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ORIGINAL RESEARCH

Associations of Central Adiposity With Subclinical Coronary Calcification and Disease Progression

Meta-Analysis of 68,629 Participants

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ABSTRACT

BACKGROUND Coronary artery calcium (CAC) is a robust marker of subclinical atherosclerosis and cardiovascular risk. Body mass index (BMI) may inadequately reflect central adiposity, which may be more strongly associated with CAC burden and progression.

OBJECTIVES The objective of the study was to compare associations of BMI, waist circumference (WC), and waist-to-hip ratio (WHR) with CAC presence and progression in individuals without established cardiovascular disease.

METHODS A systematic search of PubMed, MEDLINE, Embase, CINAHL, Scopus, and gray literature (January 2000–March 2025) identified studies reporting anthropometric measures by CAC status (CAC = 0 vs >0) or progression. Two reviewers applied Preferred Reporting Items for Systematic reviews and Meta-Analyses-based criteria. Standardized mean differences (SMDs) were pooled using random-effects models. Heterogeneity was assessed with I-squared statistic (I^2), and between-group differences with chi-square interaction testing.

RESULTS Forty-nine studies were included (33 CAC presence; 16 progression) across 10 countries. For CAC presence (n = 43,490), WHR (SMD: 0.46; 95% CI: 0.35-0.56; $I^2 = 81\%$) and WC (SMD: 0.32; 95% CI: 0.23-0.40; $I^2 = 86\%$) were higher in those with CAC >0, whereas BMI was not significant (SMD: 0.13; 95% CI: -0.03-0.29; $I^2 = 98\%$). WHR showed a stronger association than WC ($P = 0.04$). Meta-regression indicated attenuation with higher hypertension prevalence. For CAC progression (n = 25,141; mean follow-up 4.9 ± 3.0 years), WC (SMD: 0.34; 95% CI: 0.27-0.42; $I^2 = 64\%$) was more strongly associated than BMI (SMD: 0.21; 95% CI: 0.16-0.27; $I^2 = 29\%$) ($P = 0.01$).

CONCLUSIONS Central adiposity measures (WHR and WC) show stronger associations with CAC presence and progression than BMI, supporting their use as markers of cardiovascular risk while highlighting limitations of BMI alone. (Anthropometrics and subclinical coronary artery calcification: a systematic review and meta-analysis; CRD420251083057) (JACC Adv. 2026;5:102844) © 2026 Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

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ABBREVIATIONS AND ACRONYMS

ASCVD = atherosclerotic cardiovascular disease

BMI = body mass index

CAC = coronary artery calcium

CT = computed tomography

HDL = high-density lipoprotein cholesterol

I^2 = I-squared statistic

LDL = low-density lipoprotein cholesterol

NOS = Newcastle-Ottawa Scale

SBP = systolic blood pressure

SFA = subcutaneous fat area

SMD = standardized mean difference

VAT = visceral adipose tissue

VFA = visceral fat area

WC = waist circumference

WHR = waist-to-hip ratio

Obesity is a well-established risk factor for atherosclerotic cardiovascular disease (ASCVD), yet the utility of various anthropometric markers and their association with cardiovascular risk remains debated.[1,2] Body mass index (BMI), the most commonly used measure, fails to differentiate between fat and lean mass and does not account for fat distribution, limiting its precision in cardiovascular risk stratification.[3] Emerging evidence suggests that central adiposity, reflected by waist circumference (WC) and waist-to-hip ratio (WHR), is more strongly associated with cardiometabolic complications, including insulin resistance, dyslipidemia, and endothelial dysfunction, compared to generalized obesity.[4-6] Furthermore, WC and WHR have been shown to be associated with major adverse cardiovascular events.

Coronary artery calcium (CAC) scoring is a sensitive, noninvasive marker of subclinical coronary atherosclerosis that independently predicts major adverse cardiovascular events.[7,8] Understanding the relationship between anthropometric markers and CAC burden may provide insights into early atherogenesis and help primary screening strategies. Although individual studies have explored associations between body composition indices and CAC, results have been inconsistent, and a comprehensive synthesis of these associations is lacking.

Similarly, measures of central adiposity have been linked to cardiovascular outcomes and adverse cardiometabolic profiles,[9] but its relationship with CAC progression remains incompletely understood.[10] Understanding how these anthropometric indices relate to CAC progression may provide additional insights into cardiovascular risk and inform targeted prevention strategies.

This systematic review and meta-analysis aimed to quantify and compare the strength of association between various anthropometric measures of obesity: BMI, WC, and WHR with the presence of CAC and CAC progression. We hypothesized that markers of central adiposity (WC and WHR) would demonstrate stronger associations with subclinical CAC and CAC progression than BMI, supporting a shift toward using distribution-sensitive body composition measures in cardiovascular risk assessment.

METHODOLOGY

SEARCH STRATEGY AND SELECTION. This systematic review and meta-analysis is reported according to the Meta-analysis Of Observational Studies in Epidemiology and Preferred Reporting Items for Systematic reviews and Meta-Analyses guidelines and checklist.[11] Preferred Reporting Items for Systematic reviews and Meta-Analyses checklist is available in Supplemental Appendix. The review protocol was registered on the International Prospective Register of Systematic Reviews, registration number PROSPERO CRD420251083057. This study was conducted using aggregated data from previously published studies and did not involve access to identifiable individual participant data. Therefore, formal institutional ethics approval was not required in accordance with local guidelines.

We conducted a comprehensive literature search to identify studies examining the association between CAC and anthropometric measures. The search strategy was developed using a combination of Medical Subject Headings and relevant keywords grouped under 2 conceptual domains: 1) CAC and subclinical atherosclerosis; and 2) anthropometric markers of adiposity. Our full search strategy is available in Supplemental Appendix. These 2 domains were combined using the Boolean operator AND, and the search was applied to PubMed/MEDLINE, EMBASE, CINAHL, and Scopus from 2000 to 2025. Gray literature (inclusive of abstracts) was also searched. Only human studies published in English were considered. Reference lists of included studies were also manually screened to identify additional relevant publications. Please see inclusion and exclusion criteria given subsequently.

INCLUSION CRITERIA. 1) Population: Adult human participants (≥18 years) from observational cohorts or cross-sectional studies; 2) reported at least 1 anthropometric measure such as BMI, WC, or WHR; 3) reported stratification of anthropometric measure by CAC score comparing CAC = 0 and CAC >0; 4) reported stratification of anthropometric measure by CAC progression and CAC nonprogression; and 5) published in English.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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EXCLUSION CRITERIA. 1) Non-human or animal studies; 2) studies without quantitative data comparing anthropometric indices; 3) studies in which CAC was not measured using CT-based imaging techniques; 5) case reports and case series.

ENDPOINTS. The primary endpoint was strength of association between anthropometric measures and CAC presence in standardized mean differences (SMDs). We performed a sensitivity analysis of the primary endpoint to look at comparative strength of association (WHR vs WC vs BMI). The secondary endpoint was the strength of association between anthropometric measures and CAC progression (categorical variable) in SMD.

DEFINITIONS OF CAC PROGRESSION VARIED ACROSS INCLUDED STUDIES. Some studies defined progression as any increase in Agatston score, whereas others used absolute or percentage increases exceeding predefined thresholds to account for interscan variability. For the purposes of this analysis, CAC progression was accepted as defined by the original study authors.

Additional secondary analyses included differences in demographic and cardiometabolic variables such as age, sex, ethnicity, blood pressure (systolic blood pressure [SBP] and diastolic blood pressure), lipid profile (low-density lipoprotein cholesterol [LDL], high-density lipoprotein cholesterol [HDL], total cholesterol, and triglycerides), glycemic indices (hemoglobin A1c and diabetes mellitus), lipid lower therapy, hypertension status, smoking status, high-sensitivity C-reactive protein, and total body fat.

DATA EXTRACTION. Two investigators (S.K. and G.M.) independently screened records retrieved from the search by title and abstract. Details including year of publication, study design, study location, participant characteristics, patient demographics, outcome measures, results, and author's conclusion were collated and examined. Selected records were further screened for eligibility in full text by the same investigators (S.K. and G.M.). Data collection was performed independently by 2 investigators (S.K. and G.M.) using the same predetermined template. Discrepancies at any stage of selection were arbitrated by a third author (S.V.).

QUALITY APPRAISAL OF INCLUDED STUDIES. The quality and risk of bias of included studies were independently assessed by 2 reviewers (S.K. and G.M.) using the Newcastle-Ottawa Scale (NOS).[12] Both reviewers were trained in the application of this tool before commencing the assessment. The NOS evaluates studies across 3 domains: selection (maximum 4 points), comparability (maximum 2 points), and outcome (maximum 3 points), with a total possible score of 9. Discrepancies were resolved through discussion and consensus. Full NOS analysis in Supplemental Appendix.

STATISTICAL ANALYSIS. For each anthropometric measure of interest (i.e. BMI, WHR, and WC), a random-effects meta-analysis was performed when data from 3 or more studies were available. Studies were included if they reported quantitative estimates (means with SDs) or sufficient summary statistics (eg, medians with IQRs) to enable derivation of means and SDs, stratified by CAC score (CAC = 0 vs CAC >0) or CAC progression. Pooled SMDs with 95% CIs were calculated to compare anthropometric indices between groups. SMDs were used rather than absolute mean differences because included studies reported anthropometric variables using heterogeneous summary statistics (means with SDs or medians with IQRs) and demonstrated differences in variability and measurement protocols (WC at midpoint vs umbilicus vs lower waist) across study populations. For studies reporting medians and IQRs, means and SDs were estimated using established methods (Wan et al[13] and Luo et al[14]) to enable inclusion in pooled analyses. Where studies reported binary outcomes, ORs and 95% CIs were extracted directly when available. For comparability across studies, ORs per 1-SD increase were derived from SMDs using established transformation methods when not directly reported.[15]

As most studies reported raw anthropometric values stratified by CAC status without multivariable adjustment, pooled estimates represent unadjusted SMD. Adjusted effect estimates were not pooled because the covariate adjustment strategies varied substantially between studies, which would have introduced methodological heterogeneity and limited comparability of pooled estimates. Accordingly, residual confounding from demographic and metabolic covariates could not be excluded. Between-study heterogeneity was assessed using the I-squared statistic (I^2), with $I^2 \geq 50\%$ considered indicative of substantial heterogeneity. Publication bias was assessed through Egger tests and visual inspection of funnel plots.

A test for subgroup interaction was conducted to compare the relative strength of association between anthropometric indices (WHR vs BMI, WC vs BMI) and the presence of CAC. Where fewer than 3 studies were available or data were insufficient, findings were summarized qualitatively.

To explore sources of heterogeneity and effect modification, meta-regression analyses were

TABLE 1 Summary of Cross-Sectional and Longitudinal Studies Examining the Relationship Between Anthropometric or Fat Distribution Indices and Coronary Artery Calcium

First Author (Year)	Study Cohort	Study Design	Country	CT Scanner	Follow-Up Period (y)	Anthropometric Measures
CAC incidence (cross-sectional/ baseline studies)						
Snell-Bergeon (2004)	Asymptomatic patients	Cross-sectional	United States	EBCT	-	BMI, WC, WHR
Yu (2013)	Asymptomatic patients	Cross-sectional	South Korea	MDCT	-	BMI, WC, WHR
Horwich (2023)	Asymptomatic patients	Cross-sectional	United States	MDCT	-	WC
Lee (2016)	Nondialysis CKD patients	Cross-sectional	South Korea	MDCT	-	BMI, WC, WHR
Lee (2014)	Asymptomatic patients	Cross-sectional	South Korea	MDCT	-	BMI, WC
Kommuri (2015)	Asymptomatic patients	Cross-sectional	United States	EBCT/MDCT	-	BMI, WC, WHR
Ahmadi (2010)	Asymptomatic patients	Cross-sectional	United States	EBCT	-	BMI
Ashen (2021)	Asymptomatic firefighters	Cross-sectional	United States	ECG-gated noncontrast CT	-	BMI
Boakye (2023)	Obese asymptomatic	Cross-sectional	United States	EBCT/MDCT	-	BMI
Ding (2008)	Asymptomatic patients	Cross-sectional	United States	EBCT	-	BMI, WC
Eun (2020)	Postmenopausal asymptomatic women	Cross-sectional	South Korea	MDCT	-	BMI, WHR
Eun (2021)	Asymptomatic patients	Cross-sectional	South Korea	MDCT	-	BMI, WC, WHR
He (2012)	CKD patients	Cross-sectional	United States	EBCT/MDCT	-	BMI, WC, WHR
Hughes-Austin (2014)	Asymptomatic patients	Cross-sectional	United States	EBCT/MDCT	-	BMI, WC, WHR
Kirsch (2012)	Asymptomatic patients	Cross-sectional	United States	MDCT	-	BMI
Marques (2010)	Suspected CAD	Cross-sectional	Brazil	64-slice MDCT	-	BMI, WC, WHR
Mizia-Stec (2008)	Cardiac syndrome X	Cross-sectional	Poland	64-slice MDCT	-	BMI, WC, WHR
Oikawa (2015)	Suspected CAD	Cross-sectional	Japan	64-slice MDCT	-	BMI
Pokharel (2014)	Retired NFL athletes	Cross-sectional	United States	EBCT	-	BMI, WC
Sosnowski (2012)	Symptomatic patients	Cross-sectional	Poland	MDCT	-	BMI
Wong (2021)	Asymptomatic patients	Cross-sectional	Singapore	MDCT	-	BMI
Agatisa (2005)	Asymptomatic patients	Cross-sectional	United States	EBCT	-	BMI, WHR
Bundy (2018)	CKD patients	Prospective (baseline analysis)	United States	EBCT	-	BMI
Haider (2024)	Asymptomatic patients	Cross-sectional	United States	MDCT	-	BMI, WC
Higo (2024)	Asymptomatic patients	Cross-sectional	Japan	EBCT/MDCT	-	BMI, WC, WHR
Kang (2024)	MASLD patients	Cross-sectional	Korea	MDCT	-	BMI, WC
Liu (2012)	Asymptomatic patients	Cross-sectional	United States	MDCT	-	BMI
Nunes (2020)	Asymptomatic patients	Cross-sectional	Brazil	64-slice MDCT	-	BMI, WC
Onuma (2016)	Asymptomatic patients	Prospective (baseline CAC)	United States	MDCT	-	BMI
Opperman (2019)	Female asymptomatic	Cross-sectional	Brazil	128-slice MDCT	-	BMI, WC, WHR
Radford (2016)	Asymptomatic patients	Prospective (baseline CAC)	United States	EBCT	-	BMI
Yoo (2024)	Asymptomatic patients	Prospective (baseline CAC)	Korea	64-slice MDCT	-	BMI

Continued on the next page

performed using study-level covariates. The following moderator variables were assessed individually: mean age, sex distribution, prevalence of hypertension, diabetes mellitus, dyslipidemia, mean SBP, LDL, hemoglobin A1c, and smoking prevalence. Meta-regression was conducted using a random-effects model framework. All statistical analyses were conducted using Review Manager (RevMan;

TABLE 1 Continued

First Author (Year)	Study Cohort	Study Design	Country	CT Scanner	Follow-Up Period (y)	Anthropometric Measures
CAC progression (longitudinal studies)						
Anand (2007)	Asymptomatic T2DM patients	Prospective cohort	Canada	EBCT	2.5 ± 0.4	BMI, WHR
Antonio-Villa (2023)	Asymptomatic patients	Prospective cohort	Mexico	64-slice MDCT	4.8 (4.5-5.3)	BMI, WC
Cho (2016)	Asymptomatic patients	Prospective cohort	Korea	64-slice MDCT	4	BMI
Costacou (2007)	Asymptomatic T1DM patients	Prospective cohort	United States	EBCT	4	BMI, WHR
Gao (2025)	Asymptomatic patients	Prospective cohort	China	256-slice CT	6.3 ± 3.3	BMI, WC
Lee (2019)	Asymptomatic patients	Prospective cohort	Korea	64-slice MDCT	9	BMI, WC
Lee (2021)	Asymptomatic patients	Prospective cohort	Korea	64-slice MDCT	3.25	BMI, WC
Maahs (2005)	Asymptomatic T1DM patients	Prospective cohort	United States	EBCT	2.6 (1.6-3.3)	BMI, WC
Oh (2016)	Asymptomatic patients	Prospective cohort	Korea	64-slice MDCT	4	BMI, WC
Okwuosa (2012)	Asymptomatic patients	Prospective cohort	United States	EBCT	2.5	BMI
Park (2019)	Asymptomatic patients	Prospective cohort	Korea	64-slice MDCT	4.2	BMI
Shen (2020)	Asymptomatic patients	Prospective cohort	Taiwan	64-slice MDCT	5.71 ± 2.68	BMI
Snell-Bergeon (2003)	Asymptomatic T1DM patients	Prospective cohort	United States	EBCT	2.7 ± 0.3	BMI
Sung (2016)	Asymptomatic patients	Prospective cohort	Korea	64-slice MDCT	2.3	BMI
Varma (2022)	Asymptomatic patients	Prospective cohort	United States	EBCT/MDCT	2.6	BMI
Xie (2024)	Asymptomatic patients	Prospective cohort	United States	EBCT/MDCT	13.58 ± 2.25	BMI

Data include study cohort, design, country, CT modality, follow-up duration (for longitudinal studies), and anthropometric measures assessed. Cross-sectional studies evaluated CAC incidence at baseline, whereas prospective cohorts assessed CAC progression over follow-up.

BMI = body mass index; CAC = coronary artery calcium; CAD = coronary artery disease; CKD = chronic kidney disease; CT = computed tomography; EBCT = electron beam computed tomography; ECG = electrocardiogram; MASLD = metabolic dysfunction-associated steatotic liver disease; MDCT = multidetector computed tomography; NFL = National Football League; T1DM = type 1 diabetes mellitus; T2DM = type 2 diabetes mellitus; WC = waist circumference; WHR = waist-to-hip ratio.

version 5.4) and Stata/MP (version 18.5, StataCorp LLC). A 2-tailed P value <0.05 was considered statistically significant.

RESULTS

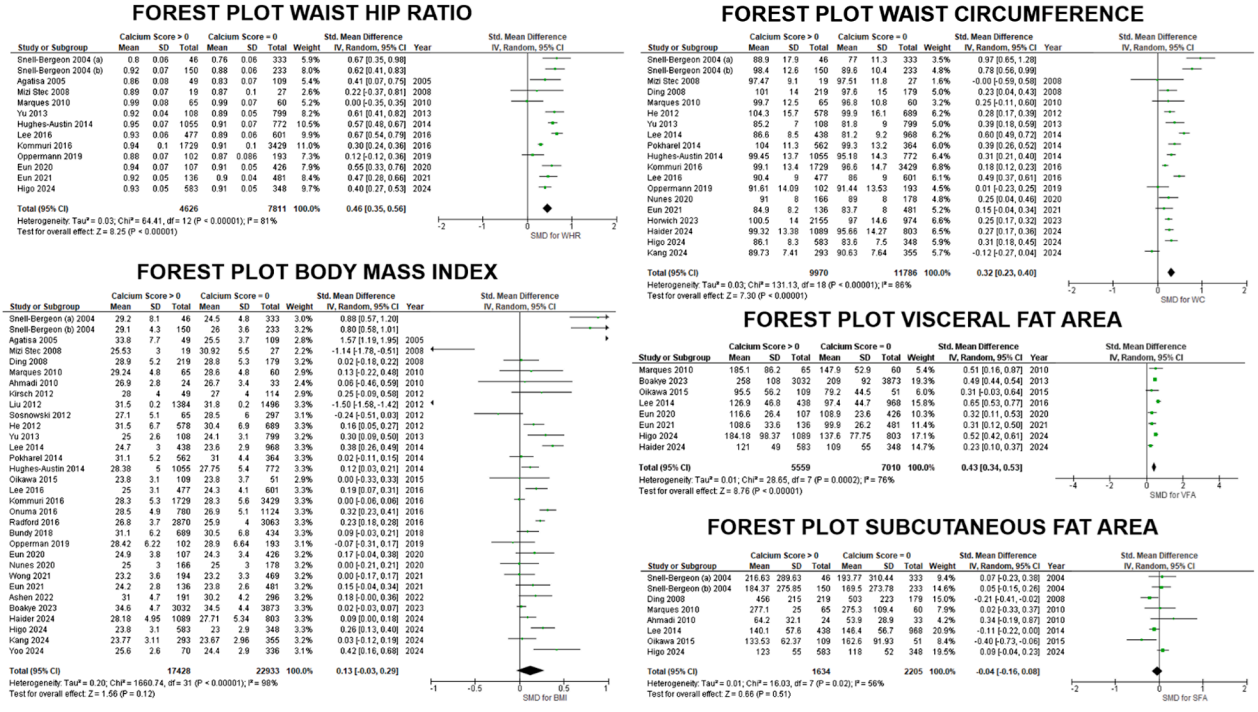
SEARCH RESULTS. The initial literature search identified 2,267 articles from multiple databases. Following a rigorous screening process, 49 studies met the predefined inclusion criteria and were incorporated into the meta-analysis. Of the 49 studies, 33 studies assessed CAC differentiation, and 16 studies assessed CAC progression. Our search identified studies from 10 countries: Brazil, Canada, China, Japan, Mexico, Poland, Singapore, South Korea, Taiwan, and the United States, allowing for pooled analysis across diverse international populations. Study summary available in **Table 1**.

These studies, published between January 2000 and January 2025, were systematically reviewed and quantitatively synthesized. Of the included studies assessing presence of CAC, all studies were cross-sectional observational studies.[16-48] Similarly, all studies assessing CAC progression were prospective cohort studies.[47,49-63] Follow-up periods ranged from 2.3 to 13.6 years, with a pooled mean of 4.86 ± 3.04 years.

CORONARY ARTERY CALCIUM MEASUREMENT. CAC was consistently measured using noninvasive cardiac imaging techniques, primarily electron-beam computed tomography (CT)or multidetector CT, to quantify the burden of calcified atherosclerotic plaque in the coronary arteries. The Agatston score was the most employed metric, calculated from non-contrast CT images gated to the cardiac cycle. Scanning protocols varied slightly between studies but typically included acquisition during mid-diastole for optimal image quality. All studies analyzed CAC as a categorical variable to assess associations with adiposity, metabolic markers, or cardiovascular risk. CAC progression was defined according to study-specific definitions, generally reflecting increases in Agatston score at follow-up.

MEASUREMENT OF SUBCUTANEOUS AND VISCERAL FAT AREA. Across the majority of studies, subcutaneous fat area (SFA) was quantified using CT at the level of the L4-L5 intervertebral disc. At this anatomical landmark, adipose tissue was identified within an attenuation range of −190 to −30

FIGURE 1 Meta-Analysis of Anthropometric Indices and Fat Depots Comparing Individuals With and Without Coronary Artery Calcium



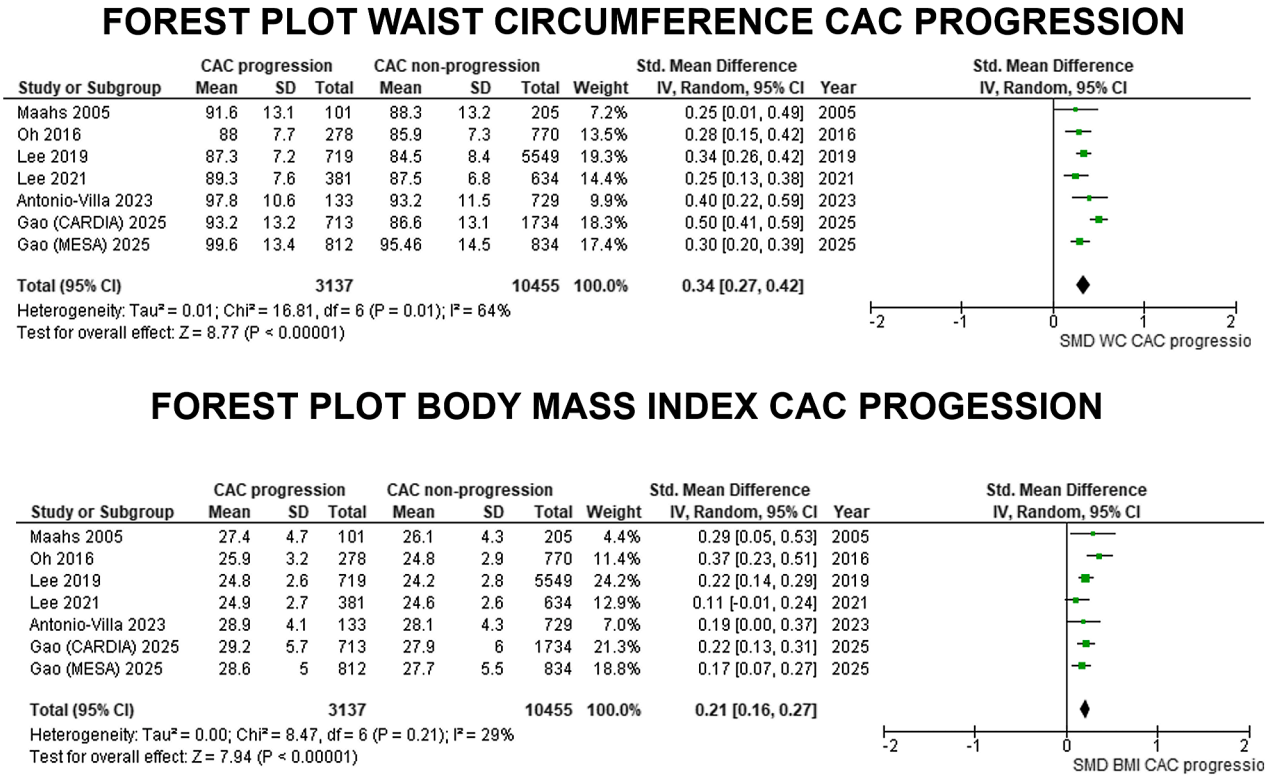
Adiposity indices and coronary artery calcium (CAC) presence. Forest plots show pooled standardized mean differences (SMDs) for individuals with vs without CAC across adiposity measures. Waist-to-hip ratio (SMD 0.46; 95% CI: 0.35-0.56), waist circumference (SMD 0.32; 95% CI: 0.23-0.40), and visceral fat area (SMD 0.43; 95% CI: 0.34-0.53) were consistently higher in those with CAC. Body mass index showed only a weak, nonsignificant association (SMD 0.13; 95% CI: −0.03 to 0.29), while subcutaneous fat area was unrelated to CAC (SMD −0.04; 95% CI: −0.16 to 0.08). WHR = waist-to-hip ratio.

Hounsfield units (HU), corresponding to the radiodensity of adipose tissue. The subcutaneous compartment was defined as adipose tissue located external to the abdominal wall musculature and internal to the dermis, separated from visceral fat by the abdominal wall fascia. Planimetric tracing was performed on axial images, and the total SFA (cm^2) was calculated using a semiautomated segmentation technique.

Similarly, across the majority of studies, visceral fat area (VFA) using CT at a single cross-sectional slice at the umbilical level or the L4-L5 vertebral level, a reliable surrogate for total abdominal visceral fat. Adipose tissue was identified using HU thresholds between −190 and −30 HU, and visceral fat was defined as the fat enclosed by the inner aspect of the abdominal wall or peritoneal membrane, excluding subcutaneous tissue. The area (in cm^2) was then automatically or manually calculated using specialized image-analysis software.

META-ANALYSIS SUMMARY. Across multiple anthropometric indices, individuals with CAC demonstrated higher central adiposity compared with those without CAC. WHR was significantly higher in the CAC >0 group (pooled SMD = 0.46; 95% CI: 0.35-0.56; $P < 0.00001$; $I^2 = 81\%$), indicating a moderate association between WHR and CAC presence. WC was also significantly greater in the CAC >0 group (pooled SMD = 0.32; 95% CI: 0.23-0.40; $P < 0.00001$; $I^2 = 86\%$), although the effect size was smaller than for WHR. In contrast, BMI showed only a small, nonsignificant pooled difference between groups (SMD = 0.13; 95% CI: -0.03 to 0.29; $P = 0.12$; $I^2 = 98\%$). VFA was consistently higher in those with CAC (pooled SMD = 0.43; 95% CI: 0.34-0.53; $P < 0.00001$; $I^2 = 76\%$). In contrast, SFA did not differ significantly between groups (SMD = −0.04; 95% CI: -0.16-0.08; $P = 0.51$; $I^2 = 56\%$). Similarly, body weight (kg) did not differ significantly between individuals with CAC >0 and those with CAC = 0 (SMD = 0.12; 95% CI: 0.00-0.20; $P = 0.054$; $I^2 = 59\%$). Figures 1 and 2 illustrate meta-analysis forest plots of CAC incidence and CAC progression. When stratified by measurement site, WC assessed at the umbilicus demonstrated a stronger association with the

FIGURE 2 Meta-Analysis of Anthropometric Measures Comparing Individuals With vs Without Coronary Artery Calcium Progression

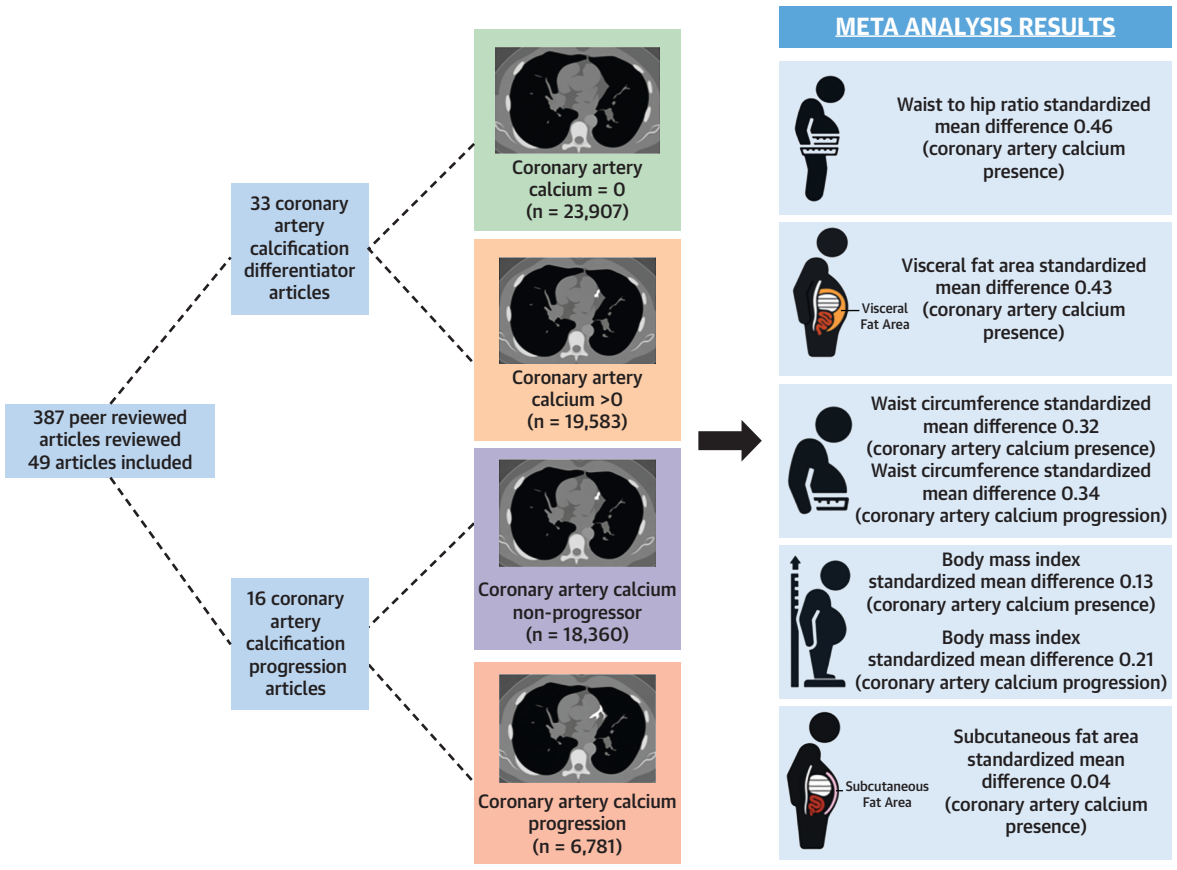


Waist circumference and BMI in relation to CAC progression. Pooled analyses show that both waist circumference (SMD 0.34; 95% CI: 0.27-0.42) and BMI (SMD 0.21; 95% CI: 0.16-0.27) are significantly higher in individuals with CAC progression compared with those without. The association was stronger for waist circumference, with moderate heterogeneity (I^2 = 64%), whereas BMI showed weaker but consistent effects with low heterogeneity (I^2 = 29%). CAC = coronary artery calcium; SMD = standardized mean difference; WC = waist circumference.

presence of CAC (pooled SMD = 0.36; 95% CI: 0.23-0.48; I^2 = 79%) compared with the midpoint method (pooled SMD = 0.31; 95% CI: 0.20-0.41; I^2 = 67%; P = 0.03 for interaction). Both methods, however, had an increased association with CAC compared to BMI alone. Additional qualitative analyses of alternative fat depots and indices, including thoracic adipose tissue, epicardial adipose tissue, intramuscular fat, conicity index, and visceral-to-subcutaneous fat ratio, are summarized in **Table 2**.

CAC PROGRESSION. A total of 16 studies that assessed CAC progression were included. In random-effects meta-analysis of studies reporting BMI, patients with CAC progression had significantly higher BMI than those without progression (pooled SMD = 0.22; 95% CI: 0.18-0.26; P < 0.00001; I^2 = 38%). This corresponded to an estimated OR of 1.50 (95% CI: 1.40-1.62) per 1-SD increase in BMI. For WC, CAC progression was likewise associated with higher values (pooled SMD = 0.34; 95% CI: 0.26-0.42; P < 0.00001; I^2 = 65%), equivalent to an estimated OR of 1.85 (95% CI: 1.61-2.13) per 1-SD increase. These ORs were derived from pooled SMDs using established transformation methods and were not directly extracted from individual studies. Pooled analysis of 7 head-to-head studies (n = 13,592) demonstrated a significant positive association between WC and CAC progression (SMD = 0.34; 95% CI: 0.27-0.42; P < 0.001), with moderate heterogeneity (I^2 = 64%). Pooled analysis of the same 7 studies also showed a significant association with BMI (SMD = 0.21; 95% CI 0.16-0.27; P < 0.001), with low heterogeneity (I^2 = 29%). Although WHR was not able to be meta-analyzed for CAC progression, the 2 reporting studies both demonstrated significant differences in the CAC progression group (WHR 0.95 ± 0.08 vs 0.93 ± 0.07 and 1.03 ± 1.2 vs 1.00 ± 1.2). VFA was significantly associated of CAC progression (180; 108, 251) cm^2 vs (128; 81, 189) cm^2, P < 0.001.[47] Similarly, visceral adipose tissue (VAT)-subcutaneous adipose tissue ratio was also significantly associated with

CENTRAL ILLUSTRATION **Central Obesity and Association With Subclinical Coronary Artery Calcium and Progression of Coronary Artery Calcium**



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Of the 387 studies screened, 49 met the inclusion criteria (33 for CAC presence, 16 for progression). Pooled analyses showed stronger associations for waist-to-hip ratio (WHR) (SMD 0.46) and waist circumference (WC) (SMD 0.32) than for body mass index (BMI) (SMD 0.13). Imaging substudies by computed tomography also identified higher visceral fat area (VFA) (SMD 0.43) vs subcutaneous fat area (SFA) (SMD 0.04). For CAC progression, WC (SMD 0.34) exceeded BMI (SMD 0.21), highlighting central adiposity as a stronger correlate of subclinical atherosclerotic burden. CAC = coronary artery calcium.

CAC progression (1.10 ± 0.46 vs 1.03 ± 0.45; $P < 0.001$).[55] Please see **Central Illustration**.

CORRELATION OF ANTHROPOMETRICS WITH FAT DEPOT. Across the 4 included studies, WC showed the strongest and most consistent association with VFA, with correlation coefficients ranging from 0.68 (Ding 2008)[25] to 0.84 (Lee 2014[20]). WC was also strongly associated with SFA in most studies (0.61-0.83), although the strength of association was generally lower than that for VFA. BMI was moderately to strongly correlated with both VFA and SFA, with slightly higher values for SFA in most data sets. For example, in Snell-Bergeon 2004,[16] BMI-SFA correlation was 0.88 compared to 0.70 for BMI-VFA; similar patterns were seen in Ding 2008 (0.73 vs 0.54). WHR correlations with VFA were modest (0.43-0.47) and generally weaker for SFA, with one study (Marques 2010) reporting a negative association (−0.13).

META REGRESSION. Meta-regression was performed to explore potential sources of heterogeneity in the associations between adiposity measures (BMI, WC, and WHR) and CAC. Hypertension prevalence was inversely related to effect size for all three-adiposity metrics: BMI (−0.013; 95% CI: -0.023 to −0.004; $P = 0.01$), WC (−0.011; 95% CI: -0.019 to −0.004; $P = 0.01$), and WHR (−0.008; 95% CI: −0.015 to −0.000; $P = 0.05$). Increasing age was significantly associated with smaller effect sizes for BMI (coefficient = −0.023; 95% CI: −0.045 to −0.001;

TABLE 2 Qualitative Analysis of Fat Depots in Relation to Coronary Artery Calcium

Qualitative Analysis of Fat Depots	Fat Depot/Index	Comparison (CAC + vs CAC-)	Key Findings
Ahmadi (2010)	Thoracic adipose tissue	146.1 ± 59.1 cm^3 vs 125.6 ± 68.6 cm^3	Higher in CAC+ ($P = 0.001$)
	Epicardial adipose tissue	79.3 ± 38.5 cm^3 vs 68.3 ± 35.6 cm^3	Higher in CAC+ ($P = 0.001$)
	Framingham Risk Score	9.5 ± 4.1% vs 6.8 ± 3.9%	Higher in CAC+ ($P = 0.001$); addition of SAT, PAT, EAT improved AUC (0.88 ± 0.02 vs 0.63 ± 0.04, $P = 0.001$)
Ding (2018)	Intramuscular fat	22.0 ± 13.0 cm^2 vs 19.1 ± 13.0 cm^2	Higher in CAC+ ($P = 0.03$)
	Nonsubcutaneous fat index	0.58 ± 3.0 vs −0.76 ± 2.6	Higher in CAC+ ($P < 0.0001$)
Eun (2021)	Fat-to-muscle ratio	0.76 ± 0.27 vs 0.69 ± 0.28	Higher in CAC+ ($P = 0.02$)
Kommuri (2015)	Conicity index	1.32 ± 0.10 vs 1.30 ± 0.10	Higher in CAC+ ($P < 0.001$)
Lee (2014)	Visceral-to-subcutaneous fat ratio	1.0 ± 0.4 vs 0.7 ± 0.4	Higher in CAC+ ($P < 0.001$)

Comparisons of adipose tissue depots and composite indices between individuals with CAC (CAC+) and without CAC (CAC-) across selected studies. multiple fat compartments—including thoracic, epicardial, intramuscular, and visceral adipose tissue—were consistently higher in CAC + individuals. composite indices (eg, non-subcutaneous fat index, fat-to-muscle ratio, conicity index, and visceral-to-subcutaneous fat ratio) also discriminated CAC presence, with several studies reporting significant improvements in risk prediction when fat depots were added to traditional scores.

AUC = area under the curve; CAC+ = presence of coronary artery calcium; CAC- = absence of coronary artery calcium; EAT = epicardial adipose tissue; PAT = pericardial adipose tissue; SAT = subcutaneous adipose tissue.

$P = 0.04$) and WC (−0.021; 95% CI: −0.034 to −0.007; $P = 0.01$), but not WHR. Higher SBP was associated with smaller BMI effect sizes (−0.034; 95% CI: −0.053 to −0.016; $P < 0.001$), but not WC or WHR. No statistically significant associations were found for sex distribution, diabetes, smoking prevalence, diastolic blood pressure, LDL, HDL, or triglycerides with any adiposity measure after adjustment in meta-regression Table 3.

PUBLICATION BIAS. Assessment of small-study effects using Egger regression test did not reveal significant publication bias for most pooled analyses, including BMI (SMD = 0.13; $P = 0.42$), WC (SMD = 0.32; $P = 0.18$), WHR (SMD = 0.46; $P = 0.09$), SFA (SMD = 0.04; $P = 0.66$), and VFA (SMD = 0.43; $P = 0.14$). Similarly, analyses restricted to participants with CAC progression vs nonprogression showed no evidence of small-study effects for BMI (SMD = 0.21; $P = 0.27$) or WC (SMD = 0.34; $P = 0.19$). In addition, funnel plot assessment showed that studies were symmetrically distributed around the mean effect, with only 1 or 2 studies as outliers with a balanced spread. These findings suggest that the pooled estimates are unlikely to be substantially influenced by publication bias.

SEX-SPECIFIC DIFFERENCES. Due to the limited number of studies reporting sex-stratified anthropometric data, formal meta-analysis stratified by sex was not feasible. Therefore, sex-specific comparisons are presented descriptively based on available study-level data. In the study by Snell-Bergeon (2004), among women, those with CAC were older and demonstrated substantially greater adiposity compared with women without CAC. Women with CAC had higher BMI (29.2 ± 8.1 vs 24.5 ± 4.8 kg/m^2), greater VAT (48.5 vs 27.1 cm^2), higher SFA (188.3 vs 129.6 cm^2), and larger WC (88.9 vs 77.0 cm). WHR was also higher in women with CAC (0.80 vs 0.76). In contrast, men exhibited higher absolute adiposity regardless of CAC status. Men with CAC had higher BMI (29.1 vs 26.0 kg/m^2), VFA (71.7 vs 47.6 cm^2), subcutaneous fat (158.5 vs 113.3 cm^2), and WC (98.4 vs 89.6 cm) compared with men without CAC. However, the relative differences between CAC-positive and CAC-negative men were smaller than those observed in women. In analyses of CAC progression in the study by Costacou (2007),[52] women who progressed exhibited higher adiposity compared with women without progression, including higher BMI (25.5 vs 24.6 kg/m^2) and WHR (0.80 vs 0.79). Although absolute differences were modest, progression occurred at lower absolute body size compared with men. Among men, CAC progression was associated with higher BMI (26.4 vs 24.7 kg/m^2) and slightly higher WHR (1.10 vs 1.10, minimal difference), with consistently higher absolute anthropometric values across both progression groups.

DISCUSSION

In this first and largest systematic review and meta-analysis to date, we evaluated the association between key anthropometric markers (BMI, WC, and WHR) and the presence of CAC, a well-established marker of subclinical atherosclerosis. Across 49 studies encompassing over 68,000 participants, we found that measures of central adiposity (WC and WHR) demonstrated a greater association with CAC compared to BMI. Our analysis also demonstrated a stepwise gradient in association strength: WHR showed the highest SMD between those with and

TABLE 3 Meta-Regression of Clinical Moderators on the Association Between Adiposity Indices and CAC

Moderator Variable	BMI Coefficient (95% CI)	BMI *P* Value	WC Coefficient (95% CI)	WC *P* Value	WHR Coefficient (95% CI)	WHR *P* Value
Age	−0.023 (−0.045 to −0.001)	**0.04**	−0.021 (−0.034 to −0.007)	**0.01**	−0.009 (−0.023 to 0.005)	0.18
Male	0.013 (−0.002 to 0.027)	0.08	−0.006 (−0.022 to 0.009)	0.36	-	-
DM	−0.004 (−0.024 to 0.015)	0.66	−0.001 (−0.016 to 0.014)	0.84	0.012 (−0.020 to 0.044)	0.25
HTN	−0.013 (−0.023 to −0.004)	**0.01**	−0.011 (−0.019 to −0.004)	**0.01**	−0.008 (−0.015 to −0.000)	**0.05**
Smoker	0.001 (−0.010 to 0.011)	0.92	−0.006 (−0.014 to 0.002)	0.11	−0.002 (−0.010 to 0.006)	0.56
SBP	−0.034 (−0.053 to −0.016)	**0.00**	−0.011 (−0.035 to 0.012)	0.29	−0.020 (−0.060 to 0.020)	0.23
DBP	−0.000 (−0.025 to 0.025)	1.00	0.009 (−0.007 to 0.026)	0.18	-	-
LDL	−0.002 (−0.021 to 0.017)	0.81	−0.015 (−0.034 to 0.005)	0.12	−0.006 (−0.026 to 0.014)	0.46
HDL	0.000 (−0.046 to 0.046)	0.99	0.003 (−0.061 to 0.067)	0.91	−0.005 (−0.055 to 0.046)	0.83
Triglycerides	0.003 (−0.006 to 0.011)	0.48	−0.003 (−0.020 to 0.013)	0.62	0.002 (−0.010 to 0.013)	0.54

Meta-regression coefficients (95% CI) and *P* values are shown for BMI, waist circumference (WC), and waist-to-hip ratio (WHR) across potential moderator variables. Negative coefficients indicate attenuation of the adiposity–CAC association with increasing levels of the moderator. **Bold** values indicate statistical significance ($P < 0.05$).

DBP = diastolic blood pressure; DM = diabetes mellitus; HDL = high-density lipoprotein cholesterol; HTN = hypertension; LDL = low-density lipoprotein cholesterol; SBP = systolic blood pressure.

without CAC, followed by WC, with BMI showing a nonsignificant association. Although heterogeneity was anticipated to be high, this reflects the diverse study populations, clinical characteristics and anthropometric measurement (ie umbilicus vs iliac crest); importantly, the direction of associations remained consistent, supporting the robustness of our findings. These results should, however, be interpreted in the context of unadjusted pooling; therefore, the observed differences reflect associations at the population level rather than fully adjusted, causal effects.

Although our findings demonstrate directionally consistent associations across studies, the substantial between-study heterogeneity warrants careful interpretation. Heterogeneity likely stems from variation in participant demographics, ethnicity, cardiometabolic burden, and measurement techniques. Thus, the pooled results indicate broadly consistent directional associations, although the exact magnitude of effect should be interpreted with caution. The uniformity of direction across studies supports consistency of associations; however, substantial heterogeneity limits the precision of pooled effect estimates.

Our meta-regression results suggest a potential role of baseline cardiovascular factors in influencing between-group differences in adiposity measures. Specifically, higher prevalence of hypertension was consistently associated with attenuated differences across BMI, WC, and WHR, suggesting that individuals with more advanced cardiometabolic comorbidity may demonstrate less pronounced adiposity. This is likely due to shared pathophysiological pathways for vascular disease and visceral obesity. Furthermore, older age was also a significant moderator for differences in WC, highlighting age-related fat distributive changes, particularly plateau in central adiposity. Collectively, these findings highlight the need to account for cardiovascular risk factor burden and age when interpreting adiposity metrics in clinical practice. Correlation data further supported this pattern: WC and WHR were more strongly linked to VFA, a metabolically active fat depot with established proatherogenic effects, than BMI, which correlated more with subcutaneous fat. This is in keeping with other published studies which demonstrate a strong relationship with VFA and WHR and WC, even in healthy adults.[64]

Although sex-stratified meta-analysis was not feasible, descriptive analyses revealed clear sex-specific patterns. Women with CAC exhibited greater adiposity than women without CAC, despite having lower absolute fat mass than men, suggesting a lower threshold at which adiposity translates into calcific disease. In contrast, men demonstrated higher absolute adiposity irrespective of CAC status, with smaller relative differences between affected and unaffected individuals. Similar patterns were observed for CAC progression, with women showing progression at lower levels of adiposity. Collectively, these findings may suggest that adiposity could confer disproportionate coronary risk in women; however, these observations are based on limited descriptive data and should be interpreted cautiously.

Among nonanthropometric correlates, expected associations were observed for male sex, hypertension, diabetes, and smoking, whereas HDL was inversely associated with CAC. Notably, lipid-lowering therapy was associated with approximately 2-fold higher odds of CAC presence (OR: 2.37), which likely reflects confounding. In more detail, this can be explained by individuals prescribed statins or

other lipid-lowering drugs generally would have had higher baseline cardiovascular risk or established subclinical disease prompting therapy. Furthermore, high-sensitivity-C-reactive protein showed a positive trend toward significance (SMD = 0.083; 95% CI: −0.006 to 0.172; $P = 0.068$), suggesting that higher systemic inflammatory burden may accompany the presence of CAC. The consistent direction across studies supports the biologic plausibility that low-grade inflammation contributes to early atherogenesis.[65]

CAC PROGRESSION. In the sub-analysis of 16 studies, both BMI and WC were significantly associated with CAC progression, with a stronger association for WC. These findings suggest that central adiposity is associated with the progression of coronary atherosclerosis, although the effect magnitude appears moderate and likely multifactorial. WC reflects VAT burden more directly than BMI, which is a composite measure of lean and fat mass, and may therefore better capture the metabolic and inflammatory effects that may be involved in plaque progression.[66] Visceral fat is metabolically active, releasing proinflammatory cytokines, free fatty acids, and adipokines that promote endothelial dysfunction, oxidative stress, and vascular calcification.[67] Clinically, these results suggest that anthropometric measures that capture central fat distribution may improve identification of individuals at higher risk of CAC progression beyond BMI alone and may also highlight a modifiable target for intervention. This has implications for primary prevention strategies, where lifestyle and pharmacological approaches aimed at reducing visceral adiposity could attenuate calcification progression.

Overall, our findings support the growing recognition that conventional reliance on BMI alone underestimates cardiometabolic risk related to body fat distribution. These findings are consistent with prior literature indicating that visceral fat, rather than total body weight, plays a more direct role in the pathogenesis of atherosclerosis through mechanisms such as low-grade inflammation, insulin resistance, and endothelial dysfunction.[68-72] It is well known that central fat accumulation, particularly visceral adiposity, is more metabolically active and inflammator.[73] These observations have important clinical implications. Despite their greater predictive value, WC and WHR remain underutilized in clinical risk stratification frameworks, including most guidelines.[1] Our findings suggest that incorporating these measures may provide additional information relevant to early cardiovascular risk assessment, particularly in populations with normal BMI but high central adiposity. Across included studies, WC correlated more strongly with VFA than with SFA underscoring its utility in capturing metabolically active fat depots that drive atherosclerosis. In contrast, BMI showed a tendency to correlate more closely with SFA than VFA, reflecting its insensitivity to fat distribution. This limitation means that individuals with a normal BMI but disproportionately high visceral fat may be misclassified as low risk, despite having a high WC and corresponding elevated cardiometabolic risk. Given that VFA, rather than SFA, is more closely linked to proinflammatory cytokine release and vascular calcification, WC provides a more relevant measure of atherogenic adiposity than BMI alone.

Several prior studies have compared the relative associations of BMI, WC, and WHR with atherosclerotic burden. Large cohort analyses, such as those from the Multi-Ethnic Study of Atherosclerosis (MESA) and the Framingham Heart Study, have consistently demonstrated that central adiposity measures outperform BMI in their association with subclinical and clinical cardiovascular outcomes. For example, MESA reported that WC and WHR were independently associated with CAC after adjusting for BMI, highlighting the incremental prognostic value of fat.[18,29] Similarly, the Dallas Heart Study and EPIC-Norfolk cohorts found that visceral adiposity quantified by imaging correlated more strongly with CAC and incident cardiovascular events than BMI.[74,75] A recent meta-analysis of over 250,000 participants also confirmed that WHR and WC were more robust predictors of cardiovascular mortality and coronary disease than BMI,[76] and finding further supported by a later meta-analysis of 15,000 participants.[77] Collectively, these findings underscore the limitations of BMI as a sole measure of adiposity and support the clinical relevance of incorporating central adiposity indices into cardiovascular risk assessment.

Our qualitative synthesis also highlights that beyond traditional anthropometric indices, specific fat depots and composite adiposity measures demonstrate stronger associations with subclinical atherosclerosis. Measures such as thoracic adipose tissue, epicardial adipose tissue, and the visceral-to-subcutaneous fat ratio strongly reflect pathogenic ectopic fat accumulation, which is metabolically active and proinflammatory, contributing to coronary CAC. The observed improvement in predictive performance when subcutaneous adipose tissue, pericardial adipose tissue and epicardial adipose tissue were incorporated into Framingham Risk Score

underscores the value of integrating imaging-derived adiposity metrics into established cardiovascular risk models. Similarly, parameters such as intramuscular fat, nonsubcutaneous fat index, and fat-to-muscle ratio capture broader patterns of adverse body composition, including sarcopenic obesity, that are not fully captured by BMI or WC alone. The strong associations observed for the conicity index and visceral-to-subcutaneous fat ratio reinforce the notion that central adiposity distribution, rather than total fat mass, is more closely linked to CAC burden. These findings collectively suggest that phenotyping of fat distribution may enhance risk stratification and could inform targeted prevention strategies.

CLINICAL IMPLICATIONS AND FUTURE DIRECTIONS

Our findings provide supportive evidence that central adiposity measures, particularly WC and WHR, are associated with ASCVD risk and may complement existing risk assessment approaches, although recognizing the incremental improvement beyond BMI is modest. Current widely used algorithms, such as the pooled cohort equations and the Framingham Risk Score, rely heavily on age, sex, lipid levels, blood pressure, diabetes, and smoking status, but do not account for body fat distribution.[78] Similarly, current global definitions of obesity, including those proposed by the World Health Organization, rely primarily on BMI thresholds. However, contemporary consensus statements increasingly recognize that BMI alone inadequately reflects adiposity and related health risk. A recent international commission published in *The Lancet Diabetes & Endocrinology* proposed redefining obesity based on excess adiposity and its functional consequences.[79] The commission recommends that BMI be used primarily as a population-level screening tool, with anthropometric measures such as WC or WHR incorporated to better characterize cardiometabolic risk. These findings contribute to a growing body of literature suggesting that reliance on BMI alone may be insufficient for cardiometabolic risk assessment, and that incorporation of central adiposity measures alongside BMI may provide a more comprehensive characterization of obesity-related cardiovascular risk. Although BMI provides a simple and widely applicable measure of body mass, it does not capture body fat distribution or differentiate between lean mass and adiposity. Given that WC and WHR demonstrated stronger associations with both prevalent CAC and CAC progression than BMI, incorporating these simple, inexpensive metrics may provide additional information regarding cardiovascular risk, particularly among individuals with normal BMI but high central adiposity who may be underclassified by current tools.

For WC, widely used thresholds are ≥102 cm (men) and ≥88 cm (women), with lower ethnic-specific cutoffs endorsed by the International Diabetes Federation (South/East Asian men ≥90 cm, women ≥80 cm); WHR thresholds commonly used are >0.90 (men) and >0.85 (women).[80] Clinical measurement should be standardized (nonstretch tape, at end-expiration; WHO protocol at the midpoint between the lower rib and iliac crest) given small landmark differences can shift classification.[81] Importantly, however, the clinical utility of WC and WHR may be greatest among younger or intermediate-risk individuals, in whom adiposity-related vascular risk is less likely to be captured by traditional markers such as diabetes or hypertension. Despite its simplicity and low cost, WC remains underutilized in routine clinical practice, although both American College of Cardiology/American Heart Association and European Society of Cardiology prevention guidelines recommend measuring WC alongside BMI to better stratify obesity-related cardiometabolic risk.[82]

Risk stratification models such as the body rounded index and a body shape index have been assessed for their prediction of cardiovascular disease beyond BMI. Body rounded index, validated against imaging in >11,000 adults, has been shown to correlate strongly with visceral adiposity outperforming BMI in predicting cardiometabolic risk.[83] Similarly, body shape index, tested in European cohorts, independently predicted all-cause and cardiovascular mortality beyond BMI and WC.[84] Incorporating central adiposity metrics, which are simple, low-cost measures of central adiposity, is therefore crucial as they capture visceral fat distribution more directly than BMI and enhance cardiovascular risk association in routine clinical practice.

From a public health perspective, this evidence may inform targeted screening strategies and reinforce the importance of simple, low-cost anthropometric assessments in primary prevention settings. Prior large-scale cohorts, including MESA and the Dallas Heart Study, have shown that adding WC or WHR to traditional risk factors has been shown to improve model calibration and reclassification indices for ASCVD events in prior studies.[85,86] Interventions aimed at reducing central adiposity, through dietary modification, physical activity, and pharmacologic approaches, could be prioritized in individuals identified as high-risk based on WC and WHR rather than BMI alone.[87] Given the high prevalence of obesity and

metabolic syndrome globally, incorporating these indices may improve early identification of individuals with subclinical atherosclerosis, ultimately enabling earlier and more targeted preventive strategies. Finally, our findings should be strictly interpreted within the context of subclinical disease; our analyses do not establish incremental predictive value for hard cardiovascular outcomes.

STUDY LIMITATIONS. Although this study has several strengths, including the inclusion of a large and diverse population, rigorous methodology, and formal interaction testing across anthropometric indices, there are some limitations that must be acknowledged. Firstly, a key limitation is that the pooled analyses were based only on unadjusted SMDs, as most studies did not provide effect estimates corrected for clinical covariates. Therefore, the observed associations may partly reflect confounding by demographics and clinical factors. Secondly, substantial heterogeneity was observed across studies, likely reflecting differences in population characteristics, CAC measurement protocols, and anthropometric cutoffs. These findings likely reflect differences in participant demographics, risk factor profiles, and imaging protocols. Although univariate meta-regression identified several study-level covariates, such as diabetes prevalence, SBP, age, and male composition, as contributors to between-study variability, these analyses were exploratory and based on a limited number of studies per model. Thirdly, definitions of CAC progression were also not uniform across the included studies, with some defining progression as any increase in Agatston score and others using absolute or percentage thresholds. This lack of standardization in the definition and quantification of CAC progression has been previously highlighted in the literature.[88] Fourth, the inability to perform sex-stratified meta-analyses was also a limitation. Although sexual dimorphism is well established in the distribution and metabolic consequences of central vs overall adiposity, the majority of included studies did not report sufficient sex-specific data to permit pooled analyses by sex. As such, we could not fully assess whether the associations between WC, WHR, and CAC differ between men and women. Finally, variations in measurement techniques for WC and WHR across studies may introduce measurement bias.

CONCLUSIONS

This meta-analysis demonstrates that central adiposity markers, particularly WHR and WC, have a stronger association with subclinical coronary atherosclerosis than BMI. These findings underscore the need to shift clinical and research emphasis toward distribution-sensitive measures of adiposity to better reflect associations with cardiovascular risk. Future prospective studies are warranted to evaluate whether incorporating WC and WHR into clinical practice improves risk prediction in coronary artery disease.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Bhatt served in the advisory board of Angiowave, Antlia Bioscience, Bayer, Boehringer Ingelheim, CellProthera, Cereno Scientific, E-Star Biotech, High Enroll, Janssen, Level Ex, McKinsey, Medscape Cardiology, Merck, NirvaMed, Novo Nordisk, Repair Biotechnologies, Stasys, and Tourmaline Bio; was a member of board of directors of American Heart Association New York City, Angiowave (stock options), Bristol Myers Squibb (stock), DRS.LINQ (stock options), and High Enroll (stock); served as a consultant for Alnylam, Altimmune, Broadview Ventures, Corcept Therapeutics, Corsera, GlaxoSmithKline, Hims, Sanofi, SERB, SFJ, Summa Therapeutics, and Worldwide Clinical Trials; served in the data monitoring committees of Acesion Pharma, Assistance Publique-Hôpitaux de Paris, Baim Institute for Clinical Research, Boston Scientific (Chair, PEITHO trial), Cleveland Clinic, Contego Medical (Chair, PERFORMANCE 2), Duke Clinical Research Institute, Mayo Clinic, Mount Sinai School of Medicine (for the ABILITY-DM trial, funded by Concept Medical; for ALLAY-HF, funded by Alleviant Medical), Novartis, Population Health Research Institute, and Rutgers University (for the NIH-funded MINT Trial); received honoraria from American College of Cardiology (Senior Associate Editor, Clinical Trials and News, ACC.org; Chair, ACC Accreditation Oversight Committee), Arnold and Porter law firm (work related to Sanofi/Bristol-Myers Squibb clopidogrel litigation), Baim Institute for Clinical Research (AEGIS-II executive committee funded by CSL Behring), Belvoir Publications (Editor in Chief, Harvard Heart Letter), Canadian Medical and Surgical Knowledge Translation Research Group (clinical trial steering committees), CSL Behring (AHA lecture), Duke Clinical Research Institute, Engage Health Media, HMP Global (Editor in Chief, Journal of Invasive Cardiology), Medtelligence/ReachMD (CME steering committees), MJH Life Sciences, Oakstone CME (Course Director, Comprehensive Review of Interventional Cardiology), Philips (Becker's Webinar on AI), Population Health Research Institute, WebMD (CME steering committees), and Wiley (steering committee); served as deputy editor for Clinical Cardiology and Progress in Cardiovascular Diseases; received patent for sotagliflozin (named on a patent for sotagliflozin assigned to Brigham and Women's Hospital who assigned to Lexicon; neither I nor Brigham and Women's Hospital receive any income from this patent); received research funding from Abbott, Acesion Pharma, Afimmune, Alnylam, Amarin, Amgen, AstraZeneca, Atricure, Bayer, Boehringer Ingelheim, Boston Scientific, CellProthera, Cereno Scientific, Chiesi, Cleerly, CSL Behring, Faraday Pharmaceuticals, Fractyl, Idorsia, Janssen, Javelin, Lexicon, Lilly, Medtronic, Merck, MiRUS, Moderna, Novartis, Novo Nordisk, Pfizer, PhaseBio, Regeneron, Reid Hoffman Foundation, Roche, Sanofi, Stasys, and 89Bio; received royalties from Elsevier (Editor, Braunwald's Heart Disease); and is served as a site co-investigator at Cleerly. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS body mass index, coronary calcification, waist circumference, waist-to-hip ratio

APPENDIX For an expanded Methods section, please see the online version of this paper.