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Which measures of adiposity predict subsequent left ventricular geometry? Evidence from the Bogalusa Heart Study

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Abstract

Background and aims—Left ventricular (LV) hypertrophy increases the risk of future cardiovascular events. The relationship between obesity in young adulthood and later LV geometry is unknown. We examined the association between long-term changes in measures of adiposity and subsequent LV geometry among 1073 young adults from the Bogalusa Heart Study.

Methods and results—Echocardiography-measured LV geometry was classified into normal ($N = 796$), concentric remodeling ($N = 124$), eccentric hypertrophy ($N = 99$), and concentric hypertrophy ($N = 54$) by integrating relative wall thickness and LV mass index. The mean age of our population was 38 years when the LV geometry was measured. Body mass index (BMI) increased by a mean of 4.9 kg/m² over a median of 20 years, waist circumference (WC) by 10.9 cm over 17 years, waist/hip ratio by 0.02 over 10 years, waist/height ratio by 0.06 over 17 years, abdominal height by 0.9 cm over 10 years, body fat (BF) percentage by 12.7% over 20 years, and Visceral Adiposity Index by 0.30 over 17 years. In polytomous logistic regression models corrected for multiple comparisons, participants with one-standard-deviation increases in BMI, WC, waist/height ratio, and BF had 2.00 (95% confidence interval (CI): 1.53–2.61), 1.33 (1.06–1.68), 1.35 (1.07–1.70), and 1.60 (1.26–2.03) times the risk of eccentric hypertrophy, respectively, after adjustment for demographic, lifestyle, metabolic risk factors, and follow-up time. Likewise, the rates of change in BMI, WC, waist/height ratio, and BF were associated with eccentric hypertrophy. There was no association with concentric remodeling or concentric hypertrophy.

Conclusions—Our findings suggest that increases in BMI, WC, waist/height ratio, and BF were strong predictors of eccentric hypertrophy in middle age.

Keywords

Left ventricular geometry; Obesity; Epidemiology

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Disclosures

None.

Introduction

Overweight and obesity affect approximately 68% of U.S. adults [1], and obesity is an independent risk factor for heart failure and other cardiovascular diseases [2,3]. The prevalence of overweight and obesity increases from young adulthood to middle age in both men and women [4]. It is now recognized that the development of heart failure and other cardiovascular events is often preceded by structural alteration of the left ventricle, for instance, left ventricular (LV) hypertrophy [5]. Physiological mechanisms underlying the relationship between obesity and LV hypertrophy may include excessive lipid accumulation within the myocardium [6]; increase in the hemodynamic load; and/or influences on proinflammatory cytokines, the extracellular matrix, and fibrosis [7].

Many epidemiologic studies [8–13], but not all [14], have suggested that obesity is more often related to eccentric LV hypertrophy, where the LV wall thickness increases normally in proportion to the increase in chamber radius among adults. By contrast, concentric LV hypertrophy, where the increase in LV wall thickness is greater than the chamber radius, is more often related to hypertension [15]. Both diet-induced weight loss and sustained weight loss after bariatric surgery may reverse these changes in LV geometry [16,17]. Yet it remains unclear which measures of adiposity and obesity are most strongly associated with subsequent changes in LV geometry [15]. Determining which measure is most strongly associated may help in the early identification and prevention of disease.

Longitudinal data on the relationship between changes in adiposity and subsequent LV geometry are very limited. In a study of 132 young men and women, an increase in body mass index (BMI) of >5.5 kg/m² between 13 and 27 years of age was associated with a greater increase in the LV mass index than BMI change <5.5 kg/m² [18]. Likewise, Lorber and colleagues examined the relationship between the rate (or velocity) of BMI change and LV mass index and relative wall thickness (RWT), over 10 years among 1358 generally healthy adults. They reported that the rate of BMI change was not consistently associated with LV geometry [19]. However, neither study accounted for potential confounding factors such as blood pressure, lipids, and physical activity in their analyses [18,19]. Moreover, these studies used only BMI as a measure of adiposity. Very few studies have examined other measures of adiposity, which are simple to collect and may help improve early prevention of LV hypertrophy and geometry changes [20,21]. Therefore, we took advantage of the multiple measures of adiposity collected throughout young adulthood in the Bogalusa Heart Study, a biracial community-based cohort, to prospectively examine the association between changes, in both magnitude and velocity, in measures of adiposity and subsequent LV geometry measured in middle age.

Methods

Study population

The Bogalusa Heart Study is a long-term prospective cohort study of a semirural biracial (65% white and 35% black) community in Bogalusa, Louisiana. The population and study design of the Bogalusa Heart Study have been previously described [22,23]. Beginning in 1973, a variety of adiposity measures were collected on individuals in this cohort throughout

young adulthood. Participants included in this analysis are individuals who had measures of adiposity at least two times in young adulthood and underwent LV examination by echocardiography during follow-up in 2000–2005. Participants with coronary heart disease, myocardial infarction, and congestive heart failure at baseline were excluded ($N = 38$). The final sample included 1073 individuals. All protocols were approved by the Institutional Review Board of Tulane University and written informed consent was obtained from all participants.

Measurement

Trained examiners collected all data using standardized protocols of the Bogalusa Heart Study [23,24]. At each examination, anthropometric indices were measured in duplicate or triplicate and then averaged. Height and weight were measured twice to the nearest 0.1 cm and nearest 0.1 kg, respectively. Averaged values were used to calculate BMI as weight in kilograms divided by height in meters squared. Waist circumferences were measured twice from the horizontal plane at 1 cm above the navel. Hip circumferences were measured twice to the nearest centimeter (maximum posterior extension between the iliac crest and buttocks). The waist/hip ratio was calculated by dividing averaged values for waist circumference by hip circumference. The waist/height ratio was calculated by dividing waist circumference by body height. Abdominal height, defined as the thickness of the abdomen at waist level, was measured with a portable sliding-beam abdominal caliper while the subject lay supine on an examining table (Holtain-Kahn Abdominal Caliper; Holtain Ltd., Dyfed, Wales, UK) [25]. Body fat as a percentage of body weight was calculated based on triceps and subscapular skinfold thickness using Slaughter's body fat equation [26]. Visceral Adiposity Index (VAI) was a sex-specific index based on BMI and waist circumference, corrected for levels of high-density lipoprotein cholesterol and triglycerides [27].

Two-dimensional guided M-mode echocardiography was performed to assess LV dimensions with 2.25- and 3.5-MHz transducers, according to the recommendations of the American Society of Echocardiography [28]. Parasternal long- and short-axis views were measured in duplicate and then averaged. LV internal dimension, ventricular septal thickness, and posterior wall thickness were measured at end diastole. Images were recorded and repeated observations were obtained by trained technicians on a randomized sample of 6% of the participants 10–12 days apart. Coefficients of variation for inter- and intra-reader variability for all measures of cardiac anatomy were <10% in our sample. All echocardiograms were digitized and measured on Tomtec/Freeland Cardiology Workstation digitizing systems (Tomtec/Freeland Systems, Broomfield, CO, USA).

To evaluate the geometry of the left ventricle, both LV mass index and RWT were collected. The LV mass was calculated as $0.8 * (1.04 * ((LV \text{ internal dimension} + \text{posterior wall thickness} + \text{ventricular septal thickness}) [3] - (LV \text{ internal dimension}) [3])) + 0.6 \text{ g}$ using the formula of Devereux and normalized by indexing to $\text{height}^{2.7}$ [29]. The LV mass index was categorized using gender-specific normal values of $50 \text{ g/m}^{2.7}$ for men and $47 \text{ g/m}^{2.7}$ for women [30]. The RWT of the left ventricle was calculated as $(\text{ventricular septal thickness} + \text{posterior wall thickness}) / LV \text{ internal dimension}$ [31]. A partition value of 0.42 for RWT was used for both men and women to represent the 95th percentile value in normal subjects.

Based on the values of the LV mass index and RWT, four LV geometric patterns were defined as follows: (1) normal geometry was present when the LV mass index and RWT were within normal limits; (2) normal LV mass index and increased RWT was classified as concentric remodeling; (3) increased LV mass index, but normal RWT, was classified as eccentric LV hypertrophy; and (4) increases in both LV mass index and RWT was classified as concentric LV hypertrophy [32].

At baseline, self-reported current smoking status (yes/no) was defined as the use of cigarettes within the past year. Self-reported regular alcohol drinking (yes/no) was defined as drinking alcohol beverages more than twice a week. Self-reported leisure-time physical activity was assessed on a scale of 1 (very inactive) to 5 (very active) based on a validated questionnaire [33,34]. Venipuncture was performed after a 12-h fast. Serum cholesterol and triglyceride levels were assayed using an enzymatic procedure as part of a lipid panel (Laboratory Corporation of America, Burlington, NC, USA). Fasting plasma glucose levels were measured as part of a multiple chemistry profile (SMA20, Laboratory Corporation of America, Burlington, NC, USA) by a glucose oxidase method. Serum uric acid levels were determined using standard uricase methods. Replicate blood pressure measurements were obtained by trained observers on the right arm of the participants in a relaxed, sitting position. Arm measurements, length and circumference, were carried out during the examination to ensure proper cuff size. Systolic and diastolic blood pressure levels were recorded as the first and fifth Korotkoff phases using mercury sphygmomanometers. Blood pressure levels were reported as the mean of six fifth-phase replicate readings, taken by each of two randomly assigned observers. Hypertension was defined as a systolic blood pressure ≥ 140 mmHg or a diastolic blood pressure ≥ 90 mmHg or a use of antihypertensive medication.

Statistical analysis

Descriptive statistics were calculated for all study variables. Changes in measures of adiposity and their association with LV geometry were assessed in two ways. First, the magnitude of change was computed by subtracting the earliest value from the latest value for each measure of adiposity. Second, the rate (velocity) of change was obtained from the slope of a separate regression line fitted to the adiposity measures over the length of follow-up for each individual. For both analyses, we performed polytomous logistic regression analyses, with the outcomes defined as the four categories of LV geometry: normal, concentric remodeling, eccentric hypertrophy, and concentric hypertrophy. Given the approximately normal distributions of both the magnitude and the rate of increase of all measures of adiposity, we obtained the odds ratio for the associations as a 1-SD increase in each measure of adiposity. Models were adjusted for age, race, gender, time-dependent covariates including systolic blood pressure, diastolic blood pressure, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides (log-transformed), fasting blood glucose, uric acid, current smoking, regular alcohol drinking and leisure-time physical activity, and follow-up time. As VAI was corrected for high-density lipoprotein cholesterol and triglycerides, the models were not adjusted for these two covariates. Furthermore, gender and race differences were tested for interaction. When there was significant interaction, the results were present separately. For all of the above analyses, we

used the false discovery rate method to correct for the multiple comparisons [35]. Analyses were performed using SAS 9.3 for Windows (SAS Institute, Cary, NC, USA).

Results

Of 1073 adults, 796 (74.0%) had normal LV geometry, 124 (11.8%) had concentric remodeling, 99 (9.2%) had eccentric hypertrophy, and 54 (5.0%) had concentric hypertrophy. Among African Americans, the prevalence of concentric remodeling, eccentric hypertrophy, and concentric hypertrophy were 9.9%, 14.5%, and 8.7%, respectively. Among Caucasians, the prevalence of concentric remodeling, eccentric hypertrophy, and concentric hypertrophy were 12.3%, 6.9%, and 3.4%, respectively. The mean (standard deviation (SD)) age of the population was 22 (3.6) years at their earliest measurement of adiposity and 38 (4.8) years when LV geometry was measured. Table 1 shows population characteristics according to LV geometry categories as measured in 2005 (middle age). Compared to the participants with normal LV geometry, those who had either eccentric or concentric LV hypertrophy were more likely to be African American and hypertensive, and these participants had higher levels of systolic blood pressure, diastolic blood pressure, and fasting blood glucose. Participants with eccentric hypertrophy had significantly lower levels of high-density lipoprotein cholesterol and significantly higher levels of uric acid, and they were more often women, compared to those with normal LV geometry. Meanwhile, those with eccentric or concentric hypertrophy had significantly higher measures of adiposity than those with normal LV geometry, with the exception of the waist/hip ratio and VAI.

The mean (SD) of the earliest and latest measures of adiposity, absolute changes in measures of adiposity, and median (interquartile range (IQR)) of follow-up years are shown in Table 2. Increases were seen in all measures of adiposity. The time between the earliest and latest measures was longer on average for BMI and body fat percentage, and shorter for waist/hip ratio and abdominal height.

Multivariable adjusted odds ratios (95% confidence intervals) for the associations between a 1-SD absolute change in each measure of adiposity and eccentric and concentric LV hypertrophy are shown in Table 3. One-SD increases in BMI, waist circumference, waist/height ratio, and body fat percentage were significantly associated with a higher risk of eccentric LV hypertrophy. Likewise, we observed significant associations for 1-SD increases in the rate (velocity) of change in BMI, waist circumference, waist/height ratio, and body fat percentage with eccentric LV hypertrophy (Table 4). By contrast, a 1-SD change, in either magnitude or rate, in waist/hip ratio, abdominal height, or VAI was not significantly associated with the presence of eccentric LV hypertrophy. None of the measures of adiposity was significantly associated with the presence of concentric LV hypertrophy after correction for multiple comparisons. One-SD increases in magnitude and rate of changes in BMI, waist circumference, waist/height ratio, and body fat percentage were significantly associated with higher LV mass index, LV diastolic posterior wall thickness, LV end-diastolic diameter, and interventricular septal thickness (Appendix Tables 1 and 2). We tested for interactions by race and gender. There was no significant interaction between race or gender and any measure of adiposity in the association with LV hypertrophy.

Discussion

In this sample of 1073 community-based young adults from the Bogalusa Heart Study, long-term increases, in both magnitude and rate, in BMI, waist circumference, waist/height ratio, and body fat percentage were associated with subsequent eccentric LV hypertrophy. To our knowledge, this is the first study to examine and compare the associations between changes in various measures of adiposity and LV geometry. These associations were independent of age, race, gender, blood pressure, serum lipids, fasting blood glucose, uric acid, current smoking status, regular alcohol drinking, and leisure-time physical activity levels. By contrast, changes in waist/hip ratio, abdominal height, or VAI were not significantly associated with LV hypertrophy. Our findings that greater increases in some measures of adiposity and faster rates of change are strongly related to eccentric LV hypertrophy are unique in that few studies on weight changes have examined this question in a cohort with longitudinal data, and none of these included multivariable adjusted models. The few previous studies that examined this topic mostly used a single measure of adiposity, such as BMI or waist circumference [9,11,13]. Our study further expands current knowledge by assessing the magnitude and velocity of changes in a variety of measures of adiposity during young adulthood.

The mechanisms by which changes in adiposity affect LV geometry are not entirely clear [2], and the current evidence is limited to pathophysiological mechanisms linking obesity status to LV geometry. One major mechanism by which obesity may influence LV geometry and the subsequent development of eccentric hypertrophy is through the enlargement of the left ventricle [36]. This process may be influenced by increased hemodynamic load [37], decreased LV myocardial contractility, and inflammatory activity that promotes cardiac remodeling [38]. In comparison with eccentric LV hypertrophy, concentric LV hypertrophy is most often associated with increased pressures primarily from hypertension rather than obesity [39]. As it is well known that obesity is highly correlated with hypertension, untangling the causal effects of obesity from hypertension on LV geometry is often difficult. In this study, we adjusted for systolic and diastolic blood pressure to examine the association of changes in adiposity with LV geometry independent of blood pressure.

It is well known that BMI is a validated measure of adiposity and has been widely used in many epidemiologic studies. Only a few studies have evaluated the long-term change in BMI as a predictor of LV geometry [19]. One study reported the correlation of rates (velocity) of change in BMI over a 10-year interval with LV geometry among adults [19]. However, unlike our study, the latter study failed to control for important confounders such as blood lipids, smoking, and physical activity. The present study assessed both the magnitude and velocity of long-term changes in BMI, and it extended the analyses to other measures of adiposity.

Our data showed that increases in waist circumference or waist/height ratio were significantly associated with the presence of eccentric LV hypertrophy. By contrast, we did not observe a significant association using changes in waist/hip ratio, abdominal height, or VAI. Waist circumference is a validated measurement of abdominal obesity, and it has been highly correlated with total abdominal fat (correlation coefficients: 0.87–0.93) and

abdominal visceral fat (correlation coefficients: 0.84–0.93) by performing computed tomographic (CT) scans [40]. The waist/height ratio, used as a measure of central adiposity adjusting for body size, is simple and conceptually appealing, although there is no evidence that it is better than waist circumference in predicting morbidity and mortality [41]. The interpretation of results with regard to waist/hip ratio or abdominal height may be hindered by a relatively shorter duration of follow-up in our study. In addition, the fact that waist/hip ratio can reflect both visceral fat mass and/or gluteofemoral muscle mass adds complexity to its interpretation. Indeed, waist/hip ratio has been shown to be less predictive of total abdominal fat or abdominal visceral fat than waist circumference [40]. The VAI has been used to represent adipose tissue function and distribution. However, its application to large population-based studies, especially those among persons who are obese and/or have dyslipidemia, has been cautioned because of low reliability due to extremes of triglycerides, BMI, and/or waist circumference [42]. In our study, almost half of the population were obese, and the mean BMI was 29.3 kg/m².

Changes in body fat percentage were also associated with eccentric hypertrophy; however, these results should be interpreted with caution. Thus far, there has been no consensus on which equations for the calculation of body fat percentage should be used in epidemiological studies [43,44]. The formula for the conversion of skinfold measures to body fat percentage that has been conventionally used is particularly suitable for children and young adults, with inconclusive evidence of its predictive value in middle-aged adults [26]. In addition, skinfold measurement is prone to interobserver variations [45].

There is no consensus on the criteria for normalizing LV chamber measurements to body size parameters such as height or body surface area [46–48]. In the setting of obesity, evidence from population-based prospective cohort studies has suggested that LV hypertrophy should be routinely indexed to height^{2.7}, as indexing to body surface area, another widely used parameter, underestimates the prevalence of LV hypertrophy among obese and overweight persons [49]. Because our purpose is to identify markers of adiposity that may assist in the early identification of LV hypertrophy, and a substantial proportion of our population is obese, overweight, or hypertensive, we chose to index LV hypertrophy to height^{2.7} to avoid misclassification in LV geometry and specifically underestimation among the obese and overweight, which has been demonstrated with indexing to body surface area [46].

The conclusions from this study are subject to some limitations. This is an observational study, so residual confounding cannot be excluded. The present study incorporated data from multiple decades, and so it may be affected by factors that vary over long periods. However, we controlled for multiple time-dependent variables in the data analyses. Our study also has several strengths. First, the Bogalusa Heart Study has been using standardized protocols and well-established quality control procedures for data collection since its inception in 1973 [50,51]. Second, this study includes a number of repeated examinations for each participant, which enables us to longitudinally assess the magnitude and velocity of the changes in measures of adiposity and their relationship with cardiovascular disorders such as LV hypertrophy. Finally, this study was conducted in a biracial community-dwelling

population, enhancing the generalizability of the results; thus, it has important public health implications for future cardiovascular disease prevention strategies.

In summary, data from this large, population-based cohort study suggest that both magnitude and velocity of increases in BMI, waist circumference, waist/height ratio, and body fat percentage in young adulthood are strong predictors of eccentric LV hypertrophy in middle age among a community-based population. These findings suggest that persons with larger and/or faster increases in measures of adiposity from young adulthood to middle age may benefit from closer monitoring and targeted prevention strategies to avoid cardiovascular sequelae. Primordial and early prevention strategies should focus on minimizing weight gain in young adulthood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.numecd.2014.11.001>.

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Table 1

Population characteristics according to the left ventricular geometry.^a

Variable	Left ventricular hypertrophy		Concentric remodeling	Normal geometry	P value ^b
	Eccentric hypertrophy	Concentric hypertrophy			
No. of participants	99	54	124	796	...
Age, year	39.0 (4.1)	39.2 (3.9)	38.3 (4.7)	37.9 (4.5)	0.03
Female, %	52.5	79.6 ^c	40.3 ^c	55.7	<0.001
Blacks, %	48.5 ^c	53.7 ^c	26.6	27.9	<0.001
Systolic blood pressure, mmHg	128.0 (17.8) ^c	131.8 (20.0) ^c	115.6 (12.8)	114.5 (12.9)	<0.001
Diastolic blood pressure, mmHg	85.4 (1.6) ^c	87.3 (13.0) ^c	78.1 (8.8)	77.2 (9.6)	<0.001
LDL cholesterol, mg/dl	122.8 (39.0)	130.5 (33.3)	122.9 (33.9)	124.2 (33.8)	0.54
HDL cholesterol, mg/dl	45.6 (12.5) ^c	47.9 (12.6)	46.4 (12.4)	50.2 (14.0)	0.01
Triglyceride, mg/dl	135.41 (93.3)	143.4 (87.4)	139.7 (111.0)	129.7 (102.4)	0.60
Fasting glucose, mg/dl	96.9 (3.1) ^c	107.3 (48.0) ^c	88.9 (22.3)	88.4 (24.9)	<0.001
Hypertension, %	48.5 ^c	59.3 ^c	27.4	21.4	<0.001
Current smoking, %	29.3	37.0	29.0	30.3	0.73
Regular alcohol drinking, %	39.1	27.0	43.4	38.6	0.38
Physically inactive, % ^d	72.4	77.5	69.4	67.7	0.45
Uric acid, mg/dL	5.58 (1.58) ^c	5.21 (1.54)	5.50 (1.57) ^c	5.06 (1.53)	<0.001
Measures of adiposity					
BMI, kg/m ²	36.9 (7.5) ^c	36.6 (8.4) ^c	29.0 (5.9)	27.9 (5.9)	<0.001
Waist circumference, cm	107.9 (17.5) ^c	104.0 (16.7) ^c	93.5 (13.0)	90.1 (14.7)	<0.001
Waist/hip ratio	0.89 (0.10) ^c	0.87 (0.09)	0.87 (0.08)	0.85 (0.08)	<0.001
Waist/height ratio	0.64 (0.09) ^c	0.63 (0.09) ^c	0.54 (0.07)	0.53 (0.08)	<0.001
Abdominal height, cm	26.4 (4.7) ^c	25.9 (4.6) ^c	22.8 (4.1)	21.6 (4.2)	<0.001
Body fat, %	40.4 (12.0) ^c	38.9 (13.0) ^c	33.6 (13.9)	30.2 (12.3)	<0.001
Visceral Adiposity Index	2.13 (1.76)	2.10 (1.97)	2.28 (1.87)	1.91 (1.71)	0.12
Obesity status, %					<0.001

Variable	Left ventricular hypertrophy		Concentric remodeling		Normal geometry	P value ^b
	Eccentric hypertrophy	Concentric hypertrophy				
Normal weight (BMI < 25 kg/m ²)	5.1	7.4	28.3		34.4	
Overweight (BMI 25–30 kg/m ²)	14.1	9.3	33.3		34.7	
Obesity (BMI > 30 kg/m ²)	80.8	83.3	38.4		30.9	

BMI: body mass index; HDL: high-density lipoprotein; LDL: low-density lipoprotein.

^a All data are mean (SD) or percentage of participants; ellipses indicate not applicable.

^b From one-way ANOVA test for continuous variables and from chi-squared test for categorical variables.

^c $P < 0.05$ (using false discovery rate method), compared to normal geometry.

^d Self-reported leisure-time physical activity, scaled 1–2 (inactive) or 3–5 (active).

Table 2

Measures of adiposity over follow-up in 1073 participants in the Bogalusa Heart Study.

Variables	Mean of earliest measures (SD)	Mean of latest measures (SD)	Mean absolute changes in adiposity measures (SD)	Median follow-up in years (IQR)
Body mass index, kg/m ²	24.5 (5.6)	29.3 (7.0)	4.9 (4.9)	20 (6)
Waist circumference, cm	82.1 (14.1)	92.8 (16.0)	10.9 (11.5)	17 (10)
Waist/hip ratio	0.83 (0.18)	0.85 (0.08)	0.02 (0.17)	10 (0)
Waist/height ratio	0.49 (0.08)	0.55 (0.09)	0.06 (0.07)	17 (10)
Abdominal height, cm	21.5 (4.0)	22.5 (4.5)	0.9 (2.9)	10 (7)
Body fat, %	18.9 (11.4)	31.6 (12.8)	12.7 (12.3)	20 (6)
Visceral Adiposity Index	1.70 (1.66)	1.98 (1.75)	0.30 (1.49)	17 (10)

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Table 3

Multivariate adjusted^a odds ratio (95% confidence interval) of abnormal left ventricular geometry by one-standard-deviation changes in measures of adiposity.

	Left ventricular hypertrophy			
	Eccentric		Concentric	
	Odds ratio (95% confidence interval)	<i>P</i> value ^b	Odds ratio (95% confidence interval)	<i>P</i> value ^b
Body mass index, kg/m ^{2a}	2.00 (1.53, 2.61)	<0.001	1.45 (1.02, 2.03)	0.08
Waist circumference, cm	1.33 (1.06, 1.68)	0.03	1.34 (0.93, 1.91)	0.13
Waist/hip ratio	1.00 (0.76, 1.32)	0.99	0.98 (0.71, 1.35)	0.96
Waist/height ratio	1.35 (1.07, 1.70)	0.03	1.38 (0.99, 1.92)	0.10
Abdominal height, cm	1.11 (0.85, 1.44)	0.68	0.96 (0.70, 1.31)	0.96
Body fat, %	1.60 (1.26, 2.03)	<0.001	1.47 (1.07, 2.04)	0.05
Visceral Adiposity Index	1.02 (0.77, 1.36)	0.96	0.95 (0.57, 1.59)	0.96

^a Adjusted for age, race, gender, follow-up time, and the following time-dependent covariates: systolic blood pressure, diastolic blood pressure, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglyceride, fasting blood glucose, smoking status, regular alcohol drinking, leisure-time physical activity, and serum uric acid; models of Visceral Adiposity Index were not adjusted for high-density lipoprotein cholesterol or triglyceride.

^b $P < 0.05$ (using false discovery rate method), comparing to normal geometry.

Table 4

Multivariate adjusted^a odds ratio (95% confidence interval) of abnormal left ventricular (LV) geometry by rate (velocity) of one-standard-deviation changes in measures of adiposity.

	LV hypertrophy			
	Eccentric		Concentric	
	Odds ratio (95% confidence interval)	<i>P</i> value ^b	Odds ratio (95% confidence interval)	<i>P</i> value ^b
Body mass index, kg/m ² per year	1.74 (1.32, 2.29)	<0.001	1.29 (0.93, 1.80)	0.22
Waist circumference, cm per year	1.97 (1.40, 2.77)	<0.001	1.44 (0.91, 2.27)	0.23
Waist/hip ratio, per year	1.02 (0.73, 1.45)	0.96	0.99 (0.68, 1.45)	0.96
Waist/height ratio, per year	1.99 (1.41, 2.82)	<0.001	1.46 (0.92, 2.30)	0.23
Abdominal height, cm per year	0.98 (0.79, 1.21)	0.95	0.98 (0.72, 1.33)	0.95
Body fat, % per year	1.42 (1.10, 1.81)	0.02	1.56 (1.07, 2.26)	0.07
Visceral Adiposity Index, per year	1.30 (0.76, 2.22)	0.52	0.98 (0.75, 1.28)	0.96

^a Adjusted for age, race, gender, follow-up time, and the following time-dependent covariates: systolic blood pressure, diastolic blood pressure, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglyceride, fasting blood glucose, smoking status, regular alcohol drinking, leisure-time physical activity, and serum uric acid; models of Visceral Adiposity Index were not adjusted for high-density lipoprotein cholesterol or triglyceride.

^b *P* < 0.05 (using false discovery rate method), comparing to normal geometry.