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Body surface area is a predictor of coronary artery calcium, whereas body mass index is not

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Abstract

Introduction—We sought to establish whether elevated BMI and body surface area (BSA), two measures of obesity, are predictors of coronary artery calcium (CAC).

Methods—We retrospectively analyzed 3172 consecutive patients who underwent calcium scoring at our center. We applied a multiple logistic regression model to estimate the independent association between BMI of at least 25 kg/m² and incidence of CAC with adjustment for covariates. We carried out the same analysis to find out if there is an independent association between BSA of at least 1.71m² (commonly used definition for abnormally elevated BSA) and incidence of CAC. We also performed a sex subanalysis based on BMI and BSA.

Results—There were 2105 patients in the cohort with BMI of at least 25 kg/m² compared with 1067 patients with BMI of less than 25 kg/m². After adjustment for covariates, a significant association was not found between increased BMI and incidence of CAC. In addition, no significant findings were found in the sex subanalysis. A total of 2760 patients had a BSA of at least 1.71m² compared with 412 patients with BSA of less than 1.71m². After adjustment for covariates, a significant association (odds ratio 2.08, 95% confidence interval 1.16–3.73, $P = 0.014$) was found between elevated BSA and CAC incidence. There were 89 men with BSA of at least 1.9m² and 2248 with BSA of at least 1.9m². After adjustment for covariates, the logistic regression model showed a significant association (odds ratio 2.24, 95% confidence interval 1.19–4.21, $P = 0.012$) between BSA of at least 1.9m² and incidence of CAC.

Conclusion—Elevated BSA is a predictor of CAC incidence, whereas elevated BMI is not. Moreover, elevated BSA is a predictor of CAC incidence particularly in men.

Keywords

coronary artery calcium; coronary artery disease; obesity

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There are no conflicts of interest.

Conflicts of interest

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Introduction

Obesity is an epidemic in the United States, as a majority of Americans are overweight and one-third are obese [1]. In addition, obesity is believed to be an independent predictor of atherosclerotic plaque [2], as well as mortality [3].

BMI is a commonly used measure of obesity, which is calculated based on a person's height and weight [4]. Body surface area (BSA) is a lesser known measure based on height and weight that can also be used to predict obesity [5]. Its current major clinical use is for chemotherapy drug dosing [6].

Coronary artery calcium (CAC) score is a marker for atherosclerotic plaque burden. A clear association has been shown between CAC score and conventional cardiovascular risk factors including premature family history of coronary artery disease (CAD), diabetes, and lipid values [7–9]. Although pathologic specimens have shown that the amount of CAC only weakly correlates with the degree of luminal narrowing in the coronary arteries [10–12], the likelihood of obstructive disease certainly increases with increasing CAC scores [13–15]. In addition, elevated CAC scores have been shown to be a predictor of incidence of myocardial infarction and death from cardiovascular disease [16]. Patients with 0 CAC scores may still have atherosclerosis in the form of noncalcified plaque, but their coronary event rates have been shown to be low [17].

We did a retrospective study to see whether elevated BMI and BSA can accurately predict incidence of CAC.

Methods

Study patients

We retrospectively analyzed 3172 patients who underwent either electron beam computed tomography (EBCT) or multidetector computed tomography (MDCT) at our center (UCLA Harbor Hospital) between May 1999 and January 2009 for a variety of indications. Blood draws and detailed surveys regarding symptoms and past medical history were performed before scanning. No exclusion criteria were applied. The study was approved by the institutional review board at our institution, and informed consent was obtained from each participant.

Baseline characteristics collected included age, sex, weight, height, BMI, self-identified ethnicity (Caucasian, African-American, Hispanic/Latino, Asian, or other), comorbid conditions (hyperlipidemia, hypertension, diabetes), current smoking status, and family history. Reported comorbidities were verified by physical examination, chart review, and laboratory data.

Scan protocol

All MDCT scans were performed at our center using a 64-multidetector row Lightspeed VCT scanner (GE Healthcare, Waukesha, Wisconsin, USA). The EBCT scans were performed on an Imatron C-100 scanner (Imatron, San Francisco, California, USA). Patients

were given β blockers as needed for a goal heart rate of less than 60 beats per minute. Both the MDCT and EBCT scanning protocols and interpretation methods for this study were identical to our core CT reading laboratory function in the Multi-Ethnic Study of Atherosclerosis [18].

Calcification was quantified as previously described by Agatston *et al.* [19]. Total CAC score was calculated by the Agatston method as the sum of lesion scores from the four major coronary arteries (left main, left anterior descending, left circumflex, and right coronary arteries). A single experienced interpreter interpreted all studies based on the commercially available software (AccuImage Diagnostics Corporation, San Mateo, California, USA) at the time of the study. CAC scores were not remeasured for the purposes of this study.

Statistical analysis

Patients initially were divided into two groups: BMI of at least 25 kg/m² and BMI less than 25 kg/m². We applied a multiple logistic regression model to estimate the independent association between BMI of at least 25 kg/m² (consistent with the WHO definition of supranormal BMI) [20] and incidence of CAC (defined as CAC score ≥ 1). We first did this using an unadjusted model, and then used a second model adjusted for age, sex, ethnicity, and cardiac risk factors. A sex subanalysis was performed similarly using the same BMI categories.

Patients were then divided into two groups based on BSA: BSA of at least 1.71m² and BSA less than 1.71m² (normal BSA 1.7m²) [21]. We applied a multiple logistic regression model to estimate the independent association between BSA of at least 1.71m² and incidence of CAC. We first did this using an unadjusted model, and then used a second model adjusted for age, sex, ethnicity, and cardiac risk factors.

A sex subanalysis was performed similarly for BSA as well, but with different groups. Men were divided into two groups: BSA of more than 1.9m² and BSA of 1.9m² or less. Women were divided into two groups: BSA more than 1.6m² and BSA 1.6m² or less. These cut-offs were based on average BSAs for each sex, and are the commonly used cut-offs for BSA by sex [21].

BMI was calculated by dividing the weight (kg) by the square root of the height (m) squared [20]. BSA was calculated by the square root of the product of the weight (kg) and height (cm) divided by 3600 [21].

All data were exported to SAS (version 9.2; SAS Institute Inc., Cary, North Carolina, USA) for analysis.

Results

There were 2105 patients in the cohort with BMI of at least 25 kg/m² compared with 1067 patients with BMI of less than 25 kg/m². The group with the larger BMI had significantly more incidence of hypertension ($P<0.001$), diabetes ($P<0.001$), and hyperlipidemia ($P=0.03$). Furthermore, there were significant differences in age, sex, and ethnicity ($P<0.001$) (see Table 1 for full results).

No significant statistical association [odds ratio (OR) 1.18, 95% confidence interval (CI) 0.99–1.41, $P=0.07$] was found between BMI of at least 25 kg/m² and incident CAC in the unadjusted logistic regression model. After adjustment for age, sex, ethnicity, hypertension, diabetes, and hyperlipidemia, a significant association was still not found (OR 1.14, 95% CI: 0.90–1.44, $P=0.28$) (see Table 2 for full results).

For the sex subanalysis, there were 2337 men in our cohort. A total of 1640 had BMI of at least 25 kg/m² compared with 697 with BMI less than 25 kg/m². The group with the larger BMI among the men had significantly more hypertension ($P<0.001$) and hyperlipidemia ($P=0.001$). Moreover, there was a significant difference in ethnicity ($P<0.001$). See Table 3 for full results of the patient characteristics for each sex. After adjustment for ethnicity, hypertension, and hyperlipidemia using a logistic regression model, a significant association was not found (OR 1.10, 95% CI: 0.81–1.41, $P=0.54$) between BMI of at least 25 kg/m² and incident CAC in the male population (see Table 4 for full results).

There were 835 women in our cohort, of whom 465 had BMI of at least 25 kg/m² compared with 370 with BMI less than 25 kg/m². The group with larger BMI among the women had significantly more hypertension ($P<0.001$) and diabetes ($P<0.001$). There was also a significant difference in ethnicity ($P=0.014$). See Table 3 for full results of the patient characteristics for each sex. After adjustment for ethnicity, hypertension, and hyperlipidemia using a logistic regression model, a significant association was not found (OR 1.26, 95% CI: 0.86–1.87, $P=0.24$) between BMI of at least 25 kg/m² and incident CAC in the female population (see Table 4 for full results).

A total of 2760 patients had a BSA of at least 1.71m² compared with 412 with BSA less than 1.71m². There were significant differences between the two groups in terms of age, sex, and ethnicity ($P<0.001$). The two groups did not have any other significant differences in cardiac risk factors (see Table 5 for details).

The unadjusted logistic regression model showed a significant association (OR 1.76, 95% CI: 1.41–2.20, $P<0.001$) between BSA of at least 1.71m² and incident CAC. After adjustment for age, sex, and ethnicity, this significant association was retained (OR 2.08, 95% CI: 1.16–3.73, $P=0.014$) (see Table 6 for full results).

For the sex subanalysis, there were 1831 men with BSA of at least 1.9m² and 506 with BSA<1.9m². There was a significant difference between the two male groups in terms of ethnicity ($P<0.001$). There were no significant differences in cardiac risk factors. See Table 7 for full results of the patient characteristics for each sex. After adjustment for ethnicity, the logistic regression model showed a significant association (OR 2.24, 95% CI: 1.19–4.21, $P=0.012$) between BSA of at least 1.9m² and incidence of CAC in the male group (see Table 8 for full results).

There were 687 women with BSA of at least 1.6m² and 148 with BSA less than 1.6m². There were significant differences between the two groups in terms of age ($P<0.001$) and ethnicity ($P<0.001$). See Table 7 for full results of the patient characteristics for each sex. After adjustment for ethnicity and hypertension, the logistic regression model showed no

significant association (OR 1.19, 95% CI: 0.49–2.89, $P=0.71$) between BSA of at least 1.6m^2 and incidence of CAC in the female group (see Table 8 for full results).

Discussion

The few studies focusing at the connection between elevated BMI and CAD have revealed mixed results. In 2000, Kim *et al.* [22] showed that increased BMI was a risk factor for incidence of CAD in a cohort of 5209 Framingham Heart Study participants, as well as a risk factor for CAD mortality in women. Subsequently in a 2004 prospective study of 906 women, Wessel *et al.* [23] found that elevated BMI was not associated with obstructive CAD or adverse cardiovascular events. In 2011, investigators analyzed the Systematic Coronary Risk Evaluation dataset and found a graded relationship between increasing BMI and cardiovascular mortality [24]. In the same year, however, Coutinho *et al.* [25] found that central obesity and not BMI is associated with increased mortality in a large meta-analysis. There have not been any significant studies analyzing BSA and CAD.

Our large retrospective study shows that elevated BSA significantly predicts incidence of CAC, whereas elevated BMI does not. BSA is likely a better predictor of CAD, because it is a more accurate measure of obesity than BMI. BMI is known to overestimate obesity in patients with a muscular build [26]. Muscle is more dense than fat, and BMI is not able to differentiate increased weight secondary to muscle versus fat [27]. One pound of muscle obviously weighs the same as one pound of fat, but takes up less area. Therefore, BSA is a more accurate measure of obesity, including central obesity, as it is a measurement of area and is able to account for the difference between muscle and fat better than BMI.

Interestingly, in the sex subanalysis of our cohort, elevated BSA significantly predicted CAC incidence in men, but not women. This is likely because men typically have a greater percentage of muscle mass than women [28], making BSA particularly useful in males as a measure of obesity.

This is only a single-center retrospective study on this topic, so that is an obvious limitation. Prospective studies analyzing BSA and cardiovascular outcomes would be interesting and provide us more information. Perhaps, BSA should be used by primary-care physicians as the measure for obesity in the general patient population, and increased BSA treated as a cardiovascular risk factor.

References

1. Selassie M, Sinha AC. The epidemiology and aetiology of obesity: a global challenge. *Best Pract Res Clin Anaesthesiol.* 2011; 25:1–9. [PubMed: 21516909]
2. Eckel RH. Obesity and heart disease: a statement for healthcare professionals from the nutrition committee, American Heart Association. *Circulation.* 1997; 96:3248–3250. [PubMed: 9386201]
3. Lewis CE, McTigue KM, Burke LE, Poirier P, Eckel RH, Howard BV, et al. Mortality, health outcomes, and body mass index in the overweight range: a science advisory from the American Heart Association. *Circulation.* 2009; 119:3263–3271. [PubMed: 19506107]
4. Kushner RF, Blatner DJ. Risk assessment of the overweight and obese patient. *J Am Diet Assoc.* 2005; 105 (5 Suppl 1):S53–S62. [PubMed: 15867897]

5. Body surface area for adjustment of drug dose. *Drug Ther Bull.* 2010; 48:33–36. [PubMed: 20200147]
6. Pinkel D. The use of body surface area as a criterion of drug dosage in cancer chemotherapy. *Cancer Res.* 1958; 18:853–856. [PubMed: 13573353]
7. Hecht HS, Superko HR, Smith LK, McColgan BP. Relation of coronary artery calcium identified by electron beam tomography to serum lipoprotein levels and implications for treatment. *Am J Cardiol.* 2001; 87:406–412. [PubMed: 11179523]
8. Kuller LH, Matthews KA, Sutton-Tyrrell K, Edmunowicz D, Bunker CH. Coronary and aortic calcification among women 8 years after menopause and their premenopausal risk factors: the Healthy Women Study. *Arterioscler Thromb Vasc Biol.* 1999; 19:2189–2198. [PubMed: 10479662]
9. Daviglus ML, Pirzada A, Liu K, Yan LL, Garside DB, Dyer AR, et al. Comparison of low risk and higher risk profiles in middle age to frequency and quantity of coronary artery calcium years later. *Am J Cardiol.* 2004; 94:367–369. [PubMed: 15276108]
10. Simons DB, Schwartz RS, Edwards WD, Sheedy PF, Breen JF, Rumberger JA. Noninvasive definition of anatomic coronary disease by ultrafast computed tomographic scanning: a quantitative pathologic comparison study. *J Am Coll Cardiol.* 1992; 20:1118–1126. [PubMed: 1401612]
11. Mautner GC, Mautner SL, Froelich J, Feuerstein IM, Proschan MA, Roberts WC, et al. Coronary artery calcification: assessment with electron beam CT and histomorphometric correlation. *Radiology.* 1994; 192:619–623. [PubMed: 8058924]
12. Detrano R, Tang W, Kang X, Mahaisavariya P, McCrae M, Garner D, et al. Accurate coronary calcium phosphate mass measurements from electron beam computed tomograms. *Am J Card Imaging.* 1995; 9:167–173. [PubMed: 7549356]
13. Wexler L, Brundage B, Crouse J, Detrano R, Fuster V, Maddahi J, et al. Coronary artery calcification: pathophysiology, epidemiology, image methods and clinical implications. A scientific statement from the American Heart Association. *Circulation.* 1996; 94:1175–1192. [PubMed: 8790070]
14. Budoff MJ, Georgiou D, Brody A, Agatston AS, Kennedy J, Wolfkiel C, et al. Ultrafast computed tomography as a diagnostic modality in the detection of coronary artery disease – a multicenter study. *Circulation.* 1996; 93:898–904. [PubMed: 8598080]
15. Guerci AD, Spadaro LA, Popma JJ, Goodman KJ, Brundage BH, Budoff M, et al. Electron beam tomography of the coronary arteries: relationship of coronary calcium score to arteriographic findings in asymptomatic and symptomatic adults. *Am J Cardiol.* 1997; 79:128–133. [PubMed: 9193010]
16. Budoff MJ, Gul KM. Expert review on coronary calcium. *Vasc Health Risk Manag.* 2008; 4:315–324. [PubMed: 18561507]
17. Raggi P, Callister TQ, Cooil B, He Z, Lippolis NJ, Russo DJ, et al. Identification of patients at increased risk of first unheralded acute myocardial infarction by electron beam computed tomography. *Circulation.* 2000; 101:850–855. [PubMed: 10694523]
18. Carr JJ, Nelson JC, Wong ND, McNitt-Gray M, Arad Y, Jacobs DR, et al. Calcified coronary artery plaque measurement with cardiac CT in population based studies: standardized protocol of Multi-Ethnic Study of Atherosclerosis (MESA) and Coronary Artery Risk Development in Young Adults (CARDIA) study. *Radiology.* 2005; 234:35–43. [PubMed: 15618373]
19. Agatston AS, Janowitz WR, Hildner FJ, Zusmer MR, Viamonte M, Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. *J Am Coll Cardiol.* 1990; 15:827–832. [PubMed: 2407762]
20. World Health Organization (WHO). WHO Technical Report Series 894. Geneva: World Health Organization; 1995. Obesity: preventing and managing the global epidemic Report of a WHO Consultation.
21. Mosteller RD. Simplified calculation of body-surface area. *N Engl J Med.* 1987; 317:1098. [PubMed: 3657876]
22. Kim KS, Owen WL, Williams D. A comparison between BMI and conicity index on predicting coronary heart disease: the Framingham Heart Study. *Ann Epidemiol.* 2000; 10:424–431. [PubMed: 11018345]

23. Wessel TR, Arant CB, Olson MB, Johnson BD, Reis SE, Sharaf BL, et al. Relationship of physical fitness vs body mass index with coronary artery disease and cardiovascular events in women. *JAMA*. 2004; 292:1179–1187. [PubMed: 15353530]
24. Dudina A, Cooney MT, De Bacquer D, De Backer G, Ducimetiere P, Jousilahti P, et al. Relationships between body mass index, cardiovascular mortality, and risk factors: a report from the SCORE investigators. *Eur J Cardiovasc Prev Rehabil*. 2011; 18:731–742. [PubMed: 21642320]
25. Coutinho T, Goel K, Correa de Sa D, Kragelund C, Kanaya AM, Zeller M, et al. Central obesity and survival in subjects with coronary artery disease: a systematic review of the literature and collaborative analysis with individual subject data. *J Am Coll Cardiol*. 2011; 57:1877–1886. [PubMed: 21545944]
26. Rothman KJ. BMI-related errors in the measurement of obesity. *Int J Obes*. 2008; 32:S56–S59.
27. Hamdy O, Porramatikul S, Al-Ozairi E. Metabolic obesity: the paradox between visceral and subcutaneous fat. *Curr Diab Rev*. 2006; 2:367–373.
28. Maughan RJ, Watson JS, Weir J. Strength and cross-sectional area of human skeletal muscle. *J Physiol*. 1983; 338:37–49. [PubMed: 6875963]

Table 1

Clinical and demographic characteristics of the cohort stratified by body mass index

	BMI < 25, <i>n</i> = 1067	BMI ≥ 25, <i>n</i> = 2105	<i>P</i> -value
Age (years)	67 ± 19	64 ± 19	< 0.001
Women, <i>n</i> (%)	370 (34.7)	465 (22.1)	< 0.001
Ethnicity, <i>n</i> (%)	–	–	< 0.001
Caucasian	660 (76.7)	1392 (79.9)	–
African-American	34 (4.0)	78 (4.5)	–
Hispanic/Latino	57 (6.6)	160 (9.2)	–
Asian	95 (11.0)	86 (4.9)	–
Other	14 (1.6)	27 (1.5)	–
Smoking	148 (14.0)	328 (15.8)	0.20
Hypertension, <i>n</i> (%)	294 (28.3)	889 (43.5)	< 0.001
Family history, <i>n</i> (%)	616 (59.2)	1217 (58.7)	0.13
Diabetes, <i>n</i> (%)	67 (6.4)	223 (10.8)	< 0.001
Hyperlipidemia, <i>n</i> (%)	567 (56.8)	1210 (60.9)	0.03

Table 2
Association between BMI and incidence of coronary artery calcium in full population^a

	Unadjusted			Adjusted ^a		
	OR	95% CI	P-value	OR	95% CI	P-value
BMI < 25 kg/m ²	Referent (1.0)	–	–	Referent (1.0)	–	–
BMI ≥ 25 kg/m ²	1.18	0.99–1.41	0.07	1.14	0.90–1.44	0.28

CI, confidence interval; OR, odds ratio.

^a Adjusted for age, sex, ethnicity, hypertension, diabetes, and hyperlipidemia.

Table 3

Clinical and demographic characteristics of the male and female cohort stratified by body mass index

	Men			Women		
	BMI < 25, n = 697	BMI 25, n = 1640	P-value	BMI < 25, n = 370	BMI 25, n = 465	P-value
Age (years)	66 ± 20	64 ± 19	0.012	69 ± 18	67 ± 18	0.06
Ethnicity, n (%)	–	–	< 0.001	–	–	0.014
Caucasian	447 (79.5)	1113 (82.0)	–	213 (71.5)	279 (72.3)	–
African-American	23 (4.1)	53 (3.9)	–	11 (3.7)	25 (6.5)	–
Hispanic/Latino	24 (4.3)	107 (7.9)	–	33 (11.1)	53 (13.7)	–
Asian	58 (10.3)	64 (4.7)	–	37 (12.4)	22 (5.7)	–
Other	10 (1.8)	20 (1.5)	–	4 (1.3)	7 (1.8)	–
Smoking	91 (13.2)	262 (16.2)	0.066	57 (15.7)	66 (14.4)	0.597
Hypertension, n (%)	182 (26.6)	633 (39.9)	< 0.001	112 (31.5)	256 (55.8)	< 0.001
Family history, n (%)	381 (55.5)	910 (56.5)	0.289	235 (66.2)	307 (66.7)	0.52
Diabetes, n (%)	47 (6.9)	153 (9.5)	0.042	20 (5.6)	70 (15.5)	< 0.001
Hyperlipidemia, n (%)	349 (53.6)	940 (61.2)	0.001	218 (62.6)	270 (60.0)	0.447

Table 4
Association between BMI and incidence of coronary artery calcium in male and female population^a

	Men			Women		
	OR	95% CI	P-value	OR	95% CI	P-value
BMI < 25 kg/m ²	Referent (1.0)	–	–	Referent (1.0)	–	–
BMI ≥ 25 kg/m ²	1.10	0.81–1.41	0.54	1.26	0.86–1.87	0.24

CI, confidence interval; OR, odds ratio.

^aMale cohort adjusted for age, race, hypertension, and hyperlipidemia. Female cohort adjusted for ethnicity, hypertension, and diabetes.

Table 5

Clinical and demographic characteristics of the cohort stratified by body surface area

	BSA < 1.71, <i>n</i> = 412	BSA ≥ 1.71, <i>n</i> = 2760	<i>P</i> -value
Age (years)	69 ± 18	65 ± 19	< 0.001
Women, <i>n</i> (%)	323 (78.4)	512 (18.6)	< 0.001
Ethnicity, <i>n</i> (%)	–	–	< 0.001
Caucasian	220 (64.1)	1832 (81.1)	–
African-American	7 (2.0)	105 (4.6)	–
Hispanic/Latino	45 (13.1)	172 (7.6)	–
Asian	64 (18.7)	117 (5.2)	–
Other	7 (2.0)	34 (1.5)	–
Height (cm)	159.5 ± 6.4	176.2 ± 8.8	< 0.001
Weight (kg)	58.2 ± 5.2	85.91 ± 13.9	< 0.001
BMI (kg/m ²)	23.0 ± 2.7	27.6 ± 3.8	< 0.001
Smoking	65 (16.1)	411 (15.0)	0.57
Hypertension, <i>n</i> (%)	156 (38.8)	1027 (38.3)	0.29
Family history, <i>n</i> (%)	247 (62.2)	1586 (58.4)	0.09
Diabetes, <i>n</i> (%)	41 (10.3)	249 (9.2)	0.49
Hyperlipidemia, <i>n</i> (%)	234 (59.8)	1543 (59.5)	0.88

BSA, body surface area.

Table 6
Association between body surface area and incidence of coronary artery calcium in full population^a

	Unadjusted			Adjusted ^a		
	OR	95% CI	P-value	OR	95% CI	P-value
BSA < 1.71 m ²	Referent (1.0)	–	–	Referent (1.0)	–	–
BSA ≥ 1.71 m ²	1.76	1.41–2.20	<0.001	2.08	1.16–3.73	0.014

BSA, body surface area; CI, confidence interval; OR, odds ratio.

^a Adjusted for age, gender, and ethnicity.

Table 7
Clinical and demographic characteristics of the male and female cohort stratified by body surface area

	Men			Women		
	BSA < 1.9, n = 506	BSA 1.9, n = 1831	P-value	BSA < 1.6, n = 148	BSA 1.6, n = 687	P-value
Age (years)	65 ± 19	64 ± 20	0.17	73 ± 17	66 ± 18	< 0.001
Ethnicity, n (%)	–	–	< 0.001	–	–	< 0.001
Caucasian	288 (67.9)	1272 (85.1)	–	72 (62.1)	420 (73.9)	–
African-American	16 (3.8)	60 (4.0)	–	3 (2.6)	33 (5.8)	–
Hispanic/Latino	35 (8.3)	96 (6.4)	–	15 (12.9)	71 (12.5)	–
Asian	77 (18.2)	45 (3.0)	–	23 (19.8)	36 (6.3)	–
Other	8 (1.9)	22 (1.5)	–	3 (2.6)	8 (1.4)	–
Height (cm)	169.7 ± 7.0	180.3 ± 6.5	< 0.001	158.1 ± 4.9	164.6 ± 7.0	< 0.001
Weight (kg)	68.2 ± 5.7	91.4 ± 12.4	< 0.001	53.4 ± 4.0	74.7 ± 12.6	< 0.001
BMI (kg/m ²)	23.8 ± 2.6	28.1 ± 3.5	< 0.001	22.0 ± 1.9	27.5 ± 4.5	< 0.001
Smoking	68 (13.6)	285 (15.7)	0.24	23 (15.9)	100 (14.8)	0.74
Hypertension, n (%)	156 (31.6)	659 (37.1)	0.07	56 (38.1)	312 (46.7)	0.14
Family history, n (%)	265 (53.3)	1026 (57.0)	0.30	95 (66.0)	447 (66.6)	0.89
Diabetes, n (%)	53 (10.7)	147 (8.2)	0.07	16 (11.0)	74 (11.1)	0.95
Hyperlipidemia, n (%)	283 (59.5)	1006 (58.8)	0.79	93 (66.9)	395 (59.9)	0.13

BSA, body surface area.

Table 8
Association between body surface area and incidence of coronary artery calcium in male and female population^a

	Men			Women		
	OR	95% CI	P-value	OR	95% CI	P-value
BSA < 1.9 m ² for men, BSA < 1.6 m ² for women	Referent (1.0)	–	–	Referent (1.0)	–	–
BSA 1.9 m ² for men, BSA 1.6 m ² for women	2.24	1.19–4.21	0.012	1.19	0.49–2.89	0.71

BSA, body surface area; CI, confidence interval; OR, odds ratio.

^aMale cohort adjusted for ethnicity. Female cohort adjusted for age and ethnicity.