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ORIGINAL ARTICLE

Aerobic or resistance exercise performed the previous day does not attenuate postprandial hyperglycemia-induced endothelial dysfunction in overweight/obese adults

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Abstract

Introduction Postprandial hyperglycemia (PPH) impairs vascular endothelial function (VEF). A single bout of aerobic exercise (AE) attenuates PPH-induced decreases in brachial artery flow-mediated dilation (FMD), a non-invasive measure of VEF, in healthy adults for up to 17 h post-exercise. Studies examining the effects of resistance exercise (RE) on postprandial FMD responses are lacking.

Purpose We hypothesized that a single bout of exercise performed the prior evening would attenuate PPH-induced decreases in FMD, independent of exercise modality.

Methods In a randomized, cross-over design, overweight/obese adults [$n = 11$ (8 women); 22 ± 4 years; 32.3 ± 5.8 kg m^{-2}] completed 3 separate trials: control (seated rest), AE (30 min at ~ 60% VO_{2max}), or whole-body RE (30 min, 6 exercises, 3×10-repetition maximum). Each trial occurred 14–17 h prior to an oral glucose tolerance test (OGTT). Brachial artery FMD and plasma glucose and insulin were measured prior to and at 30-min intervals for 2 h following the OGTT. Repeated-measures ANOVA and Bonferroni post hoc tests were used to evaluate differences within and between trials.

Results Trials occurred 15.3 ± 1.0 h prior to the OGTT. Relative to baseline, FMD transiently decreased ($P < 0.05$) at 30–60 min post-ingestion, plasma glucose increased ($P < 0.01$) at 30–90 min post-ingestion, and plasma insulin increased ($P < 0.01$) at 30–120 min post-ingestion. No between trial differences were observed for FMD, glucose, or insulin.

Conclusions Aerobic or resistance exercise performed the evening prior to an OGTT does not attenuate postprandial decreases in brachial artery FMD in overweight/obese adults.

Keywords Flow-mediation dilation · Insulin · Glucose · Acute exercise

Abbreviations

AE	Aerobic exercise
ANOVA	Analysis of variance
AUC	Area under the curve
BMI	Body mass index
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
ELISA	Enzyme-linked immunosorbent assay
FMD	Flow-mediated dilation
HDL-C	High-density lipoprotein cholesterol
HOMA-IR	Homeostatic model assessment of insulin resistance
HR	Heart rate
LDL-C	Low-density lipoprotein cholesterol
NO	Nitric oxide
OGTT	Oral glucose tolerance test
PPH	Postprandial hyperglycemia
RE	Resistance exercise
RER	Respiratory exchange ratio
RM	Repetition maximum
RPE	Rating of perceived exertion
SBP	Systolic blood pressure
TC	Total cholesterol
TG	Triglycerides
VEF	Vascular endothelial function
VO_{2max}	Maximal oxygen consumption

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Introduction

Epidemiological studies support that postprandial hyperglycemia (PPH), the transient increase in blood glucose levels following a carbohydrate challenge, better predicts cardiovascular disease (CVD)-related morbidity and mortality compared to fasting glucose levels (Tominaga et al. 1999; DECODE Study Group 2001). Studies in healthy adults show that PPH transiently impairs vascular endothelial function (VEF) (Ceriello et al. 2002; Mah et al. 2011), suggesting that repeated postprandial insults to the vasculature might contribute to the development of CVD. Thus, strategies that lower PPH may mitigate its adverse vascular effects and reduce CVD risk.

Regular exercise decreases CVD risk (Pedersen and Saltin 2015), in part, by reducing PPH-mediated impairment of VEF. In support, PPH responses to an oral glucose tolerance test (OGTT) were lower in regular aerobic or resistance exercisers compared to healthy sedentary adults (Das et al. 2018). Further, VEF (assessed non-invasively via brachial artery flow-mediated dilation (FMD)) transiently decreased following ingestion of the OGTT in sedentary participants, an effect not observed in exercisers (Das et al. 2018). To our knowledge, only one study has examined the efficacy of a single bout of exercise to attenuate PPH-induced decreases in brachial artery FMD. In healthy adults, brachial artery FMD decreased after ingestion of a high carbohydrate meal (Weiss et al. 2008). Compared to a non-exercise control trial, the postprandial brachial artery FMD response curve was shifted upward and the plasma glucose and insulin response curves were shifted downward when a 60 min bout of aerobic exercise (AE) was performed 17 h prior to the high carbohydrate meal (Weiss et al. 2008). These data (Weiss et al. 2008) suggest that AE performed the previous day improves postprandial vascular and metabolic responses. However, the high percentage (70%) of participants who performed regular AE (Weiss et al. 2008) may have attenuated the negative influence of PPH on brachial artery FMD (Das et al. 2018), lessening the generalizability of these findings to physically inactive adults. Current physical activity guidelines recommend that adults participate in 30 min of moderate-intensity AE at least 5 days/week, as well as resistance exercise (RE) at least 2 days/week (Garber et al. 2011). It is currently unknown if single bouts of AE that meet current physical activity recommendations (Garber et al. 2011) influence postprandial FMD responses.

Studies examining the effects of a single bout of RE on PPH are equivocal, with most (Chapman et al. 2002; Mitchell et al. 2011; Fluckey et al. 1994), but not all (Andersen and Hostmark 2007), studies showing no effect of prior (i.e., 14–24 h) RE on PPH. In overweight/obese adults, neither 30 min of AE nor 30 min of RE (consistent with Australian physical activity recommendations) performed 14 h prior to a mixed meal challenge lowered postprandial blood glucose and insulin responses or altered the postprandial decrease in central augmentation index, a measure of arterial stiffness (Ho et al. 2011). To our knowledge, no studies have determined the influence of a single bout of RE on PPH-induced changes in FMD. In the present study, we examined the efficacy of a single 30 min bout of AE or RE to prevent reductions in brachial artery FMD responses to an OGTT in overweight/obese adults. By addressing this knowledge gap, our rationale for this study is that it will lead to the development of specific exercise strategies that alleviate the adverse metabolic and vascular effects during the postprandial period.

The purpose of this study was to determine if 30 min bouts of exercise attenuate PPH-induced decreases in brachial artery FMD in physically inactive overweight/obese adults. In addition, we sought to determine whether differences in postprandial glycemic and FMD responses existed between exercise modalities. Compared to a non-exercise control trial, we hypothesized that a single bout of AE or RE performed the evening (i.e., 14–17 h) prior to an OGTT would attenuate PPH-induced decreases in brachial artery FMD, independent of exercise modality.

Methods

Study design

The protocol for this study was approved by the Institutional Review Board at Miami University and written informed consent was obtained from all participants. Following screening and familiarization visits, participants completed three randomized study trials in a cross-over design: (1) AE, (2) RE, and (3) seated rest (control). Each study trial consisted of two visits to the laboratory. The first visit occurred in the late afternoon/early evening (e.g., 4:00 p.m.) and was preceded by a 2-h fast and no exercise for 48 h. During this visit, subjects performed either AE, RE, or seated rest (control). Following the first visit, participants returned the next morning (e.g., 8:00 a.m.) after an overnight fast. The morning visit occurred 14–17 h following the completion of AE, RE, or control. During each morning visit, brachial artery FMD was assessed and blood collected prior to and at 30-min intervals for 2 h following ingestion of the glucose beverage.

Participants

Eleven overweight/obese men and premenopausal women were recruited to participate in this study. A phone

screening was conducted to determine initial eligibility. Participants were required to meet the following inclusion criteria: 18–50 years of age; non-smokers, body mass index (BMI) ≥ 25 kg m^{-2}; not taking any anti-hypertensive, lipid-lowering, and/or anti-diabetic medications; not taking any dietary supplements; no musculoskeletal injuries or physical limitations affecting ability to exercise; weight stable (± 2 kg) over the past 3 months; no history or symptoms of cardiovascular, pulmonary, metabolic, or neurological disease; and < 2 days/week of moderate-intensity physical activity over the past 6 months.

Screening visit

Following the initial phone screening, individuals determined to be eligible for the study were scheduled for an in-person screening visit after an overnight fast and no exercise for 48 h. During this visit, participants were given a detailed explanation of the study, provided informed consent, and completed a health history and physical activity questionnaire. Additionally, women completed a questionnaire at the screening visit and at each trial to assess their menstrual history and oral contraceptive use. Next, height and waist circumference were measured using standard procedures. Body mass and composition were measured using bioelectrical impedance analysis (InBody 770, Cerritos, CA, USA). Heart rate (HR) and blood pressure were recorded two times following 5 min of seated rest using an automated blood pressure monitor (Omron HEM907XL, Bonnockburn, IL, USA). A fasted blood sample was obtained from an antecubital vein for the determination of blood lipid concentrations (Cholestech LDX Analyzer, Abbott, Abbott Park, IL, USA) (Table 1). Participants then completed a graded exercise test to volitional fatigue on a motor-driven treadmill to determine maximal oxygen consumption (VO_{2max}). Expired air was collected and analyzed using a calibrated Parvomedics True One 2400 Metabolic System (ParvoMedics, Sandy, UT, USA). Participants must have met two of the following criteria to determine if VO_{2max} was achieved: respiratory exchange ratio (RER) ≥ 1.10, 10 bpm of age-predicted maximum heart rate HR, rating of perceived exertion (RPE) ≥ 18, and plateau of VO_2 with an increase in work rate.

Familiarization visit

Approximately, 1 week following the screening visit, participants reported to the laboratory for a familiarization session with the RE machines and determination of 10-repetition maximum (RM) in the following exercises: leg press, seated chest press, seated leg curl, lat pulldown, seated shoulder press, and seated row. All RE machines, with the exception of leg press (plate loaded), were selectorized weight stack design. Participants were instructed to refrain from strenuous physical activity for 48 h prior to the familiarization visit. Participants completed a 5 min treadmill warm-up at self-selected speeds upon arrival to the laboratory. For each exercise, an initial set of 10 repetitions was performed with a light load. The participant then performed successive sets with increasing loads until the study personnel determined the 10-RM load following the protocol from the National Strength and Conditioning Association (National Strength and Conditioning Association. Essentials of Strength Training and Conditioning, 4th edition 2016). Each participant's 10-RM load was determined within 3–5 sets.

Table 1 Participant characteristics

Variable	Value
Age (years)	21.8 ± 3.8
BMI (kg m^{-2})	32.3 ± 5.8
Body fat (%)	39.2 ± 8.8
Waist circumference (cm)	100.2 ± 19.7
SBP (mmHg)	113 ± 7
DBP (mmHg)	77 ± 5
VO_{2max} (ml kg min^{-1})	31.7 ± 5.8
TC (mmol L^{-1})	4.5 ± 1.0
HDL-C (mmol L^{-1})	1.5 ± 0.5
LDL-C (mmol L^{-1})	2.3 ± 0.9
TG (mmol L^{-1})	1.3 ± 0.9

Data are mean ± SD (n = 11; 8 women)

BMI body mass index, *DBP* diastolic blood pressure, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *SBP* systolic blood pressure, *TC* total cholesterol, *TG* triglycerides, VO_{2max} maximal oxygen consumption

Study trials

Participants completed three randomized study trials in a cross-over design: (1) AE, (2) RE, and (3) seated rest (control). The first study trial occurred approximately 1 week following the familiarization visit. Study trials were separated by ≥ 1 week in men and approximately 1 month in women to ensure that women were tested at approximately the same phase of their individual menstrual cycle (Thijssen et al. 2011). Each study trial was conducted over 2 days and consisted of a late afternoon/early evening visit and a morning visit 14–17 h later. During the late afternoon/early evening visit, participants completed either AE, RE or seated rest. The morning visit consisted of the completion of a 2 h OGTT. Within participants, the three late afternoon/early evening visits and the three morning visits occurred at the same time of day (≤ 1 h).

Participants completed a 1 day food log prior to their first study trial and were instructed to replicate their dietary intake the day prior to their second and third trials. Food

records were assessed for energy and nutrient intake (Food Processor Nutrition Analysis software, version 11.2, ESHA Research, Salem, OR, USA) by study personnel.

A 5 min treadmill warm-up at a self-selected speed preceded each exercise bout. During the AE bout, participants completed 30 min of continuous treadmill exercise at approximately 60% VO_{2max}. During the AE trial, expired air was collected and analyzed (ParvoMedics) every 5 min during the first 15 min of exercise to confirm that participants were within ± 5% of their individual 60% VO_{2max} (Harris et al. 2008). Adjustments were made to treadmill speed and/or grade to maintain the correct intensity. Once a stable VO_2 within the desired range was observed, the mouthpiece was removed for the remaining duration of the exercise session. During the RE bout, participants completed six exercises in the following order: leg press, seated chest press, seated leg curl, lat pulldown, seated shoulder press, and seated row. Each exercise was performed for 3 sets of 10 repetitions per set, with the load corresponding to the previously determined 10-RM load. Rest periods between sets and exercises were 90 and 120 s, respectively. The RE bout lasted approximately 30 min. A 5 min cool-down on the treadmill at a self-selected speed concluded each exercise bout. The non-exercise control trial consisted of 40 min of quiet rest in the laboratory. Heart rate and RPE were assessed at rest during each trial, at 10-min intervals during the control trial, at 5-min intervals during the AE trial, and following each set during the RE trial. At the conclusion of each trial (exercise and control), participants were instructed to avoid strenuous exercise for the remainder of the day, continue recording their dietary intake, and report back to the laboratory the next morning (14–17 h later) after an overnight fast. At each morning visit, participants were asked if they had complied with all pre-testing instructions. No morning visits were rescheduled due to non-compliance with the pre-testing instructions.

The next morning, body mass and composition were reassessed, and a flexible catheter was inserted into an antecubital vein followed by ≥ 15 min of supine rest in a quiet, darkened, temperature-controlled room. Next, baseline brachial artery FMD was assessed and blood was collected, immediately followed by ingestion of a flavored, glucose beverage (1 g glucose kg^{-1} body mass; TRUTOL, Fisher Diagnostics). Brachial artery FMD was assessed and blood samples were obtained at 30-min intervals during the 2 h postprandial period. At least 15 min of quiet, supine rest preceded each FMD measurement during the postprandial period.

Brachial artery flow-mediated dilation

Brachial artery FMD was assessed using high-frequency ultrasonographic imaging as previously described (Ballard et al. 2013). Briefly, the rapid inflation cuff was placed on the forearm just distal to the elbow, and the brachial artery was imaged on the upper arm using a 5- to 12-MHz multifrequency linear array transducer connected to a high-resolution ultrasound (uSmart 3300; Terason, Burlington, MA, USA). Pre-occlusion recording of blood velocity and arterial diameter were taken for 1 min. The cuff was then rapidly inflated to a pressure of 200 mmHg for 5 min using a rapid cuff inflator (Hokanson E20, Bellvue, WA, USA), and then released. Blood velocity and arterial diameter were recorded for the last minute of cuff inflation, and for 3 min following deflation. Ultrasound recordings were analyzed using automated edge-detected software with end-diastolic gating (Medical Imaging Applications, Iowa City, IA, USA). Shear rate area under the curve (AUC) was calculated to determine the hyperemic stimulus responsible for FMD. Flow-mediated dilation AUC was measured using the trapezoidal method. The ultrasonographer for these experiments (CJV) has calculated between day ($n = 12$; ≥ 1 week) reproducibility of fasting brachial artery FMD in our laboratory; the intra-class correlation coefficient and coefficient of variation (CV) was 0.80 and 14.3%, respectively.

Blood analysis

Whole blood was collected at each time point into evacuated tubes. Plasma was obtained by centrifugation (2000×*g*, 15 min, 4 °C) and transferred to cryogenic vials, which were stored at − 80 °C until analyses were completed. Plasma glucose was measured using a commercially available clinical assay (Pointe Scientific, Canton, MI, USA; inter-assay CV = 3.3%) on a microplate reader (BioTek Instruments, Synergy HT, Winooski, VT, USA). Plasma insulin was measured via enzyme-linked immunosorbent assay (ALPCO, Salem, NH, USA; inter-assay CV = 4.8%). Total insulin and glucose AUC were measured using the trapezoidal method. Whole-body insulin sensitivity during the OGTT was determined by calculating the insulin sensitivity index (ISI) (Matsuda and DeFronzo 1999). The homeostatic model assessment of insulin resistance (HOMA-IR) was calculated to assess insulin resistance (Matthews et al. 1985).

Statistical analyses

Sample size was determined from a previous cross-over study in 16 healthy men showing that ingestion of an OGTT significantly decreased brachial artery FMD by 43% (from 6.8% at baseline to 3.9% at 60 min postprandial) (Mah et al. 2011). Assuming that prior exercise would attenuate the postprandial decrease in FMD by ~ 50% (i.e., from 6.8% at baseline to 5.3% at 60 min postprandial), our calculations indicated that 11 participants provided 80% power ($P < 0.05$) to detect a 1.5% absolute difference in FMD at

60 min post-glucose ingestion between the exercise and control trials. Guidelines suggest that a 1.5–2% absolute change in FMD is the minimal statistically significant improvement that can be detected with intervention (Corretti et al. 2002). One-way analysis of variance (ANOVA) was used to evaluate between trial differences in fasting measures, dietary intake, ISI, HOMA-IR, and glucose and insulin AUC. Two-way repeated measures ANOVA were used to evaluate differences due to time, trial, and their interaction. In the presence of significant main or interaction effects, pairwise differences within and between groups were evaluated using Bonferroni post hoc tests. Effect size (ES) was calculated using Cohen's *d*. An α-level of $P \leq 0.05$ was considered statistically significant for all analyses. Data are means ± SD and/or 95% confidence intervals (CI).

Results

Participants and Dietary intakes

Eleven overweight/obese individuals completed the study (Table 1). Participants were physically inactive, overweight/obese based on BMI, and of poor cardiorespiratory fitness on the basis of VO_{2max} (American College of Sports Medicine. Guidelines for Exercise Testing and Prescription, 10th edition 2018). Of the 11 participants, 7 had elevated waist circumference (≥ 102 cm for men and ≥ 88 cm for women), 3 had impaired fasting glucose (≥ 5.6 mmol L^{-1}), 3 had low HDL-cholesterol (< 1.0 mmol L^{-1} for men and < 1.3 mmol L^{-1} for women), and 3 had elevated fasting triglycerides (≥ 1.7 mmol L^{-1}) (Alberti et al. 2009). No participant had hypertension (systolic blood pressure ≥ 130 mmHg and diastolic blood pressure ≥ 85 mmHg) (Alberti et al. 2009). Dietary intakes of energy and macronutrients did not differ between trials (Table 2).

Study trials

Body mass did not differ between trials (97.2 kg, 97.9 kg, and 98.4 kg for trials 1, 2, and 3, respectively; $P > 0.05$). All exercise and control visits were conducted between 3:00 and 6:00 p.m., which was 15.3 ± 1.0 h prior to the OGTT. Participants completed the 30 min of AE at 58.7 ± 5.5% VO_{2max}. Average RPE did not differ between the AE and RE trials (12.6 ± 1.8 and 13.5 ± 1.1 for AE and RE, respectively; $P > 0.05$), indicating similar perception of intensity between the exercise bouts.

Table 2 Participants' dietary intakes

Variable	Resistance	Aerobic	Control
Total energy (kcal day^{-1})	2218 ± 843	2153 ± 744	2131 ± 781
Carbohydrate (%)	44.9 ± 7.1	45.8 ± 7.6	43.5 ± 6.6
Protein (%)	15.8 ± 5.7	15.1 ± 3.8	15.6 ± 4.2
Fat (%)	38.7 ± 6.7	38.3 ± 6.3	39.8 ± 5.2
Saturated fat (g day^{-1})	32.4 ± 16.6	30.2 ± 12.2	30.2 ± 13.4

Data are means ± SD. Dietary intakes were determined from food records for the 1 day preceding each oral glucose tolerance test

Plasma glucose and insulin

Fasting plasma glucose and insulin did not differ between trials ($P > 0.05$). Plasma glucose increased ($P < 0.001$) by 30 min following glucose beverage ingestion and remained elevated from baseline through 90 min (Fig. 1a). Plasma glucose AUC did not differ between trials (Fig. 1b). The 95% CI for plasma glucose AUC were 816–1065; 801–979; and 783–975 mmol min L^{-1} for control, AE, and RE, respectively. The ES for plasma glucose AUC was 0.63 for AE versus control and 0.59 for RE versus control. Plasma insulin increased ($P < 0.01$) by 30 min following glucose beverage ingestion and remained elevated from baseline through 120 min (Fig. 2a). Plasma insulin AUC did not differ

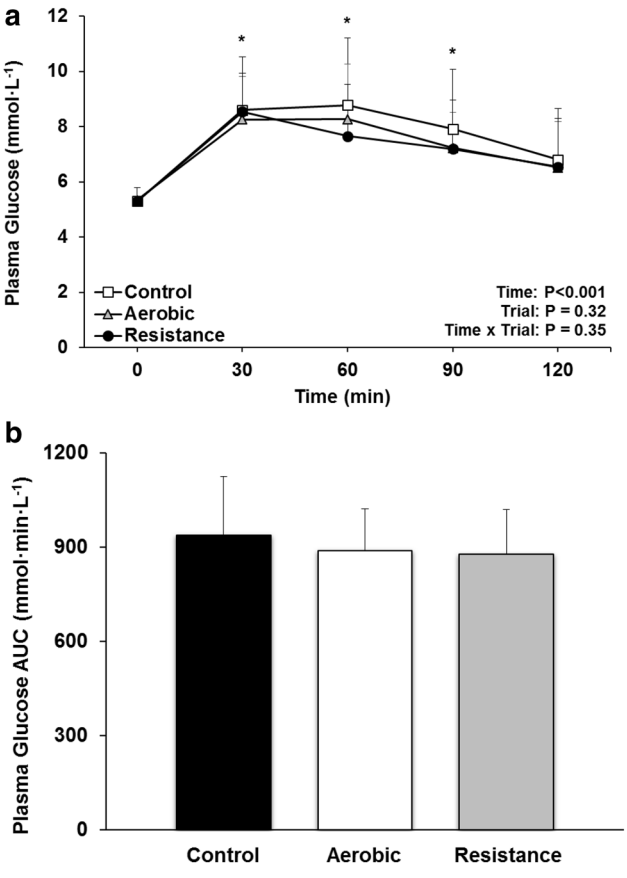

Fig. 1 Absolute plasma glucose (**a**) and AUC (**b**) responses for the control and exercise trials. Data are means ± SD. *$P < 0.05$ from 0

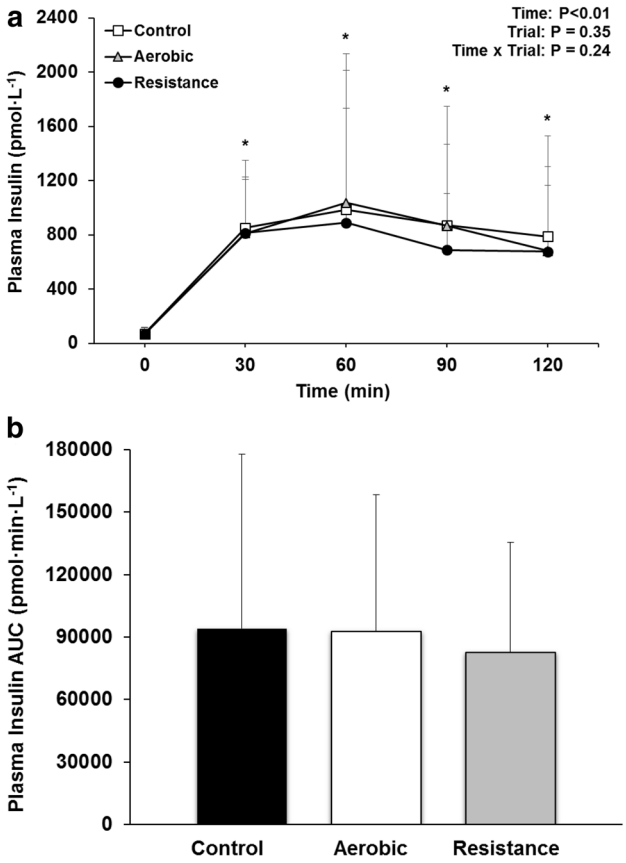

Fig. 2 Absolute plasma insulin (**a**) and AUC (**b**) responses for the control and exercise trials. Data are means ± SD. *$P < 0.05$ from 0

between trials (Fig. 2b). The 95% confidence intervals for plasma insulin AUC were 37,716–150,433; 48,392–136,856; and 47,369–118,245 pmol min L^{-1} for control, AE, and RE, respectively. The ES for plasma insulin AUC was 0.09 for AE versus control and 0.43 for RE versus control. Change from baseline was calculated for plasma glucose and insulin but findings did not differ from absolute values. The ISI (3.50 ± 2.03; 3.39 ± 1.63; and 3.78 ± 2.08 and for control, AE, and RE, respectively) and HOMA-IR (2.37 ± 1.58; 2.42 ± 1.68; and 2.28 ± 1.56 and for control, AE, and RE, respectively) did not differ between trials.

Brachial artery FMD

Pre-occlusion brachial artery diameter did neither differ throughout the postprandial period nor between trials (Table 3). Peak brachial artery diameter decreased ($P < 0.01$) from baseline at 30 min, with no differences detected between trials (Table 3). Brachial artery FMD responses (Fig. 3) decreased ($P < 0.05$) from baseline at 30 and 60 min, with no differences detected between trials. Absolute FMD values showed similar responses. Flow-mediated dilation AUC did not differ between trials (898 ± 287; 893 ± 260; and 973 ± 266% min^{-1} for control, AE, and RE, respectively). The 95% confidence intervals for brachial artery FMD AUC were 705–1090; 718–1068; and 795–1152% min^{-1} for control, AE, and RE, respectively. The ES for FMD AUC was 0.02 for AE versus control and 0.34 for RE versus control.

Table 3 Changes in brachial artery diameter and shear rate

Variable	Resistance	Aerobic	Control	Time	Trial	Time × trial
Pre-occlusion diameter (mm)				0.65	0.44	0.06
0 min	3.64 ± 0.46	3.61 ± 0.49	3.60 ± 0.41			
30 min	3.65 ± 0.47	3.65 ± 0.48	3.57 ± 0.39			
60 min	3.70 ± 0.49	3.63 ± 0.42	3.61 ± 0.41			
90 min	3.75 ± 0.49	3.61 ± 0.47	3.65 ± 0.37			
120 min	3.68 ± 0.47	3.63 ± 0.47	3.60 ± 0.44			
Maximal post-occlusion diameter (mm)				0.01	0.14	0.67
0 min	4.02 ± 0.48	3.95 ± 0.48	3.93 ± 0.38			
30 min*	3.90 ± 0.43	3.86 ± 0.44	3.81 ± 0.35			
60 min	3.96 ± 0.53	3.88 ± 0.42	3.83 ± 0.37			
90 min	3.97 ± 0.49	3.89 ± 0.47	3.92 ± 0.37			
120 min	3.98 ± 0.44	3.91 ± 0.48	3.90 ± 0.41			
Shear rate AUC				0.01	0.97	0.94
0 min	27,667 ± 7392	26,289 ± 8127	27,418 ± 6063			
30 min	23,625 ± 8529	22,608 ± 7488	21,940 ± 6593			
60 min	22,599 ± 8400	24,091 ± 9363	23,309 ± 5512			
90 min	22,433 ± 7368	23,393 ± 11,726	22,000 ± 6375			
120 min	23,877 ± 9760	24,848 ± 9887	24,415 ± 6987			

Data are means ± SD

*$P \leq 0.01$ from 0 min. Abbreviations: AUC, area under the curve

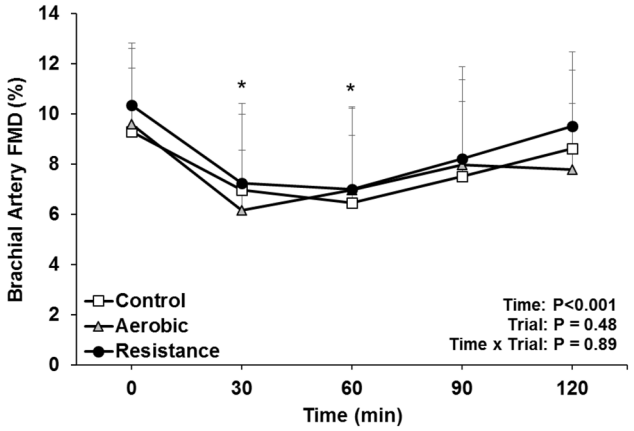

Fig. 3 Brachial artery flow-mediation dilation (FMD) responses for the control and exercise trials. Data are means ± SD. *$P < 0.01$ from 0

A main effect for time ($P < 0.01$) was observed for shear rate AUC (Table 3), but no pairwise differences were detected.

Discussion

To our knowledge, our study is the first to examine the effect of different exercise modalities on postprandial FMD responses to an OGTT in overweight/obese individuals. Overweight/obese adults were chosen for this study as they make up approximately two-thirds of the U.S. adult population (Fryar et al. 2016). Additionally, obese individuals exhibit impaired glucose tolerance as evidenced by exaggerated PPH responses compared to lean individuals (Jonk et al. 2011), suggesting that exercise may attenuate PPH to a greater extent in overweight/obese individuals compared to previous observations in healthy, physically active adults (Weiss et al. 2008; Andersen and Hostmark 2007). Consistent with prior studies (Ceriello et al. 2002; Das et al. 2018; Mah et al. 2011), we showed that PPH transiently decreased brachial artery FMD, a non-invasive measure of VEF that reflects nitric oxide (NO)-dependent dilation (Green et al. 2014) and predicts future CVD risk (Yeboah et al. 2009; Inaba et al. 2010). In contrast to our hypothesis, performing a single bout of AE or RE approximately 15 h prior to an OGTT did not attenuate PPH responses or postprandial decreases in brachial artery FMD compared to a non-exercise control trial.

Few studies have simultaneously evaluated the effects of a single bout of exercise on postprandial metabolic and vascular responses. In one study, 13 healthy men and women (48 years, BMI = 24 kg/m^2) consumed a high carbohydrate meal (101 g carbohydrate) on two separate occasions: (1) 17 h after performing a 60 min bout of AE, and (2) after 48 h of no exercise (Weiss et al. 2008). Blood samples were collected and brachial artery FMD was assessed at baseline and every 30 min for 150 min after consumption of the high carbohydrate meal. AE did not eliminate postprandial decreases in brachial artery FMD (time effect: $P < 0.01$), but did shift the FMD response curve upward (treatment effect: $P < 0.001$). Furthermore, AE shifted the postprandial plasma glucose and insulin response curves downward (treatment effect: $P < 0.05$), increased the plasma ISI, and decreased HOMA-IR. These data suggest that performing AE the evening prior to a high carbohydrate challenge attenuates postprandial decreases in brachial artery FMD and improves insulin sensitivity in healthy adults (Weiss et al. 2008). Compared to the present study, the smaller postprandial decrease in brachial artery FMD reported by Weiss et al. (Weiss et al. 2008) may be explained by differences in habitual exercise participation between studies that influence PPH responses (Das et al. 2018). Greater PPH responses in sedentary individuals compared to regular exercisers (Das et al. 2018) may have induced more oxidative stress that reduced NO bioavailability (Mah et al. 2011), potentially explaining the greater postprandial decrease in FMD in our physically inactive participants. In addition, differences in BMI between our participants and others (Weiss et al. 2008) may have increased PPH (Jonk et al. 2011), and thus contribute to our greater reduction in postprandial FMD responses. Further, others using a high-fat meal have shown that higher exercise intensity (Tyldum et al. 2009), age of participants (Sedgwick et al. 2013), and performing exercise closer (e.g., 1 h) to meal ingestion (Short et al. 2012) are important considerations when examining postprandial metabolic and/or vascular changes. Thus, future research is warranted to more definitively determine the most efficacious intensity, duration, and timing of exercise to attenuate postprandial disturbances in healthy and at-risk populations.

A separate study in 10 healthy resistance-trained men (24 years, BMI = 24 kg m^{-2}) reported that RE performed 14 h before consumption of a carbohydrate-rich meal (1 g carbohydrate kg^{-1} body mass) did not significantly lower 2 h postprandial blood glucose or insulin responses compared to a non-exercise control trial (Andersen and Hostmark 2007). However, peak blood glucose values and incremental area under the blood glucose curve in the period 0–60 min after carbohydrate ingestion were lower when the carbohydrate-rich meal was preceded by RE (Andersen and Hostmark 2007). Others have shown that RE performed 15–18 h prior to a high-carbohydrate challenge does not significantly attenuate postprandial blood glucose responses (Fluckey et al. 1994; Chapman et al. 2002). To our knowledge, only one study has examined the effects of prior AE and RE of equal duration on postprandial glucose tolerance in obese individuals. In 10 obese, untrained men (24 years, BMI = 33 kg m^{-2}), neither 60 min of AE or 60 min of RE performed 24 h prior to an

100 g OGTT-lowered postprandial blood glucose or insulin responses compared to a non-exercise trial (Mitchell et al. 2011). Brachial artery FMD was not measured in this study (Mitchell et al. 2011). The present study in overweight/obese men and women advances existing knowledge by showing that neither 30 min of AE nor RE performed ~ 15 h prior attenuate the adverse effect of PPH on brachial artery FMD. However, we acknowledge that any beneficial effect of performing exercise closer to the postprandial challenge may have been negated by the shorter exercise duration.

Studies comparing the effects of prior AE and RE on postprandial changes in vascular function are scarce. In 18 middle-aged overweight/obese individuals (59 years; BMI = 32 kg m^{-2}), no between trial differences were found for the decrease in central augmentation index (a measure of arterial stiffness) in response to a mixed meal challenge (50% carbohydrate, 15% protein, 35% fat) when comparing a non-exercise control condition to 3 separate exercise trials performed 14 h prior to the meal: (1) 30 min of AE (60% of heart rate reserve), (2) 30 min of RE (8–12 reps at 10-RM), and (3) 30 min of combined AE and RE (Ho et al. 2011). Further, blood glucose and insulin AUC did not differ between trials (Ho et al. 2011). Our results are in agreement as they demonstrate that performing AE and RE the day prior to an OGTT has a minimal effect on postprandial vascular and metabolic responses.

Studies in humans support that PPH transiently impairs VEF, at least in part, by inducing oxidative stress responses that reduce NO bioavailability (Mah et al. 2011). Oxidative stress and NO bioavailability were not measured in the present study as changes in postprandial responses of these variables are dependent on PPH (i.e., glucose AUC) (Mah et al., 2011), which did not differ between trials. Our sample size was similar to others examining a single bout of exercise on postprandial glucose tolerance and/or VEF (Weiss et al. 2008; Chapman et al. 2002; Mitchell et al. 2011). Only young, overweight/obese individuals were tested in the present study. The young age of our participants may have resulted in normal glucose tolerance due to compensatory hyperinsulinemia. Thus, future studies are needed to determine if age and/or insulin resistance influences the effect of exercise on postprandial VEF responses in overweight/obese adults. Sleep time and hydration status were not assessed prior to each OGTT in the present study. Future postprandial studies should attempt to standardize sleep time and hydration status as both short-term sleep deprivation (Spiegel et al. 1999) and hypohydration (Johnson et al. 2017) have been shown to decrease glucose tolerance. Finally, postprandial vascular and metabolic responses to a relative, rather than an absolute (i.e., 75 g) glucose dose were assessed in the present study. Future studies are warranted to determine differences in postprandial responses to ingesting an absolute versus a relative glucose dose in healthy and clinical populations.

Conclusion

This study advances existing knowledge regarding exercise and postprandial VEF responses (Weiss et al. 2008) by showing that performing a single bout of AE or RE approximately 15 h prior to an OGTT does not attenuate PPH or postprandial decreases in brachial artery FMD in overweight/obese adults. Findings from our study suggest that improvements in postprandial VEF responses in healthy adults performing AE (Weiss et al. 2008) does not extend to physically inactive overweight/obese individuals. The reason for these differences between populations remains to be explored, but may be due to differences in habitual physical activity levels that influence PPH responses (Das et al. 2018) and/or exercise duration. No studies have been conducted to directly examine the influence of exercise bouts differing in duration on postprandial responses. A better understanding of the most efficacious exercise conditions to attenuate PPH and postprandial vascular endothelial dysfunction in different populations will advance our understanding of lifestyle strategies contributing to the decrease in CVD risk. Future research should be conducted to examine the efficacy of various strategies, including the timing of prior exercise and the combination of exercise and dietary alterations, to attenuate the adverse effects of PPH on VEF. Further, studies are warranted examining the influence of age, sex, and/or health status on postprandial cardiometabolic responses to exercise.

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Author contributions KB, CB, and CV conceived and designed research. KB, CB, CV, and KA conducted experiments and analyzed data. KB and KT wrote the manuscript. All the authors read and approved the manuscript.

Conflict of interest The authors declare that they have no conflict of interest.

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