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Visceral adiposity and risk of coronary heart disease in relatively lean Chinese adults

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Abstract

Background—The hypertriglyceridemic waist phenotype (defined using both elevated waist circumference and triglycerides) and visceral adiposity index (VAI, defined using waist circumference, body mass index, triglycerides, and high-density lipoprotein cholesterol) have been suggested to be inexpensive yet effective markers of visceral (intra-abdominal) obesity and related dysmetabolic state. These markers may be particularly useful to Asian populations who generally have a low body weight but are prone to visceral adiposity.

Methods—We examined associations of the hypertriglyceridemic waist phenotype and VAI with risk of coronary heart disease (CHD) in a nested case-control study conducted within two prospective cohort studies of Chinese adults. We identified 355 incident cases of CHD and 697 controls matched for sex, age, and date and time of baseline sample collection. Anthropometric and lipid measurements were performed and used to define the hypertriglyceridemic waist phenotype and VAI according to published methods. Conditional logistic regression was used to evaluate the associations.

Results—Cases had a higher prevalence of the hypertriglyceridemic waist phenotype and higher VAI score than controls in both sexes. Adjusted odds ratios of CHD associated with hypertriglyceridemic waist were 5.18 (95% CI, 2.46–10.9) and 4.63 (2.03–10.5) for women and men, respectively. Adjusted odds ratios of CHD comparing the highest vs. lowest quartile of VAI were 4.44 (95% CI, 2.24–8.82) and 4.23 (1.99–9.00) for women and men, respectively.

Conclusion—Our study demonstrates, for the first time, that the hypertriglyceridemic waist phenotype and high VAI score are associated with substantially elevated risk of CHD in Chinese men and women.

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Keywords

visceral adiposity; heart disease; Chinese

Introduction

Substantial variation in cardiometabolic risk has been observed among individuals with similar levels of adiposity as measured by body mass index (BMI) [1]. Some of this variation may originate from differences in the distribution and function of adipose tissue [2, 3]. Intra-abdominal or visceral fat accumulation has been suggested to be directly involved in the pathogenesis of insulin resistance and associated metabolic diseases and cardiovascular disease (CVD) [2–4]. Waist circumference (WC), a measure of abdominal adiposity, has been indicated to be a better predictor of cardiometabolic risk than BMI, a measure of general adiposity [5]. Elevated WC has been included as one of the key criteria to define the metabolic syndrome [6]. Nevertheless, the inability of WC to distinguish between subcutaneous and visceral adiposity and the increasing recognition of the differential metabolic characteristics between these two fat depots have stimulated interest in evaluating additional markers readily available in clinical settings that may better capture visceral obesity and related dysmetabolic state [2, 4, 7, 8]. Two such markers have emerged, the hypertriglyceridemic waist phenotype and the visceral adiposity index (VAI) [7, 8]. These markers use combinations of anthropometric and lipid parameters and have been suggested to be inexpensive yet effective indicators of adipose tissue dysfunction and cardiometabolic risk [7–9].

Some Asian populations including Chinese are known to be prone to visceral fat accumulation and insulin resistance despite having generally low BMI [10, 11]; markers of visceral obesity may thus be particularly useful to Asians. We evaluated, for the first time, whether the hypertriglyceridemic waist phenotype and VAI are associated with risk of coronary heart disease (CHD) in Chinese men and women.

Methods

Study population

We conducted a nested case-control study within the Shanghai Women's Health Study (SWHS) and the Shanghai Men's Health Study (SMHS), two prospective, population-based cohort studies. Both studies were approved by the Institutional Review Boards of all institutes involved, and informed consent was obtained from all participants. The designs and methods used in the two studies are similar and have been described in detail previously [12, 13]. Participants were recruited from typical urban communities of Shanghai, China. From 1996 to 2000, the SWHS recruited 74 941 women aged 40 to 70 years (participation rate: 92.7%); from 2002 to 2006, the SMHS recruited 61 491 men aged 40 to 74 years (participation rate: 74.1%). Baseline surveys were carried out at participants' homes by trained interviewers in both studies. Using structured questionnaires, information was obtained on demographic factors, diet and other lifestyle habits, medical history, and other characteristics. Blood samples were collected at baseline from the majority of the cohort members (76% in SWHS and 75% in SMHS). Fasting was not required. All samples were processed within 6 hours of collection and stored at -70°C . After enrollment, cohort members were actively followed through biennial home visits (overall response rate: 96% in both cohorts). The mean follow-up was 10 years for the SWHS cohort and 5 years for the SMHS cohort.

Anthropometry

Body measurements, including weight, height, and circumferences of the waist and hips, were conducted at baseline by trained interviewers according to a standard protocol [14]. Waist circumference was measured at 2.5 cm above the umbilicus with the subject in a standing position. Circumference and height were measured to the nearest 0.1 cm and weight to the nearest 0.1 kg. All measurements were taken twice. A tolerance limit of 1 cm was set for height and circumference measurements and 1 kg for weight measurement. A third measurement was taken if the difference of the first two measurements was greater than the tolerance limit. The average of the two closest measurements was used for analysis. BMI was calculated as weight in kilograms divided by the square of height in meters.

Selection of CHD cases and controls

Subjects with a prior history of CHD were excluded from this nested case-control study. Cases were defined as subjects with incident CHD, including non-fatal myocardial infarction (MI) and fatal CHD, that occurred after the baseline survey but before December 31, 2009. The diagnosis of MI was confirmed through medical record review, using the World Health Organization criteria (i.e., symptoms plus either diagnostic electrocardiographic changes or elevation of cardiac enzyme levels) [15]. Death from CHD was confirmed by reviewing medical records whenever possible, referring to death certificates, and interviewing the next of kin. For each confirmed incident case of CHD, two controls were selected from the eligible cohort members who were free of CHD at the time of diagnosis of the index case. Controls were matched to cases by sex, age, menopausal status (women only), and date and time of blood sample collection. Using these criteria, 196 incident cases of CHD and 392 matched controls from the SWHS and 159 cases and 305 controls from the SMHS were included in the current study. All cases had 2 matched controls except for 13 cases with only one control.

Laboratory assays

Plasma samples obtained at baseline from cases and controls were assayed for total cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides (TG), lipoprotein(a), uric acid, and high-sensitivity C-reactive protein (CRP). All assays were performed by Vanderbilt Lipid Laboratory using the ACE[®] Clinical Chemistry System (Alfa Wassermann, Inc, West Caldwell, NJ). Low-density lipoprotein (LDL) cholesterol levels were calculated by using the Friedwald equation [16]. The levels of LDL cholesterol were directly measured for subjects with TG levels ≥ 400 mg/dL. All analyses were conducted in a blinded fashion with regard to case-control status.

Statistical analysis

Statistical analyses were conducted separately for women and men. We first compared baseline characteristics between case subjects and control subjects. To take into account the matched nature of the data, a mixed-effect model was used for continuous variables and conditional logistic regression model was used for categorical variables. Continuous variables with a highly skewed distribution were log-transformed for analysis. Second, we evaluated hypertriglyceridemic waist phenotype in relation to lipid profiles and other biomarkers in control subjects. Subjects were divided into four groups on the basis of the recommended criteria for hypertriglyceridemic waist [4, 7]: normal WC (<90 cm in men and <85 in women)/normal TG level (<177 mg/dL in men and <133 in women), increase in WC only, increase in TG level only, and increase in both WC and TG level. We then examined the association between hypertriglyceridemic waist phenotype and risk of CHD. Conditional logistic regression models were used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) of CHD associated with different WC/TG groups, and to adjust

for potential confounders. Subjects with both WC and TG measurements below the defined cut points served as the reference group. Covariates considered in the analyses included age, education level, occupation, family income, cigarette smoking, alcohol consumption, BMI, amount of physical activity, menopausal status and hormone therapy use (women only), LDL cholesterol level, and prior history of hypertension or diabetes. Finally, we estimated ORs of CHD associated with VAI. The VAI was derived by using the published formula [8]:

$$\begin{aligned} \text{Males: } VAI &= \left(\frac{WC}{39.68 + (1.88 \times BMI)} \right) \\ &\times \left(\frac{TG}{1.03} \right) \\ &\times \left(\frac{1.31}{HDL} \right); \text{ Females: } VAI \\ &= \left(\frac{WC}{36.58 + (1.89 \times BMI)} \right) \\ &\times \left(\frac{TG}{0.81} \right) \\ &\times \left(\frac{1.52}{HDL} \right) \text{ (both TG and HDL in mmol/L)} \end{aligned}$$

The VAI scores were categorized into quartiles based on the distribution among control subjects, with the lowest quartile serving as the reference group. Statistical analyses were performed by using SAS statistical software (version 9.2; SAS Institute Inc, Cary, NC). All statistical tests were based on 2-sided probability. The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [17].

Results

Baseline characteristics of cases and controls are shown in Tables 1 and 2 for women and men, respectively. Women and men who developed CHD (cases) during follow-up compared with those who did not (controls) had greater WC and BMI, less favorable lipid profiles, and higher levels of uric acid and CRP at baseline. Cases also had a higher VAI score and higher prevalence of hypertriglyceridemic waist phenotype, hypertension, and diabetes at baseline than did controls.

Distributions of lipid and other biomarkers across different WC/TG groups in control subjects are presented in Table 3. Individuals with both elevated WC and increased TG levels (the hypertriglyceridemic waist phenotype) had higher levels of total cholesterol, non-HDL cholesterol, lipoprotein(a), uric acid, CRP, and VAI score, but lower levels of HDL cholesterol, as compared with those with low WC and TG levels. The findings were similar for both sexes. The difference for lipoprotein(a) in men, however, did not reach statistical significance. Levels of LDL cholesterol appeared to be lower in individuals with the hypertriglyceridemic waist phenotype.

ORs and 95% CIs of CHD according to WC and TG levels are summarized in Table 4. In both women and men, the ORs for CHD increased with either elevated WC or increased TG levels alone. The positive association, however, was strongest in the presence of both elevated WC and increased TG levels (the hypertriglyceridemic waist phenotype). After adjusting for age, socioeconomic status, smoking, alcohol consumption, BMI, physical activity, and LDL cholesterol level, the ORs of CHD associated with the hypertriglyceridemic waist phenotype were 5.18 (95% CI, 2.46–10.9) and 4.63 (2.03–10.5)

for women and men, respectively. The associations persisted, although they were attenuated, after additional adjustment for potential intermediate variables, including hypertension, diabetes, and inflammatory biomarkers.

Similar to the hypertriglyceridemic waist phenotype, a strong positive association with CHD was also found for increased VAI scores (Table 5). The adjusted ORs of CHD comparing the highest vs. lowest quartile of VAI score were 4.44 (95% CI, 2.24–8.82) and 4.23 (1.99–9.00) for women and men, respectively. The associations were also attenuated after additional adjustment for potential intermediate variables. We calculated c -statistics to compare the predictive values of the hypertriglyceridemic waist phenotype and VAI. The c -statistics were 0.725 for the fully adjusted model that included the hypertriglyceridemic waist phenotype and 0.719 for the model including VAI in women. The corresponding c -statistics for men were 0.797 and 0.790.

Discussion

This is, to our knowledge, the first study that has evaluated the concept of applying the hypertriglyceridemic waist phenotype and VAI for assessment of CHD risk in a non-Western population. Our study population was predominantly non-obese and had relatively low LDL cholesterol levels, characteristics that are known to be associated with low risk for CHD. Nevertheless, we found that the hypertriglyceridemic waist phenotype was associated with an unfavorable cardiometabolic risk profile and a 4–5-fold increased risk of developing CHD in both men and women. We also found a strong positive association between VAI score and CHD risk. Our results suggest that these inexpensive markers of visceral obesity and related dysmetabolic state may be particularly useful in assessing CHD risk for Chinese and perhaps other Asian populations who have an apparently “healthy” body weight yet are predisposed to visceral fat accumulation and the development of metabolic abnormalities.

Excess visceral adiposity may increase cardiometabolic risk through multiple mechanisms [4]. Visceral obesity has been associated with increased production of free fatty acids, interleukin-6, tumor necrosis factor- α , and C-reactive protein and decreased production of adiponectin, all of which may be involved in the pathogenesis of insulin resistance, the metabolic syndrome, and related CVD risk [4]. Visceral obesity has also been proposed as a marker of dysfunctional adipose tissue and ectopic fat deposition, reflecting the relative inability of subcutaneous adipose tissue to properly handle and store excess energy [4].

Visceral adiposity has been more strongly linked to adverse metabolic risk profiles than subcutaneous adiposity [18]. The improved understanding of the critical role of visceral adiposity in the development of metabolic abnormalities and CVD emphasizes the need to quantify visceral adipose tissue. Although imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI), can distinguish visceral from subcutaneous adipose tissue and provide precise measurements, they are not readily available in clinical settings and are used primarily for research purposes [19]. Surrogate markers of visceral adiposity and adipose tissue dysfunction, such as those assessed in the present study, offer a simple and inexpensive alternative approach for assessing adiposity-related risks.

The Quebec Cardiovascular Study first introduced the hypertriglyceridemic waist concept to cost-effectively identify individuals with excess visceral adiposity and atherogenic metabolic triad (i.e., hyperinsulinemia, hyperapoprotein B and small, dense LDL particles) [7]. The study found that the presence of either increased WC or elevated TG alone was not sufficient to appropriately capture the metabolic triad [7]. Recently, data from the CHICAGO cohort showed that in subjects with type 2 diabetes, simultaneous presence

of increased WC and TG was related to significantly greater amount of visceral fat measured by CT and coronary artery calcium measured by electron-beam tomography, as compared with the presence of increased WC alone [20]. In a Danish cohort study, women with hypertriglyceridemic waist but without metabolic syndrome were found to have a greater annual progression rate of aortic calcification than did women with metabolic syndrome but without hypertriglyceridemic waist [21]. Few prospective studies have assessed the hypertriglyceridemic waist phenotype in relation to CHD risk and yielded consistent results [9, 21, 22]. The largest of these studies, the European Prospective Investigation into Cancer and Nutrition (EPIC)-Norfolk study, reported that the hypertriglyceridemic waist phenotype was associated with crude hazard ratios for CHD of 2.4 in men and 3.8 in women [9]. The positive association persisted in multivariable analysis adjusting for traditional risk factors for CHD. These results are similar to those of our study, despite different study settings. Collectively, these data support the notion that the hypertriglyceridemic waist phenotype confers an elevated risk of CHD and could be used as a simple, inexpensive tool for CHD risk assessment.

The Alkam Metabolic Syndrome Study recently introduced the VAI, a new surrogate marker for adipose tissue dysfunction [8]. The VAI score assesses visceral fat mass based on both anthropometric (BMI and WC) and metabolic (TG and HDL cholesterol) parameters. The VAI was found in the Alkam study to be correlated positively with visceral adiposity tissue assessed by MRI and inversely with insulin sensitivity assessed by the euglycemic-hyperinsulinemic clamp. In contrast, no correlation with insulin sensitivity was found for either WC or BMI. The VAI also showed a positive association with cardiometabolic risk in the study [8]. Our study provides evidence, for the first time, that a high VAI score predicts an increased risk of CHD in a population-based setting.

The significance of our study findings is highlighted by the characteristics of our study population, i.e., the low BMI and LDL cholesterol levels. Our study extends previous research on hypertriglyceridemic waist and VAI indicating that these markers may be particularly important for some Asian populations for whom the traditional approach to risk classification for CHD based on BMI and LDL cholesterol levels may not be optimal. Overall, the hypertriglyceridemic waist and VAI appeared to be better predictors of CHD than BMI in our study. In analyses adjusting for age only, the ORs (95% CIs) for CHD were 3.6 (2.1–6.2) in women and 3.2 (1.9–5.5) in men for hypertriglyceridemic waist, and 3.5 (1.9–6.6) in women and 2.8 (1.5–5.2) in men for the highest vs. lowest quartile of VAI, compared to the ORs of 1.4 (0.9–2.3) in women and 2.7 (1.4–5.1) in men for the highest vs. lowest quartile of BMI. In multivariable-adjusted models including hypertriglyceridemic waist, BMI was no longer related to CHD in either men or women.

Limitations of our study need to be considered when interpreting these results. The sample size of our study was relatively small, resulting in wide confidence intervals around the estimates of associations. Both fasting and non-fasting plasma samples were used in the study, which may have led to some random misclassification of the lipid levels. However, studies have shown minimal changes of lipid profiles in response to usual food intake in the general population [23]. Our findings provide support for the usefulness of non-fasting lipid profiles in predicting CVD risk as demonstrated in previous studies [23]. Given that many covariates were obtained through self-report and were thus subject to error, we cannot rule out the possibility of residual confounding by inaccurately measured covariates and other unmeasured variables.

Conclusion

Our study suggests that the hypertriglyceridemic waist phenotype and VAI, validated and convenient markers of visceral obesity, can be useful tools for CHD risk assessment in Chinese men and women.

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Table 1

Baseline characteristics of cases and controls in women

	Case	Control	<i>P</i> value
No. of subjects	196	392	
Age (y), mean $\pm$ SD	62.5 $\pm$ 7.0	62.2 $\pm$ 7.0	Matched
Waist circumference (cm), mean $\pm$ SD	83.4 $\pm$ 9.2	81.1 $\pm$ 9.1	0.003
Body mass index (kg/m ²), mean $\pm$ SD	25.6 $\pm$ 3.9	24.9 $\pm$ 3.6	0.04
Physical activity (MET-h/wk), mean $\pm$ SD	113 $\pm$ 52.7	112 $\pm$ 45.8	0.70
Total cholesterol (mg/dL), mean $\pm$ SD	196 $\pm$ 41.7	183 $\pm$ 34.2	<0.001
LDL cholesterol (mg/dL), mean $\pm$ SD	112 $\pm$ 36.4	103 $\pm$ 27.4	<0.001
HDL cholesterol (mg/dL), mean $\pm$ SD	42.2 $\pm$ 9.5	44.7 $\pm$ 11.2	0.006
Non-HDL cholesterol (mg/dL), mean $\pm$ SD	153 $\pm$ 40.3	138 $\pm$ 31.5	<0.001
Triglycerides (mg/dL) *	191 (177 – 206)	162 (153 – 172)	<0.001
Lipoprotein(a) (mg/dL) *	13.4 (11.0 – 16.3)	10.4 (8.9 – 12.2)	0.05
Uric acid (mg/dL) *	5.4 (5.3 – 5.6)	5.2 (5.0 – 5.3)	0.01
C-reactive protein (mg/L) *	1.2 (0.9 – 1.5)	0.7 (0.6 – 0.9)	0.003
Visceral adiposity index *	3.7 (3.4 – 4.1)	3.0 (2.8 – 3.2)	<0.001
Hypertriglyceridemic waist, %	38.3	24.7	<0.001
Education high school, %	20.9	21.9	0.77
Professional occupation, %	19.4	19.9	0.88
Annual family income 20,000 yuan, %	32.7	34.4	0.66
Cigarette smoking [†] , %	10.2	5.1	0.03
Alcohol consumption [‡] , %	3.1	2.3	0.59
Hormone therapy, %	1.0	1.0	1.0
Hypertension, %	53.6	32.7	<0.001
Diabetes mellitus, %	19.9	8.2	<0.001

Abbreviations: MET, metabolic equivalent; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

* Data are presented as geometric means with 95% confidence intervals.

[†] Ever smoked at least 1 cigarette per day for 6 months or longer.[‡] Ever drank alcohol at least 3 times per week for 6 months or longer.

Table 2

Baseline characteristics of cases and controls in men

	Case	Control	P value
No. of subjects	159	305	
Age (y), mean $\pm$ SD	63.2 $\pm$ 8.6	63.3 $\pm$ 8.6	Matched
Waist circumference (cm), mean $\pm$ SD	88.7 $\pm$ 8.6	85.2 $\pm$ 9.3	<0.001
Body mass index (kg/m ²), mean $\pm$ SD	24.8 $\pm$ 3.3	23.7 $\pm$ 3.3	<0.001
Physical activity (MET-h/wk), mean $\pm$ SD	60.2 $\pm$ 37.3	73.1 $\pm$ 37.8	<0.001
Total cholesterol (mg/dL), mean $\pm$ SD	189 $\pm$ 37.1	174 $\pm$ 32.8	<0.001
LDL cholesterol (mg/dL), mean $\pm$ SD	109 $\pm$ 30.6	97.9 $\pm$ 26.6	<0.001
HDL cholesterol (mg/dL), mean $\pm$ SD	37.0 $\pm$ 8.7	38.3 $\pm$ 9.5	0.14
Non-HDL cholesterol (mg/dL), mean $\pm$ SD	152 $\pm$ 34.6	135 $\pm$ 31.6	<0.001
Triglycerides (mg/dL) *	211 (191 – 232)	172 (161 – 184)	<0.001
Lipoprotein(a) (mg/dL) *	13.0 (10.5 – 15.9)	9.5 (8.0 – 11.2)	0.02
Uric acid (mg/dL) *	6.8 (6.5 – 7.1)	6.5 (6.3 – 6.6)	0.03
C-reactive protein (mg/L) *	2.1 (1.7 – 2.6)	1.1 (0.9 – 1.4)	<0.001
Visceral adiposity index *	3.3 (3.0 – 3.8)	2.6 (2.4 – 2.8)	<0.001
Hypertriglyceridemic waist, %	34.6	19.7	<0.001
Education high school, %	49.1	54.8	0.21
Professional occupation, %	35.2	34.1	0.76
Annual per capita family income 12,000 yuan, %	43.4	42.6	0.97
Smoking >10 cigarettes daily †, %	47.8	36.1	0.02
Alcohol consumption ‡, %	28.9	30.5	0.78
Hypertension, %	64.8	37.4	<0.001
Diabetes mellitus, %	14.5	12.1	0.59

Abbreviations: MET, metabolic equivalent; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

* Data are presented as geometric means with 95% confidence intervals.

† Ever smokers, usually smoking more than 10 cigarettes per day.

‡ Ever drank alcohol at least 3 times per week for 6 months or longer.

Table 3

Lipid profiles and other biomarkers by waist circumference and triglyceride levels in control subjects *

Women									
Variable	Group 1: WC < 85 cm /TG < 133 mg/dL	Group 2: WC 85 cm /TG < 133 mg/dL	Group 3: WC < 85 cm /TG < 133 mg/dL	Group 4: WC 85 cm /TG < 133 mg/dL	133	133	133	133	P** value
No. of subjects	124	32	139	97					
Total cholesterol (mg/dL)	172 (166 – 178)	174 (162 – 185)	191 (186 – 197)	188 (181 – 194)					<0.001
LDL cholesterol (mg/dL)	103 (98.2 – 108)	108 (98.1 – 117)	104 (99.0 – 108)	99.2 (93.7 – 105)					0.31
HDL cholesterol (mg/dL)	49.9 (48.0 – 51.8)	45.2 (41.6 – 48.9)	43.6 (41.9 – 45.4)	39.5 (37.3 – 41.6)					<0.001
Non-HDL cholesterol (mg/dL)	122 (117 – 127)	129 (119 – 139)	148 (143 – 152)	148 (142 – 154)					<0.001
Triglycerides (mg/dL)	93.1 (87.2 – 99.4)	104 (91.3 – 118)	217 (204 – 231)	254 (236 – 273)					<0.001
Lipoprotein(a) (mg/dL)	8.2 (6.2 – 10.9)	7.5 (4.3 – 13.0)	11.6 (9.0 – 15.1)	13.6 (9.9 – 18.6)					0.02
Uric acid (mg/dL)	4.4 (4.2 – 4.5)	5.0 (4.7 – 5.4)	5.4 (5.2 – 5.6)	6.1 (5.9 – 6.4)					<0.001
C-reactive protein (mg/L)	0.4 (0.3 – 0.5)	0.9 (0.5 – 1.7)	0.8 (0.6 – 1.0)	1.5 (1.1 – 2.2)					<0.001
Visceral adiposity index	1.5 (1.4 – 1.6)	1.9 (1.6 – 2.3)	4.0 (3.7 – 4.3)	5.5 (5.0 – 6.1)					<0.001
Men									
Variable	Group 1: WC < 90 cm /TG < 177 mg/dL	Group 2: WC 90 cm /TG < 177 mg/dL	Group 3: WC < 90 cm /TG < 177 mg/dL	Group 4: WC 90 cm /TG < 177 mg/dL	177	177	177	177	
No. of subjects	127	40	78	60					
Total cholesterol (mg/dL)	168 (162 – 173)	156 (146 – 165)	185 (178 – 192)	184 (176 – 192)					<0.001
LDL cholesterol (mg/dL)	102 (97.1 – 106)	96.6 (88.4 – 105)	95.9 (90.0 – 102)	93.5 (86.8 – 100)					0.05
HDL cholesterol (mg/dL)	42.7 (41.2 – 44.2)	35.2 (32.5 – 37.9)	35.8 (33.8 – 37.7)	34.4 (32.2 – 36.6)					<0.001
Non-HDL cholesterol (mg/dL)	125 (120 – 130)	120 (111 – 129)	150 (143 – 156)	150 (142 – 157)					<0.001
Triglycerides (mg/dL)	111 (104 – 117)	114 (103 – 126)	278 (259 – 300)	312 (287 – 340)					<0.001
Lipoprotein(a) (mg/dL)	8.9 (6.8 – 11.5)	13.0 (8.2 – 20.5)	9.1 (6.6 – 12.7)	9.5 (6.5 – 13.8)					0.78
Uric acid (mg/dL)	5.8 (5.6 – 6.0)	6.0 (5.6 – 6.3)	7.1 (6.8 – 7.4)	7.6 (7.2 – 8.0)					<0.001
C-reactive protein (mg/L)	0.8 (0.6 – 1.1)	0.7 (0.4 – 1.2)	1.5 (1.1 – 2.2)	2.1 (1.4 – 3.3)					<0.001
Visceral adiposity index	1.4 (1.3 – 1.6)	1.9 (1.7 – 2.2)	4.4 (4.0 – 4.9)	5.5 (4.9 – 6.1)					<0.001

Abbreviations: WC, waist circumference; TG, triglycerides; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

* Data are presented as age-adjusted means with 95% confidence intervals for total cholesterol, LDL cholesterol, HDL cholesterol, and non-HDL cholesterol, and geometric means for all other variables.

** Pvalue for comparing group 4 vs. group 1.

Table 4
Odds ratios of coronary heart disease by waist circumference and triglyceride levels

	Cases/controls	Model 1 ¹	Model 2 ²	Model 3 ³	Model 4 ⁴
Women					
Group 1: WC < 85 cm/TG < 133 mg/dL	33/124	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)
Group 2: WC 85 cm/TG < 133 mg/dL	15/32	2.19 (1.03 – 4.67)	2.83 (1.08 – 7.41)	2.04 (0.75 – 5.55)	1.99 (0.71 – 5.55)
Group 3: WC < 85 cm/TG 133 mg/dL	73/139	2.60 (1.53 – 4.43)	2.65 (1.50 – 4.66)	2.23 (1.25 – 4.01)	1.77 (0.90 – 3.48)
Group 4: WC 85 cm/TG 133 mg/dL ("hypertriglyceridemic waist")	75/97	3.61 (2.09 – 6.24)	5.18 (2.46 – 10.9)	4.15 (1.94 – 8.89)	3.71 (1.60 – 8.59)
Men					
Group 1: WC < 90 cm/TG < 177 mg/dL	37/127	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)
Group 2: WC 90 cm/TG < 177 mg/dL	23/40	1.88 (0.99 – 3.60)	2.20 (0.89 – 5.43)	1.96 (0.77 – 5.01)	2.43 (0.89 – 6.64)
Group 3: WC < 90 cm/TG 177 mg/dL	44/78	2.10 (1.21 – 3.64)	2.47 (1.23 – 4.95)	2.17 (1.06 – 4.43)	2.21 (0.96 – 5.08)
Group 4: WC 90 cm/TG 177 mg/dL ("hypertriglyceridemic waist")	55/60	3.19 (1.85 – 5.51)	4.63 (2.03 – 10.5)	4.00 (1.67 – 9.59)	4.48 (1.60 – 12.6)

Abbreviations: WC, waist circumference; TG, triglycerides.

¹Model 1: conditioned on matching variables and adjusted for age.

²Model 2: model 1 additionally adjusted for socioeconomic status, cigarette smoking, alcohol consumption, body mass index, amount of physical activity, level of low-density lipoprotein, and for women only, menopausal status and hormone therapy use.

³Model 3: model 2 additionally adjusted for history of hypertension or diabetes.

⁴Model 4: model 3 additionally adjusted for lipoprotein(a), uric acid, and C-reactive protein.

Table 5

Odds ratios of coronary heart disease by visceral adiposity index

Visceral adiposity index	Cases/ controls	Model 1 ¹	Model 2 ²	Model 3 ³	Model 4 ⁴
Women					
Quartile 1	24/98	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)
Quartile 2	48/98	2.21 (1.21 – 4.03)	2.38 (1.25 – 4.54)	2.20 (1.12 – 4.32)	2.20 (1.07 – 4.53)
Quartile 3	58/98	2.99 (1.63 – 5.48)	3.34 (1.75 – 6.38)	3.00 (1.52 – 5.90)	2.59 (1.20 – 5.61)
Quartile 4	66/98	3.51 (1.87 – 6.59)	4.44 (2.24 – 8.82)	4.09 (2.01 – 8.33)	2.95 (1.22 – 7.15)
<i>P</i> for trend		0.0001	<0.0001	<0.0001	0.03
Men					
Quartile 1	22/76	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)	1.0 (Reference)
Quartile 2	40/76	1.94 (1.03 – 3.66)	2.30 (1.11 – 4.78)	1.78 (0.83 – 3.79)	1.61 (0.72 – 3.56)
Quartile 3	39/77	1.81 (0.96 – 3.43)	1.85 (0.88 – 3.88)	1.48 (0.69 – 3.18)	1.16 (0.50 – 2.73)
Quartile 4	58/76	2.79 (1.50 – 5.21)	4.23 (1.99 – 9.00)	2.88 (1.31 – 6.35)	2.09 (0.82 – 5.33)
<i>P</i> for trend		0.003	0.0007	0.02	0.21

¹Model 1: conditioned on matching variables and adjusted for age.

²Model 2: model 1 additionally adjusted for socioeconomic status, cigarette smoking, alcohol consumption, amount of physical activity, level of low-density lipoprotein, and for women only, menopausal status and hormone therapy use.

³Model 3: model 2 additionally adjusted for history of hypertension or diabetes.

⁴Model 4: model 3 additionally adjusted for lipoprotein(a), uric acid, and C-reactive protein.