

THE EFFECTS OF CARDIORESPIRATORY FITNESS AND TOTAL BODY FATNESS
ON ARTERIAL STIFFNESS IN HEALTHY OVERWEIGHT AND NON-OVERWEIGHT
CHILDREN AND ADOLESCENTS

A Thesis
by
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Abstract

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Objective: To determine whether increased central aortic stiffness is associated with decreased cardiorespiratory fitness or increased adiposity in healthy children. **Study Design:** seventy 7-to-17 y.o children (14.1 ± 3.0 y.o) were studied. Carotid-femoral pulse wave velocity (cfPWV) and brachial systolic blood pressure were compared according to quartiles of ratio-scaled VO_{2VT1} , %BF, and BMI. A separate ANOVA with Dunnett's T3 post-hoc tests for multiple comparisons of quartiles was conducted for each dependent variable, with each comparison evaluated at an alpha level of 0.05. **Results:** There was a significant difference between the quartiles on cfPWV, $F(3, 66) = 4.372, p = 0.007$, partial $\eta^2 = 0.166$, with First quartile (5.0 ± 0.8 m/sec) having greater cfPWV than the Fourth quartile (4.2 ± 0.7 m/sec) ($p = 0.021$). **Conclusion:** Our results are in line with previous literature suggesting that higher levels of cardiorespiratory fitness and lower BMI are associated with lower central arterial stiffness. In particular, VO_{2VT1} normalized for fat-free mass was inversely associated with cfPWV. Moreover, contrary to some of the present literature, our findings suggest there is no difference in arterial stiffness between the lowest and the highest %BF.

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My time at Appalachian State University truly began on the day Dr. Marco Meucci acknowledged me a mentee – I will continue to treasure your mentorship moving forward. You helped me become a better person through countless thins.

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Table of Contents

Abstract	iv
Acknowledgement	v
List of Tables	vii
List of Figures	viii
Foreword	ix
Abbreviation	x
Introduction.....	1
Subjects and methods.....	3
Results.....	7
Discussion.....	12
References.....	17
Vita.....	26

List of Tables

Table 1. Descriptive characteristics of subjects	8
Table 2. Descriptive characteristics of subjects: OW vs. NW	8
Table 3. Means and standard deviations by quartiles: cfPWV (m/sec)	9
Table 4. Means and standard deviations by quartiles: BSBP (mmHg).....	9

List of Figures

Figure 1. VO_{2VT1} (ml/FFM kg/min) by Quartiles	10
Figure 2. BMI by Quartiles.....	10
Figure 3. Body fat percentage (%) by Quartiles	11
Figure 4. Body fat percentage (%) distribution	11

Foreword

This thesis will be submitted to *The Journal of Pediatrics*, an international peer-reviewed journal owned and operated Elsevier Inc.; it is formatted according to the instructions to authors for that journal.

Abbreviations

Body fat percentage (%BF)

Body mass index (BMI)

Brachial diastolic blood pressure (BDBP)

Brachial systolic blood pressure (BSBP)

Pulse wave velocity (PWV)

Carotid-femoral pulse wave velocity (cfPWV)

Fat-free mass (FFM)

Lean mass (LM)

First ventilator threshold (VT1)

Volume of oxygen uptake (VO_2)

Volume of oxygen uptake at first ventilatory threshold (VO_{2VT1})

Normal weight (NW)

Overweight (OW)

Introduction

This is a part of Pediatric Exercise Physiology Lab's Pediatric Observational Study, led by Marco Meucci, PhD.

Obesity is a risk factor for cardiovascular disease (CVD) associated with a multitude of cardiac complications and known comorbidities and metabolic risk factors in adults^{1,2} that stem from childhood.³⁻⁵ Although physical manifestation is less prevalent and obscured in youth, childhood obesity has been associated augmented risk for adulthood CVD.⁶

Carotid-femoral pulse wave velocity (cfPWV) is considered the gold standard assessment for estimating central arterial stiffness and a marker and predictor of cardiovascular risk in hypertensive patients and a strong indicator of cardiovascular health in young adults and children.^{7,8} Several risk factors such as aging, body fatness, hypertension, diabetes, and dyslipidemia have been identified as determinants of arterial stiffness in adults.⁹⁻¹¹ In adolescents, body fatness has been associated with increased arterial stiffness.¹² In children, higher levels of cardiorespiratory fitness (CRF) ratio-scaled by lean mass (LM) or fat-free mass (FFM) is related to lower levels of arterial stiffness independent of %BF.¹³⁻¹⁵

Studies have alluded to potential mechanisms whereby improved CRF may improve arterial stiffness in young adults¹⁶ and children,^{13-15,17} although the nature of their associations is yet to be conclusive. Further, CRF may affect arterial stiffness through a beneficial impact on body composition¹⁸ which itself is a strong determinant of arterial stiffness and overall cardiovascular health in young individuals.^{3,19}

Peak oxygen uptake (VO_{2peak}) is still considered the gold standard measurement of CRF. However, successful attainment of true VO_{2peak} can be difficult in children and obese individuals since it depends heavily on the ability to overcome effort, physical discomfort, and shortness of

breath, and fatigue that occurs at high exercise intensities.^{20,21} Thusly, submaximal indicators of CRF may be better tolerated and more reflective of the exercise intensity at which obese children would undertake in the real world.²² Research showed that an efficient and reproducible way to assess CRF in children is measuring the VO₂ at the point of first ventilatory threshold (VO_{2VT1}).^{23,24}

In view of these considerations, we investigated how VO_{2VT1} and %BF are associated with cfPWV in 7-to-17 year-old boys and girls of varying %BF. We hypothesized to observe an inverse association between VO_{2VT1} and cfPWV, and a direct association between %BF and cfPWV.

Subjects and Methods

Study Population

Seventy children aged 7-to-17 years were recruited from the local community. Both the children and parents/guardians gave written informed consent for the project, which was approved by the Ethics Committee of Appalachian State University. Subjects were admitted to the study if they were between the ages of 7 and 17 years of age. Subjects did not take any medications at the moment of recruitment or had any medical conditions including diabetes, heart, respiratory or renal disease as reported on the health history questionnaire. There was no control for female menstrual cycle. Participants were asked to avoid drinks containing caffeine such as soft drinks or soda 3 hours before the test, not perform exercise the day of the test or eat large meals 4 hours prior to the test. Assessments were performed in the Pediatric Exercise Physiology laboratory, at Appalachian State University, Boone NC. Total time required for the measurements was approximately 90 minutes. Measurements were performed between 8:00 to 11:00 AM or 4:00 to 6:00 PM.

Anthropometrics and Body Composition

Height was measured with a stadiometer to the nearest 0.1 cm. BMI was calculated using the body weight and height ($BMI = (kg/m^2)$). Fat mass (FM) and fat free mass (FFM) was assessed in all subjects by using air displacement plethysmography (BodPod technology, COSMED, Italy). Subjects wore tight fitting clothes, a swimmers cap, and no shoes or jewelry during the body composition assessment. Body weight (kg) was obtained using the BodPod scale and body volume was measured inside the BodPod chamber. Children were instructed to keep feet flat on the floor, hands flat on their thighs and to remain as still as possible throughout the test. This measurement was taken twice and results were averaged.

Cardiovascular Measurement

Blood pressure and arterial stiffness were assessed with an automated system SphygmoCor XCEL device (SphygmoCor, AtCor Medical, Inc.). Brachial blood pressure was taken from right arm after 5-minutes of quiet rest lying supine in a dimly lit room. Three measurements were made at 1-minute intervals and the mean of the last two was used for analysis. After an additional 1-minute of rest, the distance between the two arterial sites were measured on the body using a tape measure, and cfPWV was calculated as the distance (m) divided by the pulse wave transit time (sec) measured via applanation tonometry at the carotid site and an automatic oscillometric blood pressure cuff positioned around the middle of the right thigh. Three separate distances were measured to determine the vascular distance (cm): 1) carotid artery to sternal notch, 2) sternal notch to cuff, and 3) femoral artery to the top of the thigh blood pressure cuff. This measurement was taken in triplicate with the average used for analysis. Each subsequent measurement was taken with minimal time between. Valid waveform consistency and amplitude were assessed for passing quality control before an average was taken to ensure accuracy between trials. If values differed by more than ± 0.3 m/sec, an additional measurement was performed.

Cardiopulmonary Exercise Test

CRF was assessed by a maximal incremental exercise test on an electronically braked cycle ergometer (Lode Corival, Lode BV, Groningen, Netherlands). The protocol included 2-minute resting period sitting on the ergometer, a 1-minute unloaded pedaling at 0 W, and an incremental exercise period with increase of workload by 15 W/min until voluntary exhaustion. The participants were asked to keep the cadence of 65-70 during the test. The test was terminated

when the participant was unable to keep the cadence of 50 or required to stop. Participants were verbally encouraged to exercise until voluntary exhaustion.

Respiratory gas exchange was assessed directly by breath-by-breath method via calibrated respiratory gas analysis system (K5 Wearable Metabolic Technology, Cosmed, Chicago, IL, USA) which was calibrated per the manufacturer's recommendations. Heart rate (HR) during the exercise test was recorded using GARMIN HR chest belt (GARMIN, USA). Rate of perceived exertion was assessed using a modified Borg 0-10 scale was recorded at 15s prior to the end of every stage.

First Ventilatory Threshold

VO_2 (ml/kg/min) at VT1 was graphically determined by the V-slope method as employed by Beaver et al. (1986).²⁵ Ventilation data were employed as a secondary predictor of $\text{VO}_{2\text{VT1}}$ to identify the first rise in the ventilatory equivalent for O_2 (VE/VO_2) without a concomitant rise in ventilatory equivalent for CO_2 . The first point of consistent departure of VCO_2 from linearity will be visually selected by two investigators and the VO_2 corresponding to this point will be used as the VT1. When the choice of the threshold differs by more than 30-s between the investigators, a third independent investigator was consulted. $\text{VO}_{2\text{VT1}}$ was defined as $\text{VO}_{2\text{VT1}}$ ml/BM/min and $\text{VO}_{2\text{VT1}}$ ml/FFM/min. Breath-by-breath data were smoothed in 6-steps and time-averaged by 10s. Peak aerobic capacity ($\text{VO}_{2\text{peak}}$) was averaged over 30s during the last minute of the exercise test when the highest VO_2 value was achieved.

Statistical Analysis

Differences in cfPWV and blood pressure measurements between quartiles of ratio-scaled $\text{VO}_{2\text{VT1}}$, %BF, and BMI were assessed using a separate ANOVA with Dunnett's T3 post-hoc tests for multiple comparisons of quartiles for each dependent variable, with each comparison

evaluated at an alpha level of 0.05. Independent differences were assessed using independent samples T-test without assumption for equal variances. All analyses were performed using SPSS Software, IBM. The level of significance was set at $p < 0.05$. Results are expressed as mean \pm SD.

Results

Seventy subjects (36 girls, 34 boys) were included in this study. Eighteen subjects were considered overweight or obese per US Centers for Disease Control and Prevention BMI-for-age growth charts. See table 1 for subject characteristics.

There was a significant difference between the VO_{2VT1} normalized for FFM quartiles on cfPWV, $F(3, 66) = 4.372$, $p = 0.007$, partial $\eta^2 = 0.166$, with First quartile (4.99 ± 0.73 m/sec) having greater cfPWV than the Fourth quartile (4.24 ± 0.69 m/sec) ($p < 0.05$) (Figure 1). Post-hoc multiple comparisons yield non-significant differences between the VO_{2VT1} normalized for BM quartiles ($p > 0.05$) (Table 3).

There was a significant difference between the BMI quartiles on cfPWV, $F(3, 66) = 4.680$, $p = 0.005$, partial $\eta^2 = 0.175$, with Fourth (5.1 ± 0.9 m/sec) and Third (4.9 ± 0.7 m/sec) quartiles having greater cfPWV than the First quartile (4.3 ± 0.5 m/sec) ($p < 0.05$) (Figure2). Conversely, there was not a significant difference between the %BF quartiles on cfPWV, $F(3, 66) = 1.643$, $p = 0.188$, $\eta^2 = 0.069$ (Figure 3).

BSBP was not significantly associated with any of the parameters (Table 4).

Table 1

<i>Table 1. Descriptive characteristics of subjects</i>			
Characteristics (N=70)	All (N=70)	Children (N=16)	Adolescents (N=54)
Age (years)	14.1 ± 3.0	10.0 ± 2.3	15.3 ± 1.10**
Height (cm)	161.2 ± 15.5	144.0 ± 16.1	166.2 ± 11.3**
Body mass (BM; kg)	57.3 ± 21.2	43.2 ± 18.8	61.5 ± 20**
Body fat percentage (%)	23.4 ± 11.5	25.7 ± 15.8	22.7 ± 10.0
Body mass index	21.5 ± 5.4	19.8 ± 5.4	21.9 ± 5.3
Fat free mass (FFM; kg)	42.8 ± 13.2	30.2 ± 10.7	46.5 ± 11.5**
VO _{2peak} ml/BM kg/min	34.1 ± 8.4	34.8 ± 11.3	33.9 ± 7.5
VO _{2peak} ml/FFM kg/min	44.4 ± 7.3	46.6 ± 8.5	43.7 ± 6.8
VO _{2VT1} ml/BM kg/min	18.8 ± 5.5	21.8 ± 7.3	17.9 ± 4.6
VO _{2VT1} ml/FFM kg/min	24.6 ± 5.8	29.4 ± 6.7	23.2 ± 4.8**
Resting BSBP (mmHg)	112.0 ± 10.9	109.3 ± 9.5	112.8 ± 11.3
Resting BDBP (mmHg)	63.2 ± 7.7	63.1 ± 5.8	63.3 ± 8.3
cfPWV (m/sec)	4.73 ± 0.73	4.2 ± 0.7	4.90 ± 0.71**

***. Sig. (2-tailed) at the 0.001 level; independent samples test, equal variances not assumed*

Table 2

<i>Table 2. Descriptive characteristics of subjects: OW vs NW</i>		
Characteristics (N=70)	OW (N=18)	NW (N=52)
Age (years)	13.5 ± 2.8	14.3 ± 3.1
Height (cm)	163.9 ± 14.7	160.2 ± 15.8
Body mass (BM; kg)	76.4 ± 26.6	50.7 ± 14.1**
Body fat percentage (%)	35.8 ± 10.8	19.1 ± 8.2**
Body mass index	27.7 ± 6.1	19.3 ± 2.8**
Fat free mass (FFM; kg)	48.5 ± 15.8	40.8 ± 11.7
VO _{2peak} ml/BM kg/min	29.2 ± 6.7	35.8 ± 8.4**
VO _{2peak} ml/FFM kg/min	45.3 ± 5.8	44.0 ± 7.7
VO _{2VT1} ml/BM kg/min	15.8 ± 3.2	19.9 ± 5.8**
VO _{2VT1} ml/FFM kg/min	24.5 ± 6.1	24.9 ± 5.2
Resting BSBP (mmHg)	116.6 ± 14.7	110.4 ± 8.9
Resting BDBP (mmHg)	65.9 ± 9.1	62.3 ± 7.1
cfPWV (m/sec)	5.0 ± 1.0	4.6 ± 0.6

***. Sig. (2-tailed) at the 0.001 level; independent samples test, equal variances not assumed*

Table 3

<i>Table 3. Means and Standard Deviations By Quartiles: cfPWV (m/sec)</i>				
N=70	First	Second	Third	Fourth
VO _{2peak} ml/BM kg/min	5.1 ± 1.0	4.6 ± 0.7	4.9 ± 0.5	4.4 ± 0.6**
VO _{2peak} ml/FFM kg/min	4.9 ± 0.8	4.9 ± 0.7	4.6 ± 0.8	4.6 ± 0.6
VO _{2VT1} ml/BM kg/min	5.1 ± 0.9	4.9 ± 0.6	4.6 ± 0.5	4.3 ± 0.7
VO _{2VT1} ml/FFM kg/min	5.0 ± 0.7	4.8 ± 0.5	4.9 ± 0.8	4.2 ± 0.7*
Body fat percentage (%)	4.6 ± 0.6	4.7 ± 0.6	4.6 ± 0.5	5.1 ± 1.0
BMI	4.3 ± 0.5	4.7 ± 0.6	4.9 ± 0.7*	5.1 ± 0.9*

*. The mean difference compared with respective First is significant at the 0.05 level when equal variance is not assumed.

**. The mean difference compared with respective First is significant at the 0.05 level when equal variance is assumed.

Table 4

<i>Table 4. Means and Standard Deviations By Quartiles: BSBP (mmHg)</i>				
N=70	First	Second	Third	Fourth
VO _{2peak} ml/BM kg/min	114.4 ± 15.9	109.8 ± 8.8	111.9 ± 7.9	112.2 ± 9.9
VO _{2peak} ml/FFM kg/min	110.8 ± 10.2	112.6 ± 14.5	113.2 ± 10.0	111.5 ± 8.7
VO _{2VT1} ml/BM kg/min	113.8 ± 16.1	113.9 ± 8.5	110.7 ± 8.1	109.7 ± 9.7
VO _{2VT1} ml/FFM kg/min	113.3 ± 10.2	114.2 ± 9.1	110.7 ± 14.9	110.0 ± 10.9
Body fat percentage (%)	114.8 ± 8.5	108.9 ± 9.5	108.1 ± 8.7	116.1 ± 14.7
BMI	108.7 ± 10.5	111.2 ± 8.4	110.2 ± 7.3	118.2 ± 14.7

*. The mean difference compared with respective First is significant at the 0.05 level when equal variance is not assumed.

**. The mean difference compared with respective First is significant at the 0.05 level when equal variance is assumed.

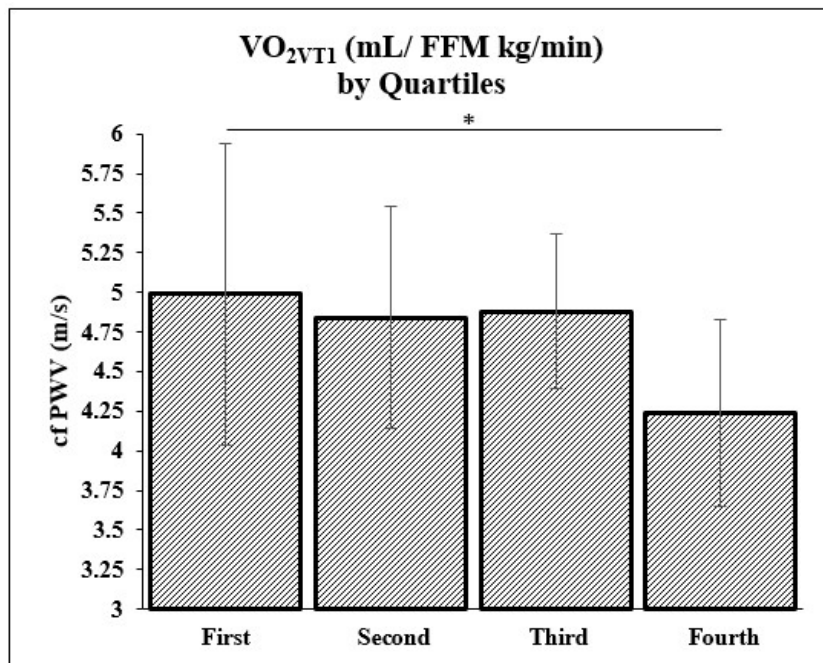


Figure 1. Differences in cfPWV between VO_{2VT1} normalized for FFM quartiles. * $p < 0.05$

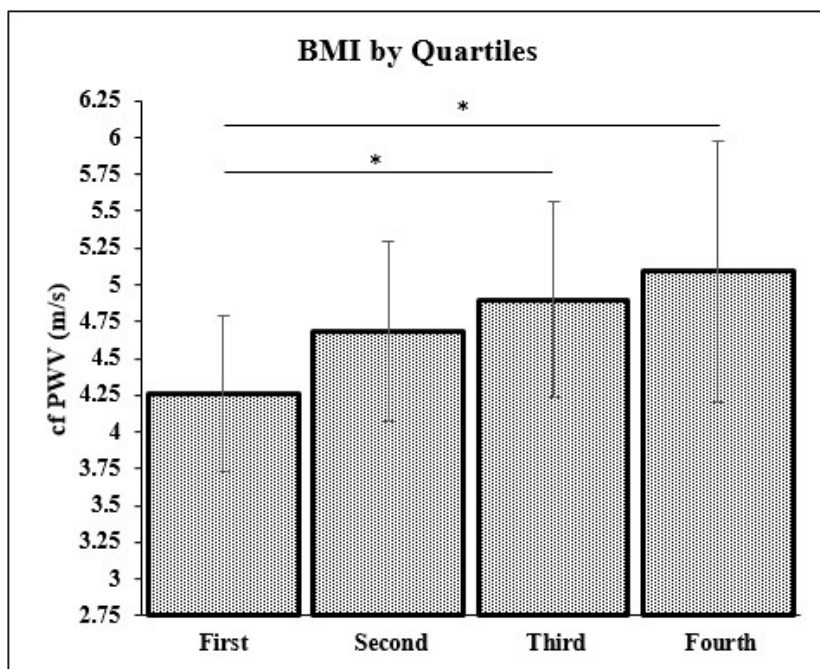


Figure 2. Differences in cfPWV between body mass index (BMI) quartiles. * $p < 0.05$

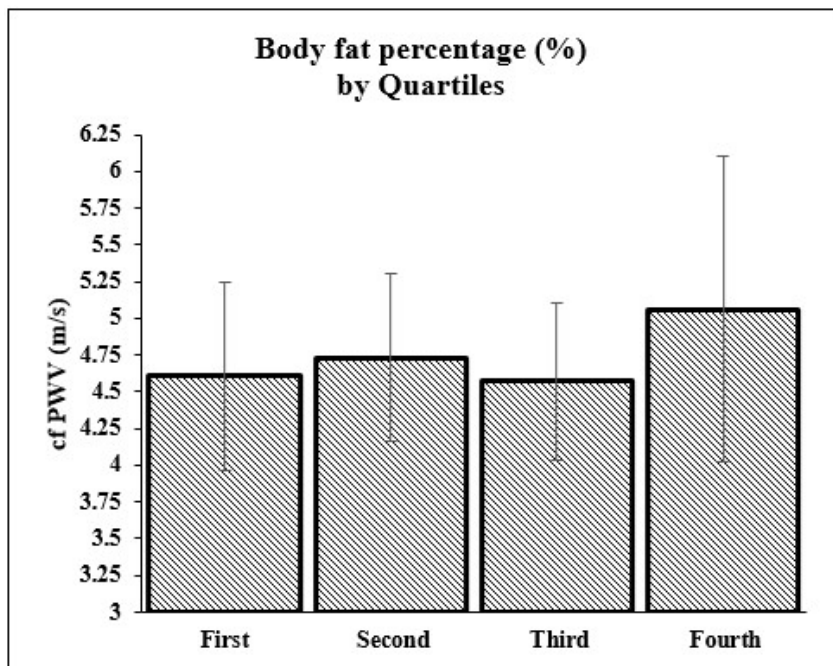


Fig. 3. Differences in cfPWV between body fat percentage (%) quartiles. * $p < 0.05$

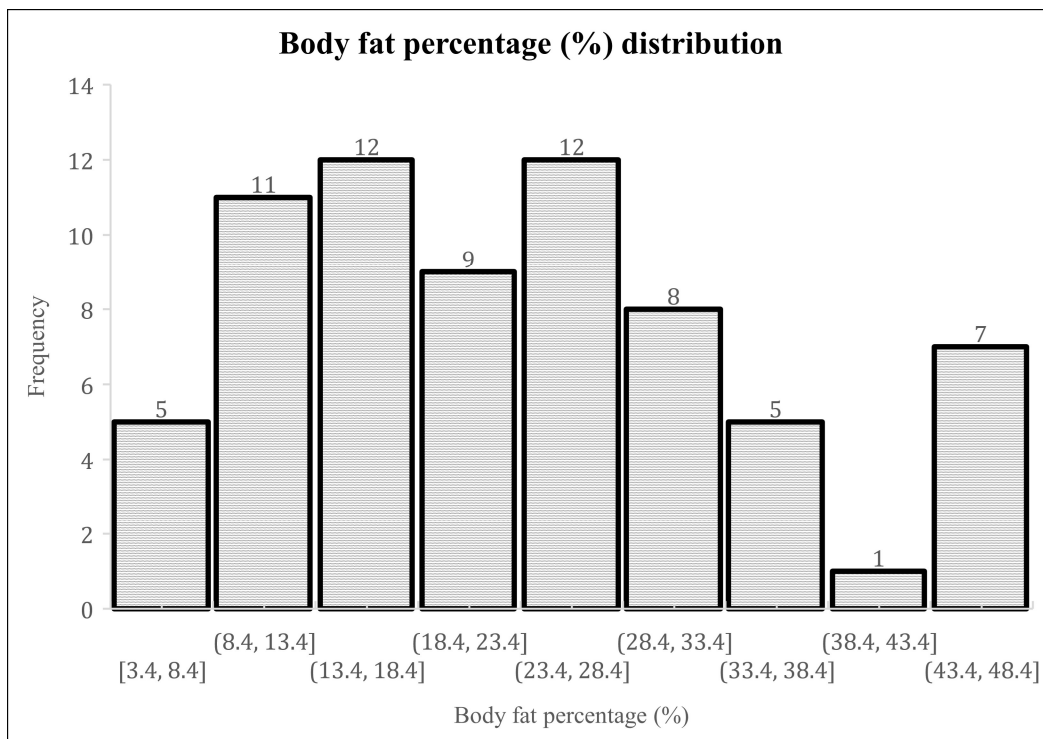


Fig. 4. Sample distribution based on body fat percentage (%).

Discussion

This study aimed to investigate the associations of arterial stiffness with CRF and body fatness in children aged 7-to-17-years. Our results are in line with previous literature showing that higher levels of CRF and lower BMI are associated with lower central arterial stiffness.^{13-15,17} Moreover, contrary to some of the present literature,¹¹ our findings show there is no difference in arterial stiffness between the lowest and the highest %BF. Furthermore, our results reported no differences with SBP with regards to CRF or %BF, which is in contrast with previous literature.^{26,27}

In children, higher levels of CRF ratio-scaled by LM or FFM is related to lower levels of arterial stiffness independent of %BF,¹³⁻¹⁵ which may allude to the most physiologically relevant associations of CRF with arterial stiffness. In healthy children, muscle mass is responsible for increased energy demands during exercise and enhanced venous return which allows left-ventricular end-diastolic volumes and cardiac output to be maintained.²⁸ On the other hand, lower arterial stiffness may increase myocardial flow reserve and myocardial perfusion²⁹ and thereby improving CRF, although the corresponding evidence in children is yet to be conclusive.³⁰ Likewise, Farpour-Lambert et al. (2009)³¹ reported that a 6-month long aerobic exercise program can improve VO_{2peak} and reduce arterial stiffness, carotid intima-media thickness, SBP and total and abdominal fat in overweight and obese prepubertal children, further supporting the potential %BF-independent cardioprotective properties of CRF in children. In line with this hypothesis, previous studies conducted in our laboratory showed that 8 weeks of play-based physical activity can decrease arterial stiffness and mean blood pressure, and increase VO_{2peak} in overweight children even without changes in body weight or body composition.³² However, given the short duration of most studies, it was suggested that the exercise-related

changes in arterial stiffness in children may have been primarily related to the beneficial changes in BP⁷ and rather than due to changes in the arterial structure composition. Noteworthy, while previous studies have also reported inverse and no correlation between self-reported physical activity and arterial stiffness in children,^{9,17} such findings are not contrary to our narrative as voluntary activity behavior has been suggested to lack the duration and intensity associated with adequate stimulus for the enhancement of VO_{2peak} in children.³³ Present study is limited by its cross-sectional design, and the mechanisms whereby improved VO_{2VT1} may improve arterial compliance in children cannot be determined. While the occurrence of the VT1 at a higher intensity is an indicator of an enhanced CRF,³⁴ its mechanistic basis in children remains to be elucidated. In particular, little normalcy regarding VT1 is known in healthy children.²⁴ Investigations have reported an age-related trend of the VO_{2VT1} occurring at a higher % of VO_{2peak} in youth.³⁵

Potential inadequacy of ratio scaled CRF parameters in growing children should be acknowledge.³⁶ VO_{2max} is a function of FFM in adults³⁷ as well as it is in children,²⁸ although regardless of the cause, high total body weight has shown to be detrimental to VO_{2max} .³⁸ While scaling VO_{2peak} by total lean body mass is superior to scaling by total body mass³⁹ and, as previously mentioned, may provide the most physiologically relevant information on the associations of CRF with arterial stiffness in children,²⁸ it still ultimately produces a size-dependent variable that penalizes heavier, more mature or overweight, individuals. In view of these considerations, exacerbated cfPWV among the first VO_{2VT1} normalized by FFM quartile (17 adolescents), relative to the fourth quartile (10 preadolescents, 8 adolescents), may be a function of age-related increases in FFM and cfPWV. Our sample of only adolescents was not large enough to produce an adequate effect size (partial $\eta^2=0.034$). Nonetheless, as determination

of FFM in youth can be relatively time-consuming and expensive, BM will probably continue as the primary scaling variable. This potential inverse association between cfPWV and VO_{2VTI} normalized by FFM requires further investigation.

Present studies report equivocal results alluding to the role that childhood obesity may play in the progression of PWV, although the discrepancy in results between studies may be attributable to differences in the methodology for the assessment of PWV.^{9,40} More closely related to our methodology, Sakuragi et al. (2010)⁴¹ reported that %BF and BMI are positively and independently associated with cfPWV; furthermore, the authors stated that increased body mass and adiposity and decreased CRF indicated by a 20-m shuttle run – susceptible to a significant error compared to the measured VO_{2peak} ⁴² – were independently associated with arterial stiffening in healthy prepubescent children. Moreover, in spite of equivocal evidence, the weight of evidence seems to align with the findings that fat mass and arterial stiffness are directly associated in youth.^{11,12,43}

Investigators have previously discussed several possibilities, such as endothelial dysfunction,^{44,45} as mechanisms whereby increased adiposity may promote arterial stiffening in children.^{3,46,47} In particular, high adiposity and sedentary lifestyle in childhood⁴⁷ are associated with the early development of arterial stiffness, dyslipidemia and insulin resistance³ which can increase the risk of future CVD in adulthood.⁴⁸ However, since insulin resistance has been shown to increase greatly after puberty,⁴⁹ its effects may be dampened in particularly younger children. Additionally, obesity-related decrease in adiponectin⁵⁰ and increases in inflammatory cytokines derived from adipose tissue may also be involved in the development of endothelial dysfunction.^{51,52} Increased BMI and adiposity are accompanied by increases in HR, ambulatory blood pressure^{27,53} and metabolic risk factors that mediate alterations in arterial function.^{54,55}

Evidently, elevated blood pressure and hypertension are a well-documented finding in children with obesity⁵⁶⁻⁵⁸ and has also been shown to be associated with an increase with PWV in adults.^{59,60} It was not unexpected that our results reported no significant differences in cfPWV between %BF quartiles, because our subjects were otherwise healthy without known medical conditions indicative of premature CV and metabolic disease, and this may be further supported by the non-significant changes in BSBP with regards to %BF.

Due to the multifaceted nature of studying cardiovascular and CRF in children during physiological development, some limitations need to be addressed. First and foremost, subjects were not separated by sex or biological age. Puberty denotes the onset of rapid physical development and maturation⁶¹ including cardiovascular and cardiorespiratory functions and age-related increases in PWV. Prepubertal girls have been shown to possess intrinsically greater central PWV when compared to age-matched boys when normalized by female sex hormones 17 β -Estradiol and Progesterone.⁶² This sexual dimorphism diminished in the postpubertal group with an increase in boys' central PWV and a decrease in girl's central PWV. Thusly, boys may increase cfPWV while girls may decrease cfPWV during puberty. Therefore, a blunting effect within groups could be responsible for the minimal differences overserved in cfPWV due to both males and females being grouped. In parallel, boys' VO_{2peak} increases almost linearly with age, and similarly but less consistently in girls' values, independent of body mass^{63,64} with some studies indicating a plateauing in girls' VO_{2peak} from ~14 years, where such of this sex difference can be attributed to intrinsic sex differences in muscle mass development during adolescent years.⁶⁵

The novelty of our findings lies atop our reproducible and modifiable methodology that employs the field's gold standard tools. Future studies are needed to investigate the effects of sex

differences and pubertal status on relationships between VO_{2VT1} , body fatness and central arterial stiffness. Controlling for biological maturational status, anthropometric parameters, and exercise test protocols within used methodology is necessary. Recognizing whether the subjects were experiencing, or already have experienced puberty as well as being able to control for the menstrual cycle will provide important information alluding to the effects of age and overweight status with cardiovascular health in clinical pediatrics. Further adjustments for other traditional cardiovascular risk factors such as fasting LDL, HDL and total cholesterol, triglycerides and plasma glucose levels, should also be considered. Additionally, longitudinal data are necessary to analyze causality and confounder effects.

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Vita

On November 29, 1992, Hwan Kim was born in Seoul, South Korea, to Young Mi Jo and Seong Joon Jo. He graduated from Athens Drive High School in Raleigh, NC, in June 2012. The following fall, he entered Appalachian State University to study Mathematics; and by the fall of 2014, he had officially begun studying Exercise Science.

After graduating in May 2017, he decided to take a short hiatus from his academics. In January 2018, he began a graduate assistantship in research in the Exercise Science Department at Appalachian State University and began his study toward a Master of Science degree. In particular, he involved himself with projects in part of Human Performance Lab and Pediatric Exercise Physiology Lab under Dr. Marco Meucci's supervision. In December 2019, he will be graduating with a Master of Science in Exercise Science.