

Abdominal obesity and coronary artery calcification in young adults: the Coronary Artery Risk Development in Young Adults (CARDIA) Study¹⁻³

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ABSTRACT

Background: Whether abdominal obesity is related to coronary artery calcification (CAC) is not known.

Objective: We investigated the relations of waist girth and waist-hip ratio (WHR) to CAC in 2951 African American and white young adults from the Coronary Artery Risk Development in Young Adults Study.

Design: The present study was a cross-sectional and observational cohort study. Using standardized protocols, we measured CAC in 2001–2002 by using computed tomography and measured waist and hip girths in 1985–1986 (baseline), 1995–1996 (year 10), and 2001–2002 (year 15, waist girth only). CAC was classified as present or absent, whereas waist girth and WHR were placed in sex-specific tertiles.

Results: After adjustment for age, sex, race, clinical center, physical activity, cigarette smoking, education, and alcohol intake, baseline waist girth and WHR were directly associated with a higher prevalence of CAC 15 y later (*P* for trend < 0.001 for both). The odds ratios (ORs) for CAC in the highest versus lowest tertiles of waist girth and WHR were 1.9 (95% CI: 1.36, 2.65) and 1.7 (1.23, 2.41), respectively. Waist girth and WHR at year 10 and waist girth at year 15 similarly predicted CAC. These associations persisted after additional adjustment for systolic blood pressure, fasting insulin concentrations, diabetes, and antihypertensive medication use but became nonsignificant after additional adjustment for blood lipids.

Conclusions: Abdominal obesity measured by waist girth or WHR is associated with early atherosclerosis as measured by the presence of CAC in African American and white young adults. This is consistent with an involvement of visceral fat in the occurrence of coronary artery calcium in young adults. *Am J Clin Nutr* 2007; 86:48–54.

KEY WORDS Coronary artery calcification, abdominal obesity, waist girth, waist-hip ratio

INTRODUCTION

Atherosclerosis is a major cause of coronary heart disease (CHD) and ischemic stroke. The progression of plaque formation and calcium deposition is associated with the accumulation of macrophages, smooth muscle cells, fibrosis, necrosis, and lipids in the arteries (1, 2). Coronary artery calcification (CAC) is a risk marker for atherosclerosis and is positively associated with CHD and cardiovascular disease (CVD) events (3–6). In general, the established risk factors for coronary calcification are the same as

those for clinical CVD: male sex, age, body mass index, elevated blood pressure, diabetes mellitus, cigarette smoking, and LDL and HDL cholesterol (3, 7–9).

Abdominal obesity is also a significant risk factor for atherosclerosis. Excessive accumulation of visceral fat is associated with insulin resistance and compensatory hyperinsulinemia, which contributes to atherosclerotic progression (10). An elevated insulin concentration is a risk marker for dyslipidemia and hypertension, and it promotes smooth muscle cell proliferation and cholesterol ester accumulation in the artery (11). However, there has been little research on the relation between abdominal obesity and subclinical atherosclerotic vascular disease across race, sex, and age groups in population-based studies. We therefore investigated the relation of visceral fat measured by waist girth or waist-hip ratio (WHR) to CAC in young African American and white men and women from the Coronary Artery Risk Development in Young Adults (CARDIA) Study.

SUBJECTS AND METHODS

Study population

The CARDIA study is a population-based cohort study designed to investigate the causes of atherosclerosis in a young biracial population from 4 communities in the United States: Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA. The baseline study population comprised 5115 African American and white men and women aged 18–30 y, who were recruited in 1985 and 1986. The participants were selected according to a balanced design by race, sex, educational attainment

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² The Coronary Artery Risk Development in Young Adults (CARDIA) Study was supported by contracts N01-HC-48047, N01-HC-48048, N01-HC-48049, N01-HC-48050, N01-HC-95095, and N01-HC-45134 from the National Heart, Lung, and Blood Institute.

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Received October 6, 2006.

Accepted for publication February 16, 2007.

(less than high school, completion of high school, or more than high school), and age groups (18–24 and 25–30 y old). The complete study design, sampling strategy, and examination techniques were reported previously (12). The participants were re-examined during 1987–1988 (year 2), 1990–1991 (year 5), 1992–1993 (year 7), 1995–1996 (year 10), and 2000–2001 (year 15), when the rate of retention was 90%, 86%, 81%, 79%, and 74%, respectively. The current study is based on those 3043 persons who underwent a coronary artery scan at the year 15 examination to determine the presence or absence of calcium deposits in the arteries.

All subjects gave written informed consent for the clinical examination. The institutional review boards at all sites approved the protocol.

Measurements

All participants were asked to fast for 12 h before the clinical examination. Body height and weight were measured with a calibrated scale and a vertical ruler, respectively, and body mass index (BMI; in kg/m²) was calculated. Waist girth was measured laterally at the point midway between the iliac crest and the lowest lateral portion of the rib cage and anteriorly at the point midway between the xiphoid process of the sternum and the umbilicus. The hip girth was measured anteriorly at the level of the symphysis pubis and posteriorly at the level of the maximal protrusion of the gluteal muscles by using a spring-gauge plastic tape measure. Seated blood pressure was measured after 5 min of rest with the use of a random-zero sphygmomanometer, and the average of the last 2 of 3 consecutive measurements was used for analysis. Serum, plasma, and whole blood samples were drawn from an antecubital vein. Plasma concentrations of total cholesterol, HDL cholesterol, and triacylglycerol were measured with an enzymatic method (13). HDL cholesterol was measured after dextran-magnesium precipitation (14), and LDL cholesterol was calculated by using the Friedewald equation (15). Fasting insulin was measured with the use of a radioimmunoassay (Linco Research, St Charles, MO). Test-retest reliability coefficients for total, HDL, and LDL cholesterol and triacylglycerol were >0.98 (16). Additional details of examination procedures were published previously (16, 17).

CAC was determined at the year 15 examination by using computed tomography (CT). Trained technicians scanned the root of aorta to the apex of the heart and obtained 40 contiguous 2.5–3.0-mm-thick transverse images by using electron beam CT (Chicago and Oakland field centers) and multidetector CT (Birmingham and Minneapolis field centers). All participants were scanned twice over a hydroxyl-apatite phantom to allow monitoring of image brightness and noise and adjustment for scanner differences. Scans were electronically sent to the CARDIA Reading Center, and a radiologist identified the courses of the coronary arteries by using specially developed image-processing software (developed at the Harbor-UCLA Medical Center CT Reading Center, Los Angeles, CA) programmed to define a calcific focus as 4 adjacent pixels composing an area of ≥ 1.87 mm². A calcium score was calculated for each calcified lesion by multiplying the area of focus by a coefficient based on the peak CT number in the focus. The coefficient ranged from 1 to 4 [1 = 131–200 Hounsfield units (HU), 2 = 201–300 HU, 3 = 301–400 HU, and 4 = ≥ 401 HU]. Total calcium scores were obtained by summing all lesions within a given artery and across all arteries (left main, left anterior descending, left circumflex, and right

coronary arteries). Each scan set with ≥ 1 nonzero coronary calcium score and a random sample of those with 0 score were reviewed by an expert investigator who had no knowledge of the scan scores, to verify the presence of CAC. The overall score was calculated as the average of the 2 scans if the investigator adjudicated the scans as being positive and was set to zero for those scans adjudicated as negative. Details of the CAC examination techniques and procedures were published previously (18).

Cigarette smoking, alcohol intake, physical activity, and educational level were assessed by means of standardized questionnaires. Smoking status was classified as never smoker, former smoker, or current smoker, and alcohol intake was classified as a continuous variable (ethanol intake, in mL/d). Self-reported physical activity scores were computed by multiplying the frequency of participation by the intensity of the activity (19). Educational level was classified by the number of years of education: less than high school, completion of high school, or at least some college. The use of antihypertensive or cholesterol-lowering medication was assessed by the self-reported questionnaire. Diabetes mellitus was defined as a fasting glucose concentration ≥ 126 mg/dL or the use of hypoglycemic agents.

Statistical analysis

After the exclusion of 4 pregnant women at baseline, 2 pregnant women at year 15 examination, and subjects who were missing dependent or independent variables or covariate values, we included 2951 men and women aged 33–45 y who underwent a coronary artery scan at the year 15 examination. Coronary calcium scores were classified as dichotomous variable (0 = absence; 1 = presence if calcium score was >0), and the waist girth and WHR were classified by using sex-specific tertiles. General linear models were used to test mean differences across waist girth categories after adjustment for age, sex, race, and field center. The chi-square test was used to compare frequency differences across waist girth categories. Multivariate logistic regression models were used to investigate the associations of waist girth (baseline, year 10, and year 15) and WHR (baseline and year 10) with the year 15 presence of CAC after adjustment for age, sex, race, and clinical center (model 1); after additional adjustment for physical activity, cigarette smoking, education, and alcohol intake (model 2); after additional adjustment for systolic blood pressure, fasting insulin, diabetes, and antihypertensive medication use from model 2 (model 3); and after additional adjustment for blood lipids (HDL and total cholesterol and triacylglycerol) from model 3 (model 4). The additional adjustment variables in models 3 and 4 could be in the causal pathway between waist girth and CAC; we regarded these models as explanatory, rather than deconfounding. The smallest waist girth or the lowest WHR tertile was the reference category. Trends across exposure categories were tested by treating those categories as a categorical scale with adjustment for covariates. We also examined the race- and sex-adjusted partial Pearson correlations among waist girth, WHR, and BMI across baseline, year 10, and year 15 examinations. All statistical procedures were performed by using SAS software (version 9; SAS Institute, Cary, NC).

RESULTS

As shown in **Table 1**, the prevalences or means of several baseline variables differed according to baseline waist girth. Prevalences of diabetes and antihypertensive medication use

TABLE 1

Characteristics of the study participants across waist girth categories at baseline and coronary artery calcification at year 15: the CARDIA Study, 1985–2001¹

	Sex-specific waist girth tertiles ²			<i>P</i>
	1 (<i>n</i> = 968)	2 (<i>n</i> = 994)	3 (<i>n</i> = 989)	
Baseline				
Age (y) ³	24.3	25.3	26.0	
African American (%)	43.9	41.5	50.1	
Adjusted values ^{3,4}				
BMI (kg/m ²)	20.8	23.4	28.8	< 0.001
Systolic blood pressure (mg/dL)	108.4	109.9	113.3	< 0.001
Total cholesterol (mg/dL)	172.4	176.7	183.7	< 0.001
HDL cholesterol (mg/dL)	56.8	54.4	48.4	< 0.001
LDL cholesterol (mg/dL)	103.7	109.1	117.3	< 0.001
Triacylglycerol (mg/dL)	60.0	66.3	89.7	< 0.001
Fasting insulin (μU/mL)	7.6	9.0	14.4	< 0.001
Alcohol intake (mL/d)	11.2	11.6	13.8	0.01
Physical activity score	427.3	446.9	401.2	0.002
Diabetes (%)	0.1	0.4	1.1	0.008
Antihypertensive medication use (%)	0.4	1.7	4.0	< 0.001
Current smoking (%)	27.0	23.5	28.8	0.03
Educational level (<high school) (%)	8.7	5.1	7.9	0.006
Year 15 outcome variable				
Coronary artery calcification (%)	6.4	8.0	13.8	< 0.001

¹ *n* = 2951. CARDIA, Coronary Artery Risk Development in Young Adults. All characteristics except coronary artery calcification were measured at CARDIA year 0 when the participants were aged 18–30 y.

² Waist girth cutoffs: <77.5, 77.5–<84.3, and ≥84.3 cm (men) and <68, 68–<75.5, and ≥75.5 cm (women) for tertiles 1, 2, and 3, respectively.

³ Values are \bar{x} .

⁴ Adjusted for age, sex, race, and clinical center.

were progressively higher across rising sex-specific waist girth tertiles. Means of BMI, systolic blood pressure, LDL and total cholesterol, triacylglycerol, fasting insulin, and alcohol intake were also progressively higher, and that of HDL cholesterol was lower across rising waist girth categories. Physical activity, smoking, and educational level showed inconsistent relations with waist girth. The prevalence of year 15 coronary calcification increased progressively across rising waist girth tertiles (*P* < 0.001).

The associations of baseline waist girth and WHR with the year 15 presence of CAC are shown in **Table 2**. After adjustment for baseline age, sex, race, and clinical center, there was a direct association between baseline waist girth and year 15 CAC and between baseline WHR and year 15 CAC (*P* for trend < 0.001 for both). Associations persisted after additional adjustment for baseline physical activity, cigarette smoking, educational level, and alcohol intake. For subjects in the highest tertile of baseline waist girth (≥84.3 and ≥75.5 cm in men and women, respectively) versus subjects in the lowest tertile, CAC risk over 15 y was 1.9 (95% CI: 1.36, 2.65). We observed a similar association between baseline WHR and CAC risk (*P* for trend = 0.001) after adjustment for multiple risk factors (model 2). For persons in the highest versus lowest tertile of WHR (≥0.85 and ≥0.75 in men and women, respectively), the OR for CAC risk was 1.7 (95% CI: 1.23, 2.41). Associations persisted after additional adjustment for systolic blood pressure, fasting insulin, diabetes, and antihypertensive medication use. As Table 2 shows, the association between waist girth and CAC risk was significant (*P* for trend = 0.01) after adjustment for these factors (model 3). A similar association was observed between baseline WHR and CAC risk (*P* for trend = 0.03). However, the associations were attenuated and became nonsignificant after additional adjustment for blood

lipids (HDL and total cholesterol and triacylglycerol) (model 4). Further adjustment for the ratio of total to HDL cholesterol did not alter the association of waist girth or WHR to CAC (data not shown).

We also examined the associations of year 10 waist girth and WHR and year 15 waist girth with the presence of CAC at year 15. Substantial concordance of these findings with those using baseline waist girth or WHR as the exposure variable was expected, given the relatively high race- and sex-adjusted correlations of these variables with each other and over time (**Table 3**). The odds of having CAC, after adjustment for age, sex, race, clinical center, physical activity, cigarette smoking, educational level, and alcohol intake (adjustments analogous to those of model 2 in Table 2), in subjects in the highest tertile of waist girth at year 10 and year 15 were 1.7 (95% CI: 0.73, 1.47) and 1.8 (95% CI: 1.28, 2.47) times, respectively, the odds in subjects in the lowest waist girth category, as shown in **Figure 1**. Also shown in Figure 1 is that subjects in the highest tertile of WHR at year 10 had odds of year 15 CAC 1.8 times (95% CI: 1.25, 2.51) those of subjects in the lowest tertile of WHR at year 10.

Several supplemental analyses were run. First, further analyses studied the association of change in waist girth or WHR with year 15 CAC. Changes in waist girth (from baseline to year 15) and WHR (from baseline to year 10) were unrelated to year 15 CAC, whether in models that included the corresponding baseline measure or in those that excluded it. Second, we assessed a possible race × sex interaction for baseline waist girth in predicting year 15 CAC in model 2; the *P* value for the race × sex interaction was 0.37. Significant trends toward increasing year 15 CAC across baseline waist girth or WHR categories were seen in models stratified by sex or by race. Third, we examined the



TABLE 2

Odds ratios (95% CI) of the presence of coronary artery calcification at year 15 by baseline (1985–1986) waist girth and waist-hip ratio (WHR) in white and African American young adults: the CARDIA Study¹

Variables	Cases	Sex-specific tertiles of predictor variables ²			P for trend
		1	2	3	
Waist girth (n)	277	62	79	136	
Adjusted for age, sex, race, and clinical center (model 1)		1.00	1.08 (0.76, 1.54)	1.86 (1.33, 2.58)	< 0.001
Multivariate models					
Model 2 ³		1.00	1.17 (0.82, 1.68)	1.90 (1.36, 2.65)	< 0.001
Model 3 ⁴		1.00	1.12 (0.78, 1.62)	1.61 (1.12, 2.31)	0.01
Model 4 ⁵		1.00	1.03 (0.71, 1.49)	1.21 (0.83, 1.77)	0.36
WHR (n)	277	62	77	138	
Adjusted for age, sex, race, and clinical center (model 1)		1.00	1.08 (0.76, 1.55)	1.92 (1.38, 2.68)	< 0.001
Multivariate models					
Model 2 ³		1.00	1.03 (0.72, 1.48)	1.72 (1.23, 2.41)	0.001
Model 3 ⁴		1.00	0.98 (0.68, 1.42)	1.47 (1.03, 2.09)	0.03
Model 4 ⁵		1.00	0.89 (0.61, 1.29)	1.19 (0.83, 1.72)	0.32

¹ n = 2951 for both measurements. CARDIA, Coronary Artery Risk Development in Young Adults.

² Tertile cutoffs are <77.5, 77.5–<84.3, and ≥84.3 cm (men) and <68, 68–<75.5, and ≥75.5 cm (women) for waist girth and <0.81, 0.81–<0.85, and ≥0.85 (men) and <0.71, 0.71–<0.75, and ≥0.75 (women) for WHR.

³ Adjusted for variables in model 1 plus physical activity, cigarette smoking, education, and alcohol intake.

⁴ Adjusted for variables in model 2 plus systolic blood pressure, fasting insulin concentrations, diabetes, and antihypertensive medication use.

⁵ Adjusted for variables in model 3 plus blood lipids (HDL- and total cholesterol and triacylglycerol concentrations).

associations categorizing sex-specific waist girth quintiles at baseline and found that the ORs (95% CI) compared with the smallest waist girth quintile (quintile 1) in model 2 were 1.06 (0.59, 1.88) for quintile 2, 1.21 (0.68, 2.14) for quintile 3, 1.50 (0.86, 2.62) for quintile 4, and 2.52 (1.47, 4.32) for quintile 5 (P for trend < 0.001). Analysis of baseline WHR in sex-specific quintiles yielded a similar association in model 2: 1.00 (lowest quintile), 1.08 (0.66, 1.74), 0.94 (0.58, 1.54), 1.54 (0.98, 2.42), and 1.99 (1.28, 3.08) (P for trend = 0.001). Fourth, we also considered categories based on National Institutes of Health guidelines (20). Men and women with baseline waist girth > 102 or > 88 cm, respectively, had odds of year 15 CAC that were 2.4 times (95% CI: 1.54, 3.79) those of men and women with smaller waist girth at baseline in model 2. Fifth, we found that associations of year 15 CAC with BMI were similar to those reported

here for baseline waist girth or WHR, but each fatness variable became nonsignificant when any 2 of those variables were in the model simultaneously (data not shown).

DISCUSSION

Although abdominal obesity is considered a risk factor for CVD incidence and mortality (21–25), little research has been conducted on the association between abdominal obesity and early atherosclerosis. Our major finding was that abdominal obesity measured by waist girth or WHR is directly associated with a higher prevalence of CAC over a period of 15 y. Persons in the highest tertiles of baseline waist girth and WHR had year 15 CAC odds, after adjustment for age, sex, race, center, physical activity, cigarette smoking, educational level, and alcohol intake, 1.9

TABLE 3

Race- and sex-adjusted partial correlations among waist girth, waist-hip ratio (WHR), and BMI: the CARDIA Study, 1985–2001 (n = 2767)¹

Variables	Waist girth			WHR		BMI		
	Baseline	Year 10	Year 15	Baseline	Year 10	Baseline	Year 10	Year 15
Waist girth								
Baseline	1.00	0.79 ²	0.71 ²	0.68 ²	0.57 ²	0.90 ²	0.74 ²	0.67 ²
Year 10	—	1.00	0.86 ²	0.50 ²	0.75 ²	0.76 ²	0.91 ²	0.82 ²
Year 15	—	—	1.00	0.44 ²	0.60 ²	0.68 ²	0.82 ²	0.89 ²
WHR								
Baseline	—	—	—	1.00	0.66 ²	0.45 ²	0.36 ²	0.30 ²
Year 10	—	—	—	—	1.00	0.43 ²	0.52 ²	0.44 ²
BMI								
Baseline	—	—	—	—	—	1.00	0.83 ²	0.76 ²
Year 10	—	—	—	—	—	—	1.00	0.91 ²
Year 15	—	—	—	—	—	—	—	1.00

¹ CARDIA, Coronary Artery Risk Development in Young Adults.

² P < 0.01.



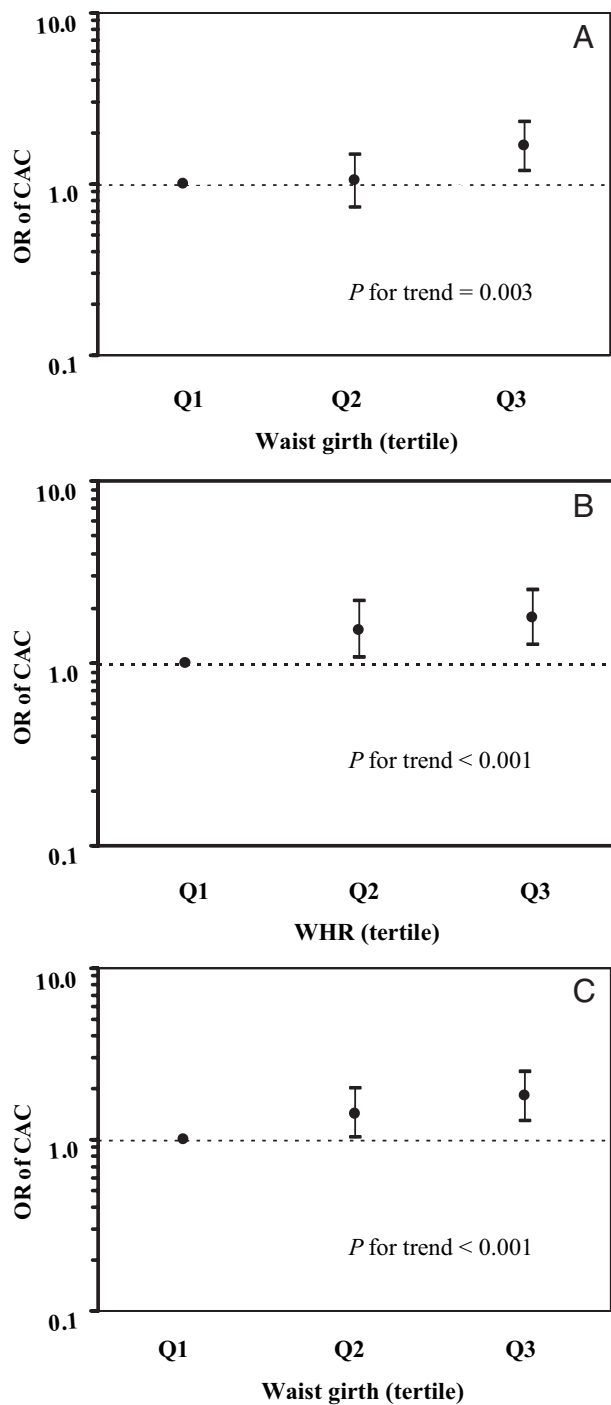


FIGURE 1. Odds ratios (ORs) of coronary artery calcification (CAC) by sex-specific tertile of year 10 waist girth (A) and waist-hip ratio (WHR; B) (referent: lowest tertile) and of year 15 waist girth (C) examinations, adjusted for age (single year), sex, race, clinical center, physical activity, cigarette smoking, education, and alcohol intake. Error bars represent 95% CIs for each OR.

(95% CI: 1.36, 2.65) and 1.7 (95% CI: 1.23, 2.41) times, respectively, those in persons in the lowest tertile. Our findings were consistent with the results from the St Francis Heart Study, in which abdominal adiposity measured by WHR or intraabdominal fat was positively correlated with CAC in 50–70-y-old US men and women (26). Our data also show that subjects in the

highest tertiles of waist girth and WHR at year 10 or year 15 had greater odds of having CAC after adjustment for multiple risk factors (model 2) than did those in the lowest tertiles. The Muscatine Study also showed that body weight during childhood (ages 8–18 y) or BMI during early young adulthood (ages 20–34 y) was positively associated with CAC in later young adulthood (ages 29–37 y) (27).

Our findings were similar after further adjustment for baseline blood pressure, insulin, diabetes, and antihypertensive medication use (P for trend = 0.01) but were further attenuated to nonsignificance after additional adjustment for blood lipids. Thus, of the factors that could well be in the causal pathway between adiposity and CAC presence, blood lipids appeared to be the ones that explained the mechanisms underlying the association between adiposity and the presence of CAC.

In our study, a dose-response relation between abdominal obesity and the presence of CAC persisted across baseline, year 10, and year 15 examinations. Abdominal obesity measured in 1985–1986, 1995–1996, and 2000–2001 was positively associated with the odds of having CAC in 2000–2001. Our findings suggest that waist girth and WHR are risk factors for early atherosclerosis, which is consistent with the National Institutes of Health guidelines (20).

Several studies have reported that abdominal obesity is associated with metabolic risk factors and CVD mortality (21–25, 28, 29). In our data, waist girth is directly associated with systolic blood pressure, blood lipids, and blood concentrations of glucose and insulin. Although we found that blood lipids were the factors that statistically explained the association of abdominal adiposity and the presence of CAC, elevated blood lipids have complex relations with blood pressure, blood glucose, and insulin. It is plausible that greater abdominal obesity may enhance atherosclerosis through disturbances in any of these variables. Greater visceral fat is associated with insulin resistance and hyperinsulinemia, and that association contributes to the progression of atherosclerosis (10). Greater visceral fat also is associated with perturbations of the hemostatic and coagulation systems. For instance, greater visceral fat elevates the release of plasminogen activator inhibitor-1 (PAI-1), increases the markers of hypercoagulability and hypofibrinolysis, and disturbs PAI-1 activity, which impairs the fibrinolytic system and contributes to thrombotic vascular disease in the artery (30, 31). Abdominal obesity is positively associated with LDL cholesterol and oxidized LDL, and that association contributes to endothelial cell injury and subsequent thrombus formation (1, 2). In general, abdominal obesity can play a prothrombotic role by increasing plasma lipid and lipoprotein concentrations (28, 29) and inflammatory markers [eg, C-reactive protein, tumor necrosis factor- α (TNF- α), and interleukin-6 (IL-6); 32–34] and by elevating blood viscosity (35) and impairing fibrinolysis (36), all of which may contribute to atherosclerotic vascular disease.

A strength of this study is that our data represent population-based samples of US African American and white young men and women. To our knowledge, this is the first study to investigate the relation of abdominal obesity measured by waist girth or WHR to the presence of CAC in young adults. Further studies are needed to determine whether abdominal obesity is associated with presence of CAC across different ethnic groups or in diabetic or hypertensive persons. One limitation of our study is that we did not assess CAC before year 15, and thus we cannot tell whether the presence of CAC is incident or prevalent. However,

other studies found that CAC was present in higher proportions in the older samples (eg, The Multi-Ethnic Study of Atherosclerosis Study), and it is likely that much of the CAC seen in the CARDIA Study developed between baseline (average age: 25 y) and year 15. Limitations of our study also include the relatively small numbers of persons with CAC, which restricted our power to examine subgroups. Furthermore, it is known that CT images of the heart are noisier and more difficult to interpret in fatter than in thinner persons. However, whereas conservative reading in the face of greater noise could explain the lack of relation cross-sectionally between BMI and CAC presence (9), both concurrent and baseline waist girth were related to the presence of CAC. Another possible limitation is that some, but not all, subjects weighing >360 lb were excluded because the CT table could not hold a person of that weight, which may have created an unknown bias. Finally, adjustment for BMI attenuated the association of waist girth with CAC presence. Whereas BMI is a measure of total-body fatness and it seems that waist girth would be a measure of fatness more specific to the abdominal region, Table 3 shows the very high correlation between waist girth and BMI. Apparently BMI carries a considerable amount of information about fatness in the abdomen; we interpret the attenuation as partially adjusting waist girth for itself.

In conclusion, we found that abdominal obesity measured by waist girth or WHR is associated with early coronary calcification in young adults. Our study adds to the body of knowledge suggesting that waist girth in young adulthood may help to identify persons with potential future CHD risk that is most likely due to associated lipid and other metabolic abnormalities.

The authors thank the staff of and participants in the CARDIA Study for their important contributions.

The authors' responsibilities were as follows—CDL and DRJ: proposed the study, analyzed the data, and wrote the manuscript; and PJM, CI, and AH: assisted with statistical analyses and revision of the manuscript. None of the authors had a personal or financial conflict of interest.

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