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Stronger associations of sagittal abdominal diameter with atherogenic lipoprotein subfractions than waist circumference in middle-aged US white and Japanese men

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Abstract

Both sagittal abdominal diameter (SAD) and waist circumference (WC) highly correlate with visceral adipose tissue (VAT) being linked to an atherogenic lipoprotein profile. However, it is uncertain whether SAD is a better correlate of atherogenic lipoprotein subfractions than WC. We examined relative associations of SAD vs WC with lipoprotein subfractions for US white and Japanese men, concurrently examining the associations of VAT vs subcutaneous adipose tissue with lipoprotein subfractions. A population-based sample of 260 white and 282 Japanese men aged 40 to 49 years was examined for VAT and subcutaneous adipose tissue by computed tomography; SAD and WC by a portable sliding-beam caliper and a measuring tape, respectively; and lipoprotein subfractions by nuclear magnetic resonance spectroscopy. Both SAD and WC were significantly and positively associated with large very low-density lipoprotein and total and small low-density lipoprotein particle concentrations, and inversely associated with large high-density lipoprotein particle concentration for both white and Japanese men. In body mass index–adjusted regression models, the significant associations of SAD remained for both white and Japanese men, whereas those of WC became nonsignificant for white men. When SAD and WC were simultaneously included into the body mass index–adjusted models, the associations of SAD remained significant and statistically stronger than those of WC for both white and Japanese men. Furthermore, the pattern of the associations of SAD with those lipoprotein subfractions was comparable to that of the associations of VAT. Sagittal abdominal diameter was comparable to VAT and stronger than WC in the associations with atherogenic lipoprotein subfractions for middle-aged, nondiabetic, white and Japanese men.

1. Introduction

Lipoprotein subfractions may provide an atherogenic feature for prediction of the risk for coronary heart disease (CHD) [1,2]. Epidemiologic studies have demonstrated that total low-density lipoprotein (LDL) particle concentration is a more predictive measure for CHD risk than LDL cholesterol [1]. Large high-density lipoprotein (HDL) particle concentration is protective against carotid atherosclerosis, coronary stenosis, and cardiovascular events [1,3,4]. Large very low-density lipoprotein (VLDL) and small LDL particle concentrations may be related to an increased prevalence of coronary calcification and cardiovascular risk [1,5].

Institutional approval: The study was approved by the Institutional Review Boards of University of Pittsburgh, Pittsburgh, PA, and Shiga University of Medical Science, Otsu, Japan.

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Increased abdominal fat contributes to CHD risk [6,7]. Of abdominal fat compartments, visceral adipose tissue (VAT) is an independent predictor of CHD risk [8,9], closely linked with metabolic abnormalities including elevated triglyceride levels [10]. Compared with subcutaneous adipose tissue (SAT), VAT is more strongly associated with metabolic and cardiovascular factors, independent of overall obesity [11,12]. Recently, VAT was proven to be more strongly associated with elevated atherogenic lipoprotein subfractions than SAT [13], including elevated concentrations of large VLDL and total and small LDL particles and a reduced concentration of large HDL particles [14]. The elevated concentrations of atherogenic lipoprotein subfractions may be related to an increased secretion of triglyceride-rich lipoproteins induced by VAT-linked free fatty acids flux toward liver [15]. Together with an increased activity of hepatic lipase, the elevation of triglyceride-rich lipoproteins (ie, large VLDL particles) results in increased production of small LDL particles, which is concurrently responsible for decreased large HDL particles [16].

Abdominal adipose tissues (AATs) including VAT and SAT can be measured by computed tomography (CT), which is not easily available in clinical practice, to identify abdominally obese individuals. Waist circumference (WC) and sagittal abdominal diameter (SAD) are inexpensive and simple anthropometric measures of AATs. Waist circumference is a major component of the diagnosis criteria of metabolic syndrome defined by the National Cholesterol Education Program's Adult Treatment Panel III and the International Diabetes Federation [17,18]. Sagittal abdominal diameter is a strong correlate of VAT [19,20]. Several studies have suggested that SAD is a better correlate of cardiovascular and metabolic risk profile than WC, particularly including elevated triglycerides, reduced HDL cholesterol, and elevated apolipoprotein B levels [21,22]. However, no previous studies have reported the relative importance of SAD compared with WC in the associations with lipoprotein subfractions.

We hypothesized that SAD would be a better correlate of the profile of atherogenic lipoprotein subfractions than WC, concurrently examining the associations of VAT vs SAT with those lipoproteins. We tested the hypothesis in each population-based sample of US white men and Japanese men in Japan aged 40 to 49 years without cardiovascular disease and diabetes from the **E**lectron-beam tomography and **R**isk **A**ssessment in **J**apanese and **U**S men in the **p**ost–World War II birth (ERA-JUMP) cohort, a population-based cross-sectional study [23].

2. Methods

2.1. Study participants

During 2002 to 2006, a population-based sample of randomly selected men aged 40 to 49 years was obtained: 310 white men from Allegheny County, Pennsylvania, and 313 Japanese men from Kusatsu, Shiga, Japan. [23] All the participants were without clinical cardiovascular disease, type 1 diabetes mellitus, cancer except skin cancer in the past 2 years, renal failure, and genetic familial hyperlipidemia. Of the original sample, we excluded men taking lipid-lowering medications (n = 49), those with type 2 diabetes mellitus diabetes (n = 26), and those with missing values (n = 6). Type 2 diabetes mellitus was defined as fasting glucose of ≥126 mg/dL or taking diabetes medication. The final sample was 542 (260 white and 282 Japanese men).

Written informed consents were obtained from all participants. The study was approved by the Institutional Review Boards of University of Pittsburgh, Pittsburgh, PA, and Shiga University of Medical Science, Otsu, Japan.

All participants underwent a physical examination, and completed a lifestyle questionnaire (eg, smoking and alcohol consumption) and a laboratory assessment as described previously [24]. Venipuncture was performed early in the clinic visit after a 12-hour fast, and samples were stored at −80°C and shipped on dry ice to the University of Pittsburgh. Data collection was standardized across research centers.

2.2. Body mass index and abdominal adiposity indices

Body mass index (BMI) was calculated using body weight and height (in kilograms per square meter). Waist circumference and SAD were measured in underwear. Waist circumference was measured twice at the umbilical level using a measuring tape while the participant was standing upright, and an average of the 2 measurements was taken. Sagittal abdominal diameter was measured using a portable sliding-beam caliper (Holtain-Kahn Abdominal Caliper; Holtain, Dyfed, Wales). While the participant was laying supine on an examining table with legs straight, the base of the caliper was placed under the subject's back; and then the caliper's upper arm was slid down to a point midway between iliac crests without compression. Afterward, SAD, the so-called height of the abdomen (ie, anterioposterior diameter), was determined with a ruler to the nearest millimeter of the caliper. The intraobserver coefficient was 2.5%, and intraclass coefficient was 95.8% [25]. Areas of the whole AAT and VAT were determined at the level between the fourth and fifth lumbar vertebrae using CT images (GE-Imatron C150; GE Medical Systems, South San Francisco, CA). Areas of SAT were calculated as AAT minus VAT. The intraclass correlation coefficients at our reading center were 99% for SAT and 99% for VAT [26].

2.3. Lipoprotein measurements

Nuclear magnetic resonance spectroscopy (LipoScience, Raleigh, NC) was performed to quantify serum lipoproteins of different sizes [27]. Particle concentrations of the following lipoproteins were determined: VLDL (large, >60 nm; medium, 35-60 nm; and small, 27-35 nm), LDL (intermediate-density lipoprotein [IDL], 23-27 nm; large, 21.3-23 nm; small, 18.3-21.2 nm), and HDL (large, 8.8-13.0 nm; medium, 8.2-8.8 nm; and small, 7.3-8.2 nm) [28].

Weighted average particle sizes were calculated from the subclass levels.

2.4. Statistical analyses

To examine the association between each abdominal adiposity index (a primary predictor variable) and each lipoprotein (an outcome variable), the multiple linear regression analysis was performed using 3 different models. In model I, age, pack years of smoking, and alcohol consumption were adjusted for. In model II, BMI was further adjusted for. In model III, SAD and WC or VAT and SAT were simultaneously added to model II. To determine if there was any significant difference in β coefficients between SAD and WC or between VAT and SAT in associations with each lipoprotein, the linear combinations of coefficient estimators were performed. Statistical significance was considered to be $P < .05$. All statistical analyses were performed with STATA 10.0 for Windows (StataCorp, College Station, TX).

3. Results

Our cohort had mean BMIs of 28 kg/cm^2 for white men and 24 kg/cm^2 for Japanese men (Table 1). According to the BMI category defined by the World Health Organization [29], 71% of white men and 27% of Japanese men were overweight or obese. White men had significantly greater levels of VAT and SAT (both in square centimeters) than Japanese men.

Sagittal abdominal diameter and WC correlated highly with each other ($r = .82$ for white men and $r = .75$ for Japanese men) (Table 2). Age-adjusted partial correlation analysis showed that SAD appeared to be comparable with WC in the correlations with VAT, whereas SAD appeared to be weaker than WC in the correlations with SAT for both white and Japanese men. For additional information, we performed the multiple regression analysis for which SAD and WC were simultaneously included as predictor variables into the adjusted models for age and BMI. The analysis showed that both SAD and WC explained VAT significantly ($\beta = .23$ and $\beta = .43$, $P < .01$ for white men; $\beta = .23$ and $\beta = .52$, $P < .001$ for Japanese men) and that the coefficients between SAD and WC in the associations with VAT did not differ significantly. On the other hand, WC, but not SAD, explained SAT significantly ($\beta = .72$ for white men, $\beta = .71$ for Japanese men, $P < .001$) for both white and Japanese men.

Both SAD and WC were significantly and positively associated with total and large VLDL particle concentrations for both white and Japanese men (model I); after further adjustment for BMI (model II), WC was not significantly associated any more for white men, but remained significantly associated for Japanese men (Table 3). In model II for Japanese men, WC appeared to have smaller R^2 values than SAD in the associations with total and large VLDL particle concentrations ($R^2 = .10$ vs $R^2 = .12$ for total VLDL particle concentration; $R^2 = .07$ vs $R^2 = .09$ for large VLDL particle concentration). Furthermore, when SAD and WC were simultaneously included into model III, WC was not significantly associated any more even for Japanese men. Both VAT and SAT were significantly and positively associated with large VLDL particle concentrations for both white and Japanese men (model I) (Table 3). After further adjustment for BMI (model II), such associations of SAT did not remain for white or Japanese men; for white men, SAT was significantly and inversely associated with large VLDL particle concentration.

Both SAD and WC were significantly and positively associated with total and small LDL and IDL particle concentrations, and inversely associated with LDL size for both white and Japanese men (model I) (Table 4). When SAD and WC were simultaneously included into model III, the significant associations of SAD remained for both white and Japanese men, whereas those of WC became nonsignificant for white men and attenuated for Japanese men; the associations of WC with total LDL and IDL particle concentrations for Japanese men became nonsignificant, and those of WC with small LDL particle concentration and LDL size attenuated ($\beta = -10.37$ to -6.24 for small LDL particle concentration; $\beta = -.05$ to $-.03$ for LDL size). However, the associations of SAD with small LDL particle concentrations and LDL size were statistically stronger than those of WC ($\Delta\beta$ between SAD and WC, $P = .012$ for small LDL particle concentration and $P = .005$ for LDL size). Visceral adipose tissue was significantly and positively associated with total and small LDL and IDL particle concentrations, and inversely associated with LDL size for both white and Japanese men in models I, II and III (Table 4). However, when VAT and SAT were simultaneously included into BMI-adjusted models (model III), SAT was not significantly associated with total and small LDL particle concentrations for both white and Japanese men; in particular, for white men, SAT showed an inverse association with small LDL particle concentration and a positive association with LDL size.

Both SAD and WC were significantly and inversely associated with large HDL particle concentration and HDL size for both white and Japanese men (model I) (Table 5). In further-adjusted models II and III, SAD remained significantly associated, whereas WC was not significantly associated any more for white men, but was still significantly associated for Japanese men. When SAD and WC were simultaneously included into model III, the associations of SAD with large HDL particle concentration and HDL size were statistically stronger than those of WC ($\Delta\beta$ between SAD and WC, $P = .037$ for large HDL particle concentration and $P = .025$ for HDL size). Visceral adipose tissue was significantly and inversely associated with large HDL particle concentration and HDL size for both white and Japanese men in models I, II, and III. When VAT and SAT were simultaneously included into BMI-adjusted models

Table 1
Basic characteristics of the study participants in 2002-2006 (N = 542)

	Mean (SD) or median (25th-75th percentile)				
	White (n = 260)		Japanese (n = 282)		*P*
Age (y)	44.9 (2.8)		45.1 (2.8)		.343
BMI (kg/m^2)	27.5 (3.9)		23.5 (3.0)		<.001
<25	75 (28.9)		206 (73.1)		
25-<30	122 (46.9)		68 (24.1)		
≥30	63 (24.2)		8 (2.8)		
VAT (cm^2)	99.6 (42.2)		78.3 (30.8)		<.001
SAT (cm^2)	145.8 (62.9)		79.3 (34.5)		<.001
WC (cm)	97.5 (10.8)		84.7 (8.0)		<.001
SAD (cm)	20.3 (2.7)		18.7 (1.8)		<.001
Systolic BP (mm Hg)	122.6 (11.4)		124. 5 (15.7)		.107
Diastolic BP (mm Hg)	73.1 (8.9)		76.2 (11.7)		<.001
Hypertension (n [%])	33 (12.7)		68 (24.1)		.001
Hypertension medication (n [%])	15 (5.8)		12 (4.3)		.418
Pack years of smoking (y)	3.5 (8.3)		19.8 (16.7)		<.001
Alcohol consumption (g/d)	10.3 (12.3)		26.2 (27.6)		<.001
Lipids					
Total cholesterol (mg/dL)	215.4 (37.1)		216.0 (34.8)		.844
Triglycerides (mg/dL)	148.9 (102.6)	124 (91-184.5)	151.2 (73.5)	134.5 (102–180)	.077
LDL-C (mg/dL)	137.7 (33.2)		131.6 (35.9)		.042
HDL-C (mg/dL)	48.5 (12.9)		54.2 (13.8)		<.001
Lipoprotein subfractions					
VLDL (nmol/L)					
Total	92.5 (43.7)	84.0 (61.2-117.9)	89.8 (44.9)	88.1(55.5-121.5)	.695
Large	4.4 (6.6)	1.6 (0.6-5.7)	2.5 (4.6)	0.4 (0.1-2.8)	<.001
Medium	40.7 (32.0)	33.8 (16.6-57.8)	44.5 (35.4)	39.6 (18.3-58.1)	.173
Small	47.4 (21.3)	45.6 (33.8-58.4)	42.8 (24.1)	40.9 (26.1-55.4)	.007
LDL (nmol/L)					
Total	1,472.7 (398.5)	1467.1(1171.3-1738.5)	1,382.9 (439.2)	1343 (1054-1677)	
Intermediate	52.1 (49.1)	41.8 (11.2-78.7)	32.9 (41.7)	16 (0-50)	.006
Large	535.0 (278.4)	519.0 (311.6-725.0)	512.4 (230.0)	501(352-674)	<.001
Small	884.9 (505.8)	799.7 (482.2-1271.3)	837.6 (508.7)	806.5(471-1179)	.484
HDL (μmol/L)					
Total	31.3 (5.9)	30.9 (27.5-34.6)	35.2 (6.5)	34.8 (30.5-39.5)	.277
Large	5.1 (3.2)	4.7 (2.8-7.0)	8.7 (4.1)	8.4 (5.5-11.7)	<.001
Medium	1.1 (2.2)	0.03 (0-1.0)	2.8 (4.4)	1 (0-3.4)	<.001
Small	25.10 (4.47)	25.2 (22.0-28.3)	23.8 (5.5)	24.2 (20.2-27.5)	.003
Size (nm)					
VLDL	49.75 (7.76)	48.0 (44.7-53.0)	44.0 (7.5)	42.4 (39.2-48.5)	<.001
LDL	21.0 (.9)	21.0 (20.3-21.7)	21.1 (0.8)	21.1 (20.4-21.7)	.281
HDL	8.6 (.5)	8.5 (8.3-9.0)	9.1 (0.5)	9.1 (8.8-9.4)	<.001

Values are means (SD) and median (25th-75th percentile) for continuous variables and number (percentages) for categorical variables. Difference between white and Japanese men: *P* values for categorical variables were obtained from χ^2 tests. *P* values for continuous variables were obtained from the *t* test or Wilcoxon rank sum test. BP indicates blood pressure; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

(model III), the significant associations of VAT remained, whereas those of SAT remained population-specific: significant and positive associations for white men vs nonsignificant associations for Japanese men.

4. Discussion

In a cohort of middle-aged, nondiabetic men, both SAD and WC were significantly and positively associated with total and large VLDL and total and small LDL particle concentrations, and inversely associated with large HDL particle concentration and LDL and HDL sizes for both white and Japanese men. After further adjustment for BMI, the associations of SAD remained significant for both white and Japanese men, whereas those of WC became nonsignificant for white men. When SAD and WC were

Table 2
Age-adjusted partial correlations between adiposity indices for white and Japanese men (N = 542)

	r (*P* < .001 for all)							
	White (n = 260)				Japanese (n = 282)			
	VAT	SAT	BMI	WC	VAT	SAT	BMI	WC
SAD	.69	.73	.81	.82	.69	.66	.72	.75
WC	.73	.88	.87		.78	.89	.89	
BMI	.69	.82			.72	.84		
SAT	.56				.70			

Table 3
Multivariate-adjusted associations between abdominal adiposity indices and VLDL subfractions for white and Japanese men (N = 542)

	Predictor variables: abdominal adiposity indices											
	White			Japanese			White			Japanese		
	β (R^2)		P	β (R^2)		P	β (R^2)		P	β (R^2)		P
Outcome variables: VLDL particles	SAD	WC	Δ (SAD-WC)	SAD	WC	Δ (SAD-WC)	VAT	SAT	Δ (VAT-SAT)	VAT	SAT	Δ (VAT-SAT)
Total												
Model I	4.33* (.09)	.70† (.04)		8.20* (.12)	1.65* (.10)		.26* (.08)	.07 (.02)		.50* (.13)	.32* (.07)	
Model II	5.24† (.09)	.06 (.05)		7.41* (.12)	1.78‡ (.10)		.23† (.08)	−.13 (.06)		.49* (.13)	.14 (.08)	
Model III	6.21† (.09)	−.72 (.09)	.001	6.41† (.12)	.97 (.12)	.035	.23† (.09)	−.12 (.09)	.002	.49* (.13)	<.01 (.13)	.022
Large												
Model I	1.06* (.21)	.18* (.11)		.62* (.09)	.12* (.07)		.06* (.18)	.02† (.06)		.04* (.11)	.02† (.05)	
Model II	1.01* (.21)	−.04 (.16)		.64† (.09)	.16‡ (.07)		.05* (.20)	−.03† (.19)		.05* (.12)	.01 (.06)	
Model III	1.29* (.24)	−.21† (.24)	<.001	.54‡ (.09)	.09 (.09)	.094	.05* (.23)	−.03† (.23)	<.001	.06* (.12)	−.01 (.12)	.003
Medium												
Model I	2.58* (.07)	.43‡ (.04)		5.30* (.07)	1.04* (<.01)		.15† (.06)	.03 (.02)		.38* (.11)	.20† (.04)	
Model II	2.12 (.07)	−.29 (.06)		4.89† (.07)	1.08 (.06)		.09 (.06)	−.15† (.08)		.44* (.11)	.06 (.05)	
Model III	3.03‡ (.08)	−.67 (.08)	.017	4.34‡ (.08)	.53 (.08)	.067	.09 (.09)	−.15† (.09)	.005	.46* (.11)	−.07 (.11)	.002
Small												
Model I	.68 (.01)	.08 (.01)		2.28† (.06)	.49† (.05)		.05 (.01)	.02 (.01)		.08 (.04)	.11‡ (.05)	
Model II	2.11‡ (.03)	.40 (.02)		1.88 (.06)	.54 (.05)		.09‡ (.02)	.05 (.01)		−.01 (.05)	.07 (.05)	
Model III	1.89‡ (.03)	.16 (.03)	.097	1.52 (.06)	.35 (.06)	.411	.10‡ (.03)	.06 (.03)	.487	−.02 (.05)	.08 (.05)	.378
Size												
Model I	.88* (.12)	.18* (.09)		.25 (.04)	.06 (.04)		.06* (.12)	.02† (.05)		.03 (.05)	.01 (.04)	
Model II	.47 (.13)	−.05 (.12)		.21 (.04)	.08 (.04)		.04‡ (.14)	−.03‡ (.14)		.04 (.05)	−.01 (.04)	
Model III	.65‡ (.14)	−.13 (.14)	.032	.15 (.04)	.06 (.04)	.848	.03‡ (.16)	−.03‡ (.16)	.001	.04‡ (.06)	−.01 (.06)	.120

For models, the outcome variable is lipoprotein concentration or size; the primary predictor variable is each abdominal adiposity index (SAD, WC, VAT, or SAT). Model I: model adjusted for age, pack years of smoking, and alcohol consumption. Model II: model adjusted further for BMI. Model III: BMI-adjusted model in which SAD and WC were simultaneously included.

* $P < .001$.

† $P < .01$.

‡ $P < .05$.

Table 4
Multivariate-adjusted associations between abdominal adiposity indices and LDL subfractions for white and Japanese men (N = 542)

	Predictor variables: abdominal adiposity indices											
	White			Japanese			White			Japanese		
	β (R^2)		P	β (R^2)		P	β (R^2)		P	β (R^2)		P
Outcome variables: LDL particles	SAD	WC	Δ(SAD-WC)	SAD	WC	Δ (SAD-WC)	VAT	SAT	Δ (VAT-SAT)	VAT	SAT	Δ (VAT-SAT)
Total												
Model I	38.18* (.09)	7.08† (.05)		102.52* (.20)	23.19* (.21)		2.75* (.10)	.77 (.03)		6.39* (.23)	4.99* (.18)	
Model II	33.11‡ (.09)	−.91 (.07)		70.23* (.22)	19.93† (.21)		2.41* (.10)	−1.24 (.08)		4.93* (.24)	2.79‡ (.19)	
Model III	41.41‡ (.09)	−6.10 (.09)	.013	57.02† (.23)	12.73 (.23)	.061	2.38† (.11)	−1.20 (.11)	<.001	4.60* (.24)	1.45 (.24)	.104
Intermediate												
Model I	5.27* (.11)	.81† (.05)		5.56* (.07)	1.07† (.05)		.30* (.09)	.08 (.03)		.39* (.09)	.21† (.04)	
Model II	6.25† (.11)	−.15 (.07)		6.10† (.07)	1.68‡ (.06)		.24‡ (.09)	−.18‡ (.09)		.49* (.10)	.18 (.04)	
Model III	7.79* (.12)	−1.13 (.12)	<.001	5.01‡ (.08)	1.05 (.08)	.107	.24‡ (.11)	−.18‡ (.11)	.001	.48* (.10)	.03 (.10)	.026
Large												
Model I	−22.82* (.06)	−3.78‡ (.03)		−39.41* (.11)	−7.81* (.09)		−1.44* (.06)	−.30 (.01)		−2.73* (.15)	−1.38* (.06)	
Model II	−22.41‡ (.06)	1.40 (.04)		−39.20* (.11)	−10.37† (.09)		−1.14‡ (.06)	1.10‡ (.06)		−3.23* (.15)	−.46 (.07)	
Model III	−29.32‡ (.07)	5.07 (.07)	.011	−32.72† (.12)	−6.24 (.12)	.045	−1.12‡ (.08)	1.08‡ (.08)	.002	−3.34* (.15)	.51 (.15)	<.001
Small												
Model I	55.73* (.11)	10.04† (.07)		136.36* (.24)	29.93* (.23)		3.89* (.12)	.99‡ (.04)		8.74* (.29)	6.16* (.19)	
Model II	49.27† (.11)	−2.15 (.09)		103.30* (.25)	28.62* (.23)		3.31† (.13)	−2.15‡ (.11)		7.67* (.29)	3.08‡ (.20)	
Model III	62.95† (.12)	−10.05 (.12)	.002	84.69* (.27)	17.93‡ (.27)	.012	3.25† (.15)	−2.09‡ (.15)	.000	7.46* (.29)	.91 (.29)	.003
Size												
Model I	−.11* (.12)	−.02* (.07)		−.22* (.23)	−.05* (.20)		−.01* (.13)	−.01‡ (.04)		−.01* (.29)	−.01* (.16)	
Model II	−.10† (.12)	<.01 (.09)		−.19* (.24)	−.05* (.20)		−.01† (.13)	<.01‡ (.11)		−.01* (.29)	−.01 (.17)	
Model III	−.12† (.13)	.02 (.13)	.001	−.16* (.25)	−.03‡ (.25)	.005	−.01† (.15)	<.01‡ (.15)	<.001	−.01* (.29)	−.01 (.29)	<.001

For models, the outcome variable is lipoprotein concentration or size; the primary predictor variable is each abdominal adiposity index (SAD, WC, VAT, or SAT). Model I: model adjusted for age, pack years of smoking, and alcohol consumption. Model II: model adjusted further for BMI. Model II: BMI-adjusted model in which SAD and WC were simultaneously included.

* $P < .001$.
† $P < .01$.
‡ $P < .05$.

Table 5
Multivariate-adjusted associations between abdominal adiposity indices and HDL subfractions for white and Japanese men (N = 542)

	Predictor variables: abdominal adiposity indices											
	White			Japanese			White			Japanese		
	β (R^2)		P	β (R^2)		P	β (R^2)		P	β (R^2)		P
Outcome variables: HDL particles	SAD	WC	Δ (SAD-WC)	SAD	WC	Δ (SAD-WC)	VAT	SAT	Δ (VAT-SAT)	VAT	SAT	Δ (VAT-SAT)
Total												
Model I	−.35† (.13)	−.10† (.14)		−.16 (.24)	−.06 (.24)		−.02† (.13)	−.01‡ (.12)		.01 (.24)	−.01 (.24)	
Model II	−.13 (.14)	−.08 (.14)		.10 (.24)	−.03 (.24)		−.01 (.14)	<.01 (.14)		.04‡ (.26)	−.01 (.24)	
Model III	−.02 (.14)	−.08 (.14)	.843	.15 (.24)	−.05 (.24)	.565	−.01 (.14)	<.01 (.14)	.404	.04‡ (.26)	−.01 (.26)	.047
Large												
Model I	−.44* (.20)	−.09* (.15)		−1.04* (.26)	−.23* (.26)		−.03* (.19)	−.01* (.11)		−.06* (.28)	−.05* (.21)	
Model II	−.31† (.21)	.02 (.19)		−.79* (.27)	−.25* (.26)		−.02† (.21)	.01‡ (.20)		−.05* (.29)	−.03‡ (.22)	
Model III	−.41† (.22)	.07 (.22)	.001	−.61† (.29)	−.17† (.29)	.037	−.02† (.23)	.01‡ (.23)	<.001	−.05* (.29)	−.01 (.29)	.015
Medium												
Model I	.02 (.01)	−.01 (.01)		.05 (.16)	−.01 (.16)		<.01 (.01)	−.01 (.01)		.01 (.17)	<.01 (.17)	
Model II	.03 (.01)	−.05‡ (.03)		.23 (.17)	.04 (.17)		−.01 (.01)	−.01 (.02)		.03† (.19)	.02 (.18)	
Model III	.12 (.03)	−.07‡ (.03)	.080	.21 (.17)	.02 (.17)	.424	−.01 (.02)	−.01 (.02)	.416	.03† (.20)	.02 (.20)	.032
Small												
Model I	.07 (.04)	.01 (.04)		.83* (.11)	.18* (.10)		.01 (.04)	−.01 (.04)		.06* (.14)	.03† (.07)	
Model II	.15 (.04)	−.05 (.04)		.66† (.17)	.18‡ (.10)		.01 (.04)	−.01 (.04)		.06* (.14)	−.01 (.09)	
Model III	.26 (.05)	−.08 (.05)	.121	.55‡ (.11)	.11 (.11)	.161	.01 (.04)	−.01 (.04)	.289	.06* (.14)	−.02 (.14)	.001
Size												
Model I	−.07* (.22)	−.01* (.15)		−.13* (.28)	−.03* (.29)		−.01* (.19)	−.01* (.12)		−.01* (.35)	−.01* (.24)	
Model II	−.06* (.23)	−.01 (.18)		−.08* (.31)	−.03* (.30)		−.01† (.21)	<.01 (.18)		−.01* (.36)	−.01‡ (.27)	
Model III	−.07* (.23)	.01 (.23)	<.001	−.07† (.33)	−.02‡ (.33)	.025	−.01† (.21)	<.01 (.21)	.005	−.01* (.36)	−.01 (.36)	.001

For models, the outcome variable is lipoprotein concentration or size; the primary predictor variable is each abdominal adiposity index (SAD, WC, VAT, or SAT). Model I: model adjusted for age, pack years of smoking, and alcohol consumption. Model II: model adjusted further for BMI. Model II: BMI-adjusted models in which SAD and WC were simultaneously included.

* $P < .001$.

† $P < .01$.

‡ $P < .05$.

simultaneously included into the BMI-adjusted models, the associations of SAD were not only significant, but also statistically stronger than those of WC for both white and Japanese men. Furthermore, the pattern of the associations of SAD with those lipoprotein subfractions was comparable to that of the associations of VAT.

Both SAD and WC correlate with VAT with similar magnitudes [30]. However, concerning correlations with SAT, our finding suggests that SAD may correlate weakly with SAT, whereas WC may correlate strongly with SAT ("Results"). Practically at the same height of abdomen (ie, at the same SAD), an increase in WC may reflect increased fat slid transversely toward the sides of the waist in the supine position, being regarded as SAT. Kullberg et al [20] reported that transverse abdominal diameter correlated highly with SAT. The Framingham Heart Study reported that WC was a stronger correlate of SAT than VAT in 1984 middle-aged men and women [31].

We found that, compared with SAT, VAT had stronger associations with higher particle concentrations of total, large, and medium VLDL and small LDL; lower particle concentration of large HDL; and lower average sizes of LDL and HDL for both white and Japanese men. These findings may support the notion that VAT is strongly linked with altered metabolism of triglyceride-rich lipoproteins, inducing nonesterified fatty acids flux to the liver [15]. An increased secretion of triglyceride-rich lipoproteins (ie, large VLDL particles) favors the transfer of triglyceride from triglyceride-rich lipoproteins to LDL and HDL through the action of cholesteryl ester transfer protein. During the process, hepatic lipase activity is increased, progressively lipolyzing the triglyceride-rich LDL and HDL to, finally, small LDL and HDL particles, respectively [32,33]. Furthermore, such process is also related to the lowered HDL cholesterol that was noted to have a fairly linear relationship to lowered concentrations of large HDL particles [34,35].

We found that SAT showed weak associations with lipoprotein subfractions for both white and Japanese men when VAT and SAT were simultaneously included into BMI-adjusted models. Although VAT carries greater cardiovascular and metabolic risks [11,12], SAT has also been associated with the risks [11,36]. However, increasing evidence indicates that the associations of SAT may be relatively weak [11,37,38] and possibly beneficial [39]. Fox et al [11] reported that SAT showed weaker correlations with metabolic risk factors than VAT in the Framingham Heart Study Offspring and Third-Generation Study Cohorts. Oka et al [38] showed that both VAT and SAT were significantly associated with metabolic risk factors in middle-aged Japanese men and women, but SAT did not remain significant when VAT and SAT were simultaneously included in the regression models. Furthermore, Sam et al [13] reported that SAT, unlike VAT, was not significantly associated with total VLDL and LDL particle concentrations in patients with type 2 diabetes mellitus in the CHICAGO trial with white and African American participants.

In addition, we found that SAT had beneficially significant associations with large VLDL, small LDL, and large HDL for white men. Recently, the Framingham Heart Study reported that, among individuals with high VAT (the highest-tertile group of VAT), increased SAT was significantly associated with lower triglycerides levels, whereas, among individuals with low VAT (the lowest-tertile group of VAT), increased SAT was significantly associated with higher triglycerides levels [39]. Given the relation of hypertriglyceridemia to the lipoprotein metabolism as mentioned above, the beneficial associations of SAT with those lipoprotein subfractions found in our findings may parallel the inverse association of SAT with triglycerides levels found in the Framingham Heart Study. In this context, SAT may be protective against unfavorable redistribution of VLDL and LDL subfractions for white men who have a relatively greater absolute amount of VAT rather than for Japanese men who have a relatively less absolute amount of VAT.

We found that the pattern of the associations of SAD with atherogenic lipoprotein subfractions (ie, large VLDL, total and small LDL, and large HDL particle concentrations) was comparable with that of VAT for both white and Japanese men. In our finding, SAD was a strong correlate of VAT for both white and Japanese men. Previous studies have revealed that SAD is the strongest correlate of VAT among other anthropometric abdominal adiposity measures including WC [20,40,41]. Kvist et al [40] reported that, among anthropometric abdominal adiposity measures (ie, WC and abdominal transverse diameters), SAD measured by a multiscan CT was the most predictive measure for the amount of VAT in both men and women with a wide range of body weights. Therefore, increased SAD may reflect a VAT-linked elevation of atherogenic lipoprotein subfractions well.

We found that SAD had stronger associations with atherogenic lipoprotein subfractions (ie, large VLDL, total and small LDL, and large HDL particle concentrations) than WC for both white and Japanese men, independent of BMI. Previous studies have reported that SAD has stronger associations with insulin resistance and cardiometabolic risk factors known as being linked to VAT than WC in the general population, middle-aged men and women, or obese men [21,42,43]. To the best of our knowledge, our current study is the first to report stronger associations of SAD with atherogenic lipoprotein subfractions than WC compared with relative associations of VAT vs SAT across 2 ethnic population groups. This main finding may be possibly explained by relative contributions of SAD vs WC to VAT vs SAT, indicating a weaker contribution of SAD to SAT than WC as mentioned above. Therefore, to identify high-risk abdominally visceral obese individuals linked with elevated atherogenic lipoprotein subfractions, SAD may be a better measure than WC across white and Japanese men.

Strengths of our study include the incorporation of different ethnic population groups as well as the analysis of the relative associations of SAD vs WC compared with

those of VAT vs SAT. Limitations of our study include the cross-sectional nature of the study design and generalizability to female, elderly, or other race population groups.

In a cohort of middle-aged, nondiabetic men, the pattern of the associations of SAD was comparable to that of the associations of VAT. Most notably, SAD had stronger associations with atherogenic lipoprotein subfractions, that is, large VLDL, total and small LDL, and large HDL particle concentrations, than WC for both white and Japanese men.

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