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I, Brenda Mendizabal M.D., hereby submit this original work as part of the requirements for the degree of Master of Science in Clinical and Translational Research.

It is entitled:

Racial Differences in the Influence of Risk Factors in Childhood on Left Ventricular Mass in Young Adulthood

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**Racial Differences in the Influence of Risk Factors in Childhood on Left Ventricular Mass
in Young Adulthood**

A thesis submitted to the
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of the University of Cincinnati
in partial fulfillment of the
requirements for the degree of

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in Clinical & Translational Research

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by

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ABSTRACT:

Cross-sectional studies have demonstrated a relationship between cardiovascular (CV) risk factors and left ventricular mass (LVM) in adolescents. Few data exist relating the burden of those risk factors over the childhood and young adult years on LVM, a known risk factor for hard CV events later in life. Racial differences exist in the development of cardiovascular disease (CVD), with blacks being adversely affected. We sought to examine the relationship and racial differences between CV risk factors measured since age 10, and LVM/ht^{2.7} (LVMI) in adulthood in the NHLBI Growth and Health Study (NGHS), a longitudinal study examining the origins of racial differences in accumulation of adiposity and development of CV risk in girls. A total of 556 girls (mean age 10.0 years, 55.2% black, 45.8% white) had anthropometrics, blood pressure, and labs performed at up to 17 visits. LVMI was collected with 2-D guided echocardiographic imaging at a mean age of 26 years. Mean values at baseline and follow-up, and area under the curve (AUC) for CV risk factors, stratified by race, were examined by t-tests. General linear models were constructed to evaluate independent determinants of LVMI overall, and stratified by race. The initial full model contained race, body mass index (BMI), percent body fat by skinfold (PBF), systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), low density lipoprotein cholesterol (LDL-C), triglyceride/high-density lipoprotein (TG/HDL) ratio, HOMA-IR (homeostasis model assessment of insulin resistance), and c-reactive protein (CRP). As seen previously, black girls had higher LVMI compared to whites. Blacks also had higher adiposity and insulin across time and lower TG/HDL ratio (all p-values < .0001). The major determinants of LVMI were race, BMI z-score AUC, SBP z-score AUC, PBF AUC, HR AUC and an interaction between race and HR (model R² = 0.40, p-value < 0.0001). Stratifying by race, HR did not enter the model for blacks. The major determinants of LVMI in

young adult females are adiposity and SBP. The contribution of HR to LVMI in whites may relate to previously noted higher sympathetic tone in obese whites. Efforts to prevent the development of obesity and hypertension in young girls are essential to reduce future hard CV events in adulthood.

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Introduction

Cross-sectional studies have demonstrated a relationship between cardiovascular (CV) risk factors and left ventricular mass (LVM) in adolescents. However, few data exist relating the burden of those risk factors over the childhood and young adult years on LVM, a known risk factor for hard CV events later in life ¹. Additionally, there are known racial differences in the development of CVD, with blacks being adversely affected. Specifically, blacks have been shown to have increased LVMI compared to their white counterparts ².

The NHLBI Growth and Health Study (NGHS) was initiated in 1985 as a longitudinal study to identify the origins of the racial differences in development of obesity and other CV risk factors in black and white girls and the effect of these racial differences on the heart. In this study, we sought to examine the relationship between the burden of CV risk factors, measured since age 10, particularly adiposity, blood pressure (BP), and heart rate (HR), on left ventricular mass index ($LVM/ht^{2.7}$, LVMI) in adulthood. We also evaluated racial differences in the effect of CV risk factors on LVMI.

Methods

Study Population: NGHS was a longitudinal, multi-center study of the development of obesity and its effects on CV risk factors in black and white girls. The NGHS protocol, including recruitment, has been described previously ³. Eligibility criteria included ³: girls age 9 or 10 years, identified themselves as “black” or “white” (non-Hispanic), and had racially concordant parents or guardians. All girls who enrolled in the study had assented, and their parents (or guardian) had provided written informed consent. The study protocol was approved by the Institutional Review Board.

Although the original NGHS design included 3 centers, only the Cincinnati site had 7 additional years of data collection (total of 17 visits), including echocardiograms. Therefore, only the Cincinnati cohort is included in these analyses (N=556, mean age at enrollment = 10.0 ± 0.5 years; mean age at echocardiogram = 25.7 ± 1.7 years; 55.2% black, 45.8% white).

CV Risk Factor Assessment: Measurements of height, weight, percent body fat by skinfold thickness (PBF), and BP were obtained by trained examiners. Height was measured using custom-made stadiometers with the girls wearing no shoes. Weight was measured with calibrated Health-O-Meter electronic scales with girls wearing provided T-shirts or gowns. Skinfold measurements were obtained at the triceps, suprailiac, and subscapular sites with Holtain calipers. All anthropometric measurements were taken twice and repeated a third time if the first two measurements differed by more than 0.5 cm (height), 0.3 kg (weight), or 1.0 mm (skin folds). Degree of overweight and adiposity was assessed using body mass index (BMI), calculated as weight in kilograms divided by height in meters squared, and the sum of the three skinfold thicknesses. The degree of adiposity was assessed using the ratio of truncal (subscapular and suprailiac) to total sum of skin folds.

Blood pressure was obtained by trained examiners in subjects seated with feet resting on a flat surface and the arm at heart level. The appropriate size cuff was selected based on upper arm circumference. Blood pressure was measured three times with measurements at least 60 seconds apart using a standard mercury sphygmomanometer. The second and third measurements of BP were used to calculate the mean systolic and diastolic K4 and K5 blood pressure. The diastolic K5 BP measurement was substituted for the diastolic K4 measurement when K4 could not be distinguished from K5. Diastolic K5 BP measurements were used in our analyses.

The collection of fasting laboratory values (total cholesterol, low density lipoprotein (LDL-C), high density lipoprotein (HDL-C), triglycerides (TG), glucose, insulin) for the NGHS have been described previously in detail ³.

Echocardiographic Measures: Echocardiograms were performed on a GE Vivid 7 – V7916 (Milwaukee, WI, USA) ultrasound system. All images were obtained with the participant in the left lateral decubitus position to acquire parasternal long-axis, parasternal short-axis and apical 4-chamber views for a total of three cardiac cycles. Absence of structural heart disease was confirmed, and then the mean of 3 readings of left ventricular end-diastolic, end-diastolic septal thickness and end-diastolic posterior wall thicknesses were measured off-line using a Digiview Image Management and Reporting System (Digisonics, Houston, Texas). LVMI was calculated by the Devereaux formula ⁴ and normalized to height^{2.7} as recommended by De Simone ⁵.

Statistical Analysis: All data analyses were performed with SAS version 9.4. Mean values at baseline, follow-up, and AUC for CV risk factors, stratified by race, were examined by t-tests. The AUC of serial measurements of each CV risk factor was used as a measure of cumulative risk burden from childhood to adulthood. Risk factors measured at the first and last examinations were used as childhood and adulthood values, respectively.

General linear models were constructed to evaluate independent determinants of LVMI overall, and stratified by race to examine racial differences. Spearman correlation coefficients were used to assess collinearity between CV risk factors and to evaluate bi-variate correlations between CV risk factors and LVMI to construct the most parsimonious initial model. The initial full model contained race, BMI, PBF, HR, systolic blood pressure (SBP), diastolic blood

pressure (DBP), LDL-C, TG/HDL ratio, HOMA-IR (homeostasis model assessment of insulin resistance, calculated as: fasting insulin (microU/L) x fasting glucose (nmol/L)/22.5), and c-reactive protein (CRP). Stepwise regression was used removing non-significant variables, with the final model representing the independent determinants of follow-up LVMI.

Results

Subject baseline and follow-up characteristics are described in Table 1. By study design all 556 participants were female (55.2% black and 45.8% white). Mean age at baseline was 10.0 ± 0.5 years, and mean age at follow-up was 25.7 ± 1.7 years. As expected all anthropometric variables, including height, weight, BMI, PBF, and waist circumference (WC) were significantly greater at follow-up (p-value $\leq .0001$). Similarly, SBP and DBP were higher and HR was lower from baseline to follow-up (p-value $\leq .0001$). There was no significant difference between baseline and follow-up total cholesterol; however, the remaining labs were significantly different (all p-value $\leq .0001$, except LDL-C p-value < 0.02). There were fewer participants with lipids (N=355) and glucose or insulin levels (N=291). Additionally, glucose and insulin levels were obtained at a mean age of 16, unlike other baseline labs.

Racial differences in CV risk factor variables measured at childhood, adulthood, and over time (AUC) are shown in Table 2. Blacks were slightly older and taller at baseline. Overall, blacks had higher measures of adiposity at baseline, follow-up, and over time (weight, BMI, PBF, WC; p-value $\leq .0001$). There was no difference in PBF in childhood. As adults, blacks had higher SBP and DBP (p-value $< .0001$, < 0.03) and had lower HR in childhood and over time (p-value < 0.05). Blacks had lower total cholesterol, TG and TG/HDL ratio as adults and

over time (p-value $\leq .0001$). Insulin was higher in blacks in childhood, adulthood and over time, and glucose was higher in blacks in adulthood and over time (p-value ≤ 0.05).

Cardiac measures in adulthood, stratified by race, are displayed in Table 3. As adults, blacks had statistically greater LVMI compared to whites. However, there was no significant difference between blacks and whites in adult HR, stroke volume (SV), cardiac output (CO) or systemic vascular resistance (SVR). There were fewer participants that had SV (N=220) and CO or SVR (N=185).

BMI z-score AUC and SBP z-score AUC were significant predictors of LVMI in unadjusted linear regression (p-value < 0.00001 , $R^2 = 0.34$ and p-value < 0.00001 , $R^2 = 0.11$ respectively), as seen in Figures 1 and 2. In fully adjusted analyses (Table 4), the major determinants of LVMI were race, BMI z-score, SBP z-score, PBF, HR, and an interaction between race and HR (model $R^2=0.40$, p-value $< .0001$, Figure 3). However, when stratified by race, HR did not enter the model for blacks. LVMI regression on HR AUC differed by race, demonstrating significance only for white girls ($R^2=0.40$).

Discussion

In this study, we found that young black women had higher LVMI than white women at the same age. Racial differences were also found in the effect of HR on LVMI. We showed that the burden of excess adiposity and higher BP over time were major determinants of LVMI in young adult females. Given the relationship between LVMI and hard CV events in adults¹, it is important to determine the risk factors in childhood associated with LVMI in adulthood.

The influence of BP and obesity on left ventricular hypertrophy (LVH) in adults was demonstrated in the 1980s in the Framingham Heart Study (FHS)⁶. Using LVM normalized to

height for LVH, SBP was found to be an independent predictor of LVH. In analyses restricted to females (mean age 52 years), there was a 25% increase in LVH for every 20 mm Hg increase in SBP. The prevalence of LVH was also higher in obese participants with a dose-response relationship between the severity and duration of obesity and the prevalence of LVH. As with SBP, obesity was an independent predictor of LVH, with a 51% increase in the prevalence of LVH in women for every 2 kg/m² increment in BMI ⁶.

The CARDIA (Coronary Artery Risk Development in Young Adults) Study performed echocardiograms in participants younger than those in the FHS (CARDIA mean age 30 years) ⁷. In cross-sectional analyses, LVM (unadjusted for body size) correlated with body weight and SBP across sex and black-white racial subgroups ². A later CARDIA study, performed at mean age 50 years, determined that baseline and change in BMI and BP (mean arterial pressure) were significant determinants of LVMI after 20 years of follow-up ⁷. As with our study, HR was also an independent predictor ⁷. The Bogalusa Heart Study (BHS) similarly found the burden of SBP and adiposity from childhood (AUC) influenced LVMI in adulthood (mean age 33 years) ⁸. The effect of obesity on LVM may be even stronger than BP, as BHS also demonstrated that the mediation effect of adult BMI was significantly stronger than those of adult SBP on LVMI ⁹.

Differences in LVMI between black and white adults have been observed in both CARDIA ^{2,7} and BHS, ⁸ where blacks had greater LVMI compared to their white counterparts. In BHS, the greater LVMI in blacks disappeared when adjusted for BMI and SBP, suggesting that higher levels of BP and adiposity in blacks, may partly account for the racial differences observed ⁸. Gardin et al ¹⁰ also examined racial differences in predictors of change in LVM in CARDIA (mean age 35 years). For all race/sex groups, initial LVM and BMI and change in BMI were significant predictors of LVM measured at 5 years of follow-up. However, in black

women, initial SBP and change in SBP were also significant. Black women were also the only race/gender subgroup to have a significant increase in LVM over the 5 year period (mean increase of 5.9 g or 5.8%). However, whether adiposity or BP was more important in determining acquisition of LVM in black women was unclear as change in LVM was most strongly correlated with change in BMI ¹⁰.

The influence of CV risk factors on LVMI has also been examined in the children. In a retrospective study of children (mean age 14 years) with primary HTN, the overall prevalence of LVH was 15.5% by adult criteria ($\text{LVMI} > 51 \text{ g/m}^{2.7}$) and 41.1% by pediatric criteria ($\text{LVMI} > 36.88 \text{ g/m}^{2.7}$) ¹¹. Obese ($\text{BMI} \geq 95^{\text{th}}$ percentile) children with hypertension (HTN) were more likely to have LVH by either criteria. The investigators could not evaluate the effect of BP on LVMI since all subjects included in the study were hypertensive. To better evaluate the effect of BP level on LVMI, McNiece et al ¹² pooled data from subjects referred to a HTN clinic (mean age 12 years) and found the prevalence of LVH ($\text{LVMI} > 51 \text{ g/m}^{2.7}$) increased significantly from normotensive to Stage 1 to Stage 2 HTN. Importantly, the increased risk of LVH associated with Stage 2 HTN persisted after adjusting for age, gender, race, and BMI (OR 4.13). These investigators, however, did not separately evaluate the effect of obesity on LVMI. The effect of BP level on LVMI was confirmed in a larger study (N=723 subjects, mean age 18 years), where subjects with pre-HTN and sustained HTN had higher LVMI compared to normotensive controls ¹³. BP category remained an independent predictor of LVMI even after adjusting for other CV risk factors, including obesity. One limitation of this work is the inclusion of a large number of subjects with T2DM (N=258) making the results less generalizable.

Few studies have examined the effect of both adiposity and BP on LVMI in youth. In a cross-sectional, multicenter study of children with newly diagnosed primary HTN (mean age 13

years), Brady et al found the prevalence of LVH ($\text{LVMI} > 51 \text{ g/m}^{2.7}$) to be greater in obese subjects (50% versus 32% for lean), irrespective of BP category/severity¹⁴. LVMI was found to be higher in obese compared to lean adolescents in another study (mean age 16 years), and patients with both obesity and high BP had the highest prevalence of LVH ($\text{LVMI} \geq 95^{\text{th}}$ percentile), and both BMI and SBP were significant independent determinants of LVM. However, the interaction between obesity and HTN on LVH was not significant¹⁵. This study, however, was limited by only including black adolescents. A longitudinal study by Sivanandam et al recruited children at mean age 13 years, and performed echocardiography at 27 years. There was no relationship between childhood SBP and adult LVMI, but there was an correlation between adult SBP and LVMI, that became non-significant after adjustment for obesity¹⁶. In a similar study with a shorter follow-up, Urbina et al, found boys and girls (mean age 14 years) with higher adiposity at baseline had higher LVMI at 4 year follow-up. SBP was also a significant independent determinant of LVMI, but this was only seen in females¹⁷. Additionally, baseline weight and triceps skin fold thickness predicted follow-up LVMI for both sexes, but change in SBP was significantly related to follow-up LVMI only in females ($p\text{-value} \leq 0.05$)¹⁷. Clearly, larger studies are needed to elucidate the contribution of adiposity and BP to LVMI.

Pediatric studies have also looked at how CV risk factors effect LVMI differently by race. Compared to white children, Brady et al found an increased odds and prevalence of LVH ($\text{LVMI} > 51 \text{ g/m}^{2.7}$) in non-white children with primary HTN and obesity¹⁴. Similarly, Pruetete et al demonstrated the prevalence of LVH ($\text{LVMI} \geq 95^{\text{th}}$ percentile) was significantly higher in black children (49% versus 30%), however race was not independently associated with LVH¹⁸.

There are limited data examining the effect of autonomic tone and heart rate on LVH. Although low-frequency heart rate variability (LF-HRV) has been previously shown to be an

independent risk factor for LVH¹⁹, Hill et al²⁰ described a cross-sectional study of 148 black and white adults (mean age 33 years) where high-frequency heart rate variability (HR-HRV), which reflects parasympathetic tone and has previously thought to be cardioprotective²¹, was positively associated with LVMI in blacks even after accounting for age, sex, and BMI. This association was not found in whites. Race differences in heart rate variability (HRV) have been previously demonstrated in the ARIC (Atherosclerosis Risk in Communities) study which showed higher HF/LF-HRV ratio in blacks with 10-minute ECG strips.²² In this study, however, LVM was not measured and there was no difference in resting baseline HR. Urbina et al,²³ similarly demonstrated parasympathetic predominance in black versus white adolescent males in the Bogalusa Heart Study, using 24-hour Holter monitor recordings for HRV. There was also a trend for parasympathetic predominance in black subjects with higher levels of BP (higher HF/LF ratio). In this study, whites demonstrated higher baseline HR compared to blacks in the normotensive group, similar to our study. Although the observed racial differences in HR related to LVMI in our study may relate to a predominance of parasympathetic tone in blacks, we cannot conclusively determine this without a measure of autonomic tone.

Limitations

Due to the study design of the NGHS cohort, only black and white girls were included, limiting generalizability to males and other races and ethnicities. We were also limited as our echocardiograms did not include sensitive measures of cardiac strain or diastolic function. Similarly, there is no measure of fitness or autonomic tone to further elaborate on our findings. We also did not include any precise measures of body composition, such as DEXA or MRI, to assess lean versus fat mass, and its effect of LVMI. Lastly, the NGHS study cohort lacks hard CV endpoints as the final echocardiograms were performed at approximately 26 years of age.

Conclusion

The major determinants of LVMI in young adult females are adiposity and SBP. Furthermore, black girls have a higher burden of adiposity over time, contributing to the observed racial difference in LVMI, suggesting higher CV risk in blacks. The racial differences in HR and its significance on LVMI remains unclear. Efforts to prevent the development of obesity and HTN in young girls of all races and ethnicities are essential to reduce future hard CV events in adulthood.

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Tables and Figures

Table 1: Subject Characteristics at Baseline and Follow-up				
Variable	Baseline		Follow-up	
	Mean	SD	Mean	SD
Age (years)*	10.0	± 0.5	25.7	± 1.7
Height (cm)*	140.9	± 7.6	164.6	± 6.5
Weight (kg)*	36.9	± 9.9	77.7	± 23.0
BMI (kg/m²)*	18.4	± 3.7	28.7	± 8.2
PBF (%)*	21.6	± 8.7	38.4	± 15.6
WC (cm)*	64.7	± 8.9	83.9	± 15.6
SBP (mmHg)*	101.0	± 8.5	108.7	± 9.9
DBP (mmHg)*	59.4	± 10.5	68.1	± 9.5
HR (beats/min)*	83.3	± 12.8	71.7	± 10.4
Total Cholesterol (mg/dL)	172.4	± 29.9	172.6	± 35.6
TG (mg/dL)*	78.1	± 31.2	97.0	± 53.5
LDL-C (mg/dL)*	105.9	± 29.1	102.2	± 32.0
HDL-C (mg/dL)*	54.5	± 12.0	50.9	± 12.8
Glucose (mmol)*	69.1	± 15.6	88.6	± 20.7
Insulin (mmol)*	14.7	± 12.0	12.1	± 10.1
* p-value < 0.05				

Abbreviations: BMI, body mass index; PBF, percent body fat by skin fold; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; TG, triglycerides; LDL, low density lipoprotein; HDL, high density lipoprotein

Table 2

Table 2: Racial Differences in CV Risk Factors				
Variable	White		Black	
	Mean	SD	Mean	SD
Age (years)				
Childhood*	9.9	± 0.5	10.0	± 0.5
Adulthood	25.7	± 1.7	25.7	± 1.6
Height (cm)				
Childhood*	138.8	± 7.0	142.6	± 7.6

Adulthood	165.0	±	6.4	164.3	±	6.6
AUC†	148.2	±	16.3	147.7	±	16.7
Weight (kg)						
Childhood*	34.6	±	8.1	38.7	±	10.8
Adulthood*	71.0	±	19.7	83.1	±	24.0
AUC*	54.7	±	13.5	61.1	±	16.3
BMI (kg/m²)						
Childhood*	17.8	±	3.1	18.8	±	4.0
Adulthood*	26.1	±	7.1	30.7	±	8.4
AUC*	21.1	±	4.9	23.4	±	5.8
PBF (%)						
Childhood	21.0	±	7.8	22.0	±	9.4
Adulthood*	33.3	±	13.0	42.7	±	16.4
AUC*	25.8	±	8.7	29.2	±	10.6
WC (cm)						
Childhood*	63.4	±	8.2	65.8	±	9.2
Adulthood*	79.7	±	14.1	87.3	±	16.0
AUC*	67.7	±	11.6	71.3	±	12.8
SBP (mmHg)						
Childhood	100.8	±	8.3	101.2	±	8.7
Adulthood*	106.6	±	8.8	110.5	±	10.4
AUC	98.9	±	11.5	99.7	±	11.9
DBP (mmHg)						
Childhood	59.6	±	9.1	59.3	±	11.5
Adulthood*	67.2	±	8.5	68.9	±	10.2
AUC	60.8	±	7.8	61.3	±	8.0
HR (bpm)						
Childhood*	84.6	±	12.4	82.2	±	12.9
Adulthood	72.5	±	11.3	71.1	±	9.6
AUC*	71.4	±	9.7	69.8	±	9.2
Total Cholesterol (mg/dL)						
Childhood	174.3	±	28.8	170.5	±	31.0
Adulthood*	178.8	±	35.8	167.8	±	34.7
AUC*	156.8	±	32.5	151.4	±	29.8
TG (mg/dL)						
Childhood*	81.3	±	30.4	74.8	±	31.7
Adulthood*	110.2	±	54.7	86.9	±	50.5
AUC*	91.7	±	39.6	72.2	±	29.4
HDL-C (mg/dL)						

Childhood*	53.2	±	11.1	55.8	±	12.7
Adulthood*	52.5	±	14.1	49.7	±	11.5
AUC	47.8	±	10.4	49.3	±	10.3
TG/HDL-C ratio						
Childhood	1.6	±	0.8	1.5	±	0.9
Adulthood*	2.4	±	1.7	1.9	±	1.6
AUC*	2.0	±	1.2	1.5	±	0.9
LDL-C (mg/dL)						
Childhood	108.5	±	28.7	103.2	±	29.3
Adulthood	104.0	±	32.3	100.7	±	31.7
AUC	93.3	±	26.6	90.0	±	24.9
Glucose (mmol)						
Childhood	68.2	±	11.4	69.8	±	18.6
Adulthood*	86.5	±	10.3	90.2	±	25.8
AUC*	80.8	±	20.3	86.6	±	37.5
Insulin (mmol)						
Childhood*	12.1	±	9.1	17.3	±	13.8
Adulthood*	9.7	±	8.7	14.0	±	10.7
AUC*	9.0	±	6.0	13.2	±	7.5
†AUC values were divided by follow-up years.						
* p-value ≤ 0.05						

Abbreviations: LVMI, left ventricular mass index; HR, heart rate; SV, stroke volume; CO, cardiac output; SVR, systemic vascular resistance

Table 3

Table 3: <i>Racial Differences in Cardiac Measures</i>				
<u>Variable</u>	<u>White</u>		<u>Black</u>	
	<u>Mean</u>	<u>SD</u>	<u>Mean</u>	<u>SD</u>
LVMI (g/m^{2.7})*	29.9	± 8.7	33.8	± 8.8
HR (bpm)	71.7	± 10.8	71.0	± 9.8
SV (mL)	49.6	± 16.6	51.0	± 15.5
CO (L/min)	3.4	± 1.2	3.7	± 1.3
SVR (mmHg·min/L)	25.9	± 7.3	25.0	± 8.4
* p-value <0.05, N =556				

Abbreviations: LVMI, left ventricular mass index; HR, heart rate; SV, stroke volume; CO, cardiac output; SVR, systemic vascular resistance

Table 4: <i>Major Determinants of LVMI Overall and Stratified by Race</i>			
<u>Parameter</u>	<u>Combined</u>	<u>White</u>	<u>Black</u>
Intercept	3.49	14.33	26.18
Race	10.89	----	----
HR AUC	0.031	0.016	----
Race*HR AUC	-0.015	----	----
BMI z-score AUC	0.19	0.18	0.19
PBF AUC	0.013	0.014	0.013
SBP z-score AUC	0.12	0.12	0.12
R²	0.4	0.36	0.37

Abbreviations: HR, heart rate; BMI, body mass index; PBF, percent body fat by skin fold; SBP, systolic blood pressure

Figure 1

Abbreviations: LVMI, left ventricular mass index; BMI, body mass index; AUC, area under the curve

Figure 2

Abbreviations: LVMI, left ventricular mass index; SBP, systolic blood pressure; AUC, area under the curve

Figure 3

Abbreviations: LVMI, left ventricular mass index; HR, heart rate; AUC, area under the curve