0.13	0.09*	0.18	1 <sup>†</sup>	0.18	0.18	0.36
WC, cm	0.53 <sup>‡</sup>	0.26*	0.7 <sup>‡</sup>	0.10	0.44 <sup>‡</sup>	0.4	0.18 <sup>‡</sup>	0.46 <sup>†</sup>
Triglycerides, mg/dL	0.16 <sup>†</sup>	0	0.01	0.04	0.12 <sup>†</sup>	0	0.06	0.02
HDL cholesterol, mg/dL	0.09*	0	0.04	0.05	0.05	0	0	0
LDL cholesterol, mg/dL	0	0	0	0.01	0.17 <sup>†</sup>	0.2	0.05	0.09
Total cholesterol, mg/dL	0.03	0.04	0.01	0.19	0.11*	0	0.02	0.05
Non-HDL, mg/dL	0.85	-0.17	0.15	-0.57	-0.47	0.55	0.48	0.42
AST, U/L	0.01	0	0	0	0	0.19	0	0
ALT, U/L	0.13*	0.08	0.05	0.2	0.05	0	0.02	0
Gamma-GT	0.32 <sup>‡</sup>	0.24*	0.02	0	0	0	0	0
Systolic blood pressure, mmHg	0.28 <sup>‡</sup>	0.13	0.09*	0.03	0.42 <sup>‡</sup>	0.26	0.02	0.24
Diastolic blood pressure, mmHg	0.34 <sup>‡</sup>	0.12	0.06	0.12	0.33 <sup>†</sup>	0.03	0.02	0.14
Fasting plasma glucose, mg/dL	0.02 <sup>‡</sup>	0.21*	0.01	0.05	0.08	0	0.01	0
MetS	0.41 <sup>‡</sup>	0.11*	0.21 <sup>†</sup>	0.05				

ALT = alanine aminotransferase, AST = aspartate transaminase, BMI = body mass index, C = control, GGT = gamma glutamyl transferase, HDL-C = high density lipoprotein cholesterol, LDL-C = low density lipoprotein cholesterol, MetS = metabolic syndrome, non-HDL = nonhigh density lipoprotein cholesterol, SCF = subcutaneous fat, VF = visceral fat, WC = waist circumference.

\* *P* < 0.05.

† *P* < 0.01.

‡ *P* < 0.001.

The proposed mechanism for the increased metabolic risk is the possibility that the metabolically active adipose tissue found in the visceral region of be become dysfunctional, increasing the secretion of substances that alter the metabolic profile and produce chronic inflammation. Several studies have demonstrated that the VF compartment is metabolically active, secreting such vasoactive substances as inflammatory markers, adipocytokines, markers of hemostasis and fibrinolysis and growth factors which may contribute to its role in cardiometabolic risk factor manifestation.<sup>7,8,25–34</sup>

Our results are consistent with these findings character, community-based sample of men and women in that we show that all cardiometabolic risk factors examined were more strongly associated with VF and SCF.

The Dallas Heart Study, which examined metabolic risk factors relations in 1934 black and white women and men with VF and SCF as assessed by magnetic resonance imaging (MRI) are associated positively with prevalence of hypertension, but only VF provides significant information above and beyond BMI and WC.<sup>35</sup>

Other studies have demonstrated relations between VF and hypertension.<sup>15,16,36–38</sup>

Our results show that both VF are associated positively hypertension, WC and MetS.

In a Japanese study of 973 men who made a computed tomography (CT) to assess VF, a significant association was observed with metabolic risk factors. The incidences of components of metabolic risk factors were significantly higher among individuals with a greater increase in VF ( $P < 0.001$ ). Significant increases the odds ratio for the incidence of high triglycerides and low HDL-C were observed among individuals  $\geq 50$  cm<sup>2</sup> increased VF.<sup>39</sup>

In a study of 607 patients who underwent CT for evaluation of VF. In both men and women, the VF showed significant positive correlations with age, BMI, waist circumference, SCF area, VF area/SCF area (v/s) ratio, systolic blood pressure, blood pressure diastolic blood sugar fasting (FBS), hemoglobin A1c (HbA1c), high density lipoprotein cholesterol (HDL), triglycerides (TG) and significant negative correlation between the levels of HDL and adiponectin. The total cholesterol (TC), low density lipoprotein (LDL), non-HDL not, can they be correlated with VF in men or women.<sup>40</sup> We also found that both VF and SCF were associated with triglycerides, WC and MetS in women and men.

In another study of 128 Japanese Americans who were followed for a period of 10 to 11 years, who confirmed 57 cases of IGT. IGT significant predictors included VF area (odds ratio [OR] 1 SD increase of 3.82, 95% CI: 1.63–8.94 in fasting plasma glucose [g] at 4.5 mmol/L), HOMA-IR (2.41, 1.15–5.04), incremental insulin response (IIR) (0.30, 0.13–0.69 PPG at a level of 4.5 mmol/L), by the interactions VF and FPG ( $P < 0.003$ ) and IIR by FPG ( $P < 0.03$ ) after adjustment for age, sex, FPG, and BMI.<sup>11</sup> In our study was seen a significant association VF with impaired glucose tolerance in men with MetS ( $P < 0.05$ ,  $r = 0.45$ ).

Impaired fasting glucose and diabetes, multiple prior studies have demonstrated relations between The VF and pre-diabetic hyperglycemia and diabetes. Although our results show that VF is more highly correlated with MetS than is SCF, VF was an important correlate of the MetS.

Other authors studied 1511 individuals in the MESA (Multi-Ethnic Study of Atherosclerosis) with adiposity assessment by CT. A total of 253 participants without MetS at initial scan underwent repeat CT (median interval 3.3 years). Higher

VF was associated with cardiometabolic risk and coronary artery calcification, regardless of BMI. VF was more strongly associated with incident MetS than SCF regardless of weight, and was modestly associated with BMI.<sup>41</sup> The new findings in our study was the correlation of ectopic fat with non-HDL cholesterol, liver enzymes, gamma-GT, and Mets.

## Strengths and Limitations

This prospective cross-sectional study was limited by its sample size. Strengths of our study include the use of a community-based sample with participants not enriched for adiposity-related traits and high risk for CVD. Routine screening of metabolic risk factors was performed, and adjustment was made for several potential confounders. We used the highly reproducible thickness method of VF and SCF assessment, and which has a high accuracy and reproducibility as compared MRI and CT. Not a multicenter study that could allow a generalization of the data for other ethnicities.

## CONCLUSION

Both VF and SCF are associated with an adverse metabolic risk but, SCF provides better information in women.

## REFERENCES

1. Thom T, Haase N, Rosamond W, et al. Heart disease and stroke statistics–2006 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*. 2006;113:e85–e151.
2. Fox CS, Evans JC, Larson MG, et al. Temporal trends in coronary heart disease mortality and sudden cardiac death from 1950 to 1999: the Framingham Heart Study. *Circulation*. 2004;110:522–527.
3. Hu FB, Stampfer MJ, Manson JE, et al. Trends in the incidence of coronary heart disease and changes in diet and lifestyle in women. *N Engl J Med*. 2000;343:530–537.
4. Flegal KM, Carroll MD, Ogden CL, et al. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA*. 2002;288:1723–1727.
5. Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA*. 2006;295:1549–1555.
6. Klein S. The case of visceral fat: argument for the defense. *J Clin Invest*. 2004;113:1530–1532.
7. Goodpaster BH, Krishnaswami S, Resnick H, et al. Association between regional adipose tissue distribution and both type 2 diabetes and impaired glucose tolerance in elderly men and women. *Diabetes Care*. 2003;26:372–379.
8. Kanaya AM, Harris T, Goodpaster BH, et al. Adipocytokines attenuate the association between visceral adiposity and diabetes in older adults. *Diabetes Care*. 2004;27:1375–1380.
9. DeMarco VG, Aroor AR, Sowers JR. The pathophysiology of hypertension in patients with obesity. *Nat Rev Endocrinol*. 2014;10:364–376.
10. Soo L, James BM. Links between ectopic fat and vascular disease in humans. *Arterioscler Thromb Vasc Biol*. 2014;34:1820–1826.
11. Boyko EJ, Fujimoto WY, Leonetti DL, et al. Visceral adiposity and risk of type 2 diabetes: a prospective study among Japanese Americans. *Diabetes Care*. 2000;23:465–471.
12. Tulloch-Reid MK, Hanson RL, Sebring NG, et al. Both subcutaneous and visceral adipose tissue correlate highly with insulin resistance in African Americans. *Obes Res*. 2004;12:1352–1359.

13. Wagenknecht LE, Langefeld CD, Scherzinger AL, et al. Insulin sensitivity, insulin secretion, and abdominal fat: the Insulin Resistance Atherosclerosis Study (IRAS) Family Study. *Diabetes*. 2003;52:2490–2496.
14. Hayashi T, Boyko EJ, Leonetti DL, et al. Visceral adiposity is an independent predictor of incident hypertension in Japanese Americans. *Ann Intern Med*. 2004;140:992–1000.
15. Sironi AM, Gastaldelli A, Mari A, et al. Visceral fat in hypertension: influence on insulin resistance and beta-cell function *Hypertension*. 2004;44:127–133.
16. Ding J, Visser M, Kritchevsky SB, et al. The association of regional fat depots with hypertension in older persons of white and African American ethnicity *Am J Hypertens*. 2004;17:971–976.
17. Pascot A, Lemieux S, Lemieux I, et al. Age-related increase in visceral adipose tissue and body fat and the metabolic risk profile of premenopausal women. *Diabetes Care*. 1999;22:1471–1478.
18. Nagaretani H, Nakamura T, Funahashi T, et al. Visceral fat is a major contributor for multiple risk factor clustering in Japanese men with impaired glucose tolerance. *Diabetes Care*. 2001;24:2127–2133.
19. Nicklas BJ, Penninx BW, Ryan AS, et al. Visceral adipose tissue cutoffs associated with metabolic risk factors for coronary heart disease in women. *Diabetes Care*. 2003;26:1413–1420.
20. Lemieux S, Prud'homme D, Moorjani S, et al. Do elevated levels of abdominal visceral adipose tissue contribute to age-related differences in plasma lipoprotein concentrations in men? *Atherosclerosis*. 1995;118:155–164.
21. Kobayashi H, Nakamura T, Miyaoka K, et al. Visceral fat accumulation contributes to insulin resistance, small-sized low-density lipoprotein, and progression of coronary artery disease in middle-aged non-obese Japanese men. *Jpn Circ J*. 2001;65:193–199.
22. Mori Y, Hoshino K, Yokota K, et al. Increased visceral fat and impaired glucose tolerance predict the increased risk of metabolic syndrome in Japanese middle-aged men. *Exp Clin Endocrinol Diabetes*. 2005;113:334–339.
23. Goodpaster BH, Krishnaswami S, Harris TB, et al. Obesity, regional body fat distribution, and the metabolic syndrome in older men and women. *Arch Intern Med*. 2005;165:777–783.
24. Carr DB, Utzschneider KM, Hull RL, et al. Intra-abdominal fat is a major determinant of the National Cholesterol Education Program Adult Treatment Panel III criteria for the metabolic syndrome. *Diabetes*. 2004;53:2087–2094.
25. Hayashi T, Boyko EJ, Leonetti DL, et al. Visceral adiposity and the risk of impaired glucose tolerance: a prospective study among Japanese Americans. *Diabetes Care*. 2003;26:650–655.
26. Lemieux I, Pascot A, Prud'homme D, et al. Elevated C-reactive protein: another component of the atherothrombotic profile of abdominal obesity. *Arterioscler Thromb Vasc Biol*. 2001;21:961–967.
27. Azuma K, Katsukawa F, Oguchi S, et al. Correlation between serum resistin level and adiposity in obese individuals. *Obes Res*. 2003;11:997–1001.
28. Fukuhara A, Matsuda M, Nishizawa M, et al. Visfatin: a protein secreted by visceral fat that mimics the effects of insulin. *Science*. 2005;307:426–430.
29. Cigolini M, Targher G, Bergamo Andreis I, et al. Visceral fat accumulation and its relation to plasma hemostatic factors in healthy men. *Arterioscler Thromb Vasc Biol*. 1996;16:368–374.
30. Mertens I, Van Gaal LF. Visceral fat as a determinant of fibrinolysis and hemostasis. *Semin Vasc Med*. 2005;5:48–55.
31. Miyazawa-Hoshimoto S, Takahashi K, Bujo H, et al. Elevated serum vascular endothelial growth factor is associated with visceral fat accumulation in human obese subjects. *Diabetologia*. 2003;46:1483–1488.
32. Giusti V, Suter M, Verdumo C, et al. Molecular determinants of human adipose tissue: differences between visceral and subcutaneous compartments in obese women. *J Clin Endocrinol Metab*. 2004;89:1379–1384.
33. Nazare JA, Smith J, Borel AL, et al. Usefulness of measuring both body mass index and waist circumference for the estimation of visceral adiposity and related cardiometabolic risk profile (from the INSPIRE ME IAA Study). *Am J Cardiol*. 2015;115:307–315.
34. Fukuda S, Hirata A, Nishizawa H, et al. Systemic arteriosclerosis and eating behavior in Japanese type 2 diabetic patients with visceral fat accumulation *Cardiovasc Diabetol*. 2015;14:8.
35. Vega GL, Adams-Huet B, Peshock R, et al. Influence of body fat content and distribution on variation in metabolic risk. *J Clin Endocrinol Metab*. 2006;91:4459–4466.
36. Hayashi T, Boyko EJ, Leonetti DL, et al. Visceral adiposity is an independent predictor of incident hypertension in Japanese Americans. *Ann Intern Med*. 2004;140:992–1000.
37. Park YW, Allison DB, Heymsfield SB, et al. Larger amounts of visceral adipose tissue in Asian Americans. *Obes Res*. 2001;9:381–387.
38. Hayashi T, Boyko EJ, Leonetti DL, et al. Visceral adiposity and the prevalence of hypertension in Japanese Americans. *Circulation*. 2003;108:1718–1723.
39. Matsushita Y, Nakagawa T, Yamamoto S, et al. Effect of longitudinal changes in visceral fat area on incidence of metabolic risk factors: the Hitachi health study. *Obesity*. 2013;21:2126–2129.
40. Shiina Y, Homma Y. Relationships between the visceral fat area on CT and coronary risk factor markers. *Intern Med*. 2013;52:1775–1780.
41. Shah RV, Murthy VL, Abbasi SA, et al. Visceral adiposity and the risk of metabolic syndrome across body mass index: the MESA Study. *JACC Cardiovasc Imaging*. 2014;7:1221–1235.