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Original Research

The Impact of Obesity on Endothelial Function Following Maximal Aerobic Exercise

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Abstract

International Journal of Exercise Science 19(2): 2010, 2026. Obesity is associated with increased arterial stiffness and endothelial dysfunction, leading to inflammation and oxidative stress. This study aimed to investigate the effects of maximal aerobic exercise on indicators of endothelial function (C1q-TNF-related-protein-9 [CTRP9], total nitric oxide [NOx], and flow-mediated dilation [FMD]) in obese and normal-weight individuals. Twenty young male participants (11 obese and 9 normal weight) participated in a maximal graded treadmill exercise test. The serum levels of CTRP9 and NOx and FMD were measured prior to, immediately following exercise, and 1 and 2 hours into recovery. There was a significant time effect for serum CTRP9 ($p=0.049$) with a decrease from immediately post exercise to 1 hour after exercise mainly driven by the normal-weight group ($p=0.007$), followed by a return to the baseline levels at 2 hours into recovery. A significant time effect for serum NOx was also observed following exercise ($p=0.033$) with an increase mainly driven by the obese group from baseline to immediately after exercise ($p=0.006$) that returned to baseline level during recovery. No change was found in FMD in both groups following exercise ($p=0.452$), although obese participants had lower FMD compared to normal-weight participants across time ($p=0.017$). These findings suggest that maximal aerobic exercise elicits a transient alteration in biomarkers of endothelial function (NOx and CTRP9), independent of obesity status. Therefore, the novel results from this study may indicate that obesity does not impact the acute endothelial response to maximal aerobic exercise.

Keywords: Vascular Function, C1q-TNF-related-protein-9, total nitric oxide, maximal exercise, obesity

Introduction

The prevalence of obesity has rapidly increased in the United States since the 1960's, now effecting over 40% of the population.[1] Excess adiposity and dysfunctional adipose tissue characterized by an altered secretory profile and impaired metabolic function in obese individuals contribute to low-grade chronic inflammation that is linked to increased risk of inflammatory diseases, such as cardiovascular disease.[2,3] Cardiovascular complications from obesity-associated inflammatory processes involve increased intravascular reactive oxygen species (ROS) generation and leukocyte infiltration into vessel walls, leading to endothelial dysfunction.[4,5] As such, the resulting impairment of endothelium-dependent vasodilation is considered one of the first subclinical stages in the atherosclerotic process.[6]

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The endothelial cells produce various regulatory molecules in controlling vascular tone with nitric oxide (NO) being considered the primary molecule responsible for maintaining vascular homeostasis.[7] NO is produced by endothelial nitric oxide synthase (eNOS), resulting in the relaxation of vascular smooth muscle and subsequent vasodilation.[8] NO has an extremely short half-life and is primarily metabolized into nitrite/nitrate (total nitric oxide [NOx]).[9] A recent meta-analysis indicated that chronic exercise interventions, regardless of duration or type, can significantly increase serum NOx levels following the exercise program.[10] Of particular importance, NOx levels have been negatively correlated with abdominal obesity and body mass index (BMI),[11] suggesting that obesity-related endothelial dysfunction may be partially attributed to reduced NO bioavailability. In this regard, flow-mediated dilation (FMD) of the brachial artery is a reliable method to estimate endothelial cell capacity to release NO in response to physiological stimulus, such as reactive hyperemia (endothelium-dependent vasodilation).[12] Decreased FMD has been demonstrated to be predictive of incidents of cardiovascular events, independent of other cardiovascular risk factors highlighting its prognostic value.[13] It has also been demonstrated that obesity is associated with a reduced FMD, and that weight loss can improve FMD.[14] Acute exercise-induced improvements in endothelial function may represent a mechanism by which physical activity confers cardiovascular protection. It has previously been demonstrated that acute alterations in FMD following exercise are predictive of chronic changes in FMD following multi-week exercise programs.[15] Interestingly, a previous study demonstrated that there was a significant increase in FMD for 1-4 hours following high-intensity exercise in normal-weight individuals, while obese individuals did not show a significant difference from baseline,[16] suggesting obesity may impair the cardiovascular benefits of exercise.

Importantly, obesity-related oxidative stress and inflammation suppress the function of eNOS via uncoupling[17] and reduce NO bioavailability,[4] thereby impairing the endothelium's vasodilatory function. A novel adipokine C1q-TNF-related-protein-9 (CTRP9) induces an increase in endothelial NO production via AMP-activated protein kinase (AMPK) phosphorylation and subsequent activation of eNOS.[18] CTRP9 is a paralog of adiponectin that suppresses TNF-alpha induced expression of endothelial adhesion molecules, thus potentially decreasing vascular inflammation.[19] Serum CTRP9 concentrations were found to have an inverse correlation with visceral fat amount in humans.[20] In contrast, serum CTRP9 levels were higher in obese individuals compared to the lean control group and then decreased following weight-loss surgery.[21] Lastly, we recently demonstrated CTRP9 increased immediately following both continuous moderate-intensity exercise and high-intensity interval exercise in obese individuals[22] establishing its responsiveness to exercise stimuli. Given CTRP9's direct mechanistic involvement in eNOS activation and NO production it may ameliorate obesity associated vascular dysfunction through enhanced NO bioavailability.

A recent study examined the effects of acute maximal exercise on postexercise hemodynamics in obese and normal-weight individuals and found that carotid-femoral pulse wave velocity (cfPWV, a measurement of central arterial stiffness) increased immediately following maximal intensity exercise in obese participants but decreased immediately in normal-weight individuals.[23] While some literature exists on the effects of consistent exercise training on endothelial function,[16] there is limited data on the acute effects of maximal aerobic exercise on endothelial function, particularly in obese individuals. Therefore, the purpose of this study was to investigate the impact of obesity on indicators of endothelial function in response to maximal aerobic exercise. We hypothesized that serum CTRP9 would increase similarly for both obese and normal-weight participants following maximal aerobic exercise; however, only the normal-weight participants

would have an exercise-mediated increase in FMD and serum NOx. This hypothesis was based on the expectation that obesity-related endothelial dysfunction would blunt the acute vasodilatory response to exercise in the obese individuals, while the adipokine CTRP9 would respond similarly to exercise stress regardless of body composition.

Methods

Participants

Twenty (11 obese and 9 normal weight) relatively healthy young males participated in this study. Male participants were specifically recruited to control for hormonal variability across the menstrual cycle, which is known to influence endothelial function.[37] Obesity was defined as having a BMI above 30 kg/m^2, while normal weight was in the range of 18.5 to 24.9 kg/m^2. Waist to hip ratio was utilized to ensure central adiposity within the obese group. Prior to data collection, all participants completed an informed consent form, a medical history questionnaire, and a 7-day physical activity record. The study received approval from the Institutional Review Board at Florida Atlantic University and was conducted in accordance with the Declaration of Helsinki, with written informed consent obtained from each participant. This research was carried out fully in accordance with the ethical standards of the International Journal of Exercise Science.[24]

Exclusion criteria included any known or suspected cardiovascular, metabolic, rheumatologic, or other inflammatory disease. Additionally, participants were excluded if they were users of any tobacco products (cigars, cigarettes, chewing tobacco, vapors), using any medications or supplements, or if they drank on average ten or more alcoholic beverages per week. These exclusion criteria were determined based on a health history questionnaire. Participants were required to undergo a minimum 8-hour overnight fasting period before the laboratory visit. Additionally, participants refrained from consuming alcohol, caffeine, and engaging in intense physical activity for at least 24 hours prior to the laboratory visit.

Protocol

Participants came to the laboratory between 7:00-7:30AM. Upon arrival, they filled out the informed consent questionnaire, medical history questionnaire, and 7-day physical activity questionnaire and their height and weight were measured (SECA 769, Chino, CA) along with hip and waist circumference. Additionally, following 20 minutes of sitting, resting heart rate was recorded using a heart rate monitor (Polar T31, Polar Electro, Kempele, Finland) and blood pressure was measured using a sphygmomanometer (752M-Mobile Series, American Diagnostic Corporation, Hauppauge, NY). Immediately after these recordings a trained phlebotomist performed blood sampling following standard aseptic techniques. A closed IV catheter system (BD Nexiva 20GA, REF 383516, Franklin Lakes, NJ, USA) was inserted in the superficial vein of the upper arm. A total of 30 mL of blood were collected into 5mL serum separation tubes (SST) for subsequent analysis. Before beginning the exercise protocol, the participants laid supine on the ultrasound bench for 20 minutes prior to obtaining their resting FMD measurement using an ultrasound (Phillips iU22, Foster City, CA, USA).

Participants completed a graded exercise test (approximately 12 to 15 minutes) on a treadmill (Norditrack X11i) designed to assess maximal oxygen consumption (VO_{2max}) measured by open-circuit spirometry (ParvoMedics Metabolic Measurement System [ParvoMedics, Sandy, UT, USA]) and maximal heart rate (HRmax). The maximal exercise protocol began with a three-

minute warm up at 60% predicted HRmax, followed by an increase in speed until 80% predicted HRmax. Subsequently, the grade was increased by 2% every two minutes until attainment of VO_{2max}. The validation of VO_{2max} was determined by either the primary criterion of a plateau in VO_2 or 2 of the 3 secondary criteria are achieved. The secondary criteria are (1) reaching predicted HRmax, (2) achieving a respiratory exchange ratio of >1.15, and (3) reporting a rating of perceived exertion (15-point Borg Scale) of 19 or 20. Importantly, these perceived exertion scales were used to measure the perception of stress each minute during exercise. If participants reported a difficulty to maintain exercise intensity, then the exercise testing was terminated. Blood samples were collected along with FMD measurements prior to the exercise protocol, upon immediate completion of the exercise protocol, 1 hour, and 2 hours into recovery.

For each blood draw, 30 mL of blood were collected into 5mL SST for serum protein analysis and centrifuged for 10 minutes at 1300 × g at room temperature. The serum was collected and stored in aliquots at –80° C for subsequent analyses of CTRP9 (Cloud-Clone Corp., Houston, TX, USA) and NOx (Enzo Life Sciences Inc., Farmingdale, NY, USA) by enzyme-linked immunosorbent assays (ELISA).

Previously established guidelines[25] were followed for the FMD measurement and protocol. The brachial artery was identified and imaged longitudinally using a Phillips L9-3 broadband 9.0 MHz vascular probe on the medial upper arm 2–10 cm above the antecubital fossa near the level of the heart. Landmarks were identified to ensure the same location for all repeated FMD measurements. Additionally, the probe was outlined to ensure identical placement for each time-point. Diameter and blood flow velocity were recorded in Digital Imaging and Communications in Medicine (DICOM) format using a duplex mode of ultrasound that allows simultaneous B-mode imaging for diameter measurements and Doppler for blood velocity (shear rate) using a Philips iU22 ultrasound. The insonation angle was set to 60° and gate width was adjusted for an accurate measurement of blood velocity.[25]

Prior to baseline FMD measurements the participants laid supine for 20 minutes and after acclimatization baseline measurements were recorded for 1 minute. A blood pressure cuff (WelchAllyn 406920 series) was placed two centimeters distal to the antecubital fold and was then inflated to ≤250 mmHg for 5 min. Before the cuff was release, measurements were recorded for 20 seconds and after the cuff release measurements were recorded for 3 minutes.

Validated software (Medical Imaging Applications, LLC., Coralville, IA, USA) was used for offline analysis of the recorded images for brachial diameter (mm) and blood velocity (shear rate). ECG gating was utilized for consistent cardiac cycles (end diastole) for brachial artery diameter measurements. FMD (%) was quantified as the peak brachial artery diameter observed after cuff release and reported as percent change from the average baseline diameter.

Statistical Analyses

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS version 29). Differences in baseline variables between the obese and normal-weight groups were assessed using independent *t* tests. A 2 (group) × 4 (time points: Pre, Post, R1 [one hour into recovery], and R2 [two hours into recovery]) repeated measures analyses of variance (ANOVA) were utilized to examine the effect of maximal aerobic exercise on serum levels of CTRP9 and NOx and FMD. Analyses of covariance (ANCOVA) were utilized to examine exercise-mediated serum CTRP9, NOx, and FMD, independent of group differences in heart rate, mean arterial blood pressure, and

VO_{2max}. Covariates (rHR, rMAP, and relative VO_2max) were selected based on their established influence on endothelial function[35,36] and exercise responses and because they significantly differed between the obese and normal weight groups at baseline. For example, resting heart rate can reflect autonomic tone, which modulates endothelial function. Resting mean arterial pressure directly influences shear stress patterns and baseline endothelial function[36] and is necessary to include as a covariate for our statistical analysis to remove the influence of hypertension within the obese group at baseline. Lastly, relative VO_2max represents cardiorespiratory fitness, which is strongly associated with endothelial function and was therefore controlled for statistically. F-statistics from ANOVA and ANCOVA analyses served as indicators of effect magnitude, with larger F-values indicating stronger effects. For correlation analyses, Pearson correlation coefficients (r) were interpreted as secondary measures of effect size, with values of $r \geq 0.10$, 0.30, and 0.50 representing small, medium, and large effect sizes, respectively, according to Cohen's conventions. Bonferroni post hoc analysis was utilized for pairwise comparisons. Greenhouse-Geisser correction of degrees of freedom was used when sphericity assumptions were violated. Pearson product-moment correlations were used to examine the relationship among outcome variables: CTRP9, NOx, and FMD. A priori power analysis was conducted using the program G*Power (version 3.1.9.7) to determine the required sample size. Based on the estimated effect size of 0.30 with an α-level of 0.05 for the outcome measures in response to maximal aerobic exercise[22], an estimated sample size of 18 participants in this study would achieve adequate power (>80%). As such, 20 participants were enrolled in this study to ensure adequate power. Statistical significance was defined as a *p*-value ≤ 0.05.

Results

Anthropometric Measurements of the Study Participants

As shown in Table 1, the analysis revealed a significant difference in weight ($p<0.001$), BMI ($p<0.001$), waist circumference ($p<0.001$), hip circumference ($p<0.001$), waist-to-hip ratio ($p=0.003$), relative VO_{2max} ($p<0.001$), resting heart rate ($p=0.003$), resting systolic blood pressure ($p<0.001$), resting diastolic blood pressure ($p=0.004$), and resting mean arterial pressure ($p<0.001$) between obese and normal-weight participants. There were no significant differences in age ($p=0.190$) and height ($p=0.492$) between the obese and normal-weight participants.

Table 1. Characteristics of Participants.

Variable	Normal-Weight (n=9)	Obese (n=11)	p-value
Age (years)	23.9 ± 3.6	26.6 ± 4.8	0.190
Height (m)	1.79±0.04	1.77±0.07	0.492
Weight (kg)	73±10	118±17	<0.001
Body Mass Index (kg/m²)	23±2	38±5	<0.001
Waist (cm)	81±7	114±16	<0.001
Hip (cm)	96±5	120±8	<0.001
WHR (a.u.)	0.84±0.05	0.95±0.09	0.003
VO_{2max} (mL/kg/min)	50±5	36±6	<0.001
Resting Heart Rate (bpm)	64±9	77±8	0.003
rSBP (mmHg)	114±7	147±13	<0.001
rDBP (mmHg)	74±8	87±9	0.004
rMAP (mmHg)	87±7	103±9	<0.001

Data are represented as mean ± SD. WHR= waist-to-hip ratio; VO_{2max} = maximal oxygen consumption; rSBP = resting systolic blood pressure ; rDBP = resting diastolic blood pressure; rMAP = resting mean arterial pressure.

At baseline, there were no significant differences in CTRP9 (p=0.103), NOx (p=0.726), FMD (p=0.858), and the brachial artery diameter (p=0.168) between obese and normal-weight participants. These insignificant differences in CTRP9 ($F_{[1,16]}$ =0.016, p=0.901), NOx ($F_{[1,15]}$ =0.000, p=0.983), and FMD ($F_{[1,19]}$ =0.002, p=0.963) remained, even after controlling for differences in rHR, rMAP, and relative $VO_{2\,max}$.

As shown in Figure 1, repeated measures ANOVA demonstrated a significant time effect for CTRP9 following maximal aerobic exercise ($F_{[1,12]}$ =3.128, p=0.049). Furthermore, this time effect persisted after controlling for differences in rHR, rMAP, and relative VO_{2max} ($F_{[1,9]}$ =4.102, p=0.023), with a decrease from immediately post to 1 hour after exercise mainly observed in the normal-weight group (p=0.007), with a return to the baseline level at two hours into recovery.

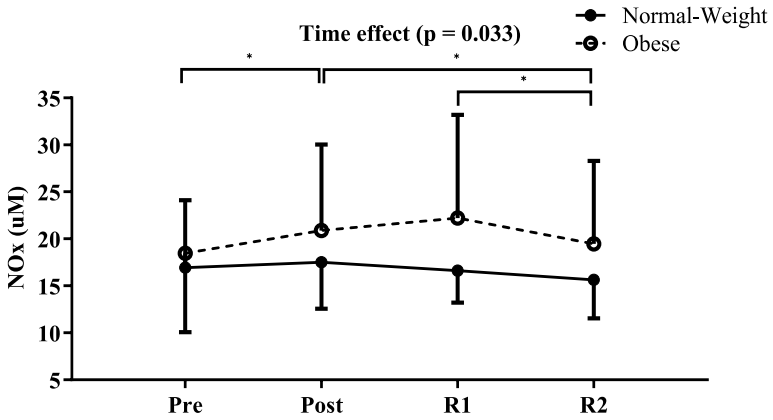

Figure 1. The CTRP9 response to maximal intensity exercise in normal-weight and obese participants. Data are presented as means ± SD. *Time effect Post vs. one hour into recovery.

A significant time effect for NOx was found following maximal aerobic exercise ($F_{[1,12]}$ =3.241, p=0.033), with a significant increase driven by the obese group from baseline to immediately after exercise (p=0.006) (Figure 2). However, after controlling for differences in rHR, rMAP, and relative VO_{2max}, the time effect for NOx following maximal exercise failed to persist ($F_{[1,9]}$ =0.1.313, p=0.290).

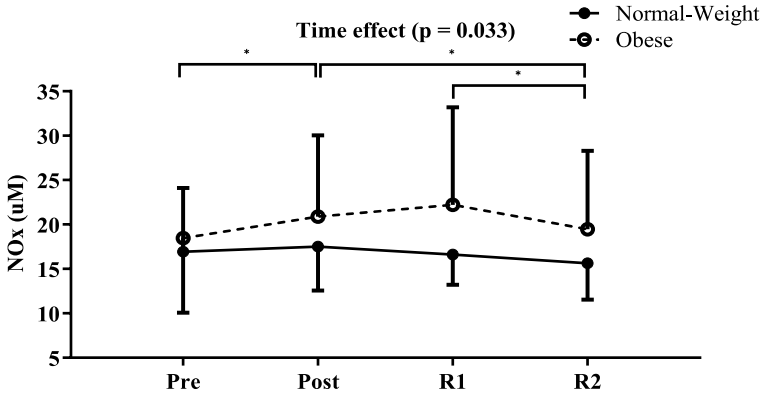

Figure 2. The NOx response to maximal intensity exercise in normal-weight and obese participants. Data are presented as means ± SD. *Time effects; Pre vs. Post, Post vs. two hours into recovery, one hour into recovery vs. two hours into recovery.

There was no significant time effect for FMD observed following maximal aerobic exercise ($F_{[1,18]}$ =0.864, p=0.452), even after controlling for rHR, rMAP, and relative VO_{2max} ($F_{[1,15]}$ =2.338, p=0.098; Figure 3). However, across all timepoints there was significantly lower FMD in the obese participants compared to the normal-weight participants ($F_{[1,18]}$ =6.981, p=0.017).

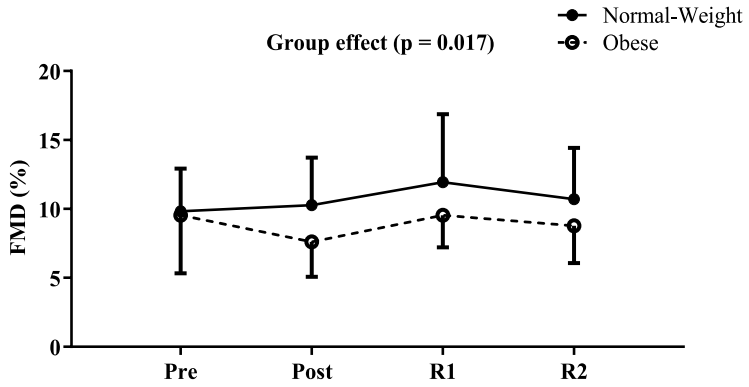

Figure 3. The FMD response to maximal intensity exercise in normal-weight and obese participants. Data are presented as means ± SD. Group effect (p = 0.017), across all timepoints.

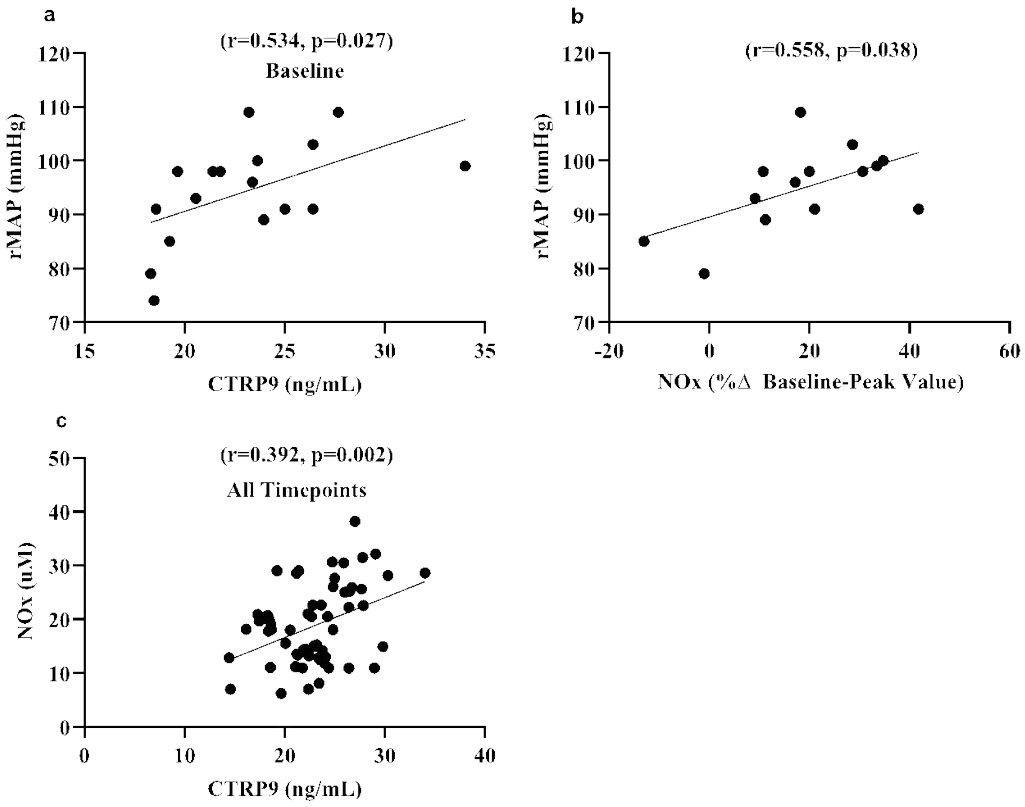

Figure 4. The relationship between baseline CTRP9 and resting mean arterial blood pressure (rMAP) in combined groups using Pearson correlation is presented in **panel a (n=17)**, the relationship between rMAP and the percent change (%Δ) in NOx from baseline to peak value in combined groups using Pearson correlation is presented in **panel b (n=14)**, and the relationship between CTRP9 and NOx and in combined groups across all time points using Pearson correlation is presented in **panel c**. Missing blood samples is the cause of the discrepancy between panel a and panel b sample size.

Correlational Analyses

As shown in Figure 4, when combined with both obese and normal-weight participants, a positive correlation was found between rMAP and CTRP9 at baseline (r=0.534, p=0.027; **panel a**). Moreover, the percent change (baseline to peak value) of NOx was positively correlated with rMAP in response to maximal aerobic exercise (r=0.558, p=0.038); **panel b**). Additionally, our analyses observed a significant positive correlation between CTRP9 and NOx when combining both groups by pairing all time points (r=0.392, p=0.002; **panel c**). However, no significant correlation was found between CTRP9 and FMD (r=-0.187, p=0.140) or NOx and FMD (r=-0.142, p=0.274) across all time points.

Discussion

The current study investigated the effect of maximal aerobic exercise on the serum levels of CTRP9 and NOx and brachial artery FMD responses in obese and normal-weight participants. We observed a significant time effect with an increase in serum NOx immediately following exercise, along with a decrease in serum CTRP9 at 1 hour into recovery particularly in the normal-weight group. When controlling for differences in rHR, rMAP, and relative VO_{2max}, the significant increase across time in serum NOx failed to persist. Furthermore, there was no significant change in FMD in response to maximal aerobic exercise in either the obese or normal-weight participants, whereas FMD was lower in obese participants compared to normal-weight participants. These findings suggest that maximal aerobic exercise elicits a transient alteration in biomarkers of endothelial function (NOx and CTRP9), independent of obesity status. It should be noted, however, that the time effect for serum NOx failed to persist after controlling for differences in rHR, rMAP, and relative VO_2max, suggesting that baseline hemodynamic differences between groups may have contributed to the observed response and that conclusions regarding obesity independence should be interpreted with caution.

We observed a significant reduction in serum CTRP9 during the recovery phase in both obese and normal-weight participants. In contrast to our previous work demonstrating increases in serum CTRP9 following both continuous moderate-intensity and high-intensity interval exercise,[22] the present study observed a reduction in CTRP9 during the recovery phase following a maximal graded exercise protocol. Whether this divergent response reflects the greater physiological stress of truly maximal exercise or the different protocol structure (graded to exhaustion vs. interval or steady-state) remains unclear. The CTRP9 response may be suppressed under conditions of extreme physiological stress, potentially due to increased catecholamine-mediated inhibition of adipokine secretion or accelerated clearance at maximal intensities. Additionally, the CTRP9 response may be dependent on longer exercise durations, however, in highly trained male rowers, a maximal rowing ergometer test to exhaustion (approximately 6 minutes in duration) transiently decreased serum adiponectin levels,[27] another anti-inflammatory adipokine with a high degree of amino acid sequence overlap to CTRP9.[26] The fact that a similar suppressive response is observed across both a brief maximal bout (~6 minutes) and the longer graded protocol in the present study (~12–15 minutes) suggests that exercise intensity, rather than duration alone, may be the primary driver of this adipokine suppression. Further research is necessary to better understand the impact of exercise intensity on the acute changes in adipokine circulation. Additionally, absence of significant differences between the obese and normal-weight groups indicates that the acute response of CTRP9 to maximal exercise is independent of body composition. We observed a weak positive correlation between CTRP9 and NOx supporting previous research that indicates CTRP9's role in the regulation of NO bioavailability.[18] However, our analyses did not observe a

significant correlation between CTRP9 and FMD, indicating that the transient reduction in CTRP9 after acute maximal aerobic exercise did not result in detectable changes in endothelial function as measured by FMD. The present findings suggest that CTRP9 may not influence endothelial function in the setting of maximal aerobic exercise or may require repeated bouts or longer exposure to elicit meaningful changes in FMD.

We observed a significant time effect for serum NOx following maximal aerobic exercise, with post-hoc analyses indicating that this increase was primarily driven by the obese group (baseline to immediately post-exercise, p=0.006), with minimal change observed in the normal-weight group. This differential response may reflect the greater resting blood pressure and sympathetic nervous system activation observed in the obese group at baseline, resulting in a larger exercise-induced shear stress stimulus and consequently greater eNOS activation[28] and NO production[29] compared to normal-weight participants. After controlling for rHR, rMAP, and relative VO_{2max}, the statistical significance of the NOx changes failed to persist, further indicating that these hemodynamic variables may have driven the transient increase. The elevation was transient as NOx decreased during recovery returning close to baseline levels. Additionally, there was no correlation between NOx and FMD, suggesting that the elevations in NOx did not improve endothelial function. This contrasts with a recent study that found a correlation between NOx and FMD in type 2 diabetes patients following a 12-week high-intensity interval training intervention.[30] This highlights that maximal aerobic exercise may acutely impair NO bioavailability through increased oxidative stress. For example, previous research has demonstrated that acute exercise results in increased oxidative stress and inflammation in an intensity-dependent manner[31] and increased oxidative stress can decrease NO bioactivity,[32] thereby limiting the degree of vasodilation. Thus, while serum NOx levels may increase, the enhanced vasodilation of the brachial artery may be attenuated by increased oxidative stress following maximal aerobic exercise.

Contrary to our expectations, we found no significant change in FMD in response to maximal aerobic exercise in either the obese or normal-weight participants. This is likely multifactorial. For example, while maximal aerobic exercise promotes NO production through increased shear stress,[28] it simultaneously increases oxidative stress[31] and increases the baseline diameter of the brachial artery that directly affects the FMD measurement. While prior studies in men and young healthy participants have similarly reported no significant acute change in FMD following maximal aerobic exercise,[33,34] those studies examined homogeneous samples and did not directly compare the FMD response between individuals with and without obesity. To the best of our knowledge, the present study is the first to directly compare the acute FMD response to maximal aerobic exercise between obese and normal-weight individuals, demonstrating that obesity does not further impair the FMD response to this exercise stimulus.

Our correlation analyses revealed a significant positive association between CTRP9 and NOx, further reinforcing the hypothesis that CTRP9 enhances NO production.[18] However, there was no significant association between CTRP9 and FMD or NOx and FMD. Although serum CTRP9 has been shown to be significantly higher in obese individuals and decreased following weight-loss surgery,[21] we speculate that additional factors such as oxidative stress and inflammation may be mediating endothelial function during acute maximal aerobic exercise. This highlights the need for future studies to simultaneously measure acute changes in inflammatory and oxidative stress biomarkers to better understand the complexities modulating the endothelial response to maximal aerobic exercise.

In conclusion, our findings indicate that maximal aerobic exercise elevates serum NOx and decreases serum CTRP9 but does not alter FMD. Some limitations of this study exist including the small sample size and inclusion of young healthy males only. The inclusion of only males limits the generalizability of these findings to female populations, in whom sex hormones are known to modulate endothelial function .[37] Additionally, the absence of direct measures of oxidative stress restricts our ability to fully describe the oxidative mechanisms at play during maximal exercise. Lastly, we did not directly measure adiposity using body fat percentage but rather classified the participants based upon their BMI and waist to hip ratio. Further studies should utilize a larger sample size to increase statistical power to detect subtle group differences, recruit a more diverse sample and explore additional biomarkers of oxidative stress and inflammation to further elucidate the mechanisms associated with endothelial function in response to acute maximal aerobic exercise.

Author Contribution Statement

CJH and BGF conceived and designed the research. BGF conducted the experiments. MS and BGF the analyzed data. MS, CJH, and BGF wrote the manuscript. All authors read and approved the manuscript.

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Conflict of Interest Notification

The authors report no financial or non-financial interests that are directly or indirectly related to the work submitted for publication.

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