Title

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Journal

Obesity, 23(11)

ISSN

1930-7381

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Publication Date

2015-11-01

DOI

10.1002/oby.21221

Peer reviewed

HHS Public Access

Author manuscript

Obesity (Silver Spring). Author manuscript; available in PMC 2016 May 18.

Published in final edited form as:

Obesity (Silver Spring). 2015 November ; 23(11): 2294–2298. doi:10.1002/oby.21221.

Associations of Body Composition Measures and C2, a Marker for Small Artery Elasticity: The Multi-Ethnic Study of Atherosclerosis

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Abstract

Objective—Lower C2, a continuous blood pressure waveform characteristic asserted to represent small artery elasticity, predicts future cardiovascular disease (CVD) events. We hypothesized that the paradoxical positive association between body mass index (BMI) and C2 may reflect muscle instead of excess fat.

Methods—In a multi-ethnic, community living cohort of 1,960 participants, we used computed tomography (CT) scans of the abdomen to measure visceral adipose tissue (VAT) and total abdominal muscle tissue (TAMT), and used applanation tonometry of the radial arteries to assess C2. We then ascertained the period cross-sectional associations between BMI, TAMT, and VAT with C2.

Results—The mean age was 62 ± 9 years and 50% male. After adjustments for age, gender, ethnicity, pack years smoking cigarettes, diabetes, hypertension, total and HDL cholesterol, higher BMI (standardized beta = 0.09, p-value < 0.01) and more TAMT (standardized beta = 0.12, p-value < .01), were significantly associated with higher C2. In contrast, more VAT (standardized beta = -0.09, p-value < .01) was associated with lower C2.

Conclusion—In multivariable analysis, VAT, in contrast to TAMT and BMI was associated with less compliant small arteries. Visceral fat may be a better marker for detrimental excess body fat than BMI.

Keywords

Body mass index; Visceral adipose tissue; Abdominal muscle tissue

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Disclosure: The authors declare no conflict of interest.

Introduction

Studies investigating correlates and outcomes of excess body fat often use body mass index [BMI, weight (kg)/height (m^2)]. The “obesity paradox” is the observation that being overweight with excess body fat may be associated with lower mortality and better cardiovascular disease (CVD) outcomes, despite the fact that obesity is a recognized risk factor for CVD. This observation initially reported over a decade ago in coronary heart disease (CHD) patients undergoing percutaneous coronary intervention, has been demonstrated in patients with hypertension, peripheral artery disease (PAD), stroke, myocardial infarction (MI), heart failure, and end stage renal disease (HF).([1–5]) Moreover, a systematic review of 40 cohort studies and 250,152 participants with known CHD, found a U-shaped relationship between body mass index (BMI) and CVD mortality, with the lowest and highest weight categories exhibiting the highest risk, while the moderate overweight category was associated with a lower risk compared to the normal weight group.([6])

Several theories for the obesity paradox have been proposed. First, BMI may indicate lean body mass rather than just fat mass. Robero-Corral et al. used body fat percentage (BF%, calculated from bioelectrical impedance analysis) as the gold standard to determine the accuracy of BMI to identify obesity in men (BF% > 25%) and women (BF% > 35%) in large sample representative of the U.S. population.([7]) They reported that BMI ≥ 30 had a low sensitivity and high specificity to detect obesity in men (46 and 95 percent respectively) and women (49 and 99 percent respectively). The also reported that BMI was more strongly correlated with lean mass (R^2=.44, P-value ≤ 0.01) than body fat percentage (R^2=0.53, P-value ≤ 0.01). Second, others have proposed that BMI may not account for excess central fat, which is thought to more adversely affect CVD outcomes than peripheral fat. For example, in a meta-analysis of 15,923 (5,696 deaths) participants with CHD, higher central fat (measured by waist circumference, and waist-hip ratio) was associated with mortality even among individuals with normal BMI.([8]) Finally, because BMI doesn't discriminate between lean body mass and fat mass, some have proposed that low BMI categories, may reflect underlying low muscle mass and poor nutritional status commonly found in chronic disease patients.([9])

Notably, most observations of the obesity paradox have been made in patients with chronic diseases. In the Multi-ethnic Study of Atherosclerosis (MESA), a cohort of community dwelling participants free of clinically manifest CVD at enrollment, a positive association was reported between BMI and C2, a continuous blood pressure waveform characteristic asserted to represent small artery elasticity.([10]) Reduced C2, indicating greater small artery stiffness, was an independent predictor of incident hypertension and future CVD events.([10–12]). In this same cohort, compared to the 1st C2 quintile, participants in the 5th quintile were at a higher risk of future hypertension (Incident Relative Risk = 2.85, 95% CI: 1.95, 4.16).([12]) Also in this cohort, a per-standard deviation increase in C2 was associated with future CVD (HR=0.71, 95% CI: 0.61, 0.83).([10]) Given the paradoxical relationship between BMI and C2, we conducted analysis that aimed to determine the relationship between computed tomographic (CT) measures of abdominal body composition with C2 in an effort to garner additional mechanistic insights to factors contributing to the obesity paradox. We hypothesized that, independent of BMI and other CVD risk factors, total abdominal muscle

tissue (TAMT, rectus abdominus, oblique, psoas, and paraspinal muscles) area would be positively associated with C2. In contrast, we hypothesized that, despite the direct association of BMI with C2 reported previously, visceral adipose tissue (VAT, intra-abdominal fat) area would be inversely associated with C2.

Methods

Study sample

MESA is a multi-center, prospective cohort designed to investigate prevalence, correlates, and progression of subclinical (asymptomatic) atherosclerosis and their associations with incident clinical events. A detailed description of the study design, recruitment methods, examination components and data collections has been published.([13]) In brief, participants included 6,814 men and women (age 45-84) of Caucasian, Hispanic-, African-, and Chinese-American descent, free from clinically manifest CVD at baseline. Participants were recruited between July 2000 and August 2002 at 6 U.S field centers; New York, NY; Baltimore, MD; Winston-Salem, NC; St Paul, MN; Chicago IL; and Los Angeles, CA. Signed informed consent was obtained for all participants, and institutional review board approval was obtained for all participating institutions.

During follow up visits between August 2002 and September 2005, a randomly selected subsample of 2202 MESA participants were invited to participate in an ancillary study that aimed to determine the presence and extent of abdominal aortic calcium (AAC) using computed tomography. Of these, 2172 agreed to participate. Individuals were excluded if they were pre-menopausal, or had a recent (within 6 months) abdominal computed tomography (CT) scan. This left 1,970 participants who underwent abdominal CT scans. At follow-up visits between August 2002 and September 2005, abdominal images were obtained using multi-detector CT scanners at Columbia University, Wake Forest University, and University of Minnesota field centers (Sensation 64 [Siemens, Malvern, Pennsylvania] and GE Lightspeed [GE Healthcare, Waukesha, Wisconsin], Siemens S4 Volume Zoom, and Siemens Sensation 16, respectively). Electron-beam CT scanners were utilized at Northwestern University and University of California, Los Angeles (Imatron C-150, Imatron Inc., South San Francisco, California). Of the 1,970 participants, complete visualization of the visceral cavity was available in 1,960 individuals who represent the analytic sample for this study.

Central body composition measurements

Among individuals who provided abdominal CT scans, an ancillary study focused on abdominal body composition was conducted that interrogated 6 transverse cross-sectional slices at and just superior to L4/L5, L3/L4, L2/L3 for different measures of adipose tissue and skeletal muscle. Slices centered at L4/L5 junction were selected to quantify TAMT and VAT. Lean tissue was identified as being between 0-100 Hounsfield units (HU), and fat tissue was identified as -190 and -30 HU. TAMT was defined by adding the area for the bilateral rectus abdominus, oblique, psoas, and paraspinal lean tissue area, VAT was defined as fat within the visceral cavity. Within each area of interest (TAMT and VAT), we assigned the density value assigned to each pixel using the MIPAV 4.1.2 software (National Institutes

of Health, Bethesda, Maryland) as fat or lean tissue and then computed the total area (as cm^2). For all analyses, image analyses were performed by technologists blinded to participant clinical information.

C2 measurement

At the baseline visit between 2000 and 2002, arterial waveforms were recorded for the entire cohort using the HDI/PulseWave CR-2000 (Hypertension Diagnostics, Inc., Eagan, Minnesota). Details of this procedure have previously been published.([10]) In brief, a solid-state pressure transducer array (tonometer) was placed over the radial artery of the dominant arm to record the pulse contour. C2 is estimated by the device from the waveform modeled as a sinusoidal function dampened by a decaying exponential. An index estimated directly from the waveform is divided by systemic vascular resistance (SVR) to obtain C2. SVR is estimated as mean arterial blood pressure/cardiac output, and cardiac output is estimated from ejection time taken from the pulse waveform, heart rate, age, height, and weight.

Risk factor assessment

Participants were given standardized questionnaires at baseline, which were used to obtain information on demographics, medical history, and smoking history. A medication inventory was also performed, and medications were grouped based on use to treat high blood pressure, or elevated blood glucose. Blood pressure was measured 3 times in the seated position with a Dinamap model Pro 100 automated oscillometric sphygmomanometer after at least 5 minutes of rest. The average of the last 2 measurements was used. Standard measurements were taken for height and weight, and blood samples were obtained after a 12h fast for measurements of total cholesterol, high-density lipoprotein (HDL) cholesterol, and glucose.

Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Hypertension was defined as systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg, or current use of anti-hypertensive medication. Diabetes was defined as fasting plasma glucose ≥ 126 mg/dL, or use of hypoglycemic medications.

Statistical analysis

This is a period cross-sectional analysis of the 1960 participants with available CT scans of the visceral cavity obtained between 2002 and 2005 and C2 measured at the baseline examination in MESA approximately 18 and 36 months previously, respectively. Descriptive statistics for the study cohort were summarized by means (SD) and ranges for continuous variables, and frequencies for categorical variables. Spearman rank correlations of BMI, TAMT, and VAT with C2 were calculated. Mean C2 for quartiles of TAMT crossed with VAT were determined using ethnic and sex specific cut points. Multivariable stepwise backward deletion (p criterion < 0.1) linear regression was used to determine the independent association of BMI, TAMT, and VAT with C2. Model 1 adjusted for BMI, TAMT, and VAT; model 2 included model 1 plus demographics (age, gender, and ethnicity), and traditional CVD risk factors (pack years smoking, diabetes, hypertension, total and HDL cholesterol). Variance inflation factor (VIF) was used to test for collinearly among variables in both models. Interactions terms, BMI* gender, TAMT* gender, VAT* gender for

estimating C2 were also tested in Model 2 (separately). All analyses were conducted using PSAW Statistics 18, version 18.0.3 (SPSS, Inc., 2009 Chicago, IL). P-value ≤ 0.05 (two-sided) was considered significant for all analyses including interaction terms.

Results

Among 1960 participants, the mean age was 62 ± 9 years, 991 (51%) were men, 40% were European-, 21 % were African-, 26% were Hispanic- and 13% were Chinese-Americans (Table 1). The median [interquartile range] for C2 was 3.8 [2.4 - 5.9 ml/mmHg ×100], mean BMI was 28 ± 5 kg/m^2, mean TAMT was 123 ± 27 cm^2, and mean VAT was 148 ± 69 cm^2. C2 was correlated with BMI (r = 0.11, P-value < .01), TAMT (r = 0.35, P-value < .01) and VAT (r = 0.05, P-value < .05), while BIM was correlated with TAMT (r = 0.37, P-value <. 01) and VAT (r = 0.58, P-value < .01) (not shown). Figure 1 represents mean C2, small artery elasticity, by sex and ethnic specific quartiles of TAMT by VAT. Within each VAT quartile, we observed higher small artery compliance with increasing TAMT quartile.

Table 2 presents independent associations with C2. In multivariable analysis adjusted for age, gender, ethnicity, pack years smoking cigarettes, diabetes, hypertension, total and HDL cholesterol, higher BMI and more TAMT were significantly associated with higher C2, indicating more compliant small arteries. In contrast, more VAT was associated with lower C2. Interaction terms: BMI × gender (Standardized Beta = 0.01, P-value = 1.0); TAMT × gender (Standardized Beta = -0.01, P-value = 0.8); and VAT × gender (Standardized Beta = 0.18, P-value = 0.3) were not significant (not shown). Variance inflation factor among body composition measures in fully adjusted models were 1.8 for BMI, 2.5 for TMT, and 1.8 for VAT (also not shown).

Discussion

In this study of a multi-ethnic cohort of men and women free of clinically apparent CVD, we observed a “BMI-C2 paradox” in a healthy, community-living, older population. Specifically, and after adjustment for CVD risk factors, as well as VAT and TAMT, higher BMI was associated with increased C2, even though lower C2 is a subclinical marker for CVD events. More TAMT was positively associated with higher C2. In contrast to TAMT and BMI, more VAT was independently associated with lower C2.

Reduced C2 is a subclinical marker for future CVD.([10]) However, BMI, a common clinical measure of excess body fat, was positively associated with C2. We hypothesized that VAT, a more precise measure of excess fat, especially around the central organs, would be inversely associated with C2, while TAMT would be positively associated with C2. We postulated that these associations may support the primary theories explaining the “obesity paradox” which are that BMI does not discriminate well between lean tissue and excess fat, or between peripheral and more harmful central fat.

In support of our hypothesis, more VAT was independently associated with lower C2. Our results corroborated a prior study by Sutton-Tyrrell et al. which reported larger VAT area was associated with higher aortic pulse wave velocity (aPWV), another subclinical marker for CVD and marker of greater arterial stiffness.([14],[15]) Notably, in that study, investigators

did not account for the potential effects of lean muscle tissue on vascular health. Thus, we confirm this important finding, and extend the data by demonstrating that associations are also evident for small artery elasticity, and independent of the quantity of lean muscle in the abdomen.

Also supportive of our hypothesis, more TAMT was associated higher C2. Our findings are supported by a prior study of 648 participants, which found lean leg mass, was inversely associated with arterial stiffness.([16]) Our findings along with others suggest that lean muscle mass may be driving the positive association between BMI and favorable cardiovascular outcomes. If true, BMI may reflect lean muscle tissue generally thought to have favorable effect on vascular health. In a population of participants with known CAD, we have previously published results which showed that greater 24 hour urine creatinine excretion (a marker of muscle mass)was strongly protective for CVD ([17]), and these findings have been corroborated in the general population.([18]) In further support of our hypothesis, BMI misclassification of body fat status has been previously reported.([7],[19])

Strengths of our study include precise body composition measures from abdominal CT scans, a community-living sample, representation of multiple ethnicities and both genders, and a relatively large sample size. Also, prevalent CVD at baseline was an exclusion criterion, while prior studies have studied the “obesity paradox” in cohorts with prevalent CVD. However, our study also has important limitations. First, the non-simultaneity of the predictor and outcome measures makes it problematic, even as a period cross-sectional study and we cannot assign temporality. However, it is unlikely that these measures would change systematically over a short period of time. Second, C2 is a quotient of the pressure waveform index and SVR which includes height and weight, components of BMI. However, results of sensitivity analysis with C2*SVR were similar but weaker than those for C2. Finally, use of abdominal CT slices to quantify areas of fat and muscle tissue may not be the best representation of total body fat and muscle composition. Though, abdominal skeletal muscle quantified from a single magnetic resonance imaging at L4/L5 is a strong marker for whole body skeletal muscle.([20])

Conclusion

In community-living individuals without clinically apparent CVD, greater visceral adipose tissue was associated with stiffer small arteries, despite the fact that greater body mass index was not. At the same time, greater abdominal muscle was associated with more compliant small arteries. The paradoxical association of BMI with compliant arteries may be because it is not specific to the type of mass (adipose or lean muscle) or the location of fat (visceral vs. peripheral). These findings suggest that lean muscle, instead of excess body fat, may be driving the favorable association between BMI and arterial compliance. Studies using more refined methods of body composition such as CT for visceral adipose tissue, and simultaneous assessment of muscle and fat may provide new insights into seemingly disparate relationships of BMI with adverse health outcomes.

Acknowledgments

Funding: This study was supported by grant HL072403 and contract N01HC95159 to NO1HC95169. Dr. Forbang was supported by a minority supplement to N01HC95190.

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What is already known about this subject?

- Excess fat adversely affects cardiovascular health.
- Being overweight, determined by your body mass index, is associated with favorable cardiovascular outcomes.

What does this study add?

- In a healthy, community living population, computed tomography measures of visceral fat was independently associated with adverse vascular health after adjusting for both BMI and lean muscle tissue.
- Abdominal muscle tissue has favorable effects on vascular health in contrast to visceral fat

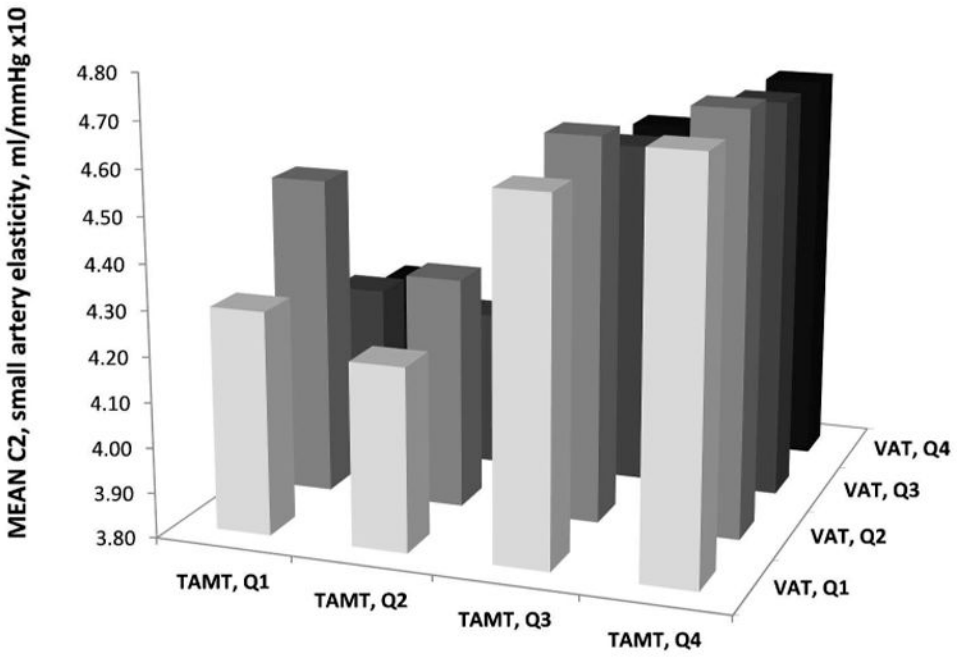

Figure 1. Mean C2, small artery elasticity by sex and ethnic specific quartiles of total abdominal muscle tissue (TAMT) and visceral adipose tissue (VAT)

Table 1

Cohort Characteristics

N = 1960	Mean ± SD [Range], N (%)
Age, years	62 ± 9 [44 - 84]
Male sex	991 (51%)
European Ethnicity	787 (40%)
African Ethnicity	410 (21%)
Hispanic Ethnicity	508 (26%)
Chinese Ethnicity	255 (13%)
Body mass index, BMI, kg/m^2	28 ± 5 [15 - 53]
Total abdominal muscle tissue, TAMT, cm^2	123 ± 27 [62 - 246]
Visceral adipose tissue, VAT, cm^2	148 ± 69 [16 - 469]
Pack Years Smoking	12 ± 21 [0 - 187]
Type 2 Diabetes Mellitus	77 (8%)
Hypertension	455 (47%)
Total Cholesterol, mg/dL	196 ± 34 [65 - 462]
HDL Cholesterol, mg/dL	51 ± 15 [21 - 121]
C2, small artery elasticity, ml/mmHg ×100	4.6 + 2.9 [1 - 20.74]

C2 = continuous blood pressure wave form characteristic asserted to represent small artery elasticity

Table 2

Multivariable Associations of Body Composition Measures and C2, Small Artery Elasticity

	Model 1			Model 2		
	Beta	**(95% CI)**	**P-value**	**Beta**	**(95% CI)**	**P-value**
Constant	0.02	(-0.79, 0.84)	0.96	8.50	(7.16, 9.89)	<.01
Body mass index, BMI, kg/m^2	0.02	(-0.03, 0.07)	.48	0.09	(0.04, 0.14)	<.01
Total abdominal muscle tissue, TAMT, cm^2	0.39	(0.34, 0.44)	<.01	0.12	(0.06, 0.18)	<.01
Visceral adipose tissue, VAT, cm^2	-0.14	(-0.19, -0.09)	<.01	-0.09	(-0.14, -0.04)	<.01
Age, years				-0.36	(-0.04, -0.31)	<.01
Male sex (vs. female)				0.21	(0.16, 0.27)	<.01
Chinese Ethnicity (vs. Caucasian)				-0.04	(-0.09, 0)	.05
African Ethnicity (vs. Caucasian)				-0.13	(-0.18, -0.09)	<.01
Hispanic Ethnicity (vs. Caucasian)				-0.07	(-0.11, -0.02)	<.01
Model summary						
R-squared		0.13			0.26	
Adjusted R-squared		0.13			0.26	
Standard Error of Estimate		2.72			2.50	
P-value		<.01			<.01	

C2 = continuous blood pressure wave form characteristic representing small artery elasticity. Model = Backward Deletion ($p < .1$ criterion), betas are per SD, 2-tailed $p < .05$ considered significant. Model 1 adjusted for BMI, TAMT, and VAT. Model 2 included model 1 plus age, gender, ethnicity, pack years smoking cigarettes, diabetes, hypertension, total and HDL cholesterol.

Obesity (Silver Spring). Author manuscript; available in PMC 2016 May 18.