Circuit Resistance Training Attenuates Acute Exertion-Induced Reductions in Arterial Function but Not Inflammation in Obese Women

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Abstract

Background: Cardiovascular disease (CVD) is a leading cause of preventable death among young women in the United States. Habitual resistance exercise training is known to have beneficial effects on endothelial function and CVD risk factors, including obesity; however, previous studies show that acute resistance exercise impairs endothelial function in obese adults who are sedentary, a response that may be linked to inflammation. We sought to determine if circuit-based resistance training (CRT) attenuates acute resistance exercise-induced reductions in endothelial function in a population of young, obese, sedentary women and whether or not inflammation plays a role in this response.

Methods: Eighteen obese [body mass index (BMI) 30.0–40.0 kg·m^{−2}] young premenopausal women were randomly assigned to either a CRT group or a no-exercise control group (CON). Conduit artery endothelial function was assessed using brachial artery flow-mediated dilation (FMD) determined by ultrasound before and after a single bout of strenuous weightlifting (SWL). In addition, circulating inflammatory mediators (tumor necrosis factor- α and C-reactive protein), blood pressure, fasting blood lipids, glucose, waist circumference, body composition, and aerobic capacity were assessed.

Results: Among participants randomized to the CRT group, 8 weeks of training led to considerable increases in FMD after SWL ($P=0.001$) compared to the CON group. However, no significant differences between the groups were observed in circulating inflammatory mediators, blood pressure, fasting blood lipids, or other physical and physiological characteristics.

Conclusions: This study shows that CRT alleviates acute exertion-induced reductions in endothelial function among obese sedentary women in the absence of changes in inflammation.

Introduction

CARDIOVASCULAR DISEASE (CVD) remains a leading cause of preventable death among young women in the United States.¹ Obesity and sedentary lifestyle are interrelated epidemics that independently contribute to increased risk of CVD morbidity and mortality.² Likewise, endothelial dysfunction, a precursor to CVD,^{3,4} is associated with a sedentary lifestyle in otherwise healthy adults⁵ and exacerbated in those who are obese.⁶ Endothelial dysfunction itself is predominantly linked to imbalances between vasodilator and vasoconstrictor substances produced by the endothelium, and the most prominent manifestation is a reduction in

endothelium-derived nitric oxide (NO) bioavailability.⁷ Because blood flow-related shear stress is a major stimulus to NO release from the endothelium,^{5,8} disturbed flow or low shear stress is the likely mechanism by which endothelial function (EF) is altered with a sedentary lifestyle.

Resistance exercise is known to have beneficial effects on CVD risk factors^{8–11} and is also associated with improvements in conduit artery flow-mediated vasodilation (FMD),¹² which is an index of EF and NO bioavailability in humans. However, previous studies performed in our laboratory and others indicate that FMD is markedly reduced following acute bouts of resistance exercise in obese women and sedentary individuals unaccustomed to exercise training.^{13–15}

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Paradoxically, cross-sectional studies indicate that exercise-induced reductions in FMD are not apparent in trained populations who regularly engage in exercise.^{12,16} Given that obesity and sedentary lifestyle independently contribute to low-grade inflammation^{17,18} and reduced NO bioavailability,^{13,19} it is plausible to hypothesize that inflammation may be a critical factor mediating postexercise reductions in FMD among obese and sedentary populations.

Circuit resistance training (CRT) has beneficial effects on CVD and its risk factors^{20–22} and has also been shown to improve FMD^{23,24} and alleviate inflammation.²⁵ However, the effects of CRT on vasodilator responses to acute exertion are unknown. Therefore, the purpose of this study was to determine if CRT attenuates acute resistance exercise-induced reductions in FMD in a population of young, obese, sedentary women and whether or not inflammation plays a role in this response. We hypothesized that 8 weeks of CRT would protect against postexercise reductions in FMD among obese sedentary women.

Methods

Study participants

Twenty premenopausal women aged 18–40 years volunteered to participate in this study and were randomly assigned to either a CRT group or a no-exercise control group (CON). Inclusion criteria were as follows: Obese [body mass index (BMI) of 30.0–40.0 kg·m⁻²]; sedentary (<150 min of moderate-intensity exercise/week); no history of CVD, hypertension, hypercholesterolemia, diabetes mellitus, sleep apnea, thyroid dysfunction, cancer, smoking, amenorrhea or irregular menses; not currently or recently pregnant or lactating; and no use of oral contraceptives or vasoactive medications. Written informed consent was obtained from all volunteers prior to participation. The study protocol was approved by the Office for the Protection of Research Subjects and the Institutional Review Board of the University of Illinois at Chicago (UIC).

Study design, physical and physiological measurements

Participants were evaluated in the UIC Clinical Interface Core (CIC) after a 12-hr overnight fast. Venous blood

samples were drawn from an antecubital vein, and plasma was separated by centrifugation for laboratory analysis of total cholesterol (TC), high-density lipoproteins (HDLs), low-density lipoproteins (LDLs), and glucose. TC and lipoproteins were measured using spectrophotometric assays. Glucose concentration was measured using the glucose oxidase procedure (Beckman Coulter Inc., Fullerton, CA). Additional blood samples were collected for measurements of tumor-necrosis factor- α (TNF- α) and C-reactive protein (CRP) as inflammatory mediators using previously described enzyme-linked immunosorbent assay (ELISA) techniques.²⁶ Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured after at least 10 min of rest using a standard manual mercury sphygmomanometer with an appropriate cuff size. Waist circumference (WC) was measured at the narrowest part of the waist (above the umbilicus and below the xiphoid process).²⁷ Body composition was determined by dual energy X-ray absorptiometry (DEXA) using established methods.²⁸ The Block Brief 2000 Food Frequency Questionnaire (NutritionQuest, Berkeley, CA) was used to obtain information about diet.

Following baseline assessments, participants randomized to the CRT group proceeded to their assigned 8-week intervention, whereas those in the control group were offered consumer education materials from the American Heart Association. The information contained in the pamphlets reflects standard clinical practice recommendations and promotes healthy living and disease prevention through nutrition and weight management, physical activity, and controlling risk factors. Once these items were offered to control participants, no further exercise instructions were provided until an 8-week follow-up, at which time all participants returned to the CIC for repeated assessments (see Fig. 1).

Determination of brachial artery vasodilator function

Assessments of EF were carried out during the baseline visit and at follow-up (approximately 1 week after the final CRT session). Brachial artery FMD was used as a noninvasive measure of endothelium-dependent vasodilator function using previously described techniques.^{13,29,30} In premenopausal women, FMD may vary during the menstrual cycle³¹; therefore, all participants underwent study procedures during

FIG. 1. Study design and procedures. All participants were screened at baseline (week 0) by way of questionnaires and a physical examination, after which brachial artery flow-mediated dilation (FMD) was assessed prior to and after performance of a single bout of bilateral lower-body strenuous weight lifting (SWL). Participants were administered nitroglycerin (NTG) after FMD for measurement of brachial artery endothelium-independent dilation. During follow-up (week 8), participants underwent reassessments of FMD prior to and after SWL. Evaluation of nitroglycerin-induced dilation was reassessed in the postexertional state.

the early follicular phase of menses. While participants were in the supine position in a quiet, temperature-controlled room, ultrasound imaging (Sonosite; Seattle, WA) of the brachial artery was performed in a longitudinal plane at a site 1–3 cm proximal to the antecubital fossa of the dominant arm¹⁵ using a high-frequency (7.5-MHz) linear array probe. Measurements were made at a depth of 30–50 mm according to each participant's frame. The same anatomical distance and examination depth were used for each assessment. The dynamic range and pulse-repetition frequency for all examinations were set at 55–60 dB (B-mode) and 3.5 kHz, respectively.

Baseline images were recorded, after which a forearm blood pressure cuff was inflated to 50 mmHg above SBP for 5 min. Brachial artery diameter (mm) was determined during peak hyperemia after release of the cuff. To assess dilation, at least 30 sec of images were captured during the first, second, and third minutes after cuff release. Baseline brachial artery flow velocity and peak velocity after cuff release were recorded using central velocity measures described previously.^{13,29} The velocity detecting range was from –30 to 140 cm/sec within a sample volume of 2 mm. Nitroglycerin-induced dilation was also assessed at baseline and follow-up. Ten minutes after recording the last diameter measurement following hyperemia, 0.4 mg of sublingual nitroglycerin (NTG) was administered and endothelium-independent dilation was assessed for 5 min.

Offline analysis of FMD and NTG responses were made using edge-detection software (Medical Imaging, Iowa City, IA), as previously described.¹³ Percent FMD and NTG responses were calculated using the averaged minimum mean brachial artery diameter at baseline compared with the largest mean values obtained after release of the forearm occlusion or administration of NTG. Peak shear rate (SR) was calculated as flow velocity (cm/sec) divided by vessel diameter. Flow-mediated dilation was normalized for the peak SR using the following equation: Normalized FMD = FMD/peak SR.¹³

Acute resistance exercise protocol

To investigate the effects of acute resistance exercise on EF, participants underwent a bout of strenuous weight lifting (SWL) during baseline and follow-up visits in accordance with a previously described protocol.¹⁵ Briefly, the exercise protocol involved performance of four to six sets of approximately 10 repetitions of bilateral lower-body weight lifting at maximum loads [approximately 85%–95% of predicted 10 repetitions maximum (10-RM)] using a variable-resistance leg press machine (Hoist HD-1610 Selectorized Leg Press, Hoist Fitness Systems, San Diego, CA).³² For each set, an isometric contraction was held during the final repetition for measurements of blood pressure. In addition, a 2-min rest interval was allotted between sets. Exercise heart rate and the 10-point Borg rated perceived exertion (RPE) scale were used as indices of intensity. Of note, the RPE scale was not used as an intensity threshold because the perceived capacity was 85%–95% of predicted 10-RM and the goal was to elicit similar increases in blood pressure between participants.¹²

CRT intervention

After baseline assessments, CRT participants underwent an orientation during which time specific exercise instructions were provided and participants were familiarized with the resistance training equipment. Aerobic capacity (YMCA submaximal cycle ergometer test) and muscular strength (10-RM) were assessed during orientation and immediately after the intervention, as previously described.^{33,34} Sessions for the CRT intervention were supervised, conducted twice weekly on nonconsecutive days for 8 weeks, and designed in accordance with established guidelines.³⁵ Each session began with a 5-min warm-up followed by performance of eight to 10 dynamic-resistance exercises in sequence (one exercise after another as a circuit) with a 30-sec rest period between exercises. Exercises were performed using free weights and isotonic variable-resistance machines to target

TABLE 1. PHYSICAL AND PHYSIOLOGICAL CHARACTERISTICS OF STUDY PARTICIPANTS

	CON (n = 8)		CRT (n = 10)		P value for interaction
	Baseline (week 0)	Follow-up (week 8)	Baseline (week 0)	Follow-up (week 8)	
Age, years	30.8 ± 9.0		30.3 ± 5.4		—
Body weight, kg	88.0 ± 18.8	87.6 ± 18.2	89.6 ± 15.4	88.8 ± 15.7	0.430
Body mass index, kg · m ⁻²	32.2 ± 6.9	32.1 ± 6.7	34.2 ± 3.0	33.7 ± 3.4	0.552
Waist circumference, cm	90.1 ± 10.4	91.1 ± 10.5	95.3 ± 7.3	92.6 ± 7.9	0.119
Body fat %	42.1 ± 7.9	42.8 ± 7.3	42.9 ± 4.2	41.9 ± 4.3	0.440
Systolic blood pressure, mmHg	119 ± 11	121 ± 14	121 ± 10	121 ± 10	0.449
Diastolic blood pressure, mmHg	76 ± 8	76 ± 7	78 ± 11	71 ± 8	1.000
Heart rate, beats/min	68 ± 16	74 ± 9	71 ± 11	70 ± 11	0.081
Total cholesterol, mg/dL	172.5 ± 27.5	164.7 ± 11.5	195.9 ± 35.6	188.7 ± 35.8	0.166
HDL-C, mg/dL	61.7 ± 11.5	57.7 ± 11.5	52.9 ± 8.9	56.7 ± 12.6	0.101
LDL-C, mg/dL	99.6 ± 23.0	92.5 ± 16.0	114.8 ± 25.9	109.3 ± 25.9	0.186
Glucose, mg/dL	84.9 ± 5.9	86.8 ± 8.6	85.6 ± 9.5	87.5 ± 12.0	0.503
TNF-α, pg/mL	4.1 ± 1.2	4.1 ± 1.6	6.1 ± 4.0	5.5 ± 4.0	0.923
CRP, pg/mL	2.7 ± 2.5	2.7 ± 3.0	5.3 ± 3.8	5.0 ± 3.2	0.945
Aerobic capacity, mL/kg p · min	31.4 ± 5.9	31.7 ± 5.7	31.7 ± 2.6	34.2 ± 2.3	0.296

All values expressed as mean (± standard deviation).

CON, control; CRT, circuit resistance training; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TNF-α, tumor necrosis factor-α; CRP, C-reactive protein.

the major muscle groups of the upper and lower body. For each exercise, a moderate-intensity training load was set at 80%–90% of 10-RM. At this initial load, participants performed two to three sets of the circuit with a 1-min rest period between circuits. Adjustments were made upon failure to complete the prescribed training load for the required repetitions. For continued progression, training loads were incremented throughout the training period. If participants could perform two or more repetitions over 10 repetitions in the last set in two consecutive workouts for a certain exercise, resistance was added to that exercise for the next session. This progression protocol ensured that volitional fatigue occurred within eight to 10 repetitions.²⁴ Each session ended with a 5-min cool-down of flexibility exercises.

Statistical analysis

Results are expressed as mean $\pm$ standard deviation (SD), unless otherwise stated. Normality of the distribution was confirmed for all data using the Shapiro–Wilk test. Nutritional questionnaires were sent to NutritionQuest for analyses of dietary characteristics, which were then compared using independent *t*-tests. Analysis of variance (ANOVA) with repeated measures was used to compare physical and physiological characteristics between CRT and CON participants before and after the 8-week intervention. Brachial artery FMD is expressed as the maximum percent change in diameter from basal blood flow to peak hyperemia. A two-way repeated measures ANOVA was performed to evaluate pre–post effects of acute resistance exercise on FMD and related vascular variables between participants at baseline and follow-up. The Tukey *post hoc* test was used for multiple comparisons. Pearson correlations were used to evaluate relationships between physiological, physical and dietary characteristics, FMD, and NTG responses. The level of statistical significance for all analyses was set at $P < 0.05$. Data were analyzed using SPSS software (v. 22.0; IBM Corp., Armonk, NY).

Results

Participant demographics and physical and physiological characteristics

A total of 18 women (10 CRT, 8 CON) completed the entire study. Two participants assigned to the CON group voluntarily dropped out of the study due to personal reasons

TABLE 2. NUTRITIONAL CHARACTERISTICS OF STUDY PARTICIPANTS

	CON (n = 8)	CRT (n = 10)	P value
Energy, kcal/day	1535.1 $\pm$ 302.9	1679.8 $\pm$ 394.8	0.47
Total fat, grams/day	58.5 $\pm$ 20.2	61.5 $\pm$ 21.3	0.87
Total carbohydrate, grams/day	134.6 $\pm$ 56.4	188.7 $\pm$ 34.1	0.41
Total protein, grams/day	54.6 $\pm$ 19.7	66.6 $\pm$ 15.0	0.37
Vitamin C, mg/day	83.0 $\pm$ 35.5	107.7 $\pm$ 48.7	0.20
Vitamin E, mg/day	6.9 $\pm$ 2.6	7.3 $\pm$ 2.8	0.89

All values expressed as mean ($\pm$ standard deviation).
CON, control; CRT, circuit resistance training.

TABLE 3. VASCULAR CHARACTERISTICS OF STUDY PARTICIPANTS ACROSS TIME POINTS

	CON (n = 8)				CRT (n = 10)			
	Baseline (week 0)		Follow-up (week 8)		Baseline (week 0)		Follow-up (week 8)	
	Before SWL	After SWL	Before SWL	After SWL	Before SWL	After SWL	Before SWL	After SWL
Baseline diameter, mm	3.4 $\pm$ 0.3	3.4 $\pm$ 0.3	3.4 $\pm$ 0.3	3.4 $\pm$ 0.3	3.4 $\pm$ 0.4	3.4 $\pm$ 0.5	3.4 $\pm$ 0.4	3.5 $\pm$ 0.5
Brachial artery FMD, %	8.4 $\pm$ 3.5	6.7 $\pm$ 3.3 [†]	8.0 $\pm$ 3.3	7.0 $\pm$ 3.3	9.5 $\pm$ 1.6	7.4 $\pm$ 1.3 [†]	9.8 $\pm$ 1.6	11.8 $\pm$ 1.7 ^{*†}
Baseline BFV, cm/sec	71.4 $\pm$ 5.7	71.2 $\pm$ 5.8	70.5 $\pm$ 6.0	68.0 $\pm$ 5.4	68.6 $\pm$ 9.2	68.7 $\pm$ 7.8	69.4 $\pm$ 7.2	69.2 $\pm$ 8.9
Peak BFV, cm/sec	131.2 $\pm$ 16.0	132.2 $\pm$ 15.1	133.0 $\pm$ 15.2	137.0 $\pm$ 13.1	131.8 $\pm$ 14.5	131.4 $\pm$ 13.1	134.9 $\pm$ 14.9	134.9 $\pm$ 15.3
Baseline SR, s ⁻¹	209.5 $\pm$ 32.9	210.4 $\pm$ 34.2	207.8 $\pm$ 30.5	200.8 $\pm$ 30.4	203.0 $\pm$ 36.4	203.5 $\pm$ 35.1	204.5 $\pm$ 32.1	202.1 $\pm$ 35.4
Peak SR, s ⁻¹	352.2 $\pm$ 45.6	360.9 $\pm$ 46.2	358.2 $\pm$ 41.8	374.0 $\pm$ 45.7	356.8 $\pm$ 62.5	358.9 $\pm$ 59.9	363.4 $\pm$ 68.9	352.2 $\pm$ 71.0
Normalized FMD Δ FMD, %	0.024 $\pm$ 0.009	0.018 $\pm$ 0.009 [†]	0.022 $\pm$ 0.008	0.019 $\pm$ 0.009 [†]	0.027 $\pm$ 0.005	0.021 $\pm$ 0.005 [†]	0.027 $\pm$ 0.007	0.034 $\pm$ 0.007 ^{*†}
NTG dilation, %	ND	-1.8 $\pm$ 1.5	ND	-0.8 $\pm$ 1.5	ND	-1.9 $\pm$ 2.3	ND	2.1 $\pm$ 1.5 ^{*†}
		19.3 $\pm$ 9.6		21.2 $\pm$ 3.5		21.2 $\pm$ 3.5		21.3 $\pm$ 2.4

All values expressed as mean ($\pm$ standard deviation).

*Statistical significance between groups after SWL ($P < 0.005$).

[†]Statistical significance within groups ($P < 0.05$).

CON, control; CRT, circuit resistance training; SWL, strenuous weight lifting; FMD, flow-mediated dilation; SR, shear rate; BFV, blood flow velocity; Δ FMD, absolute change in FMD; NTG, nitroglycerin; ND, not determined.

unrelated to adverse effects. The compliance rate for the CRT intervention was greater than 90%. Baseline and follow-up physical and physiological characteristics for all participants are presented in Table 1. Of note, circulating inflammatory mediators (TNF- α and CRP) and CVD risk factors (blood pressure, fasting blood lipids, glucose, BMI, and waist circumference) were similar between groups at both time points (Table 1). Dietary patterns of macronutrients (fats, carbohydrates, and proteins) and antioxidants (vitamins C and E) were also similar between groups (Table 2).

Brachial artery reactivity to FMD and NTG-induced dilation

Brachial artery FMD responses and related vascular characteristics of study participants at baseline (week 0) and follow-up (week 8) are presented in Table 3. Blood flows and initial arterial diameters were similar between groups and unaltered across time. At baseline, all participants exhibited substantial reductions in FMD ($P < 0.001$) after SWL (Fig. 2A). Furthermore, the magnitude of change in FMD (Δ FMD) was comparable between groups after normalization to peak SR ($P < 0.001$) (Table 3 and Fig. 2C). Among participants randomized to the CRT group, 8 weeks of training led to considerable increases in %FMD (Fig. 2B) and normalized FMD (Fig. 2D) after SWL ($P = 0.001$) compared to the CON group. In the CON group, %FMD and normalized FMD were once again reduced at follow-up (Fig. 2B, D), although the former decreased to a less-than-significant level ($P = 0.064$ and $P = 0.033$, respectively). Still, the magnitude of change in

FMD after normalization to peak SR at follow-up was significantly different between the CRT and CON groups at 8 weeks ($P < 0.001$) (Table 3). Contrary to the endothelium-dependent effects of CRT, dilations to NTG were similar (Table 3) amongst all participants across time, indicating an absence of consequential changes in endothelium-independent conduit artery function. Additionally, there was no correlation between vascular responses and physical, physiological, or nutritional characteristics.

Discussion

To our knowledge, this is the first study to determine that SWL reduces brachial artery FMD in obese sedentary, but otherwise healthy, young women and that CRT abolishes this response in the absence of weight loss. Obesity and sedentary lifestyle are associated with endothelial dysfunction and oxidative stress, which contributes to a decrease in NO bioavailability and, consequently, impaired EF.^{36,37} Our previous studies demonstrate that SWL impairs FMD in sedentary lean adults compared to resistance-trained adults despite similar FMD responses at baseline,¹³ and others have found postexercise impairments in FMD amongst sedentary overweight and obese populations.^{38,39} Existing evidence suggests that high intensities of exercise result in immediate decreases in FMD followed by a normalization period, typically within an hour after exercise cessation.¹⁴ However, the magnitude of this response is highly contingent upon numerous factors including one's training status and the type, duration, and/or intensity of exercise performed.¹⁴

FIG. 2. (A and B) the effects of a single bout of bilateral lower-body strenuous weight lifting (SWL) on brachial artery flow-mediated dilation (FMD) in sedentary controls (CON) and circuit resistance training participants (CRT) at baseline (week 0) and follow-up (week 8). (C and D) Brachial artery FMD responses to SWL after normalization to peak shear rate (SR). (*) Statistical significance between groups after SWL ($P < 0.005$). (†) Significant within-group differences observed after SWL versus before SWL ($P < 0.05$).

Similar to our findings, Harris et al.³⁸ reported an impact of training status on postexercise FMD, with physically fit overweight men exhibiting enhanced FMD and sedentary overweight men exhibiting impaired FMD an hour after exercise.³⁸ Although speculative, it is likely that the mechanism of improvement in FMD observed in CRT participants after SWL is, in part, a result of an adaptive response occurring within the vascular wall as a result of repeated episodic increases in shear stress and reductions in oxidative stress. Indeed, such factors have been shown to mediate postexercise FMD responses in various patient populations.^{38,40–42}

Repetitive increases in flow during habitual exercise training have been shown to improve FMD, which is indicative of enhanced NO-mediated EF.^{24,43,44} There is a plethora of research demonstrating that aerobic training enhances EF in overweight and obese adults,^{45,46} but few studies have investigated the specific effects of resistance training. For this study, we implemented an exercise intervention by way of CRT using progressive overload and showed that vascular protection after acute resistance exercise is afforded after 8 weeks of training.

Comparable to aerobic training, CRT provides for exercise bouts with high cardiovascular demands for a variety of muscle groups.^{20,21} CRT is performed with brief rest intervals (15–30 sec) and light-to-moderate loads that enhance muscular fitness while taxing the cardiovascular system.^{20,21} Others have found CRT interventions to have vasculoprotective effects in overweight and obese sedentary populations,^{23,24} but such effects have not been demonstrated after acute exertion, which is known to impair EF. However, similar to others,^{23,24,46} no changes in blood pressure, TC, HDLs, LDLs, or glucose were observed among participants in either the CON or CRT groups, suggesting that the effects of habitual resistance exercise may not be mediated by alterations in CVD risk factors. There were also no significant changes in CRP or TNF- α levels, suggesting that CRT does not modulate circulating proinflammatory cytokines in obesity. Although this was an unexpected finding, it is consistent with observations from other exercise studies.^{13,44} In addition, vasodilator responses to NTG after SWL were unaltered after CRT, suggesting that any changes in FMD were not related to changes in the vessels' sensitivity to NO. Resistance training may enhance conduit artery EF independently through direct mechanical effects of recurrent wall shear stress on NO production.²³ Furthermore, improvements in brachial artery reactivity after SWL may be due to adaptations to recurring shear stress associated with repeated bouts of resistance exercise as a result of training.¹⁰

Study limitations

The generalizability of the study is a limiting factor given the focus on obese women. Furthermore, statistical power may have been reduced because of the small sample size, thus limiting the ability to detect significant changes in some of the variables. Due to the pilot nature of this study, we did not perform a formal power analysis; however, a *post hoc* power analysis was conducted. With sample sizes of 10 (CRT group) and eight (CON group), the study had approximately 80% power to detect a minimum effect size of 0.42 for within-subjects factors, 0.60 for between-subjects factors, and 0.45 for within-between subjects interaction factors using

repeated ANOVA approach with an error rate of 0.05. The results of our data could provide a more precise power estimate for future studies, as larger sample sizes may be necessary to validate our findings. We only assessed peak shear rate for this study. Other studies have shown that alterations of shear rate within participants may limit and underrepresent the normalized FMD compared with area under the curve (AUC) measurements taken until peak diameter.⁴⁷ Although AUC between cuff release and peak diameter was not measured, results shown in Table 3 suggest that the magnitude of change before and after intervention using either normalized or percent FMD were similar.

Although proinflammatory cytokines were assayed, we did not assess for any circulating markers of oxidative stress; therefore, the specific roles of NO and reactive oxygen species (ROS) in acute exertion-induced impairment of EF cannot be fully elucidated. Previous studies have suggested that inflammation and oxidative stress are not altered by habitual resistance exercise,^{13,44} but direct measures of other inflammatory mediators (*e.g.*, interleukin-6 and interleukin-1 β) will require additional analysis. Although FMD is linked to NO bioavailability and impairments are likely due to reductions in NO, further studies should include measures of NO and oxidative stress.⁶ Finally, our results may have been confounded by nutritional status due to potential differences in total calories as well as macronutrient and micronutrient consumption among obese sedentary women. Although participants were required to complete a Block Brief 2000 Food Frequency Questionnaire prior to participation and no significant differences were observed (Table 2), future studies need to address the potential influence of diet.

Conclusion

The results of this study suggest that resistance training protects against acute exertion-induced reductions in brachial artery FMD in a population of young, obese sedentary women independent of alterations in inflammatory status. These findings may be important in understanding the link between obesity and EF and may have implications for designing safe exercise training programs for women and other individuals at risk of CVD.

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Author Disclosure Statement

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