HHS Public Access

Author manuscript

Atherosclerosis. Author manuscript; available in PMC 2019 April 01.

Published in final edited form as:

Atherosclerosis. 2018 April ; 271: 142–147. doi:10.1016/j.atherosclerosis.2018.02.031.

Anthropometric measures of obesity and renal artery calcification: Results from the Multi-Ethnic Study of Atherosclerosis

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Abstract

Background and aims—Renal artery calcium (RAC) has been linked to several cardiovascular disease (CVD) risk factors, including age, male gender, and hypertension. The purpose of this study was to determine whether anthropometric measures of obesity are associated with presence of RAC.

Methods—We studied 1,287 community-dwelling adults enrolled in the Multi-Ethnic Study of Atherosclerosis. Logistic regression models adjusted for CVD risk factors were used to examine body mass index (BMI), waist circumference (WC), hip circumference (HC), and waist-to-hip ratio (WHR) as primary predictors of RAC.

Results—Study participants had a mean age of 67.7 years, 55.7% were female, and 36.8% were Non-Hispanic White. Prevalence of RAC was 33.3%. WC and WHR as continuous variables were not significant with adjustment. Subjects with high WC, as defined by World Health Organization cut-offs, had significantly higher odds for RAC in the fully adjusted model. BMI and HC were not significantly associated with RAC in any models.

Conclusions—In this community-based sample of older adults, higher levels of WC are significantly associated with RAC independently of CVD risk factors. Adults who meet World

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Author contributions: Conception and design: AR and MA. Statistical expertise: MA and DR. Obtaining of funding: MA and DR. Administrative, technical, or logistic support: AR. Collection and assembly of data: AR. All authors were involved in the analysis and interpretation of the data, drafting of the article and critical revision, and final approval of the article.

Conflict of interest: The authors declared they do not have anything to disclose regarding conflict of interest with respect to this manuscript.

Health Organization criteria for high WC may be at higher risk for complications of calcified atherosclerosis in the renal arteries.

Introduction

The 2011-2012 National Health and Nutrition Examination Survey found that approximately 16.9% of youth and 34.9% of adults in the United States were diagnosed with obesity [1]. Increased weight has been linked to higher risk of hypertension, dyslipidemia, diabetes, and cardiovascular disease (CVD) [2-4]. Moreover, several studies have demonstrated an association between higher anthropometric measures and subclinical atherosclerosis [5-8].

The mechanism of calcification within atherosclerotic plaque is initiated by de-differentiation of vascular smooth muscle cells into an osteoblast/chondrocytic phenotype. Oxidized lipids promote calcification in these vascular cells via inflammatory mediators and recruitment of activated macrophages [9]. Both atherogenic dyslipidemia and the proinflammatory state have been linked to obesity [4].

Renal artery calcium (RAC) has recently emerged as an indicator of subclinical atherosclerosis. Similar to coronary artery calcium (CAC), RAC is associated with traditional CVD risk factors such as age, male gender, and hypertension. RAC itself has also been linked to increased risk of atherosclerotic calcification in the coronary arteries and abdominal aorta [11-13]. Furthermore, the presence of RAC is associated with higher all-cause mortality, independent of the presence of atherosclerotic calcium in other vascular beds [14]. To our knowledge, no previous studies have explored an association between different anthropometric measures of obesity and RAC. Therefore, we conducted a study to determine whether body mass index (BMI), waist circumference (WC), hip circumference (HC), and waist-to-hip ratio (WHR) were associated with the presence of RAC. We hypothesized that higher levels of these anthropometric measures would be independently associated with RAC.

Patients and methods

Subjects

Details regarding the study design and recruitment of the Multi-Ethnic Study of Atherosclerosis (MESA) have been previously published [15]. Briefly, MESA is a longitudinal cohort study of 6,814 community-dwelling individuals aged 45-84 years at baseline who self-identified as Black, Chinese American, Hispanic American, or Non-Hispanic White. Individuals with clinical evidence of CVD, history of major acute cardiovascular event, or prior invasive procedure for CVD were excluded from the study. Recruitment occurred at six United States communities between July 2000 and August 2002, and enrolled participants returned for follow-up clinic examinations on three subsequent examinations (visits 2, 3 and 4) at approximately 18 to 24-month intervals. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki. All participants provided written informed consent and the institutional review boards at the participating community sites approved the study.

At clinic visits 2 and 3, a random subsample of 1,978 participants from five of the six MESA field centers were enrolled in an ancillary study to determine the extent of abdominal aortic calcification using computed tomography [16]. Abdominal imaging from this ancillary study was also evaluated for the presence of RAC. The data obtained on this subset of participants comprise the analytic sample for the current study.

Data collection

At all clinic visits, standardized questionnaires were used to obtain demographic, race/ethnicity, social, and health history data. Cigarette smoking was defined as current, former or never. Total moderate and vigorous activity in metabolic equivalent (MET)-minutes per week was measured using a 28-item survey. Maximum education level was self-reported as high school graduate or less, some college, or college graduate or higher. Height, weight, and BMI were obtained using standardized measurements. Waist and hip circumferences were measured at the level of the umbilicus and at the maximal circumference of the buttocks, respectively, to the nearest 0.1 cm using a steel measuring tape (standard 4 oz tension). The calculation of blood pressure was based on the average of the second and third of three readings. Hypertension was defined as systolic blood pressure greater than or equal to 140 mm Hg, diastolic blood pressure greater than or equal to 90 mm Hg or the current use of an antihypertensive medication.

Imaging

Electron-beam CT scanners were used at two field centers, and multidetector CT mode scanners were used at the remaining 3 field centers. Images were reconstructed in a 35-cm field of view with 5-mm slice thickness. All scan scores were brightness adjusted with a standard phantom.

Noncontrast CT images were analyzed centrally using a standard protocol by the MESA CT Reading Center. RAC was assessed at the renal ostia (right or left) or renal artery (right or left and proximal, middle, or distal third). Calcification was identified as a plaque of ≥1 mm^2 with a density of greater than 130 Hounsfield units and quantified using the previously described Agatston scoring method [17].

Laboratory

At all clinic examinations, blood samples were obtained following a 12-hour fast. The blood samples were assayed for total and high-density lipoprotein (HDL) cholesterol, triglycerides, glucose, creatinine levels, C-reactive protein (CRP), Interleukin-6 (IL-6), fibrinogen, leptin, adiponectin, TNF-α, and resistin. High-sensitivity CRP and fibrinogen were measured by immuno-nephelometry using the BNII instrument (N High Sensitivity CRP and N Antiserum to Human Fibrinogen; Dade Bering Inc., Deerfield, IL, USA). IL-6 was measured by ultrasensitive ELISA (Quantikine HS Human IL-6 Immunoassay; R&D Systems, Minneapolis, MN, USA). Leptin, adiponectin, TNF-α, and resistin were measured using Bio-Rad Luminex flow cytometry (Millipore, Billerica, MA, USA) at the Laboratory for Clinical Biochemistry Research (University of Vermont, Burlington, VT, USA).

Dyslipidemia was defined as total cholesterol:HDL ratio greater than 5.0 or if the participant used medication to reduce cholesterol. Diabetes was defined as fasting glucose greater than or equal to 126 mg/dL or the use of hypoglycemic medication. Estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration equation [18].

Statistical analysis

Among 1,978 potential participants, there were 685 individuals who had scans that were either incomplete or uninterpretable for renal artery calcification and were excluded from the analysis. An additional 6 participants were missing values for the covariates and were also excluded, resulting in a final analytic sample of 1,287 participants.

Characteristics of the population were determined with a mean and standard deviation or median and interquartile range for continuous variables, whereas categorical variables were summarized as a count and percentage of the study population. Testing for group differences between those with and without any renal artery calcium was conducted using the chi-squared test for categorical variables and using ANCOVA for continuous variables after adjusting for age, gender and race. A non-parametric ANCOVA was performed by rank-transforming the continuous variable and then using that rank variable as the outcome variable in the ANCOVA. Spearman correlation coefficients were calculated to study the relation between BMI, WC, HC, and WHR after adjusting for age, gender, and race/ethnicity.

To determine the association between the anthropometric measures and the presence of RAC, we utilized logistic regression and both the continuous and categorical forms of the anthropometric variables. Categories for BMI were based on the World Health Organization Classification System as set forth by the 1993 WHO expert committee meeting: normal (<25 kg/m^2), grade 1 overweight (25-29.9 kg/m^2), grade 2 overweight (30-39.9 kg/m^2), and grade 3 overweight (≥40 kg/m^2) [19]. Cut-offs for categorization of WC, HC, and WHR were based on the recommendations of the World Health Organization Expert Consultation in 2008. Waist circumference was defined as high if greater than 88 cm for females or greater than 102 cm for males. For hip circumference, measurement greater than 104 cm for females or greater than 113 cm for males was considered high. Furthermore, WHR was categorized as high if greater than 0.85 for females or 0.90 for males [20]. For all regression analyses, the initial model 1 was adjusted for age, gender and race/ethnicity. Models were subsequently adjusted for smoking, moderate and vigorous activity level, sedentary (leisure) level, and highest level of education completed (model 2); family history of CVD, hypertension, dyslipidemia, diabetes mellitus, eGFR (model 3); C-reactive protein, IL-6, fibrinogen, leptin, adiponectin, TNF-α, and resistin (model 4).

A two-tailed p-value <0.05 was considered statistically significant and all statistical analyses were conducted using SPSS (Version 19.0; IBM Corp, Armonk, NY, USA).

Results

Among the 1,287 participants included in this study, the average age was 67.7 years (SD 9.2); 56% were female, 37% Non-Hispanic White, 14% Chinese-American, 22% African-American, and 28% Hispanic American. The mean BMI was 28.1 kg/m^2 (SD 5.5), and the mean WC was 98.1 (SD 15.1).

Approximately 33.3% of participants had a RAC score > 0 (women: 57%; men: 43%). Of the total number of participants with RAC, Non-Hispanic White individuals comprised the largest ethnic group (39%), followed by Hispanic (29%), African-American (20%), and Chinese-American (12%) (Table 1).

Compared with individuals without RAC, participants with RAC were significantly older (71.8 vs 63.5 years), more likely to be a current smoker (15% *vs.* 9%), and more likely to have hypertension (68% *vs.* 53%), dyslipidemia (48% *vs.* 35%), diabetes (19% *vs.* 13%), or a family history of CVD (20% *vs.* 13%). Moreover, those with RAC > 0 had significantly higher WC (99.3 cm *vs.* 96.9 cm) and higher WHR (0.95 *vs.* 0.94). Presence of RAC was also associated with a lower eGFR (75.0 *vs.* 77.0 mL/min/1.73 m^2), and higher IL-6 (2.2 *vs.* 1.2 pg/mL) and resistin (16.8 *vs.* 15.0 ng/mL) (Table 1). In contrast, gender, ethnicity, and BMI were similar regardless of RAC presence.

There were significant positive correlations between the various anthropometric measures of obesity. The strongest correlations occurred between BMI and WC (Spearman correlation coefficient, 0.865; $p<0.01$), BMI and HC (Spearman correlation coefficient, 0.862; $p<0.01$), and WC and HC (Spearman correlation coefficient, 0.801; $p<0.01$). WC and WHR were positively correlated as well but to a lesser degree (Spearman correlation coefficient, 0.737; $p<0.01$).

When analyzed as a continuous variable (1-SD increment), increasing WC was significantly associated with RAC in models 1 (OR 1.19; 1.04-1.37) and 2 (OR 1.20; 1.05-1.38) but was attenuated and no longer statistically significant in model 3 (OR 1.08; 0.93-1.25) (Table 2). Similarly, WHR was significantly associated with RAC in models 1 and 2 but this relationship was no longer significant in model 3. BMI and HC were not significantly associated with the presence of RAC.

Table 3 displays the results of logistic regression analyses examining the independent associations of categories of BMI, WC, HC, and WHR with the presence RAC. After adjustment for age, gender and race/ethnicity (model 1), those with high WC had 70% higher odds of RAC compared with those with lower WC (odds ratio [OR] 1.70; 95% confidence interval [CI] 1.28-2.26). Full adjustment for smoking, moderate and vigorous activity level, sedentary (leisure) level, highest level of education completed, family history of CVD, hypertension, dyslipidemia, diabetes mellitus, eGFR, C-reactive protein, IL-6, fibrinogen, leptin, adiponectin, TNF-α, and resistin (model 4) only modestly attenuated the odds of RAC (1.54; 1.12-2.12). HC, BMI, and WHR as categorical variables were not significantly associated with prevalent RAC.

Discussion

In this relatively large multiethnic cohort of older individuals from five U.S. communities, we demonstrated that only the highest category of waist circumference was associated with the presence of any RAC. Specifically, when individuals are classified using the World Health Organization categories for obesity, those defined as having high WC have increased odds of RAC independent of traditional CVD risk factors and serologic markers of inflammation. In contrast, we also found that the associations between RAC with increasing WC and WHR as continuous variables are largely mediated by the traditional CVD risk factors. While this finding may seem inconsistent with the categorical analysis of WC, this discrepancy may actually reflect that the relationship between WC and presence of RAC is non-linear. In terms of other anthropometric variables, individuals categorized as high WHR or high BMI do not exhibit increased prevalence of RAC. In addition, BMI and HC were not significantly associated with increased RAC, either as continuous or categorical variables.

Given that WHR is derived from WC, one may have expected both anthropometric measures to demonstrate comparable connections with RAC. While the relationships between WC and WHR as continuous variables with RAC were attenuated in a similar manner following adjustment for traditional CVD risk factors, only the highest category of WC remained significantly associated with RAC after full adjustment and the highest category of WHR did not. This absence of an association between the category of high WHR and RAC may be attributable to the fact that WHR cut-offs in this study were derived from predominantly Caucasian populations. Thus, the World Health Organization categories for WHR may not be directly applicable to the multi-ethnic population in this study [21]. In addition, this study found that WC had a weaker correlation with WHR when compared to BMI and HC. This may be due to the preponderance of females in this study sample, as women tend to have parallel increases in both WC and HC over time. As a result, WC changes in women do not significantly affect WHR as they age [22], and this may account for our finding of a significant relationship between the highest category of WC and RAC but no such association for a high category of WHR. A post hoc analysis of the relationship between WHR and WC using linear regression revealed a slightly higher coefficient of determination for males than females (0.57 vs 0.50), which further suggests that changes in WC have less effect on WHR in women than in men in this older cohort.

In general, our results differ from previous studies that have demonstrated significant associations between BMI and WHR with subclinical CVD. Although no prior investigations have specifically studied RAC, there are a number of studies that explore the relationship between anthropometric measures and CAC, a well-established marker of subclinical atherosclerosis. For instance, while Ho et al similarly found that individuals with WC greater than or equal to 102 had increased risk of CAC (OR = 1.5, 95% CI 1.04-2.0), they also determined that WHR greater than 0.95 had the strongest correspondence with higher levels of CAC (OR = 1.6, 95% CI 1.2-2.3) in 1,054 men without prior history of CVD [5]. Comparably, a substudy of 2,744 individuals from the Dallas Heart Study revealed that more prevalent CAC was associated with the fifth versus first quintile of WHR following multivariable adjustment for traditional CVD risk factors (OR = 1.91, 95% CI 1.30-2.80). Likewise, the highest quintile of WHR was significantly associated with aortic

plaque (OR = 2.97, 95% CI 2.28-3.87) [6]. Lastly, Snell-Bergeon et al. reported that age-adjusted continuous measurements of body mass index (BMI) (OR = 1.9, 95% CI 1.5-2.6 in females and OR = 2.2, 95% CI 1.7-2.8 in males per one standard deviation) and WC (OR = 2.0, 95% CI 1.5-2.8 in females and OR 1.9, 95% CI 1.4-2.4 in males per one standard deviation) were significantly related to CAC in 762 subjects asymptomatic for coronary artery disease [7]. In contrast, the Rancho Bernardo study did not demonstrate a significant association between BMI (OR = 1.03, 95% CI 0.77-1.39 in men and OR 1.01, 95% CI 0.75-1.37 in women by ordinal logistic regression), WC (OR = 1.06, 95% CI 0.79-1.43 in men and OR 1.10, 95% CI 0.81-1.49 in women by ordinal logistic regression), and WHR (OR = 1.21, 95% CI 0.91-1.62 in men and OR 1.22, 95% CI 0.88-1.63 in women by ordinal logistic regression) with CAC, which is more consistent with the findings of this study. Compared with the current investigation, the Rancho Bernardo study population was less ethnically diverse and consisted of elderly, middle to upper-middle class Caucasian individuals with regular access to health care [23].

Our study advances the literature regarding RAC in several important ways. First, to our knowledge this is the first study to examine the relationship between obesity and RAC. Second, we demonstrated that the relationship between continuous measurement of WC with the presence of RAC is confounded by family history of CVD and mediated by hypertension, diabetes mellitus, dyslipidemia, and eGFR. Third, in addition to traditional demographic and CVD risk factors, we included adjustments for smoking, moderate and vigorous activity level, sedentary (leisure) level, highest level of education completed, and various serologic markers of inflammation and found that the association between high categories of WC and prevalent RAC is independent of these factors.

Several limitations were also present. Due to the cross-sectional nature of this study, causality in the relationship between obesity and RAC cannot be clearly determined. Second, a significant proportion of patients without sufficient visualization of the renal arteries was excluded, which could lead to potential bias of the results. Individuals with missing data were younger (61.6 vs 67.7 years), more likely to be male (59.2 vs 44.0%), Non-Hispanic White (47.1 vs 37.0%), and have a higher BMI (28.5 vs 28.1). With the exception of younger age, these factors are traditionally associated with higher risk of atherosclerosis, suggesting that our results for RAC may actually be underestimated. Lastly, the study population consisted of individuals without clinically apparent CVD aged 45-84 years. Because of this, results may not be generalized to all other populations.

The emergence of RAC as a marker of atherosclerosis carries several implications for clinical practice. On the subclinical level, presence of RAC likely reflects unique local changes within the renal vasculature as evidenced by previously established links between RAC with hypertension [11] and with increased all-cause mortality independent of atherosclerosis in othervascular beds [14]. Given the association between the highest category of waist circumference and RAC in the current study, adults with elevated waist circumference may be at high risk for developing complications of RAC. Regardless of the presence of pre-existing radiographic evidence for renal artery calcification, older individuals with increased waist circumference may benefit from treatment specifically targeted toward the effects of calcification within the renal arteries.

Acknowledgments

Financial support: This research was supported by contracts HHSN268201500003I, N01-HC-95159, N01-HC-95160, N01-HC-95161, N01-HC-95162, N01-HC-95163, N01-HC-95164, N01-HC-95165, N01-HC-95166, N01-HC-95167, N01-HC-95168 and N01-HC-95169 from the National Heart, Lung, and Blood Institute and by grants UL1-TR-000040 and UL1-TR-001079 from NCRR. Additional support from the American Federation for Aging Research and the Medical Student Training in Aging Research Program at the University of California, San Diego.

The authors thank the other investigators, the staff, and the participants of the MESA study for their valuable contributions. A full list of participating MESA investigators and institutions can be found at http://www.mesa-nhlbi.org.

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Highlights

- High waist circumference as categorized by the World Health Organization is associated with increased odds of renal artery calcification independent of traditional cardiovascular disease risk factors and serologic markers of inflammation
- Body mass index and hip circumference were not significantly associated with increased renal artery calcification
- The relationships between increasing waist circumference and waist-hip ratio as continuous variables with renal artery calcium are largely mediated by traditional cardiovascular disease risk factors, suggesting a non-linear relationship between waist circumference and renal artery calcium

Atherosclerosis. Author manuscript; available in PMC 2019 April 01.

Table 1

Cohort characteristics stratified by the presence of RAC

Variable	Total (n = 1,287)	RAC > 0 (n = 429)	RAC = 0 (n = 858)	*p*-value
Age (years), mean (SD)[a]	67.7 (9.2)	71.8 (8.7)	63.5 (8.6)	<0.01
Gender, n (%)[b]				0.52
Female	717 (56)	244 (57)	469 (55)	
Male	570 (44)	185 (43)	389 (45)	
Race/ethnicity, n (%)[c]				0.19
Non-Hispanic White	473 (37)	169 (39)	304 (36)	
Chinese-American	179 (14)	52 (12)	127 (15)	
African-American	278 (22)	84 (20)	194 (23)	
Hispanic	357 (28)	124 (29)	233 (27)	
BMI (kg/mg2), mean (SD)d	28.1 (5.5)	28.4 (5.5)	27.8 (5.3)	0.08
WC (cm), mean (SD)[d]	98.1 (15.1)	99.3 (15.0)	96.9 (14.6)	<0.01
HC (cm), mean (SD)[d]	104.0 (11.6)	104.6 (11.5)	103.4 (11.1)	0.03
WHR, mean (SD)[d]	0.94 (0.07)	0.95 (0.08)	0.94 (0.06)	<0.01
Smoking status, n (%)[d]				<0.01
Never	600 (47)	180 (42)	420 (49)	
Former	548 (43)	187 (44)	361 (42)	
Current	139 (11)	62 (15)	77 (9)	
Total moderate and vigorous activity level (METs/week), median (interquartile range)[d]	735.0 (99.4-1762.5)	747.0 (110.4-1659.0)	798.0 (100.5-1644.0)	0.41
Total sedentary (leisure) level (METs/week), median (interquartile range)[d]	1470.0 (930.0-2280.0)	1471.4 (948.2-2270.0)	1450.7 (961.0-2575.5)	0.09
Highest level of education, n (%)[d]				<0.01
High school graduate or less	490 (38)	194 (45)	296 (35)	
Some college	217 (17)	68 (16)	149 (17)	
College graduate or higher	580 (45)	168 (39)	412 (48)	
Family history of CVD, n (%)[d]	214 (17)	86 (20)	112 (13)	<0.01
Hypertension, n (%)[d]	777 (60)	292 (68)	451 (53)	<0.01

Variable	Total (n = 1,287)	RAC > 0 (n = 429)	RAC = 0 (n = 858)	*p*-value
Dyslipidemia, n (%)[d]	535 (42)	205 (48)	304 (35)	<0.01
Diabetes, n (%)[d]	203 (16)	82 (19)	108 (13)	<0.01
eGFR (mL/min/1.73 m2), mean(SD)[d]	76.0 (15.9)	75.0 (15.8)	77.0 (15.3)	0.02
C-reactive protein (mg/L), median (interquartile range)[d]	1.5 (0.8-3.4)	1.7 (0.7-3.0)	1.4 (0.6-3.5)	0.22
Interleukin-6 (pg/mL), median (interquartile range)[d]	1.9 (1.2-3.0)	2.2 (1.0-3.5)	1.2 (1.0-2.9)	<0.001
Fibrinogen (mg/dL), median (interquartile range)[d]	431.0 (382.0-489.0)	440.5 (312.0-445.0)	436.2 (355.0-467.3)	0.32
Leptin (ng/mL), median (interquartile range)[d]	14.0 (5.8-30.4)	15.6 (6.8-32.0)	14.0 (5.9-29.5)	0.19
Adiponectin (μg/mL), median (interquartile range)[d]	17.8 (12.1-26.9)	16.8 (13.9-27.0)	18.0 (13.0-29.5)	0.70
TNF-α (pg/mL), median (interquartile range)[d]	4.6 (3.4-6.4)	4.5 (3.3-6.8)	4.6 (3.5-7.0)	0.22
Resistin (ng/mL), median (interquartile range)[d]	15.2 (11.9-19.5)	16.8 (10.5-21.2)	15.0 (10.8-20.5)	0.047

[a] Adjusted for gender and race.

[b] Adjusted for age and race.

[c] Adjusted for age and gender.

[d] Adjusted for age, gender, and race.

Table 2

Logistic regression analyses of the association of BMI, WC, HC, and WHR as continuous variables and presence of RAC

Variable	Model	OR	95% CI	*p*-value
BMI[a]	Model 1	1.12	0.98-1.29	0.10
	Model 2	1.13	0.98-1.30	0.09
	Model 3	1.00	0.86-1.16	0.98
WC[a]	Model 4	1.01	0.85-1.20	0.94
	Model 1	1.19	1.04-1.37	<0.01
	Model 2	1.20	1.05-1.38	<0.01
	Model 3	1.08	0.93-1.25	0.31
	Model 4	1.11	0.94-1.31	0.23
HC[a]	Model 1	1.13	0.98-1.29	0.09
	Model 2	1.15	1.00-1.32	0.06
	Model 3	1.05	0.91-1.22	0.49
	Model 4	1.08	0.92-1.27	0.35
WHR[a]	Model 1	1.21	1.05-1.40	<0.01
	Model 2	1.21	1.04-1.39	0.01
	Model 3	1.09	0.93-1.26	0.29
	Model 4	1.08	0.92-1.27	0.34

Model 1: Age, gender and race/ethnicity.
Model 2: Smoking, moderate and vigorous activity level, sedentary (leisure) level, and highest level of educationcompleted.
Model 3: Family history of CVD, hypertension, dyslipidemia, diabetes mellitus, eGFR.
Model 4: C-reactive protein, IL-6, fibrinogen, leptin, adiponectin, TNF-α, and resistin.

[a]Per 1-SD increment increase

Table 3

Logistic regression analyses of the association of BMI, WC, HC, and WHR as categorical variables and presence of RAC

Category (n)	Model	OR	95% CI	*p*-value
BMI[a]				
Grade 1 overweight (519)	Model 1	1.18	0.86-1.63	0.31
	Model 2	1.19	0.86-1.65	0.30
	Model 3	1.04	0.74-1.45	0.83
	Model 4	1.03	0.72-1.46	0.88
Grade 2 overweight (342)	Model 1	1.35	0.94-1.94	0.10
	Model 2	1.35	0.94-1.95	0.11
	Model 3	1.03	0.70-1.50	0.90
	Model 4	1.03	0.67-1.58	0.89
Grade 3 overweight (40)	Model 1	1.22	0.53-2.80	0.64
	Model 2	1.28	0.56-2.94	0.56
	Model 3	0.83	0.35-1.95	0.66
	Model 4	0.87	0.36-2.13	0.76
High WC (695)[b]	Model 1	1.70	1.28-2.26	<0.01
	Model 2	1.73	1.30-2.32	<0.01
	Model 3	1.49	1.10-2.01	<0.01
	Model 4	1.54	1.12-2.12	<0.01
High HC (405)[c]	Model 1	1.39	1.01-1.90	0.04
	Model 2	1.41	1.02-1.94	0.04
	Model 3	1.23	0.89-1.71	0.22
	Model 4	1.31	0.91-1.87	0.14
High WHR (1046)[d]	Model 1	1.22	0.86-1.73	0.26
	Model 2	1.21	0.84-1.72	0.31
	Model 3	0.93	0.64-1.35	0.68
	Model 4	0.89	0.60-1.31	0.54

Model 1: Age, gender and race/ethnicity.

Model 2: Smoking, moderate and vigorous activity level, sedentary (leisure) level, and highest level of education completed.

Model 3: Family history of CVD, hypertension, dyslipidemia, diabetes mellitus, eGFR.
Model 4: C-reactive protein, IL-6, fibrinogen, leptin, adiponectin, TNF-α, and resistin.

[a] Reference is BMI < 25 kg/m^2.

[b] Reference is WC ≤ 88 cm for females or ≤ 102 cm for males.

[c] Reference is HC ≤ 104 cm for females or ≤ 113 cm for males.

[d] Reference is WHR ≤ 0.85 for females or ≤ 0.90 for males.