Obesity Is Associated With Larger Arterial Diameters in Caucasian and African-American Young Adults

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Risk factors for larger arterial diameters and enhanced wall thickening have been insufficiently assessed in young adults, an age-group where preventive measures have the greatest impact. Larger vessel diameter and wall thickening may represent underlying vascular remodeling, a dynamic process by which the vascular system attempts to compensate for vascular damage (1). The purpose of the current study was to examine the association between obesity and arterial diameter and wall thickness in healthy Caucasian and African-American young adults. Associations were examined in both the muscular peripheral arteries and the more elastic central arteries.

RESEARCH DESIGN AND METHODS — Measurements were made in 205 participants aged 20–40 years. Exclusion criteria included use of antihypertensive, lipid-lowering, thyroid, blood glucose–lowering, or cardiovascular medications and history of lupus or clinical cardiovascular disease. This study was approved by the University of Pittsburgh's institutional review board. All participants provided written informed consent before study protocol initiation.

Left brachial artery adventitial diameter was determined during diastole by electronic caliper measurement of the adventitial to adventitial diameter. For common carotid measurements, the left distal common carotid artery near and far walls were imaged, and lines were electronically drawn along a 1-cm segment of the lumen-intima interface and the media-adventitia interface. The mean intima-media thickness (IMT) was averaged over the near and far walls. The lumen diameter (intima-intima distance) was calculated directly from the imaged interfaces. Common carotid interadventitial diameter was calculated by the following equation: lumen diameter plus near wall thickness plus far wall thickness.

Blood pressure and obesity measures were determined using standard examination procedures. Lipid and glucose values were determined after a 12-h fast using standard laboratory procedures.

SAS version 8.2 software (SAS Institute, Cary, NC) was used for all analyses. Diameter measures and IMT were normally distributed. Associations between diameter measures or IMT and covariates were assessed via linear regression modeling, using least squares means to calculate adjusted means by BMI categories.

RESULTS — The population was 54% women, 50% African American, and 23% current smokers. Mean values for diameter measures were 3.6 mm for brachial artery interadventitial diameter, 6.8 mm for carotid artery interadventitial diameter, and 5.5 mm for carotid artery lumen diameter. Mean carotid artery IMT was 0.6 mm. Mean values for obesity measures were 79.8 kg for weight, 84.0 cm for waist circumference, and 27.5 kg/m^2 for BMI. Mean blood pressure and lipid values were in the normal range.

Strong associations were present between obesity measures and all three diameter measures, independent of age, sex, race/ethnicity, systolic blood pressure (SBP), and height (Table 1). Greater IMT was associated with greater obesity measures in unadjusted analyses but lost significance after adjustment for age and SBP.

Individuals were categorized next by BMI, according to the World Health Organization definitions (overweight: BMI $\geq$25 but $<$30 kg/m^2; obese: BMI $\geq$30 kg/m^2) (2). Overweight individuals had a 5% larger brachial interadventitial diameter ($P = 0.017$), a 3% larger carotid interadventitial diameter ($P = 0.033$), and a 2% larger carotid lumen diameter ($P = 0.087$) compared with normal weight individuals, after adjustment for age, sex, race/ethnicity, SBP, and height. Obese individuals had a 9% larger brachial interadventitial diameter ($P < 0.01$) and 4% larger carotid interadventitial ($P < 0.01$) and lumen ($P = 0.004$) diameters compared with normal weight individuals after multivariable adjustment. IMT was not associated with categorical BMI.

CONCLUSIONS — To our knowledge, these are the first data showing strong positive relationships between arterial diameter and body weight among healthy young adults. Arterial enlargement is of concern in young individuals because the more an artery dilates, the less ability it has to compensate for future adverse conditions. This eventually results in damage to the artery wall and a gradual stiffening of the artery (1). These data suggest evidence of vascular remodeling and premature vascular aging among young overweight and obese individuals who are otherwise healthy.

The positive associations shown here between vessel diameters and obesity in African-American young adults are simi-

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Abbreviations: IMT, intima-media thickness; SBP, systolic blood pressure;

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Table 1—*Adjusted* effects of covariates on arterial diameter and wall thickness measures from regression modeling*

	Brachial artery		Carotid artery					
	interadventitial diameter (mm)		Interadventitial diameter (mm)		Lumen diameter (mm)		IMT (mm)	
Variables, increment†	β (SE)	*P*	β (SE)	*P*	β (SE)	*P*	β (SE)	*P*
Age, 5 years‡	0.03 (0.03)	0.185	0.08 (0.03)	0.002	0.04 (0.02)	0.078	0.02 (0.004)	<0.001
Male sex‡	0.50 (0.09)	<0.001	0.29 (0.09)	<0.001	0.29 (0.08)	<0.001	−0.003 (0.01)	0.835
African American‡	0.04 (0.07)	0.579	0.11 (0.07)	0.099	0.04 (0.07)	0.523	0.04 (0.01)	<0.001
Current smoker	0.13 (0.08)	0.125	0.09 (0.08)	0.264	0.11 (0.08)	0.166	−0.01 (0.01)	0.583
SBP, 10 mmHg‡	0.002 (0.02)	0.931	0.004 (0.02)	0.864	−0.02 (0.02)	0.345	0.01 (0.004)	0.001
DBP, 10 mmHg	0.01 (0.04)	0.784	0.06 (0.04)	0.125	0.03 (0.03)	0.320	0.01 (0.01)	0.033
Cholesterol, 0.26 mmol/l (10 mg/dl)	0.01 (0.01)	0.370	−0.01 (0.01)	0.291	−0.01 (0.01)	0.150	0.001 (0.001)	0.361
LDL, 0.26 mmol/l (10 mg/dl)	0.01 (0.01)	0.403	−0.004 (0.01)	0.712	−0.01 (0.01)	0.393	0.002 (0.002)	0.176
HDL, 0.13 mmol/l (5 mg/dl)	−0.03 (0.01)	0.041	−0.02 (0.01)	0.090	−0.02 (0.01)	0.049	0.001 (0.002)	0.704
Triglycerides, 0.11 mmol/l (10 mg/dl)	0.01 (0.01)	0.017	−0.002 (0.005)	0.632	−0.001 (0.005)	0.907	−0.001 (0.001)	0.284
Glucose, 0.56 mmol/l (10 mg/dl)	0.05 (0.05)	0.330	−0.001 (0.05)	0.987	−0.02 (0.05)	0.702	0.01 (0.01)	0.106
Height, 1 cm‡	0.06 (0.01)	<0.001	0.05 (0.01)	<0.001	0.05 (0.01)	<0.001	0.002 (0.002)	0.410
Weight, 5 lb	0.01 (0.005)	0.003	0.01 (0.005)	0.011	0.01 (0.005)	0.010	0.001 (0.001)	0.502
Waist circumference, 1 cm	0.01 (0.003)	0.011	0.01 (0.003)	0.001	0.01 (0.002)	<0.001	0.0001 (0.0004)	0.889
BMI, 1 kg/m^2	0.02 (0.01)	0.007	0.02 (0.01)	0.012	0.02 (0.01)	0.009	0.0004 (0.001)	0.639

*Adjusted for age, sex, race/ethnicity, supine SBP, and height, unless otherwise specified; †Values in parentheses are equivalent values in standard units. ‡Adjusted for the four remaining variables. DBP, diastolic blood pressure.

lar to those documented in middle-aged African Americans (3). African Americans suffer higher obesity rates than Caucasians (4,5). The associations with arterial diameters shown here in the context of higher obesity rates strongly suggest that cardiovascular disease rates will disproportionately increase among African Americans.

Obesity may be associated with larger diameters through a variety of factors. Obesity is accompanied by insulin resistance (6), increased levels of leptin (7), and changes to the renin-angiotensin system (8). Each of these shares a role in sodium retention and resulting increased fluid volume (9–11). The arteries then dilate to handle the increased volume, likely prompted by higher shear stress levels (12). Obesity may also be associated with vessel diameters by mechanisms unrelated to shear stress levels. Obesity is accompanied by inflammation (13), which has been suggested to be related to arterial enlargement through factors intrinsic to the vessel wall, such as macrophage infiltration (14).

Finally, more long-term chronic effects of obesity may eventually play a role, such as the arterial stiffening that accompanies arterial dilation. Arterial stiffening is associated with a degradation of elastin (15), inhibiting the artery from fully retracting during diastole, perhaps bringing about the larger diastolic lumen and adventitial diameters noted here. We have previously shown that obesity measures were strongly associated with increased central artery stiffening (16).

Future research concerning longitudinal changes in arterial diameter and wall thickening in relation to obesity is needed to determine whether the larger arterial diameters among obese individuals shown here are indeed evidence of vascular remodeling and, if so, whether adventitial diameters return to normal with weight loss. Data in middle-aged and elderly participants indicated that LDL-associated increases in vessel diameter returned to normal after lipid treatment, suggesting that certain risk factors may affect vascular tone without affecting vessel structure (17).

In conclusion, these data document strong associations between both peripheral and central artery diameter and measures of obesity among healthy young adults. Associations were equally strong for both Caucasians and African Ameri-

cans. Thus, obesity has early vascular effects which likely indicate premature vascular aging among overweight and obese individuals. Weight control among young adults should be a high priority in prevention programs.

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