



Original Research

Fitness-Fatness Index on Predicting Cardiovascular Outcomes in College-Aged Students

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Abstract

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Cardiovascular diseases (CVDs) represent a leading global health burden, with risk factors often emerging during college years. Traditional assessment methods like Body Mass Index (BMI) have significant limitations in predicting health outcomes. This study evaluated the relationship between Fitness-Fatness Index (FFI) and cardiovascular outcomes in college-aged students compared to traditional measures. A correlational-predictive non-experimental survey was conducted with 218 healthy, non-smoking young adults (109 male, 109 female; age 20.8±2.9 years). Participants underwent comprehensive anthropometric assessments, body composition analysis, aerobic fitness tests, and cardiovascular measurements, including brachial/central blood pressure and arterial stiffness parameters. Multiple linear regression analysis revealed that metabolic equivalents (METs) were the strongest predictor of arterial stiffness parameters, with the highest beta coefficients for augmentation pressure ($\beta=-0.407$, $p=0.002$), augmentation index ($\beta=-0.398$, $p<0.001$), and pulse wave velocity ($\beta=-0.234$, $p=0.006$). The waist-to-height ratio was the strongest predictor of adjusted brachial systolic blood pressure ($\beta=0.159$, $p=0.016$), while FFI was the strongest predictor of central diastolic blood pressure ($\beta=-0.306$, $p<0.001$). These findings indicate cardiorespiratory fitness is a key determinant of arterial stiffness in college-aged individuals, challenging the hypothesis that FFI would outperform traditional measures. Incorporating both METs and FFI may provide more comprehensive cardiovascular risk assessment in this population.

Keywords: Arterial stiffness, cardiorespiratory fitness, pulse wave velocity, young adults, vascular health

Introduction

Chronic diseases are responsible for 75% of global deaths, mainly driven by lifestyle-related factors such as obesity and low physical activity.¹ Cardiovascular diseases (CVDs) are a leading cause of death globally, accounting for around one-third of all deaths.² The growing prevalence of these diseases highlights the urgent need for global action.² Despite initiatives aiming to reduce premature mortality from non-communicable diseases (NCDs), the decline in death rates has not been sufficient to meet global targets.³ Lifestyle choices such as poor diet, lack of exercise, and physical inactivity are strongly linked to the development of chronic diseases, including cardiovascular disease, respiratory diseases, and diabetes.⁴ Addressing these modifiable risk factors could reduce the burden of chronic conditions. Increasing physical activity, improving diet, and reducing smoking can help lower the risk of these diseases and promote overall health, emphasizing the importance of lifestyle changes in disease prevention.⁵ Given the critical role

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of physical fitness and body composition in preventing these conditions, accurate assessment methods are crucial for identifying individuals at risk and monitoring the effectiveness of interventions.

Traditional assessment methods, such as Body Mass Index (BMI), have significant limitations in predicting health outcomes. BMI, calculated by dividing an individual's weight in kilograms by height in meters squared, categorizes individuals as underweight, normal weight, overweight, or obese.⁶ However, BMI fails to account for crucial factors such as age, sex, and overall fitness level.⁷ More importantly, BMI cannot distinguish between fat distribution and muscle mass, despite evidence that abdominal fat provides more meaningful insight into cardiovascular disease risk and obesity than total body weight alone.⁶

As an alternative measure, cardiorespiratory fitness (CRF) assessed through VO_2 max has emerged as the gold standard for determining cardiovascular health. VO_2 max measures gas exchange during strenuous physical exertion, with higher values indicating a more robust cardiovascular system and a lower risk of cardiovascular disease.⁸ However, VO_2 max also has limitations, particularly in its ability to directly quantify body composition factors such as fat distribution and the ratio of fat to lean muscle mass. Research has shown that while body fat percentage and VO_2 max are negatively correlated in female athletes, this relationship is not statistically significant,⁹ suggesting the need for more comprehensive assessment tools.

The Fitness Fatness Index (FFI) attempts to address these limitations by integrating both CRF and body composition measures. Calculated as CRF divided by the waist-to-height ratio, FFI may provide a more comprehensive assessment of physical health. Recent studies have demonstrated that FFI is associated with reduced risk of cardiovascular disease, all-cause mortality, cancer-specific mortality, and Alzheimer's-specific mortality.¹⁰ Furthermore, evidence suggests that FFI outperforms traditional measures in predicting various health outcomes,¹¹⁻¹³ making it a promising metric for comprehensive health assessment.

College students, typically aged 18 to 24, represent a critical population for early intervention strategies. The transition period from structured environments to independent decision-making often results in significant changes in diet, physical activity, and weight management behaviors.¹⁴ Although cardiovascular disease typically develops later in life, between ages 57 and 76,¹⁵ risk factors frequently emerge during college years. More than 50% of college-aged students in the United States have at least one CVD risk factor, including smoking, high blood pressure, obesity, and physical inactivity.¹⁶ Despite this high prevalence of risk factors, cardiovascular health in this population remains understudied, creating a significant gap in preventive health research.¹⁶

While FFI has demonstrated utility in predicting cardiovascular outcomes and mortality in adult populations, its effectiveness in younger populations remains inadequately explored.¹⁷ This study aims to address this research gap by evaluating the relationship between FFI and cardiovascular outcomes, specifically in college-aged students. We hypothesize that FFI will demonstrate stronger associations with cardiovascular risk factors than traditional measures such as BMI or VO_2 max alone in this population. This study represents a secondary analysis of data collected through the university's Exercise is Medicine campus health assessment program. Our findings could provide valuable insights for developing targeted early intervention strategies and improving cardiovascular health assessment protocols for young adults.

Methods

Participants

The study's methodology was quantitative, and the research design employed a non-experimental correlational-predictive study using secondary data. An a priori sample size was estimated using G*power 3.1.9.7. An *f*-test with an effect size of .15, an alpha error probability of .05, and a statistical power of .95 estimated that a sample size of 89 was needed. However, all data in the secondary data set were used.

The Institutional Review Board application underwent expedited review, and the Institutional Review Board approved all procedures at the host institution. These procedures followed the Declaration of Helsinki for human studies of the World Medical Association.¹⁸ Additionally, this research was conducted in full compliance with the ethical standards of the *International Journal of Exercise Science*.¹⁹ Written consent was required, and all participants provided written informed consent before testing. The host institution has become recognized by the American College of Sports Medicine as an "Exercise is Medicine" campus. As such, the student health clinic asks about physical activity behaviors and refers interested students who would like advice on an exercise program to participate in further health assessments to the University fitness specialists. Anthropometric, body composition, and aerobic fitness are assessed.

Healthy, nonsmoking young men and women aged 18-30 years were recruited from the University campus using the aforementioned method. Subjects were excluded if they had known cardiovascular, pulmonary, renal, peripheral vascular disease, or metabolic disease, or had symptoms suggestive of these diseases. Although the number of exclusions was not systematically documented, exclusions were anticipated to be minimal, given the young and healthy college population. Pregnant women and current smokers were excluded from the study. Biological sex was self-reported by participants from a list including male, female, or rather not say. Participants were invited to the laboratory for a single visit.

Protocol

Anthropometric assessments.

To prepare for the assessments, participants fasted for at least five hours, refrained from caffeine and alcohol for 24 hours, and avoided unaccustomed physical activity 24 hours before their visit. Body mass to the nearest 0.01 kg and stature to the nearest 0.1 cm were measured as described in the Anthropometric Standardization Reference Manual.²⁰ Height was measured with the subject standing barefoot using a stadiometer (Tree LS-PS 500). The digital scale attached to the stadiometer measured body weight with minimal clothing. Waist and hip circumference were determined while the subject stood with a Gulick II 150 cm anthropometric tape (model 67020) and reported to the nearest 0.1 cm. Waist circumference was measured with the anthropometric tape parallel to the floor and immediately above the iliac crest, with readings taken at the end of a normal exhalation.²¹ Hip circumference was assessed at the level of the most substantial protrusion of the buttocks.²⁰ Body Mass Index (BMI) was calculated using formula 1, while the fat-fit index (FFI) was calculated using formula 2.¹¹ FFI scores commonly range from 10 to 50 on a continuous scale, with higher scores being better. Waist-to-height ratio was calculated by dividing the waist in cm by height in cm.

Formula 1. $\frac{\text{Weight (kg)}}{\text{Height (m)}^2}$

Formula 2. $\frac{\text{METS}}{\text{Waist-to-height ratio}}$

Body Composition was determined via whole-body air displacement plethysmography (BOD POD, COSMED). BOD POD is valid when compared to underwater weighing.²² Participants were asked to wear a bathing suit or tight-fitting shorts and remove their shirts and jewelry to test. They were weighed again using the scale associated with the BOD POD device. They were then asked to place a silicone swim cap (Aegend) on their heads to cover their hair and sit in the BOD POD for 2 to 3 measurements of 50 seconds each.

Cardiovascular Assessments.

Participants were asked to lie supine for 20 minutes to achieve hemodynamic stability. To ensure hemodynamic stability, two blood (BP) readings were taken with an automated cuff (Welch Allyn, Connex ProBP 3400). Following this, measurements of brachial BP, central BP, pulse wave analysis (PWA), and pulse wave velocity (PWV) were assessed.

Central/brachial BP, and PWA measurements were taken using the SphygmoCor XCEL™ (AtCor Medical, Sydney, NSW, Australia) employing validated methodology.²³ Central BP is automatically derived from 10 sequential, high-quality waveforms, which undergo a validated generalized transfer function to generate the corresponding central aortic pressure waveform. Augmentation Index (Aix) is calculated as the difference between the first and second systolic peaks of the ascending aortic waveform expressed as a percentage of the central pulse pressure and normalized for a heart rate (HR) of 75 beats min^{-1} (Aix@HR75). Aix can also be expressed in absolute terms as the augmentation pressure (Ap). Three measurements were taken 5 minutes apart.

Carotid-femoral PWV was determined by recording the carotid and femoral artery waveforms simultaneously. Distances from the carotid sampling site to the suprasternal notch and from the suprasternal notch to the femoral cuff were measured. The distance from the femoral arterial pulse to the femoral cuff was obtained and subtracted from the total distance (D; in meters). The time (t; in seconds) between the onset of femoral and carotid waveforms was determined as the mean from 10 consecutive cardiac cycles. Carotid femoral PWV was calculated as follows: cfPWV = D/t ($\text{m} \cdot \text{s}^{-1}$).

Aerobic Fitness Assessment.

Participants were directed to the metabolic cart for a $\text{VO}_{2\text{max}}$ assessment following a commonly used ramp protocol.²⁴ Participants warmed up at 50 W for 5 min on a cycle ergometer. After this, power output increased by 30 W every minute until voluntary exhaustion. Oxygen consumption was analyzed continuously by the Vmax metabolic cart (CareFusion, Franklin Lakes, NJ) and averaged every 10 s. $\text{VO}_{2\text{peak}}$ was the average of the two highest consecutive 10s oxygen consumption values. True $\text{VO}_{2\text{max}}$ was determined by a plateau of oxygen consumption ($< 150 \text{ ml} \cdot \text{min}^{-1}$) with an increased intensity OR a respiratory exchange ratio above 1.15.²⁵ Heart rate (HR) was measured with a Polar HR monitor (Polar, Lake Success, NY). Ratings of perceived exertion were taken every minute throughout the test. Participants were given a five-minute cooldown on the ergometer at 30 W. $\text{VO}_{2\text{max}}$ data is expressed as $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$.

Statistical Analysis

Statistical analysis was performed using SPSS version 28 (IBM, Chicago, IL, USA). Data are expressed as means \pm SD. P value $< .05$ was considered statistically significant. All participants with missing data were removed from the analysis. Descriptive parameters were reported as mean (standard deviation). Multiple linear regression was used as the primary analysis. Predictor variables were metabolic equivalents (METs), FFI, and waist-to-height ratio. Criterion, or outcome variables, were brachial and central BP values and indicators of arterial stiffness. Age and sex were entered into the regression to adjust for these confounding variables. Model 0 represents the analysis without adjusting for these confounding variables. Model 1 represents the analysis, after entering age and sex into block 1. β (standardized regression coefficient), R^2 (adjusted determination coefficient), and p (level of significance) were obtained from these linear regression analyses. Assumptions of homoscedasticity and normality of the residuals were tested. Effect sizes (Cohen's f^2) were calculated for all significant relationships using the formula $f^2 = R^2 / (1 - R^2)$, with values of 0.02, 0.15, and 0.35 representing small, medium, and large effects, respectively.

Results

Participant Characteristics

Table 1 details the demographic and physiological characteristics of the study participants. In short, 218 ($n=109$ male, $n=109$ female) participants completed this study. Participants had a mean height of 171.6 ± 10.2 cm, weight of 74.8 ± 13.6 kg, and BMI of 25.3 ± 3.7 kg/m². Body fat percentage averaged $23.2 \pm 10.8\%$, with waist circumference of 82.4 ± 9.7 cm. Mean $\dot{V}O_2\max$ was 35.1 ± 8.7 ml \cdot kg⁻¹ \cdot min⁻¹. Despite being a college-aged population, participants demonstrated substantial heterogeneity in body composition and fitness levels. Body fat percentage ranged from 3.1% to 47.2%, representing the full spectrum from extremely lean to obese classifications. $\dot{V}O_2\max$ values ranged from 14.9 to 58.2 ml \cdot kg⁻¹ \cdot min⁻¹, spanning from poor to superior fitness categories according to age-adjusted norms. BMI ranged from 19.2 to 36.5 kg/m², encompassing normal weight through Class II obesity categories. METs values ranged from 4.2 to 16.6. Males showed significantly higher height, weight, $\dot{V}O_2\max$, FFI, METs, waist circumference, and both brachial and central systolic blood pressure than females (all $p < 0.05$). Conversely, females had significantly higher body fat percentage and augmentation pressure and index (all $p < .05$). Females had significantly lower PWV ($p < .05$).

Table 1 details the demographic characteristics of the study participants by sex. In short, 218 ($n=109$ male, $n=109$ female) participants completed this study. Participants had a mean height of 171.6 ± 10.2 cm, weight of 74.8 ± 13.6 kg, and BMI of 25.3 ± 3.7 kg/m². Body fat percentage averaged $23.2 \pm 10.8\%$, with waist circumference of 82.4 ± 9.7 cm. Mean $\dot{V}O_2\max$ was 35.1 ± 8.7 ml \cdot kg⁻¹ \cdot min⁻¹. Males showed significantly higher height, weight, $\dot{V}O_2\max$, FFI, METs, waist circumference, and both brachial and central systolic blood pressure than females (all $p < 0.05$). Conversely, females had significantly higher body fat percentage and augmentation pressure and index (all $p < .05$). Females had significantly lower PWV ($p < .05$).

Cardiovascular Risk Profile

When compared to established clinical reference values, most participants demonstrated normal cardiovascular parameters. For brachial systolic blood pressure, 50.0% had normal values (<120 mmHg), 32.1% had elevated blood pressure (120-129 mmHg), and 17.9% met criteria for hypertension (≥ 130 mmHg) according to 2017 ACC/AHA guidelines.²⁶ For diastolic blood

pressure, 91.7% had normal values (<80 mmHg), while 8.3% had hypertensive values (≥ 80 mmHg). Regarding arterial stiffness, 80.3% had normal pulse wave velocity values (<7.1 m/s), while 19.7% exceeded the 90th percentile threshold (≥ 7.1 m/s), indicating elevated arterial stiffness for their age group based on established reference values.²⁷

Table 1. Descriptive Data and Sex Differences

Variable	Male (<i>n</i> = 109)	Female (<i>n</i> = 109)	<i>p</i> -value
Age (yr)	20.9 ± 3.0	20.6 ± 2.5	0.495
Height (cm)	178.0 ± 7.5	165.2 ± 7.6	< .001
Weight (kg)	80.7 ± 11.8	68.9 ± 11.5	< .001
BMI (kg/m ²)	25.4 ± 3.4	25.2 ± 3.7	0.667
BodyFat Percentage (%)	16.5 ± 8.8	29.8 ± 9.5	< .001
Waist Circumference (cm)	84.6 ± 9.8	80.1 ± 9.4	< .001
Hip Circumference (cm)	98.3 ± 12.5	100.0 ± 11.7	0.289
VO ₂ max (ml · kg ⁻¹ · min ⁻¹)	40.2 ± 7.4	30.1 ± 7.0	< .001
FFI (METS/waist-to-height)	24.8 ± 7.0	18.2 ± 5.4	< .001
METS	11.5 ± 2.1	8.6 ± 2.0	< .001
Brachial SBP (mmHg)	122.7 ± 9.5	114.2 ± 10.2	< .001
Brachial DBP (mmHg)	68.2 ± 7.7	69.5 ± 6.7	0.26
Central SBP (mmHg)	105.1 ± 9.3	100.4 ± 8.6	< .001
Central DBP (mmHg)	69.6 ± 8.3	69.7 ± 7.8	0.938
Augmentation Pressure (mmHg)	-0.18 ± 4.0	0.90 ± 3.5	0.047
Augmentation Index (%)	-6.8 ± 11.1	-1.4 ± 14.3	0.003
Pulse Wave Velocity (m/s)	5.75 ± 1.2	5.38 ± 1.2	0.045

Note. Values are M ± SD by sex. *p* values represent tests of sex differences.

Unadjusted Associations Between Predictors and Cardiovascular Outcomes

Table 2 shows the unadjusted analysis (Model 0) results. Out of the three predictors (METS, Waist-to-height ratio, and FFI), only METS significantly predicted brachial SBP, producing a standardized beta of .140 with a small effect size ($f^2 = 0.015$, $F(1, 216) = 4.3$, $p = .039$). All predictors predicted unadjusted brachial diastolic blood pressure (DBP), with FFI predicting the greatest standardized beta value of -.271 and a small effect size ($f^2 = 0.074$, $F(1,216) = 17.1$, $p < .001$). Only the waist-to-height ratio predicted unadjusted central SBP with a beta of .188 and small effect size ($f^2 = 0.032$, $F(1,216) = 7.8$, $p = .006$). All predictors predicted unadjusted central DBP, with FFI predicting the greatest standardized beta value of -.248 and a small effect size ($f^2 = 0.060$, $F(1,216) = 17.4$, $p < .001$). All predictors significantly predicted unadjusted AP, Aix, and PWV. METS produced the strongest beta value for AP of -.284 with a small effect size ($f^2 = 0.082$, $F(1, 216) = 18.9$, $p < .001$) and Aix of -.404 with a medium effect size ($f^2 = 0.189$, $F(1,216) = 41.8$, $p < .001$). Waist-to-height ratio produced the strongest unadjusted beta value for PWV of .257 with a small effect size ($f^2 = 0.065$, $F(1, 216) = 13.7$, $p < .001$).

Table 2. Linear regression analysis of fitness, fatness, and FFI with BP and arterial stiffness parameters.

	METS			Waist-to-height ratio			FFI		
	β	R^2	<i>p</i>	β	R^2	<i>p</i>	β	R^2	<i>p</i>
Brachial SBP (mmHg)	0.14	0.015	0.039	0.13	0.012	0.056	0.081	0.002	0.235
Brachial DBP (mmHg)	-0.238	0.052	<.001	0.275	0.071	<.001	-.271	0.069	<.001
Central SBP (mmHg)	0.023	-.004	0.733	0.188	0.031	0.006	-.031	.004	0.654
Central DBP (mmHg)	-.204	0.037	0.003	0.274	0.071	<.001	-.248	0.057	<.001
Augmentation Pressure (mmHg)	-.284	0.076	<.001	0.173	0.025	0.011	-.243	0.055	<.001
Augmentation Index (%)	-.404	0.159	<.001	0.229	0.052	<.001	-.352	0.12	<.001
Pulse Wave Velocity (m/s)	-.155	0.019	0.03	0.257	0.061	<.001	-.168	0.023	0.019

The value represents Model 0. Standardized β value and adjusted R^2 value are provided.

Age and Sex-Adjusted Associations Between Predictors and Cardiovascular Outcomes

Table 3 shows adjusted analysis (Model 1) results, adjusted for sex and age. Only waist-to-height significantly predicted adjusted brachial SBP, $\beta = .159$ with a small effect size ($f^2 = 0.026$, $F(3,196) = 15.1$, $p < .001$). All predictors significantly predicted adjusted brachial DBP with waist to height producing the largest prediction, $\beta = .267$, and small effect size ($f^2 = 0.074$, $F(3,196) = 10.4$, $p < .001$). All predictors significantly predicted adjusted central SBP and DBP with waist-to-height producing the largest beta value for central SBP, $\beta = .207$, with a small effect size ($f^2 = 0.044$, $F(3,196) = 11.0$, $p < .001$), and FFI producing the largest beta value for central DBP, $\beta = -.306$, with a small effect size ($f^2 = 0.075$, $F(3, 196) = 9.7$, $p < .001$). All predictors significantly predicted adjusted AP, Aix, and PWV (all $p < .05$). METS was the strongest predictor of AP, $\beta = -.407$, with a small effect size ($f^2 = 0.049$, $F(3,196) = 8.4$, $p < .001$), Aix, $\beta = -.398$, with a small approaching medium effect size ($f^2 = 0.114$, $F(3,196) = 14.2$, $p < .001$), and PWV, $\beta = -.234$, with a small effect size ($f^2 = 0.038$, $F(3,196) = 11.2$, $p < .001$).

Table 3. Linear regression analysis of fitness, fatness, and FFI with corrected BP and arterial stiffness parameters

	METS			Waist-to-height ratio			FFI		
	β	R^2	p	β	R^2	p	β	R^2	p
Brachial SBP (mmHg)	-0.14	0.013	0.086	0.159	0.025	0.016	-0.132	0.013	0.078
Brachial DBP (mmHg)	-0.269	0.047	0.002	0.267	0.069	<.001	-0.286	0.062	<.001
Central SBP (mmHg)	-0.179	0.021	0.033	0.207	0.042	0.002	-0.18	0.025	0.02
Central DBP (mmHg)	-0.268	0.048	0.002	0.271	0.072	<.001	-0.306	0.07	<.001
Augmentation Pressure (mmHg)	-0.407	0.047	0.002	0.158	0.024	0.024	-0.196	0.029	0.013
Augmentation Index (%)	-0.398	0.102	<.001	0.199	0.039	0.004	-0.293	0.066	<.001
Pulse Wave Velocity (m/s)	-0.234	0.037	0.006	0.179	0.031	0.012	-0.19	0.028	0.017

The value represents Model 1, adjusted by sex and age. Standardized β value and adjusted R^2 value (R^2 change beyond Model 0).

Discussion

Our study aimed to evaluate the relationship between FFI and cardiovascular outcomes in college-aged students, hypothesizing that FFI would demonstrate stronger associations with cardiovascular risk factors than traditional measures such as anthropometrics or VO_{2max} alone. Contrary to our hypothesis, we found that METS emerged as the strongest predictor of arterial stiffness parameters, demonstrating the highest beta coefficients AP ($\beta = -0.407$), Aix ($\beta = -0.398$), and PWV ($\beta = -0.234$). In contrast, FFI showed weaker associations with arterial stiffness (AP: $\beta = -0.196$, Aix: $\beta = -0.293$, PWV: $\beta = -0.19$). However, distinct patterns emerged in BP measurements, where the waist-to-height ratio was the strongest predictor of adjusted brachial SBP, DBP, and central SBP. At the same time, FFI was the strongest predictor of central DBP.

Cardiorespiratory Fitness and Arterial Stiffness

The strong association between METS and arterial stiffness highlights the critical role of CRF in maintaining vascular health in young adults. Higher CRF is linked to improved endothelial function, reduced arterial inflammation, and greater vascular elasticity.^{28,29} One key mechanism is nitric oxide's enhanced production and bioavailability, a crucial vasodilator that improves endothelial function and reduces vascular resistance.³⁰ Additionally, individuals with higher CRF typically exhibit lower levels of oxidative stress and systemic inflammation, which contribute to arterial stiffening.³¹ Regular aerobic exercise promotes favorable adaptations in arterial structure,

including reduced collagen deposition and increased elastin content, which improve vascular compliance.³² Previous studies have consistently demonstrated an inverse relationship between CRF and arterial stiffness in young adults, reinforcing the importance of aerobic capacity in cardiovascular risk assessment.^{33,34} Our findings extend this understanding by demonstrating the primacy of CRF over composite measures like FFI in predicting arterial stiffness in college-aged individuals.

FFI Performance in Young Adults

Despite its established utility in predicting cardiovascular outcomes in general populations, FFI was less predictive than METS for arterial stiffness in our cohort of young adults. Several factors may explain this discrepancy. First, the physiological impact of CRF on vascular compliance may be more direct and pronounced compared to the influence of body composition in young adults.³⁵ Second, in this age group, the dynamic vascular changes driven primarily by fitness and lifestyle behaviors may not be fully captured by body composition measures incorporated in the FFI.³⁶ While FFI integrates fat mass and CRF, it does not account for other critical factors influencing BP regulation, such as autonomic nervous system activity and endothelial function. Additionally, FFI may be influenced by measurement inaccuracies in body composition assessment methods, particularly in young adults with varying muscle mass and fat distribution patterns. The stronger relationship between FFI and BP measures, particularly central DBP, suggests its potential relevance in hypertension risk assessment rather than in evaluating arterial stiffness. This finding indicates that different cardiovascular parameters may be more responsive to different assessment methods in young adults.

Clinical Applications

Our findings have important implications for cardiovascular screening protocols for college-aged students. Our findings suggest that CRF assessment may be beneficial in college health services for cardiovascular risk identification, though validation in more diverse populations is needed. Standard health assessments for this population typically focus on BMI and BP, but often neglect CRF evaluation. Incorporating simple CRF assessments, such as step tests or submaximal exercise protocols, could significantly enhance risk stratification. We recommend using both METS and FFI as complementary measures for comprehensive cardiovascular risk assessment. While METS provides critical insights into arterial stiffness and cardiovascular function, FFI contributes valuable information about BP regulation and metabolic health. This dual approach would enable more targeted intervention strategies tailored to individual risk profiles. College health programs might consider evaluating the potential role of CRF assessments in health screening protocols. Early detection and intervention during these formative years could significantly reduce long-term cardiovascular disease risk. While our findings in this relatively fit college population suggest that METS and FFI may provide complementary cardiovascular risk information, broader validation across diverse populations with varying fitness and body composition levels is essential before clinical implementation.

Sex-Specific Considerations

We observed significant sex differences in METS and arterial stiffness parameters, with males showing higher METS values (11.5 ± 2.1 vs. 8.6 ± 2.0). At the same time, females had lower PWV (5.38 ± 1.2 vs. 5.75 ± 1.2 m/s) but higher AP (0.90 ± 3.5 vs. -0.18 ± 4.0 mmHg) and Aix (-1.4 ± 14.3 vs. $-6.8 \pm 11.1\%$). These results align with existing literature on sex-based cardiovascular

differences.^{37,38} These differences likely stem from physiological, hormonal, and structural factors. Males typically have greater absolute CRF due to higher lean muscle mass, stroke volume, and oxygen-carrying capacity.³⁹ In contrast, females' smaller arterial diameters and higher resting heart rates can contribute to increased wave reflection, elevating AP and Aix.³⁸ Estrogen also protects vascular health, promoting vasodilation and reducing arterial stiffness.³⁸ These sex-based differences have important implications for cardiovascular risk assessment and intervention strategies. Current risk models may not fully account for sex-specific variations in arterial stiffness and fitness metrics.⁴⁰ Our findings suggest cardiovascular risk assessment in college-aged individuals should incorporate sex-specific reference values for METS, FFI, and arterial stiffness parameters to improve risk detection accuracy.

This study comprehensively evaluates multiple cardiovascular parameters in a relatively large sample of young adults, strengthening its reliability. Assessing METS, FFI, arterial stiffness, and blood pressure offers a broader perspective on cardiovascular health in this understudied population. Including central and peripheral hemodynamic markers improves its ability to detect early vascular dysfunction.⁴¹ With 218 participants equally distributed by sex, the study allows for meaningful sex-based comparisons. Recruiting healthy, nonsmoking individuals without known cardiovascular conditions reduces potential confounding variables. However, several limitations should be acknowledged.

While our study found statistically significant associations between fitness, fatness, and cardiovascular parameters, the effect sizes were predominantly small according to Cohen's conventions. Most significant relationships demonstrated small effect sizes ($f^2 < 0.15$), with only one relationship reaching medium effect size: METS predicting augmentation index ($f^2 = 0.189$). These small effect sizes suggest that while the relationships are statistically reliable in our sample, the practical significance may be modest. This finding aligns with the complex, multifactorial nature of cardiovascular health, where individual predictors typically account for small portions of variance. The clinical relevance of these small effects should be interpreted cautiously. In populations with greater cardiovascular risk or more diverse health profiles, these relationships might demonstrate larger effect sizes and greater clinical significance.

The cross-sectional design limits causal interpretations of the observed relationships. Longitudinal studies are needed to determine whether the observed associations persist over time and predict future cardiovascular outcomes. Additionally, while validated tools were used for all measurements, methods like air displacement plethysmography and PWV may be influenced by factors such as hydration status and recent physical activity.^{42,43} Our study focused on objective cardiovascular markers, but did not assess lifestyle factors such as diet, stress, and sleep quality, which may influence vascular health and should be considered in future research. Additionally, as this was a secondary analysis of existing data, the original data collection protocols were not specifically designed to test our research hypotheses, which may have influenced variable selection and measurement protocols. Ethnicity was also not taken into consideration and could influence the appropriateness of these assessments. Finally, an important limitation is the exclusion of participants with known cardiovascular, pulmonary, renal, peripheral vascular, or metabolic diseases, which may have removed cases most likely to demonstrate positive screening results and potentially underestimated effect sizes. While exclusions were expected to be minimal in this healthy college population, this restriction limits generalizability to clinical screening scenarios and more diverse populations with varying fitness levels, body compositions, and cardiovascular risk profiles.

Several important questions emerge from our findings that warrant further investigation. Longitudinal studies are needed to assess how changes in METS and FFI influence arterial stiffness and BP across different life stages, particularly as young adults transition into middle age. Such studies could help determine whether early intervention based on these parameters modifies long-term cardiovascular risk. Randomized controlled trials evaluating various exercise interventions could provide insights into optimal strategies for improving vascular health in young adults. These studies should investigate whether targeted interventions to strengthen CRF yield greater benefits for arterial stiffness than interventions focused on improving body composition. Future research should also explore potential mechanisms underlying the sex differences observed in our study, particularly regarding the relationship between hormonal factors, CRF, and arterial stiffness. Additionally, studies incorporating genetic factors, inflammatory markers, and more detailed body composition analyses could further elucidate the complex relationships between fitness, fatness, and vascular health in young adults.

Our findings challenge the hypothesis that FFI would outperform traditional measures in predicting cardiovascular outcomes in college-aged students. Instead, we found that CRF, measured as METS, is a key determinant of arterial stiffness in this population. At the same time, FFI and waist-to-height ratio demonstrate stronger associations with specific BP parameters. This suggests cardiovascular risk factors may respond differently to various assessment methods in young adults. The strong relationship between METS and arterial stiffness underscores the importance of incorporating CRF assessment into cardiovascular screening protocols for college-aged individuals. Using both METS and FFI may provide a more comprehensive evaluation of cardiovascular health, enabling more targeted intervention strategies. These preliminary findings in a college population provide initial insights into cardiovascular risk relationships in young adults, though broader validation is needed.

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