ORIGINAL ARTICLE

WILEY Obesity Science and Practice

Association of fitness and body fatness with left ventricular mass: The Heart Health Study

Renee J. Rogers[1] | Erik B. Schelbert[2] | Wei Lang[3] | Yaron Fridman[4] | Nalingna Yuan[1] | John M. Jakicic[1]

[1] Department of Health and Physical Activity, Physical Activity and Weight Management Research Center, Healthy Lifestyle Institute, University of Pittsburgh, Pittsburgh, Pennsylvania

[2] Department of Medicine, School of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania

[3] Department of Biostatistical Sciences, Wake Forest University School of Medicine, North Carolina

[4] Mission Health, Asheville Cardiology Associates, North Carolina

Correspondence

John M. Jakicic, Department of Health and Physical Activity, Physical Activity and Weight Management Research Center, Healthy Lifestyle Institute, University of Pittsburgh, 32 Oak Hill Court, Pittsburgh, PA 15261. Email: jjakicic@pitt.edu

Summary

Objective: Left ventricular mass (LVM) is a clinical prognostic indicator of cardiovascular disease. Left ventricular mass is associated with body size (body mass index [BMI], weight, and body surface area [BSA]). This study examined if the association between body size (weight, BMI, and BSA) and LVM is influenced by body composition and cardiorespiratory fitness in adults who are overweight or obese.

Methods: This study included cross-sectional baseline data from a randomized clinical trial. Participants included 379 adults (age, 45.6 ± 7.9 y) who were overweight or obese (BMI, 32.4 ± 3.8 kg·m^{-2}). Measures included weight, height, BMI, BSA, body composition, cardiorespiratory fitness, and LVM by cardiac magnetic resonance imaging (CMR).

Results: Left ventricular mass was positively associated with weight, BMI, BSA, and fitness ($P < .0001$) and inversely associated with percent body fat ($P < .0001$). Stepwise multiple regression models showed that body fatness was inversely associated and cardiorespiratory fitness was positively associated with LVM even after considering weight, BMI, or BSA in the analyses.

Conclusions: These cross-sectional findings support that in adults who are overweight or obese but otherwise relatively healthy, LVM is associated with both body composition and cardiorespiratory fitness. This may indicate the need to reduce body fatness and improve fitness for patients with obesity to enhance cardiovascular structure and function.

KEYWORDS

Cardiac magnetic resonance imaging (CMR), exercise, fitness, obesity

1 | INTRODUCTION

In adults over the age of 20 years, the prevalence of obesity (body mass index [BMI] ≥ 30 kg·m^{-2}) in the United States has increased from 30.5% in 1999-2000 to 39.6% in 2016 on the basis of National Health and Nutrition Examination Survey data.[1] These data also show that the prevalence of obesity was 37.9% for men and 41.1% for women. Excess body weight is of public health concern because of its association with chronic diseases, with a major concern being the association with cardiovascular disease (CVD).[2,3]

Left ventricular mass (LVM) of the heart is a clinically important prognostic indicator of CVD, with early data reported from the Framingham Heart Study supporting this conclusion.[4,5] Larger body size, as measured BMI, is associated with greater LVM measured with echocardiography.[6] Echocardiography employs assumptions to estimate LVM, and cardiac magnetic resonance imaging (CMR) is considered by some as the clinical gold standard for LVM measurement. Studies using CMR have shown larger LVM at higher levels of BMI.[7] Given that BMI is a clinical indicator of overweight or obesity, these findings may suggest that a pathway by which overweight and obesity contribute to CVD is through a maladaptive larger LVM. However, many of these studies have not included measures of body composition to quantify body fatness, which may be a more sensitive measure of obesity than BMI alone, and may further contribute to the risk of CVD beyond what is observed with BMI or other measures of body size alone (eg, body weight and body surface area [BSA]).

Studies have shown that cardiorespiratory fitness is an important physiological indicator of CVD risk.[8-10] Additional evidence suggests that even in the presence of overweight and obesity, higher levels of cardiorespiratory fitness may provide protection from CVD.[11-17] However, studies that have examined the association between indices of body size (eg, BMI, body weight, and BSA) and LVM have not considered the contribution of cardiovascular fitness as an important factor that may also contribute to LVM. Athletes have greater LVM than less active individuals,[18] and individuals who are more active have a lower CVD risk,[19-23] possibly suggesting that the higher LVM found in more active and fit individuals represents a favourable adaptive response. The CARDIA Study, a prospective observational study, has examined cardiorespiratory fitness as a predictor of LVM while also considering other lifestyle factors such as BMI, and it showed that LVM measured by echocardiography was not associated with cardiorespiratory fitness.[24] However, there are few additional studies that have examined these relationships, with few examining the combined contributions of body composition and fitness on LVM in adults clinically defined as overweight or obese.

Baseline assessments from the Heart Health Study included measures of LVM using CMR, body composition using dual-energy X-ray absorptiometry (DXA), and cardiorespiratory fitness using a graded exercise test in adults who are overweight or obese. With the use of these data, analyses were conducted to examine whether the association between body size (weight, BMI, and BSA) and LVM is influenced by both body composition and cardiorespiratory fitness in adults who are overweight or obese.

2 | MATERIALS AND METHODS

2.1 | Subjects

Subjects (N = 383) were recruited for participation into the Heart Health Study, which is a randomized clinical trial to examine the effects of weight loss through diet and physical activity on measures of CMR and other CVD risk factors. Data presented in this manuscript reflect measures assessed at baseline and prior to engagement in the weight loss intervention. Subjects were considered eligible if their age was 18 to 55 years and BMI was 25 to <40 kg·m^{-2} at the baseline assessment when eligibility was determined. Ineligibility criteria included (a) self-reporting ≥ 60 min/wk of structured moderate-to-vigorous intensity physical activity; (b) weight loss of ≥5% within the prior 6 months or a history of bariatric surgery; (c) history of cardiometabolic disease, diabetes mellitus, or cancer; (d) taking medication that could affect heart rate or blood pressure; (e) taking medication that could influence body weight; (f) treatment for psychological conditions that included medication or counselling; (g) currently pregnant, pregnant within the prior 6 months, or planning a pregnancy within the next 12 months; (h) planning on geographical relocation outside of the region within 12 months; (i) inability to comply with the components of the interventions; or (j) had a contraindication that would prohibit magnetic resonance imaging (MRI) scanning. Participants provided written informed consent and medical clearance from their physician prior to engaging in this study. All procedures were approved by the University of Pittsburgh's Institutional Review Board and Human Research Protection Office.

2.2 | Measures

2.2.1 | Descriptive characteristics

Information on gender, race, and ethnicity was collected via questionnaire. Age was confirmed from the birth date contained on a government issued identification card (eg, driver's license and passport).

2.2.2 | Weight, height, BMI, and BSA

Weight and height were assessed with the subject clothed in a lightweight hospital gown with shoes removed. Weight was assessed using a calibrated digital scale to the nearest 0.1 kg with duplicate measures differing by ≤0.5 kg. Height was assessed using a wall-mounted stadiometer to the nearest 0.1 cm with duplicate measures differing by ≤0.5 cm. Weight and height were used to compute BMI (kg·m^{-2}). Body surface area was computed as BSA = 0.0235 × Height in cm$^{0.42246}$ × Weight in kg$^{0.51456}$.

2.2.3 | CMR measures

Participants were scanned by dedicated CMR technologists on a 1.5-tesla Siemens Magnetom Espree scanner (Erlangen, Germany) with a 32-channel phased array cardiovascular coil. Standard long axis cines were acquired in the two-, three-, and four-chamber orientations. Measurements without geometric assumptions from manual end-diastolic and end-systolic endocardial and epicardial traces of short axis stacks of cines acquired with 6-mm slice thickness using commercially available software were taken for LVM. There was no inter-slice gap to improve spatial resolution in the long axis direction and accurately measure left ventricular myocardium at the base of the heart. Papillary muscles were excluded from LVM measures. Typical

parameters were as follows: field of view 380 × 340 cm, matrix 256 × 144, 1.5 × 2.4-mm pixels, flip angle (FA) 50°, temporal resolution 30 to 45 milliseconds, 30 frames per cardiac cycle, TR/TE = 2.9/1.2 milliseconds, pixel bandwidth = 930 Hz, and parallel imaging factor 2 or 3 (GRAPPA).

2.2.4 | Body composition

Total body composition (fat mass, lean mass, percent body fat, bone mineral content, and bone mineral density) was measured from a total body scan using DXA (GE Lunar iDXA, Madison, WI). Girth measures of the waist and hip were taken using a Gulick anthropometric measuring tape, with measures taken to the nearest 0.1 cm with duplicate measures differing by ≤1.0 cm. Waist circumference was measured horizontally at both the umbilicus and the iliac crest. Hip circumference was measured at the widest visual protrusion of the buttocks.

2.2.5 | Cardiorespiratory fitness

Submaximal cardiorespiratory fitness was assessed with a submaximal graded exercise test performed on a motorized treadmill as previously described.[25] The treadmill speed was maintained at 80.4 m·min^{-1}. The incline of the treadmill was initiated at 0% and increased by 1% at 1-minute intervals. The test was terminated when the participant first achieved or exceeded 85% of their age-predicted maximal heart rate.[25] Oxygen consumption (L·min^{-1} and mL·kg^{-1}·min^{-1}) was measured with indirect calorimetry using a metabolic cart (Carefusion Vmax Encore, Yorba Linda, CA), with submaximal fitness represented by oxygen consumption achieved during the final 20 seconds prior to test termination.

2.2.6 | Resting blood pressure, mean arterial pressure, and heart rate

Following initial circumference measures to determine appropriate cuff size, resting blood pressure and heart rate measurements were obtained using an automated blood pressure cuff (DINAMAP V100, GE Medical System Technologies; Milwaukee, WI) following a 5-minute seated rest period. Blood pressure was expressed as the mean of duplicate measures, with systolic blood pressure (SBP) measures differing by ≤10 mmHg and diastolic blood pressure (DBP) measures differing by ≤6 mmHg. If these criteria were not achieved, a third measurement of SBP and DBP was taken. Mean arterial pressure (MAP) was computed from the mean values of SBP and DBP using the following formula: MAP = DBP + ([SBP – DBP]/3). The mean of the heart rate measures obtained with the blood pressure measures was used to represent resting heart rate.

2.3 | Statistical analysis

Analyses were performed using SAS version 9.4 (SAS Institute, Cary NC). Descriptive statistics, mean ± standard deviation, or median (interquartile range) for continuous variables and frequency (percentage) for categorical variables was presented. Spearman correlation coefficient was used to examine bivariate correlation between LVM and continuous variables of interest, eg, age, weight, BMI, BSA, MAP, and fitness measures. Also presented are the Spearman partial correlation coefficient controlling for sex, age, and race. For the bivariate relationship between LVM and categorical variables of sex and race, the Wilcoxon rank sum test and the Kruskal-Wallis test were used, respectively. Linear regression models were fit to the outcome of LVM. Stepwise regression analyses were performed to select a set of significant predictors from the variables showing significant bivariate correlations, after forcing age, gender, race, MAP, and height in the models. Analyses were repeated with height removed from the analyses. The type I error rate was fixed at 5%.

3 | RESULTS

The flow of participants into this study is illustrated in Figure 1. Descriptive characteristics of the participants are shown in Table 1, and LVM by sex and race is shown in Table 2. Data for LVM, weight, height, BMI, BSA, percent body fat, and termination time on the exercise test were available for 383 participants. Data from four participants were not available, with two missing CMR data and two missing oxygen consumption (L·min^{-1} and mL·kg^{-1}·min^{-1}). Thus, complete data are available for 379 participants.

Bivariate analyses were conducted to examine the association between the descriptive characteristics and LVM (Table 1). Left ventricular mass was positively associated with weight, height, BMI, BSA, and all measures of fitness ($P < .0001$) and inversely associated with percent body fat ($P < .0001$). Median LVM was significantly different when analysed by sex ($P < .0001$) but not by race ($P < .1435$) (Table 2).

Results of stepwise multivariate analyses examining the association between measures of fitness and body size (weight, BMI, and BSA) are shown in Tables 3, 4 and 5. In these analyses, body weight was positively associated with LVM ($P < .0001$); however, percent body fat was inversely associated with LVM ($P ≤ .0001$) (Table 3). In the same models, cardiorespiratory fitness expressed as oxygen consumption (L·min^{-1}) ($P = .0098$) was positively associated with a larger LVM. This model reflected an r^2 of .6672. When additional analyses were performed with cardiorespiratory fitness expressed as mL·kg^{-1}·min^{-1} or test termination time, similar patterns of results were observed, with an r^2 of .6662 and .6659, respectively. Analyses were repeated with height removed as a variable; and the pattern of results for weight, percent body fat, and cardiorespiratory fitness was similar to the findings presented in Table 3 (data not shown).

Additional analyses were performed replacing body weight with BMI (Table 4). In these stepwise regression models, BMI was positively associated with LVM ($P < .0001$), percent body fat inversely associated with LVM, and fitness positively associated with LVM. The r^2 in these three models ranged from .6643 to .6666. Similar patterns of results were observed when the analyses were performed with BSA rather
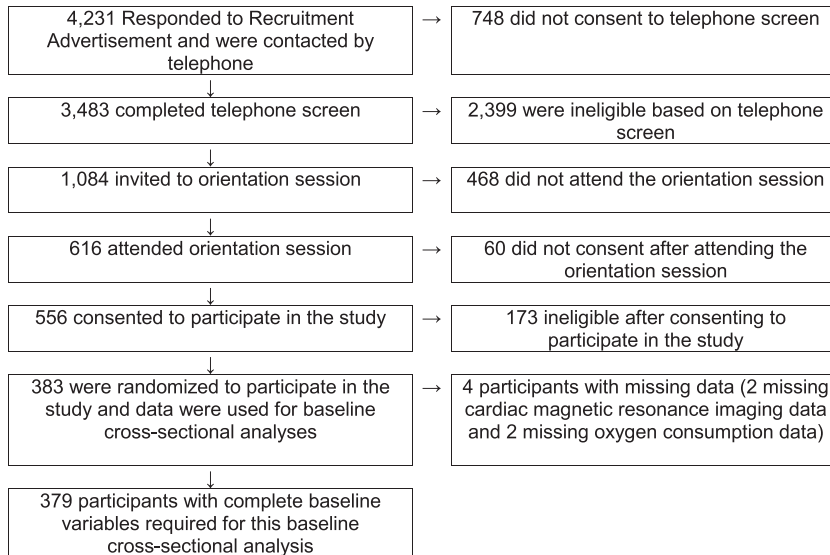
| 4,231 Responded to Recruitment Advertisement and were contacted by telephone | → | 748 did not consent to telephone screen |

| 3,483 completed telephone screen | → | 2,399 were ineligible based on telephone screen |

| 1,084 invited to orientation session | → | 468 did not attend the orientation session |

| 616 attended orientation session | → | 60 did not consent after attending the orientation session |

| 556 consented to participate in the study | → | 173 ineligible after consenting to participate in the study |

| 383 were randomized to participate in the study and data were used for baseline cross-sectional analyses | → | 4 participants with missing data (2 missing cardiac magnetic resonance imaging data and 2 missing oxygen consumption data) |

| 379 participants with complete baseline variables required for this baseline cross-sectional analysis |

FIGURE 1 Consort diagram for participant recruitment

TABLE 1 Descriptive characteristics and bivariate associations with left ventricular mass

	N	M ± SD	Spearman Correlation Coefficient (ρ)	P Value
Age (y)	379	45.6 ± 7.9	−.027	.5954
Mean arterial pressure	379	88.1 ± 9.0	.360	<.0001
Height (cm)	379	167.4 ± 8.0	.552	<.0001
Weight (kg)	379	90.8 ± 13.7	.629	<.0001
BMI (kg·m^{-2})	379	32.4 ± 3.8	.355	<.0001
% body fat	379	43.3 ± 5.5	−.282	<.0001
Body surface area	379	2.1 ± 0.2	.657	<.0001
Cardiorespiratory fitness (oxygen consumption, L·min^{-1})[a]	379	2.1 ± 0.5	.613	<.0001
Cardiorespiratory fitness (oxygen consumption, mL·kg·min^{-1})[a]	379	22.6 ± 4.4	.291	<.0001
Cardiorespiratory fitness (termination time)[a]	379	7.7 ± 3.0	.281	<.0001

Abbreviation: BMI, body mass index.

[a]Evaluation of cardiorespiratory fitness was assessed with a submaximal graded exercise test terminated at 85% of age-predicted maximal heart rate.

TABLE 2 Left ventricular mass by sex and race

Descriptive Category		N (% Total Sample)	M ± SD (Median [25th, 75th Percentile])	P Value
Total sample		379		
Sex	Female	303 (79.95%)	81.92 ± 15.09 (81.20 [72.00, 91.20])	<.0001[*]
	Male	76 (20.05%)	116.08 ± 18.44 (112.75 [103.00, 127.61])	
Race	African American/Black	86 (22.69)	88.80 ± 18.59 (86.35 [77.10, 97.70])	.1435[**]
	Asian	11 (2.90)	73.78 ± 17.07 (75.32 [59.40, 88.25])	
	Caucasian/white	271 (71.50)	89.47 ± 21.60 (84.51 [73.49, 102.50])	
	Other	4 (1.06)	93.31 ± 20.16 (88.02 [78.50, 108.12])	
	Mixed race	7 (1.85)	82.52 ± 21.73 (69.75 [67.10, 107.55])	

[*]P value based on Wilcoxon sum rank test.

[**]P value based on Kruskal-Wallis test.

than BMI (Table 5). Again, analyses were repeated with height removed as a variable; and the pattern of results for BMI, BSA, percent body fat, and cardiorespiratory fitness was similar to the findings presented in Tables 4 and 5 (data not shown).

4 | DISCUSSION

Higher levels of body fatness may be associated with a lower LVM measured by CMR, which may reflect a maladaptive response,

TABLE 3 Predictors (weight, percent body fat, and cardiorespiratory fitness) of left ventricular mass measured by cardiac magnetic resonance imaging using stepwise regression analysis

Dependent Variable	Variable	Step	β	SE	P Value	Partial r^2	Model r^2
Left ventricular mass							.6676
	Intercept		43.53	23.706	.0671		
	Age (years)[a]		0.092	0.084	.2734		
	Gender (female)[a]		−10.711	2.915	.0003		
	Race (white)[a]		−2.684	1.473	.0692		
	Height (cm)[a]		−0.149	0.132	.2601		
	Mean arterial pressure[a]		0.353	0.076	<.0001		
	Weight (kg)	1	0.787	0.095	<.0001	.1075	
	% body fat	2	−0.866	0.213	<.0001	.0263	
	Fitness (oxygen consumption, L·min^{-1})	3	5.563	2.143	.0098	.0061	
Left ventricular mass							.6662
	Intercept		35.775	25.336	.1588		
	Age (years)[a]		0.087	0.084	.3001		
	Gender (female)[a]		−11.062	2.907	.0002		
	Race (white)[a]		−2.546	1.472	.0845		
	Height (cm)[a]		−0.153	0.133	.2500		
	Mean arterial pressure[a]		0.352	0.076	<.0001		
	Weight (kg)	1	0.915	0.078	<.0001	.1075	
	% body fat	2	−0.897	0.212	<.0001	.0263	
	Fitness (oxygen consumption, mL·kg^{-1}·min^{-1})	3	0.438	0.193	.0235	.0047	
Left ventricular mass							.6659
	Intercept		48.441	23.501	.040		
	Age (years)[a]		0.080	0.084	.340		
	Gender (female)[a]		−11.622	2.872	<.0001		
	Race (white)[a]		−2.551	1.475	.0845		
	Height (cm)[a]		−0.187	0.132	.1573		
	Mean arterial pressure[a]		0.347	0.076	<.0001		
	Weight (kg)	1	0.922	0.078	<.0001	.1075	
	% body fat	2	−0.918	0.210	<.0001	.0263	
	Fitness (termination time)	3	0.590	0.269	.0293	.0043	

[a]These variables were forced in the model during the stepwise regression analysis.

whereas higher levels of cardiorespiratory fitness may be associated with a favourable adaptive response to LVM. Prior studies have shown that body size, defined by BMI or BSA, is associated with a larger LVM.[6,7] The results of this study support these findings with bivariate analyses showing associations between LVM and measures of body size that included weight, BMI, and BSA (see Tables 3, 4, and 5). However, when considered in multivariate analyses, body weight was not associated with LVM measured by CMR; however, both BMI and BSA remained significantly associated with a greater LVM (see Tables 3, 4, and 5). Moreover, in all analyses, percent body fat, measured by DXA, was inversely associated with LVM. This study also showed that cardiorespiratory fitness was positively associated with LVM after accounting for body size and body fatness.

The results of this study are of clinical importance given the association between obesity and CVD morbidity and mortality,[26,27] along with the inverse association between cardiorespiratory fitness and CVD morbidity and mortality.[8-10,12] This has resulted in debate regarding whether it is the excess body weight or low cardiorespiratory fitness that contributes to all-cause mortality and CVD. There have been reports to support both sides of this debate with some studies supporting that fitness is more highly associated with CVD,[11-14] while other studies support the position that excess weight is an important

contributor to CVD.[17,28] The results of this current study in adults who are overweight or obese, engaging in low levels of physical activity, and without known CVD suggest that both adiposity and fitness are associated with LVM. This may suggest that both prevention and treatment of excess levels of adiposity, combined with emphasis on engaging in behaviours that influence cardiorespiratory fitness, such as physical activity, may be important clinical targets that impact LVM. Moreover, while dietary change may reduce weight and adiposity, it will not have a direct influence on cardiorespiratory fitness. However, moderate-to-vigorous physical activity will result in reduced weight and adiposity along with improved cardiorespiratory fitness, with both of these factors shown to impact LVM in this current study.

The finding that measures of body size (BMI and BSA) were positively associated with LVM, whereas percent body fat was inversely associated with LVM, is an interesting observation. This may suggest that body size and total mass are associated with LVM, but when a higher percent of the mass is fat, this may reduce the quality of the myocardium. This reduced quality of the myocardium may provide partial explanation for how excess body fat may be associated with cardiac structure and potential downstream health risks. This observation warrants confirmation and further examination to potential health-related implications.

TABLE 4 Predictors (body mass index, percent body fat, and cardiorespiratory fitness) of left ventricular mass measured by cardiac magnetic resonance imaging using stepwise regression analysis

Dependent Variable	Variable	Step	β	SE	P Value	Partial r^2	Model r^2
Left ventricular mass							.6666
	Intercept		−98.252	20.503	<.0001		
	Age (years)[a]		0.097	0.084	.2489		
	Gender (female)[a]		−10.686	2.921	.0003		
	Race (white)[a]		−2.753	1.474	.0626		
	Height (cm)[a]		0.696	0.113	<.0001		
	Mean arterial pressure[a]		0.359	0.076	<.0001		
	Body mass index (kg·m^{-2})	1	2.189	0.267	<.0001	.1053	
	% body fat	2	−0.876	0.215	<.0001	.0268	
	Fitness (oxygen consumption, L·min^{-1})	3	5.819	2.135	.0067	.0067	
Left ventricular mass							.6646
	Intercept		−129.750	22.738	<.0001		
	Age (years)[a]		0.091	0.084	.2812		
	Gender (female)[a]		−11.122	2.915	.0002		
	Race (white)[a]		−2.593	1.475	.0796		
	Height (cm)[a]		0.837	0.105	<.0001		
	Mean arterial pressure[a]		0.359	0.076	<.0001		
	Body mass index (kg·m^{-2})	1	2.565	0.222	<.0001	.1053	
	% body fat	2	−0.916	0.214	<.0001	.0268	
	Fitness (oxygen consumption, mL·kg^{-1}·min^{-1})	3	0.441	0.193	.0228	.0047	
Left ventricular mass							.6643
	Intercept		−118.193	21.028	<.0001		
	Age (years)[a]		0.084	0.084	.3189		
	Gender (female)[a]		−11.684	2.880	<.0001		
	Race (white)[a]		−2.599	1.478	.0794		
	Height (cm)[a]		0.810	0.105	<.0001		
	Mean arterial pressure[a]		0.354	0.076	<.0001		
	Body mass index (kg·m^{-2})	1	2.584	0.222	<.0001	.1053	
	% body fat	2	−0.938	0.212	<.0001	.0268	
	Fitness (termination time)	3	0.595	0.270	.0281	.0044	

[a]These variables were forced in the model during the stepwise regression analysis.

The association between cardiorespiratory fitness and LVM should be interpreted with caution, given that the direction of this association is not able to be determined from this cross-sectional analysis. For example, it is possible that targeting behaviours, such as physical activity, that result in improved cardiorespiratory fitness, may contribute to an increase in LVM. Conversely, it is possible that an adult with a larger LVM may have enhanced cardiorespiratory fitness, which may allow for greater engagement in physical activity. Thus, this may indicate the need for a properly designed prospective and intervention studies to examine the magnitude of change in LVM that occurs as cardiorespiratory fitness either increases or decreases, and when these changes in cardiorespiratory fitness are accompanied by changes in body size and body fatness.

This study has numerous strengths. These strengths include the use of CMR to assess LVM, cardiorespiratory fitness assessed from a graded exercise test, and body fatness assessed using DXA. However, there are limitations that are important to recognize. This study included participants with a BMI range from 25 to <40 kg·m^{-2}, which limited the ability to examine whether the associations observed would remain with the inclusion of participants at the lower or higher ranges of BMI. Participants with known CVD were also excluded, which may have also excluded individuals with clinically relevant left ventricular hypertrophy, and therefore, it will be important to consider how the factors observed in this study would impact LVM in that clinical population. This study also included participants who self-reported low participation in regular moderate-to-vigorous physical activity, which limited the range of fitness considered in the analyses. Moreover, the sample of participants examined, aside from being overweight or obese, and engaging in low amounts of physical activity, was otherwise relatively healthy. Therefore, whether similar findings would be observed in participants who may have other risks factors or known chronic health conditions is unable to be determined. In addition, the study is unable to examine if similar findings would be observed in an older population (>55 y of age) or in participants with different demographic characteristics than those examined.

Another key consideration is that this study was initiated prior to the emergence of extracellular volume (ECV) measures. Patient vulnerability may relate more to myocardial "quality" (ie, extent of excess interstitial expansion, usually from myocardial fibrosis) than quantity (LVM).[29-31] Extracellular volume measures[32,33] the volume percent of the interstitial compartment, which represents a well-validated surrogate of myocardial fibrosis in most settings.[34]

TABLE 5 Predictors (body surface area, percent body fat, and cardiorespiratory fitness) of left ventricular mass measured by cardiac magnetic resonance imaging using stepwise regression analysis

Dependent Variable	Variable	Step	β	SE	P Value	Partial r^2	Model r^2
Left ventricular mass							.6677
	Intercept		35.238	23.175	.1292		
	Age (years)[a]		0.092	0.084	.2733		
	Gender (female)[a]		−10.552	2.918	.0003		
	Race (white)[a]		−2.637	1.473	.0743		
	Height (cm)[a]		−0.502	0.160	.0018		
	Mean arterial pressure[a]		0.352	0.076	<.0001		
	Body surface area	1	67.052	8.101	<.0001	.1071	
	% body fat	2	−0.880	0.214	<.0001	.0268	
	Fitness (oxygen consumption, L·min^{-1})	3	5.555	2.143	.0099	.0060	
Left ventricular mass							.6663
	Intercept		26.118	24.963	.2691		
	Age (years)[a]		0.087	0.084	.2999		
	Gender (female)[a]		−10.877	2.913	.0002		
	Race (white)[a]		−2.492	1.472	.0913		
	Height (cm)[a]		−0.563	0.157	.0004		
	Mean arterial pressure[a]		0.352	0.076	<.0001		
	Body surface area	1	78.029	6.686	<.0001	.1071	
	% body fat	2	−0.913	0.213	<.0001	.0268	
	Fitness (oxygen consumption, mL·kg^{-1}·min^{-1})	3	0.437	0.194	.0237	.0047	
Left ventricular mass							.6659
	Intercept		38.783	23.126	.0944		
	Age (years)[a]		0.079	0.084	.3420		
	Gender (female)[a]		−11.447	2.878	<.0001		
	Race (white)[a]		−2.491	1.475	.0921		
	Height (cm)[a]		−0.599	0.156	.0001		
	Mean arterial pressure[a]		0.347	0.076	<.0001		
	Body surface area	1	78.557	6.679	<.0001	.1071	
	% body fat	2	−0.936	0.211	<.0001	.0268	
	Fitness (termination time)	3	0.583	0.269	.0311	.0042	

[a]These variables were controlled for in the stepwise regression analysis.

5 | CONCLUSION

These cross-sectional findings support that LVM in adults who are relatively healthy, overweight, or obese and engaging in low amounts of physical activity is associated with body size (BMI and BSA), body fatness, and cardiorespiratory fitness. Given that LVM is as an important clinical indicator of CVD, these findings may provide relevant information regarding interventions that may influence LVM, such as interventions to prevent gain of weight and fat mass, to promote weight loss, and to promote improvements in cardiorespiratory fitness. Thus, future studies should investigate whether changes in weight, body composition, and fitness have differential influences on LVM. Moreover, these findings build on prior studies that have shown that body size is associated with LVM, with these findings indicating that evaluation of LVM should also consider potential associations with body composition and cardiorespiratory fitness.

CONFLICT OF INTEREST STATEMENT

No conflict of interest was declared.

DISCLOSURES

Dr. Rogers was the principal investigator on a grant awarded to the University of Pittsburgh by Weight Watchers International. Dr. Schelbert accepted contrast material from Bracco Diagnostics for

research purposes and has served on advisory boards for Merck and Bayer. Dr. Jakicic received an honorarium for serving on the Scientific Advisory Board for Weight Watchers International and was a coinvestigator on a grant awarded to the University of Pittsburgh by Weight Watchers International.

DATA SHARING

Data sharing for this cross-sectional analysis of baseline data will not be made available. Investigators interested in data from the parent study should contact Dr. Jakicic (jjakicic@pitt.edu), who is the principal investigator, for data sharing policies and procedures after publication of the primary outcomes of this study.

AUTHOR CONTRIBUTIONS

J.M.J., E.B.S., and W.L. conceived the study design. R.J.R., J.M.J., E.B.S., and Y.F. were responsible for data collection. W.L., N.Y., J.M.J., and E.B.S. were responsible for data analysis. R.J.R. and J.M.J. were involved in writing the paper, and all authors had final approval of the submitted and published versions.

ORCID

John M. Jakicic https://orcid.org/0000-0001-6800-9368

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