RESEARCH ARTICLE

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The Association Between Abdominal Fat, Cardiorespiratory Fitness, and Clinical Markers of Metabolic Syndrome

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Abstract: Our purpose was to determine the association between abdominal fat and cardiorespiratory fitness (CRF) on markers of metabolic syndrome (MetS) total cholesterol (TC), high-density lipoprotein (HDL-C), low-density lipoprotein (LDL-C), triglycerides (TG), glucose, and blood pressure. We conducted a retrospective study on 165 adults (108 men) in which correlations between waist girth (WG), CRF and blood variables were determined. The cohort was partitioned by gender into quartiles and again by WG and differences in markers of MetS were compared across quartiles by ANOVA and by ANCOVA to determine the influence of CRF. Males in the lowest WG quartile exhibited greater HDL-C and lower diastolic blood pressure vs. the highest quartile ($p < 0.05$). TG were lower in the lowest vs. the third and highest quartile ($p < 0.05$), and glucose was greater in the highest vs. the first and second quartiles ($p < 0.05$). Females in the second WG quartile exhibited higher HDL-C vs. the highest quartile, and TG, glucose, systolic and diastolic blood pressure were lower in the lowest vs. the highest quartile ($p < 0.05$). After adjusting for CRF, diastolic blood pressure across WG in males were no longer significant, and HLD-C and TG quartile differences were no longer significant in females. We confirm WG as an important correlate of clinical markers of MetS in adults. CRF mitigates relationships between WG and clinical markers of cardio-metabolic risk in men and women.

Keywords: Metabolic Syndrome, Cardiovascular Fitness, Body Fat, Waist Circumference

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1. Introduction

Metabolic Syndrome (MetS) represents a constellation of interrelated risk factors of metabolic origin that are linked to insulin resistance and strongly associated with the development and severity of type 2 diabetes mellitus, the progression of diabetic nephropathy, and with an increased risk for overt cardiovascular disease and stroke [1]. In the United States, the prevalence of MetS in the population using National Health and Nutrition Examination Survey (NHANES) data from 2011 to 2016 showed an alarming 34,7% for the general population and similar proportions for men (35,1%) and women (34,3%) [2]. There has been an increase ~10% over a decade (~80 million adults) [3], which does not represent an encouraging scenario for a country that is making efforts to combat this cluster of diseases in the population.

Clinicians and health care practitioners utilize anthropometric measures such as waist girth (WG) or waist circumference to identify individuals at increased risk for cardio-metabolic dysfunction [4]. There are WG cut-off points established for men and women according to several organizations [5, 6]; however, the deleterious effect of increasing abdominal fat on cardiometabolic health is continuous.

Cardiorespiratory fitness (CRF) is independently associated with the incidence of MetS in both, men and women [7-9]. CRF is a strong predictor of mortality even with concomitant cardiovascular and cancer disease [10, 11] and is more protective against developing cardiovascular disease outcomes than merely increasing amounts of physical activity [12]. CRF offers a protective effect in all-cause and cardiovascular disease mortality in people with MetS [13]. Meta-analytical evidence shows that CRF attenuates the effects of each MetS risk factor, thus resulting in substantial protection against its development [14]. It is known that for every 1-MET increase (3.5 $ml \cdot kg^{-1} \cdot min^{-1}$) in CRF is associated with 10 to 25% increase in survival from cardiovascular diseases [15]. Likewise, CRF has well-known strong and inverse relationship with many of the health markers described in MetS (e.g., reduced waist girth, dyslipidemia) [16-18]. Unlike WG and many cardiometabolic disease risk markers there are no known clinical cut-off points for CRF or criteria for establishing protection against cardio-metabolic dysfunction. The purpose of this study was to retrospectively characterize the association between WG, as a surrogate of abdominal fat, and relative CRF on clinical markers of MetS in men and women. Our approach was to partition male and female cohorts into quartiles based on WG and then to examine the influence of CRF as a modulator of cardio-metabolic risk across waist girth quartiles.

2. Methods

2.1 Participants

Anonymous data from information collected on 165 adults (108 males and 57 females), attending a university-based health and fitness assessment program were retrospectively reviewed. The study was

approved by the Institutional Review Board of Auburn University in accordance with expedited research described in title 45 of the Code of Federal Regulations 46.110 of the United States of America regulations. All participants completed a health history questionnaire, a medical examination, blood sampling, body composition analysis, and a graded exercise test to determine CRF and heart rate, systolic blood pressure (SBP) and diastolic blood pressure (DBP) responses to exercise of increasing intensity. The assessments were performed for the purpose of providing individual health and fitness information and exercise training recommendations for clients of a university-based community service program.

2.2 Procedures

Anthropometric and body composition. Height (cm) and body weight (kg) were measured by stadiometer and a calibrated medical scale, respectively. Waist and hip circumferences were assessed with a plastic-coated cloth measuring tape (cm). Relative (%) and absolute (g) values of total and regional lean and fat tissue were estimated using Dual-energy X-ray absorptiometry (DXA, Lunar system, General Electric, Fairfield, CT) with *enCORE* software, version 6.0. WG quartiles (Q_n) were independently created for males and females. For males, Q_1 included waist values < 89 cm, Q_2 values between 89.1 and 96.1 cm, Q_3 values between 96.2 and 103.9 cm, and Q_4 values > 104 cm. For females, Q_1 included waist values < 71.3 cm, Q_2 values between 71.4 and 75.5 cm, Q_3 values between 75.6 and 83.6 cm, and Q_4 values > 83.7 cm.

Physiological testing for cardiorespiratory fitness. The standardized Bruce treadmill protocol [6] was used to determine CRF. Total treadmill time was recorded to estimate peak absolute ($l \cdot min^{-1}$) and relative ($ml \cdot kg^{-1} \cdot min^{-1}$) oxygen consumption ($VO_2peak$) as measures of CRF. Heart rates were continuously measured during the test and SBP and DBP were obtained manually at rest and during the last 30 s of each exercise stage [6].

Blood Sampling. Blood samples were obtained in the morning after an 8- to 10-h fast and prior to undergoing physiological testing for CRF. Blood samples were collected in serum-separator tubes and analysed at a Centers for Disease Control and Prevention certified reference laboratory. Blood analyses included total cholesterol (TC), high-density lipoprotein (HDL-C), low-density lipoprotein (LDL-C), triglycerides (TG), and glucose.

2.3 Statistical analysis

Statistical analysis was performed with the IBM-SPSS Statistics, version 21 (IBM Corporation, Armonk, New York). Values are presented as mean and standard deviation (M ± SD). Independent samples t-tests were used to determine gender differences in anthropometric, physiological and blood variables.

Pearson-product moment correlations between regional body fat, WG, physiological (i.e., VO_2peak, blood pressure), blood and CRF were calculated. ANOVA tests were used to determine global differences in cardio-metabolic risk factors between WG quartiles. Tukey's *post hoc* analyses were used to follow-up global differences. ANCOVA were used to determine whether differences in the dependent variables between WG quartiles remained by using CRF as covariate. Statistical significance for all tests were set *a priori* at $p \leq 0.05$.

3. Results

Descriptive statistics for anthropometric, physiological and blood variables by gender are presented in table 1. The sample was comprised by males (mean age = 49.0 ± 10.2 yr., 95%CI = 47.1, 51.0) and females (mean age = 47.2 ± 12.5 yr., 95%CI = 43.8, 50.5). Complete data for WG were obtained for 108 males and 55 females. Physiological and blood variables were collected for 108 males and 57 females. Independent samples t-tests revealed no significant gender differences in age, gynoid fat content, TC, LDL-C, and relative CRF ($p > 0.05$) (Table 1).

Significant correlations between WG were observed in males for body fat % ($r = 0.76$, $p \leq 0.0001$, Figure 1A), android ($r = 0.90$, $p \leq 0.0001$, Figure 1B) and gynoid ($r = 0.81$, $p \leq 0.0001$) patterns of fat distribution, HDL-C ($r = -0.37$, $p = 0.01$), TG ($r = 0.29$, $p = 0.01$), blood glucose ($r = 0.37$, $p = 0.01$), SBP ($r = 0.22$, $p = 0.05$), DBP ($r = 0.27$, $p = 0.01$), and CRF ($r = -0.60$, $p \leq 0.001$, Figure 1C).

Significant correlations between WG were observed in females for body fat % ($r = 0.61$, $p \leq 0.0001$, Figure 1D), android ($r = 0.89$, $p \leq 0.0001$, Figure 1E) and gynoid ($r = 0.59$, $p \leq 0.0001$) patterns of fat distribution, TG ($r = 0.30$, $p = 0.05$), blood glucose ($r = 0.45$, $p = 0.01$), SBP ($r = 0.46$, $p = 0.01$),

DBP (r = 0.36, p = 0.01), and CRF (r = -0.49, p ≤ 0.0001, Figure 1F).

In males, TC (p = 0.362) and LDL-C (p = 0.171) did not differ between WG quartiles. HDL-C were greater in Q_1 vs. Q_4 (p = 0.002), TG concentration were lower in Q_1 vs. Q_3 (p = 0.010) and Q_4 (p = 0.008), and blood glucose were lower in Q_1 vs. Q_3 (p = 0.010), Q_1 vs. Q_4 (p = 0.002), and Q_2 vs. Q_4 (p = 0.031). SBP were similar across WG quartiles (p = 0.063); however, DBP were lower in Q_1 vs. Q_4 (p = 0.023). CRF were incrementally lower with increasing waist girth quartiles (p < 0.008 to p < 0.0001) (Table 2).

In females, TC (p = 0.512) and LDL-C (p = 0.559) did not differ between WG quartiles. HDL-C were higher in Q_2 vs. Q_4 (p = 0.042), TG were lower in Q_1 vs. Q4 (p = 0.048), and blood glucose concentration were lower in Q_1 vs. Q_4 (p ≤ 0.0001). Both, SBP (p = 0.013) and DBP (p = 0.015) were lower in Q_1 vs. Q_4. CRF was greater in Q_1 vs. Q_4 (p = 0.005) and Q_2 vs. Q_4 (p = 0.004) (Table 2).

In males, DBP was no longer significantly different between waist quartiles after adjusting for CRF (p = 0.054) (Table 3). In females, blood glucose concentrations (p = 0.004), SBP (p = 0.016) and DBP (p = 0.023) remained significantly different between waist quartiles after adjusting for CRF. HDL-C and TG were no longer significantly different between waist quartiles after adjusting for CRF (Table 3).

Table 1. Anthropometric, physiological and blood characteristics for participants (n= 165) and independent samples t-test results comparing gender.

	Males (n = 108)			**Females (n = 57)**			
Variable	**Mean ± SD**	**Min**	**Max**	**Mean ± SD**	**Min**	**Max**	**p ≤**
Age (yr)	49.0 ± 10.2	30.0	69.0	47.2 ± 12.5	22.0	73.0	0.3010
Height (cm)	176.0 ± 6.7	157.5	191.3	163.8 ± 6.0	151.1	182.9	0.0001
Weight (kg)	90.3 ± 18.6	64.5	149.8	66.8 ± 12.4	47.5	126.8	0.0001
BMI ($kg \cdot m^2$)	29.0 ± 5.1	21.0	45.70	24.9 ± 4.0	18.8	37.9	0.0001
DXA Fat (%)	28.8 ± 7.2	10.0	43.0	35.9 ± 7.7	19.0	49.0	0.0001
Android fat (kg)	2.9 ± 1.4	0.5	7.73	1.8 ± 0.9	0.4	5.6	0.0001
Gynoid fat (kg)	4.2 ± 1.8	1.2	10.8	4.8 ± 1.5	2.0	9.4	0.0580
Waist (cm)	97.4 ± 12.1	76.2	132.1	78.2 ± 10.4	60.9	118.1	0.0001
Hip (cm)	104.2 ± 10.9	85.1	147.3	99.6 ± 11.6	61.7	143.5	0.0130
VO_2peak ($l \cdot min^{-1}$)	3.3 ± 0.7	1.6	5.2	2.3 ± 0.5	1.1	3.5	0.0001
VO_2peak ($ml \cdot kg^{-1} \cdot min^{-1}$)	37.6 ± 9.7	20.5	62.2	34.8 ± 8.7	18.0	55.7	0.0780
Systolic BP (mmHg)	127.3 ± 15.1	90.0	200.0	118.9 ± 13.8	92.0	150.0	0.0010
Diastolic BP (mmHg)	79.8 ± 8.1	58.0	100.0	73.4 ± 8.2	56.0	90.0	0.0001
TC (mmol·l-1)	4.9 ± 0.9	2.8	7.1	5.1 ± 1.1	3.4	8.9	0.3200
LDL-C (mmol·l-1)	2.9 ± 0.7	1.1	5.9	2.9 ± 1.0	1.5	6.5	0.8330
HDL-C (mmol·l-1)	1.3 ± 0.3	0.7	2.4	1.7 ± 0.4	1.0	2.5	0.0001
Triglyceride (mmol·l-1)	1.6 ± 0.9	0.4	4.4	1.1 ± 0.7	0.5	4.4	0.0001
Glucose (mmol·l-1)	1.1 ± 0.2	0.8	1.6	1.0 ± 0.1	0.8	1.6	0.0001

Note: BMI = Body mass index, DXA = dual x-ray absorptiometry, VO_2peak = estimated peak oxygen consumption, BP = Blood pressure, TC = Total cholesterol, LDL-C= Low-density lipoprotein cholesterol, HDL-C = High-density lipoprotein cholesterol.

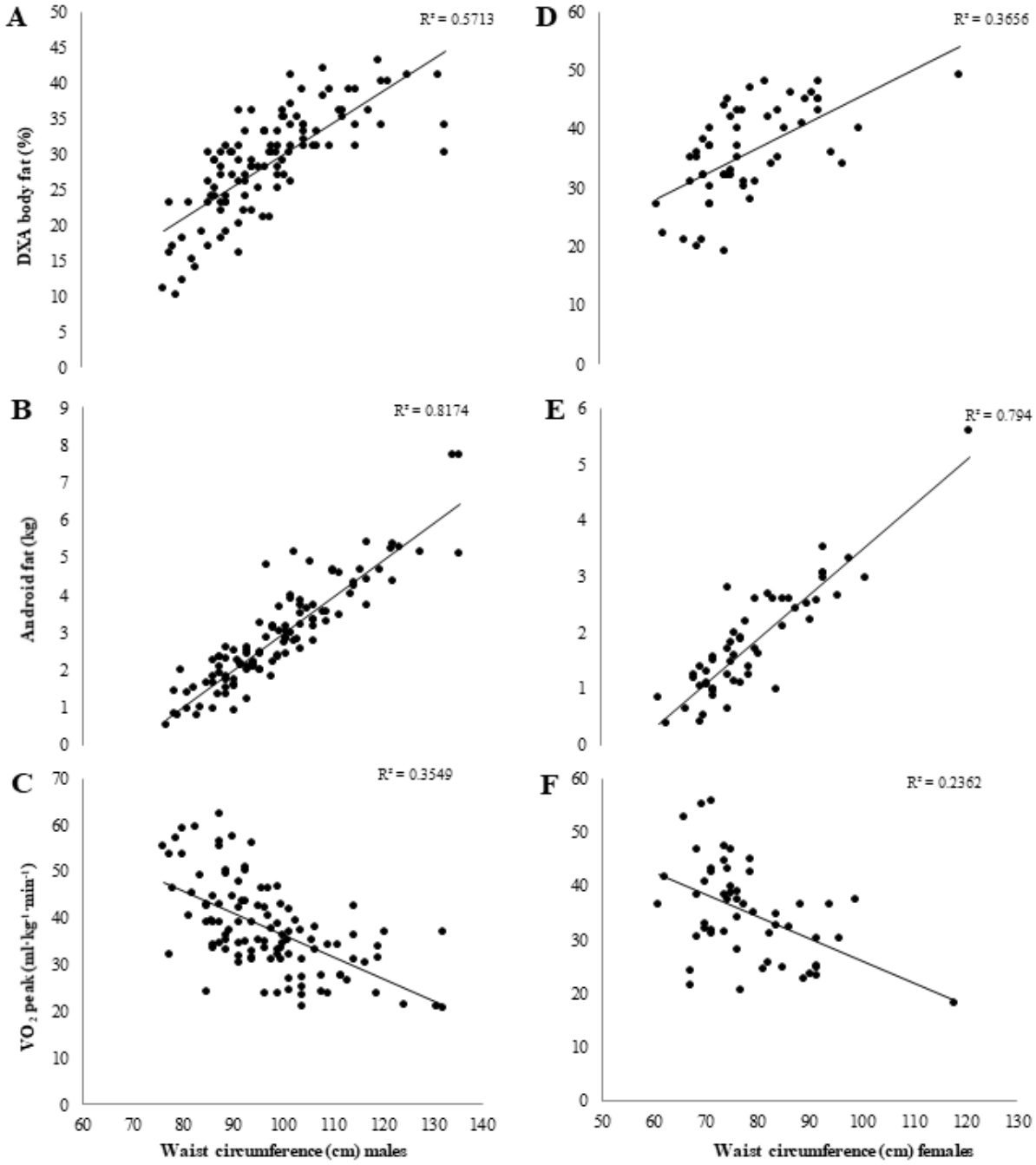

Figure 1. Scatterplot diagrams and correlations between waist circumferences and selected measures of total and abdominal body fat and cardiorespiratory fitness for males (panel A, B, C) and females (panel D, E, F).

Table 2. Selected anthropometric and blood characteristics by waist quartiles (cm) unadjusted for estimated relative ($ml \cdot kg^{-1} \cdot min^{-1}$) cardiorespiratory fitness (Values are presented as means ± 1 SD. Means with different superscript letters are significantly different at $p < 0.05$).

Males (n = 108)	**Waist quartiles (cm)**				
Variable	**Q_1 (< 89) (n = 31)**	**Q_2 (89.1-96.1) (n = 22)**	**Q_3 (96.2-103.9) (n = 27)**	**Q_4 (> 104) (n = 28)**	**p ≤**
BMI ($kg \cdot m^2$)	24.3 ± 1.8^a	26.9 ± 2.2^b	29.4 ± 1.9^c	35.6 ± 4.4^d	0.0001
DXA Fat (%)	21.7 ± 5.8^a	27.0 ± 4.9^b	31. 4 ± 4.4^c	35.6 ± 3.9^d	0.0001
Android fat (kg)	1.6 ± 0.6^a	2.3 ± 0.7^b	3.2 ± 0.7^c	4.5 ± 1.2^d	0.0001

Gynoid fat (kg)	2.8 ± 0.8^{a}	3.5 ± 0.6^{b}	4.6 ± 1.2^{c}	6.0 ± 2.0^{d}	0.0001
VO_2peak (l·min^{-1})	3.3 ± 0.8^{a}	3.3 ± 0.7^{a}	3.3 ± 0.7^{a}	3.3 ± 0.9^{a}	0.9980
VO_2peak (ml·kg^{-1}·min^{-1})	44.6 ± 9.8^{a}	40.9 ± 8.1^{a,b}	35.2 ± 6.3^{b}	29.4 ± 6.0^{c}	0.0001
SBP (mm Hg)	121.2 ± 16.1^{a}	128.1 ± 14.5^{a}	128.6 ± 15.1^{a}	130.9 ± 16.4^{a}	0.0630
DBP (mm Hg)	76.4 ± 8.9^{a}	80.8 ± 8.0^{a,b}	79.9 ± 8.3^{a,b}	82.5 ± 9.0^{b}	0.0300
HDL-C (mmol·l-1)	1.4 ± 0.3^{a}	1.3 ± 0.3^{a,b}	1.2 ± 0.3^{a,b}	1.1 ± 0.4^{b}	0.0040
LDL-C (mmol·l-1)	2.9 ± 0.6^{a}	3.2 ± 0.7^{a}	3.0 ± 0.8^{a}	2.7 ± 0.8^{a}	0.1710
TC (mmol·l-1)	4.9 ± 0.8^{a}	5.1 ± 0.9^{a}	5.1 ± 0.9^{a}	4.7 ± 1.0^{a}	0.3620
TG (mmol·l-1)	1.2 ± 1.0^{a}	1.4 ± 0.8^{a,b,c}	1.9 ± 0.8^{b,c}	1.9 ± 1.0^{c}	0.0020
Glucose (mmol·l-1)	1.0 ± 0.2^{a}	1.1 ± 0.1^{a,b}	1.1 ± 0.2^{b,c}	1.2 ± 0.2^{c}	0.0001
Females (n = 55)	**Q_1 (< 71.3)**	**Q_2 (71.4-75.5)**	**Q_3 (75.6-83.6)**	**Q_4 (> 83.7)**	
Variable	**(n = 18)**	**(n = 9)**	**(n = 13)**	**(n = 15)**	
BMI (kg·m^{2})	21.4 ± 1.4^{a}	24.9 ± 1.9^{b}	24.3 ± 2.8^{b,c}	29.9 ± 3.1^{d}	0.0001
DXA Fat (%)	30.4 ± 6.5^{a}	34.6 ± 8.1^{a,b}	37.6 ± 6.7^{b}	42.4 ± 4.6^{b,c}	0.0001
Android fat (kg)	1.0 ± 0.4^{a}	1.6 ± 0.6^{a,b}	1.8 ± 0.6^{b}	2.9 ± 0.8^{c}	0.0001
Gynoid fat (kg)	3.9 ± 0.9^{a}	4.9 ± 1.0^{a}	4.6 ± 1.7^{a}	5.9 ± 1.6^{a,b}	0.0010
VO_2peak (l·min^{-1})	2.2 ± 0.6^{a}	2.7 ± 0.3^{a}	2.2 ± 0.5^{a}	2.3 ± 0.5^{a}	0.1550
VO_2peak (ml·kg^{-1}·min^{-1})	38.6 ± 10.1^{a}	40.7 ± 5.2^{a}	33.2 ± 7.4^{a,b}	28.7 ± 6.1^{b}	0.0010
SBP (mm Hg)	110.5 ± 13.6^{a}	120.0 ± 13.5^{a,b}	122.6 ± 13.7^{a,b}	125.2 ± 13.9^{b}	0.0120
DBP (mm Hg)	69.1 ± 8.1^{a}	76.0 ± 8.1^{a,b}	72.9 ± 7.9^{a,b}	77.7 ± 8.5^{b}	0.0160
HDL-C (mmol·l-1)	1.7 ± 0.4^{a}	1.9 ± 0.4^{a,c}	1.7 ± 0.4^{a}	1.5 ± 0.4^{a,b}	0.0400
LDL-C (mmol·l-1)	2.63 ± 0.77^{a}	3.00 ± 0.98^{a}	2.98 ± 0.72^{a}	3.10 ± 1.38^{a}	0.5590
TC (mmol·l-1)	4.77 ± 0.94^{a}	5.28 ± 0.97^{a}	5.13 ± 0.87^{a}	5.29 ± 1.48^{a}	0.5120
TG (mmol·l-1)	0.9 ± 0.8^{a}	0.9 ± 0.8^{a,b}	0.9 ± 0.8^{a,b}	1.6 ± 0.8^{b}	0.0320
Glucose (mmol·l-1)	1.0 ± 0.1^{a}	1.0 ± 0.1^{a,b}	1.1 ± 0.1^{a,b}	1.1 ± 0.1^{b}	0.0020

Note: BMI = Body mass index, DXA = dual x-ray absorptiometry, VO_2peak = estimated peak oxygen consumption, SBP: Systolic blood pressure; DBP: Diastolic blood pressure, HDL-C = High-density lipoprotein cholesterol, LDL-C= Low-density lipoprotein cholesterol, TC = Total cholesterol, TG = Triglycerides.

Table 3. Blood and physiological characteristics by waist quartiles (cm) adjusted for estimated relative (ml·kg^{-1}·min^{-1}) cardiorespiratory fitness (Values are presented as adjusted means ± 1 SD. Means with different superscript letters are significantly different at $p < 0.05$).

	Waist quartiles (cm)				
Males (n = 108)	**Q_1 (< 89)**	**Q_2 (89.1-96.1)**	**Q_3 (96.2-103.9)**	**Q_4 (> 104)**	**p ≤**
Variable	**(n = 31)**	**(n = 22)**	**(n = 27)**	**(n = 28)**	
HDL-C (mmol·l-1)	1.39 ± 0.33^{a}	1.27 ± 0.33^{a,b}	1.23 ± 0.31^{a,b}	1.14 ± 0.37^{b}	0.0090
TG (mmol·l-1)	1.25 ± 0.95^{a}	1.44 ± 0.84^{a,b}	1.84 ± 0.83^{b}	1.76 ± 0.95^{b}	0.0030
Glucose (mmol·l-1)	1.05 ± 0.17^{a}	1.08 ± 0.14^{a,b}	1.14 ± 0.16^{b,c}	1.18 ± 0.16^{c}	0.0001

SBP (mmHg)	124.6 ± 16.1[a]	129.7 ± 14.5[a]	128.0 ± 15.1[a]	127.1 ± 16.4[a]	0.0090
DBP (mmHg)	76.9 ± 8.9[a]	81.0 ± 8.0[a]	79.8 ± 8.3[a]	81.9 ± 9.0[a]	0.0540
Females (n = 55)	**Q_1 (< 71.3)**	**Q_2 (71.4-75.5)**	**Q_3 (75.6-83.6)**	**Q_4 (> 83.7)**	
Variable	**(n = 18)**	**(n = 9)**	**(n = 13)**	**(n = 15)**	
HDL-C (mmol·l-1)	1.72 ± 0.38[a]	1.89 ± 0.36[a]	1.72 ± 0.40[a]	1.44 ± 0.39[a]	0.0870
TG (mmol·l-1)	0.95 ± 0.76[a]	0.99 ± 0.75[a]	0.91 ± 0.79[a]	1.52 ± 0.77[a]	0.0620
Glucose (mmol·l-1)	0.94 ± 0.13[a]	0.99 ± 0.12[a,b]	1.06 ± 0.14[b,c]	1.13 ± 0.12[c]	0.0040
SBP (mmHg)	111.6 ± 13.6[a]	121.8 ± 13.5[a,b,c]	122.0 ± 13.7[b,c]	123.3 ± 13.9[c]	0.0160
DBP (mmHg)	68.5 ± 8.1[a]	75.2 ± 8.1[b]	72.6 ± 7.9[a,b]	78.6 ± 8.5[b,c]	0.0230

Note: HDL-C= High-density lipoprotein cholesterol, TG = Triglycerides, SBP: Systolic blood pressure; DBP: Diastolic blood pressure.

4. Discussion

The aim of this study was to determine the relationship between WG and CRF, and MetS. Our results confirm the established relationship between abdominal fat as estimated from WG and cardiometabolic risk. Findings from this retrospective analysis support the notion that CRF exerts a mild influence on cardio-metabolic risk markers in opposition to that from WG in men and women. Others have found that CRF modulates the association between WG and metabolic and cardiovascular disease risk markers and that maintaining a high level of CRF is important to control cardio-metabolic health in men and women [11, 19-23].

As with any correlational research, cause- and effect between WG and CRF cannot be established. Whether an increased WG, a reflection of increased abdominal adiposity, reduces CRF or increased CRF independently prevents greater WG cannot be determined from our present results. Certainly, a reasonable argument can be made that those with greater WG are less fit because they are less inclined to increase or maintain CRF. However, biological plausibility exists for reduced cardiovascular disease risk regardless of WG with increasing CRF [11-13, 19, 24]. We also found almost normal blood pressure values, especially in Q_1 and Q_2 in males, despite low VO_2peak and almost normal lipid profile. Furthermore, the significant correlations between WG and body fat %, android and gynoid patterns of fat distribution, HDL-C, TG, blood glucose, SBP, DBP, and CRF found in males and females strongly suggest that CRF modulates MetS variables, a finding supported by previous literature [19-20].

Females with smaller WG showed higher HDL-C and lower TG, glucose, SBD, and DBP than males in every matching Q_n category (Table 3). This finding supports the association between lower relative adiposity as estimated by WG and a better metabolic and physiological profile in male and females because of exercise aimed at improving CRF. Indeed, regularly practiced exercise training reduces the prevalence of MetS in adults by 30.5% [17] and cardiorespiratory exercise training regimens appear to have significant effects in reducing abdominal adiposity, decreasing subcutaneous and visceral adipose tissue and other biochemical clinical markers of MetS [14]. Due to the considerable public health burden associated with MetS, physical activity to improve CRF should be promoted as a primary prevention strategy for ameliorating MetS [12, 13, 24]. High-intensity interval training (HIIT), aerobic and resistance training, have been recently found in meta-analytic, systematic and narrative reviews to be valid and successful strategies for reducing and controlling cardio-metabolic risk factors associated with MetS [14, 18, 25].

The exact dose-response of these exercise strategies for desirable changes in metabolic and other markers of MetS remains to be determined for several populations. Cardiorespiratory exercise reduces clinical markers of MetS in postmenopausal women in a dose-response manner [26]. For instance, women completing six months of low-to-moderate intensity cardiorespiratory exercise significantly reduced waist circumference, fasting glucose, and systolic blood pressure [26]. However, a meta-analysis suggests that HIIT interventions almost double the CRF compared to moderate-intensity continuous training and reduces TG, fasting glucose, systolic and diastolic blood

pressure, and increase HDL-C and VO_2peak [27]. Therefore, modest improvements in clinical markers of the cardio-metabolic profile might be expected on those with MetS after engaging in low-intensity exercise, and higher benefits are to be expected from increasing the intensity of their workouts.

Consistent evidence shows that physical activity ameliorates cardio-metabolic risk factors and that the dose of physical activity necessary to improve health may be much less than needed for increasing CRF. Therefore, it is possible that our present findings of only modest influence of CRF on the cardiometabolic disease risk associated with increasing waist girth is due, in part, to benefits of physical activity on health that occur at a lower dose than what is needed to improve CRF. Other major limitations of this study are that we did not account for life-style factors related to MetS such as sleep, nutrition, and stress management.

5. Conclusion

This study lends support for the notion that WG is a surrogate measure for predicting MetS regardless of gender, and that this association has been consistently found in males and females of different age and ethnic groups worldwide. We report that our primary finding provides consistency to the literature: CRF, attained primarily as a direct result of modifiable health behaviour, diminishes relationships between WG and clinical markers of cardio-metabolic risk in men and women.

References

[1] S.M. Grundy, N.J. Stone, A.L. Bailey, C. Beam, K.K. Birtcher, R.S. Blumenthal, L.T. Braun, S. de Ferranti, J. Faiella-Tommasino, D.E. Forman, R. Goldberg, P.A. Heidenreich, M.A. Hlatky, D.W. Jones, D. Lloyd-Jones, N. Lopez-Pajares, C.E. Ndumele, C.E. Orringer, C.A. Peralta, J.J. Saseen, S.C. Smith Jr, L. Sperling, S.S. Virani, J. Yeboah, AHA /ACC /AACVPR /AAPA /ABC /ACPM /ADA /AGS /APhA /ASPC /NLA /PCNA Guideline on the Management of Blood Cholesterol: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, Circulation, 139 (2019) e1046–e1081. [DOI]

[2] G. Hirode, R.J. Wong, Trends in the Prevalence of Metabolic Syndrome in the United States, 2011-2016, JAMA, 323(24) (2020) 2526-2528. [DOI] | [PubMed]

[3] United States Census Bureau. DP05: Table US Population 2021 [Available from: https://data.census.gov/cedsci/table?q=United%20States&g=0100000US&tid=ACSDP1Y2019.DP05&hidePreview=true

[4] C.S. Fox, J.M. Massaro, U. Hoffmann, K.M. Pou, P. Maurovich-Horvat, C.Y. Liu, et al., Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study, Circulation, 116(1) (2007) 39-48. [DOI] | [PubMed]

[5] World Health Organization. Waist circumference and waist-hip ratio: report of a WHO expert consultation Geneva: World Health Organization; 2008 Available from: http://apps.who.int/iris/bitstream/handle/10665/44583/9789241501491_eng.pdf;jsessionid=7A7C8D42296F177220B0755E5E306046?sequence=1

[6] American College of Sports Medicine, (2021) ACSM's guidelines for exercise testing and prescription, Wolters Kluwer, USA.

[7] A.J. Hanley, A.J. Karter, K. Williams, A. Festa, R.B. D'Agostino, Jr., L.E. Wagenknecht, et al., Prediction of type 2 diabetes mellitus with alternative definitions of the metabolic syndrome: the Insulin Resistance Atherosclerosis Study, Circulation, 112(24) (2005) 3713-3721. [DOI] | [PubMed]

[8] R. Jurca, M.J. Lamonte, T.S. Church, C.P. Earnest, S.J. Fitzgerald, C.E. Barlow, et al., Associations of muscle strength and fitness with metabolic syndrome in men, Medicine & Science in Sports & Exercise, 36(8) (2004) 1301-1307. [DOI] | [PubMed]

[9] C. Lorenzo, M. Okoloise, K. Williams, M.P. Stern, S.M. Haffner, The metabolic syndrome as predictor of type 2 diabetes: the San Antonio Heart Study, Diabetes Care, 26(11) (2003) 3153-3159. [DOI] | [PubMed]

[10] P. Kokkinos, C. Faselis, L. Sidossis, J. Zhang, I. Babu, H. Samuele, A. Ahmed, P. Karasik, A. Pittaras, M. Doumas, C. Grassos, S. Rosenberg, J. Myershi, Exercise blood pressure, cardiorespiratory fitness and mortality risk, Progress in Cardiovascular Diseases, 67 (2021) 11-17. [DOI] | [PubMed]

[11] Y. Ezzatvar, R. Ramírez-Vélez, M.L. Sáez de Asteasu, N. Martínez-Velilla, F. Zambom-

Ferraresi, F. Lobelo, et al., Cardiorespiratory fitness and all-cause mortality in adults diagnosed with cancer: systematic review and meta-analysis, Scandinavian Journal of Medicine & Science in Sports, 31(9) (2021) 1745-1752. [DOI] | [PubMed]

[12] D.C. Lee, X. Sui, F.B. Ortega, Y-S. Kim, T.S. Church, R.A. Winett, U. Ekelund, P.T. Katzmarzyk, S.N Blair, Comparisons of leisure-time physical activity and cardiorespiratory fitness as predictors of all-cause mortality in men and women, British Journal of Sports Medicine, 45(6) (2011) 504-510. [DOI] | [PubMed]

[13] P.T. Katzmarzyk, T.S. Church, S.N. Blair, Cardiorespiratory fitness attenuates the effects of the metabolic syndrome on all-cause and cardiovascular disease mortality in men, Archives of internal medicine, 164(10) (2004) 1092-1097. [DOI] | [PubMed]

[14] M.A. Wewege, J.M. Thom, K.A. Rye, B.J. Parmenter, Aerobic, resistance or combined training: A systematic review and meta-analysis of exercise to reduce cardiovascular risk in adults with metabolic syndrome, Atherosclerosis, 274 (2018) 162-171. [DOI] | [PubMed]

[15] L.A. Kaminsky, R. Arena, T.M. Beckie, P.H. Brubaker, T.S. Church, D.E. Forman, et al., The importance of cardiorespiratory fitness in the United States: the need for a national registry: a policy statement from the American Heart Association, Circulation, 127(5) (2013) 652-662. [DOI] | [PubMed]

[16] M.J. LaMonte, C.E. Barlow, R. Jurca, J.B. Kampert, T.S. Church, S.N. Blair, Cardiorespiratory fitness is inversely associated with the incidence of metabolic syndrome: a prospective study of men and women, Circulation, 112(4) (2005) 505-512. [DOI] | [PubMed]

[17] P.T. Katzmarzyk, A.S. Leon, J.H. Wilmore, J.S. Skinner, D.C. Rao, T. Rankinen, C. Bouchard, Targeting the metabolic syndrome with exercise: evidence from the HERITAGE Family Study, Medicine & Science in Sports & Exercise, 35(10) (2003) 1703-1709. [DOI] | [PubMed]

[18] N. Pattyn, V.A. Cornelissen, S.R.T. Eshghi, L. Vanhees, The Effect of Exercise on the Cardiovascular Risk Factors Constituting the Metabolic Syndrome: A Meta-analysis of Controlled Trials, Sports Medicine, 43(2) (2013) 121-133. [DOI] | [PubMed]

[19] M.H. Whaley, J.B. Kampert, H.W. Kohl, 3rd, S.N. Blair, Physical fitness and clustering of risk factors associated with the metabolic syndrome, Medicine & Science in Sports & Exercise, 31(2) (1999) 287-293. [DOI] | [PubMed]

[20] S.M. Grundy, C.E. Barlow, S.W. Farrell, G.L. Vega, W.L. Haskell, Cardiorespiratory fitness and metabolic risk, American Journal of Cardiology, 109(7) (2012) 988-993. [DOI] | [PubMed]

[21] C.H. Saely, P. Rein, H. Drexel, The metabolic syndrome and risk of cardiovascular disease and diabetes: experiences with the new diagnostic criteria from the International Diabetes Federation, Hormone and Metabolic Research, 39(9) (2007) 642-650. [DOI] PMid:17846971

[22] K. Shuval, C.E. Finley, C.E. Barlow, K.P. Gabriel, D. Leonard, H.W. Kohl, 3rd, Sedentary behavior, cardiorespiratory fitness, physical activity, and cardiometabolic risk in men: the Cooper Center Longitudinal Study, Mayo Clinic Proceedings, 89(8) (2014) 1052-1062. [DOI] | [PubMed]

[23] S. Wannamethee, A. Shaper, L. Lennon, R.W. Morris, Metabolic syndrome vs Framingham Risk Score for prediction of coronary heart disease, stroke, and type 2 diabetes mellitus, Archives of Internal Medicine, 165(22) (2005) 2644-2650. [DOI] | [PubMed]

[24] G.O. Matheson, M. Klügl, J.Dvorak, L. Engebretsen, W.H. Meeuwisse, M. Schwellnus, S.N. Blair, W. van Mechelen, W. Derman, M. Börjesson, F. Bendiksen, R. Weiler, Responsibility of sport and exercise medicine in preventing and managing chronic disease: applying our knowledge and skill is overdue, British Journal of Sports Medicine, 45(16) (2011) 1272-1282. [DOI] | [PubMed]

[25] R.B. Batacan, Jr., M.J. Duncan, V.J. Dalbo, P.S. Tucker, A.S. Fenning, Effects of high-intensity interval training on cardiometabolic health: a systematic review and meta-analysis of intervention studies, British Journal of Sports Medicine, 51(6) (2017) 494-503. [DOI] | [PubMed]

[26] C.P. Earnest, N.M. Johannsen, D.L. Swift, C.J. Lavie, S.N. Blair, T.S. Church, Dose effect of cardiorespiratory exercise on metabolic

syndrome in postmenopausal women, American Journal of Cardiology, 111(12) (2013) 1805-1811. [DOI] | [PubMed]

[27] K.S. Weston, U. Wisloff, J.S. Coombes, High-intensity interval training in patients with lifestyle-induced cardiometabolic disease: a systematic review and meta-analysis, British Journal of Sports Medicine, 48(16) (2014) 1227-1234. [DOI] | [PubMed]

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Authors Contribution

P.W. Grandjean and F. Araya-Ramírez – study design, data collection, conceptualization; J. Moncada-Jiménez – statistical analysis, interpretation of results, draft preparation; J. K. Taylor – interpretation of results, draft preparation. All the authors read and approved the final version of the draft.

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Ethics Approval

This study was approved by Institutional Review Board at Auburn University (AL, USA).

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No additional data are available.

Conflict of interest

None

Does this article screened for similarity?

Yes