European Journal of Applied Physiology (2025) 125:2209–2224
https://doi.org/10.1007/s00421-025-05754-w

ORIGINAL ARTICLE

The acute impact of resistance exercise training on cardiovascular parameters in trained and untrained adults with high blood pressure

Nile F. Banks[1] · Emily M. Rogers[1] · Anna E. Stanhewicz[1] · Kara M. Whitaker[1] · Nathaniel D. M. Jenkins[1,2,3]

Received: 11 November 2024 / Accepted: 28 February 2025 / Published online: 15 March 2025

Abstract

Individuals with elevated blood pressure or stage 1 hypertension (ES1H) are recommended to engage in lifestyle modifications, including resistance exercise training (RT), to reduce blood pressure. Twenty-five adults (age = 51.4 ± 5.2 y; 15F/10 M) with ES1H who had either recently completed 9 weeks of 3 days/week RT intervention (TR; n = 12) or a non-exercise control period (UT; n = 13) completed the study. All participants had their peripheral and central systolic (SBP and cSBP) and diastolic blood pressure (DBP and cDBP), flow-mediated dilation (FMD), carotid-femoral pulse wave velocity, c-reactive protein (CRP), cardiovagal baroreflex sensitivity (BRS), resting cardiac output, resting total peripheral resistance, and heart rate variability measures including low-frequency power, high-frequency power, and root mean square of the successive differences (lnRMSSD) collected before (T1), 20–24 h after (T2), and 72-h (T3) after a single RT session. Compared to UT, TR experienced reductions in FMD from T1 to T2 (mean change: − 2.51 ± 0.55%; p = 0.012) but were protected against reductions in BRS, which was significantly lower in UT at T2 (− 1.76 ± 1.47 ms/mmHg; p = 0.019). CRP was significantly elevated in both groups at T2 compared to T1 (+ 0.61 ± 0.29 mg/L; p = 0.037), whereas DBP (+ 3.19 ± 1.6 mmHg; p = 0.003) and lnRMSSD (− 0.29 ± 0.07 ms; p = 0.015) were significantly different at T3 compared to T1. There were no other significant effects observed. Trained individuals may experience impairments in endothelial function but be protected from impairments in cardiovagal BRS during the 24 h following a resistance exercise session performed in accordance with exercise guidelines for individuals with ES1H.

Keywords Hypertension · Blood pressure · Cardiovascular health · Acute resistance exercise · Vascular function

Introduction

While resistance exercise training (RT) is most known for its ability to prevent sarcopenia and improve the quality of life in aging adults (Tieland et al. 2018), it has also recently been added to the list of recommended lifestyle modifications to lower blood pressure in individuals with hypertension (Whelton et al. 2018). For individuals with elevated blood pressure or stage 1 hypertension (ES1H), lifestyle interventions are the first line of recommendation prior to the initiation of pharmacological treatment (Whelton et al. 2018; Williams et al. 2018). While RT lowers blood pressure in individuals with hypertension, the mechanisms by which it does so are relatively unclear (Fecchio et al. 2021). Unfortunately, studies that have examined putative vascular and autonomic mechanisms for the blood pressure-lowering effect of RT are likely confounded by the timing of measurements following the last exercise session, whereby measurements are performed within 48 h after the last session. Thus, it is possible that these studies are observing and reporting the acute, transient effects of resistance exercise and not the adaptive response to chronic resistance training. Indeed, RT has been shown to acutely reduce vascular endothelial function and central arterial stiffness (Barnes et al. 2010; Stacy et al. 2013), both of which are independent predictors of

Communicated by Paul David Chantler.

✉ Nathaniel D. M. Jenkins
nathaniel-jenkins@uiowa.edu

[1] Department of Health and Human Physiology, The University of Iowa, E118, Field House Building, Iowa City, IA 52242, USA

[2] Abboud Cardiovascular Research Center, The University of Iowa, Iowa City, IA 52242, USA

[3] Fraternal Order of Eagles Diabetes Research Center, The University of Iowa, Iowa City, IA 52242, USA

future cardiovascular disease (CVD) and mortality (Laurent et al. 2001; Lakatta and Levy 2003).

One mechanism by which RT may improve blood pressure is through improvements in vascular endothelial function (Fecchio et al. 2021). While RT has been shown to improve vascular endothelial function chronically, data suggest that the muscle damage associated with a single RT session may impair vascular endothelial function for multiple days after a single session (Stacy et al. 2013). Importantly, data suggest that training status may mitigate the acute impairment in vascular endothelial function seen following RT (Jurva et al. 2006; Phillips et al. 2011), although the data are equivocal, and no studies have been conducted in middle-aged to older (MA/O) adults with untreated ES1H.

There is consistent cross-sectional evidence suggesting that individuals who engage in RT have stiffer central arteries, even when compared to physically inactive controls (Bertovic et al. 1999; Miyachi et al. 2003; Nakamura and Muraoka 2021; Otsuki et al. 2007). Results from randomized controlled trials implementing RT interventions mostly contradict the cross-sectional evidence, with the majority reporting no changes in stiffness in a variety of populations (Casey et al. 2007; Croymans et al. 2014; Rossow et al. 2014; Yoshizawa et al. 2009). Interestingly, there are several studies that do report increases in stiffness following RT interventions (Cortez-Cooper et al. 2005; Kawano et al. 2006; Miyachi et al. 2004; Okamoto et al. 2013), and while it is unclear why these disparities exist, the time intervals at which arterial stiffness measurements are performed in relation to the last RT session may be one explanation. Notably, in almost every study that has reported an increase in arterial stiffness following RT, the arterial stiffness measurements were collected within 24 h of the last training session (Cortez-Cooper et al. 2005; Kawano et al. 2006; Miyachi et al. 2004; Okamoto et al. 2013, 2006, 2009a, 2009b). Similar to vascular endothelial function, a single RT session increases arterial stiffness for up to 72 h (Barnes et al. 2010), but it is unclear how long elevations in arterial stiffness persist in middle-aged to older adults with ES1H who likely have already experienced significant stiffening of the central arteries. It is also unclear whether the effect of an RT bout is dependent on training status. These are important questions to understand the acute effects of RT and to help understand the unclear and seemingly disparate findings regarding the chronic effects of RT on arterial stiffness in the literature.

RT also induces an inflammatory response and alters autonomic nervous system activity for up to 72 h following a session (Ihalainen et al. 2017; Smith et al. 2000; Chen et al. 2011), both of which are known to directly impair arterial stiffness and vascular endothelial function acutely (Nardone et al. 2020; Vlachopoulos et al. 2005). The acute inflammatory response following a RT session is necessary for many positive RT-induced adaptations (Markworth et al. 2014), and prior work suggests as little as one RT session can alter the inflammatory response to the 2nd session in untrained individuals (Murton et al. 2014). Interestingly, when using the same relative load, trained individuals have a significantly greater cytokine response 45 min following a single RT session compared with untrained healthy young adults (Izquierdo et al. 2009). Unfortunately, very little data exist examining the influence of training status on the inflammatory response in individuals who have hypertension and who are known to have elevated baseline levels of inflammatory markers (Hage 2014). In untrained individuals, RT acutely impairs both heart rate variability (HRV) and baroreflex sensitivity (BRS) (Vale et al. 2018; Mayo et al. 2016; Heffernan et al. 2008, 2007), both of which are already impaired in individuals with hypertension (Gokce et al. 2001; Iiyama et al. 1996). Additionally, data suggests that resistance-trained individuals may have greater muscle sympathetic nerve activity during exercise compared to individuals of lower training status (Saito et al. 2009). Thus, it is unclear whether RT will negatively or positively alter autonomic function during the recovery period.

If RT is causing a decrease in vascular endothelial function and an increase in central arterial stiffness that lasts ~ 24 h regardless of training status, it would be important to further explore alterations in RT programming variables that may limit the acute reduction to optimally protect against CVD. This is specifically important to consider for individuals with high blood pressure since both vascular endothelial function and central arterial stiffness are independent predictors of future mortality within this population (Bruno et al. 2017; Muiesan et al. 2008). Additionally, individuals with hypertension have higher baseline proinflammatory profiles and impaired autonomic function compared to age-matched controls, which could exacerbate the acute impact of RT on both vascular endothelial function and central arterial stiffness. Ultimately, while RT may be recommended to reduce blood pressure in individuals with ES1H, there is a major lack of understanding of the acute physiological effects it has following each training session and how these responses change as training status increases.

Therefore, the purpose of the current study is to examine the changes in vascular endothelial function and central arterial stiffness following a single RT session in MA/O who were previously determined to have ES1H who are either trained or untrained. Additionally, we aim to determine if alterations in inflammation or autonomic function can explain the discrepancies between groups.

Methods

Experimental design

All participants in the current study had previously completed a 9-week intervention period during which they either engaged in 3 sessions/week of RT in line with the recommendations of the ACSM for individuals with high blood pressure or served as a non-exercise control. The program consisted of 2–3 sets of 10–12 repetitions targeting the major muscle groups. Specific details regarding the exercise program utilized are included in Banks et al. (2024). The exercises utilized in the RT intervention period were the same as those listed below in the "Resistance Exercise Training Session" section. Therefore, subjects who completed the RT intervention were trained (TR), whereas subjects in the control group were untrained (UT). The first experimental visit in the current study (T1) was held during the 5–7-day period following the 9-week intervention to ensure there were no lingering acute effects of the final RT session for the individuals in the TR group (Fig. 1). During all experimental visits, participants abstained from caffeine for 12 h and food for 6 h prior to arrival and were studied at the same time of day. During each experimental visit, participants had their body mass and total body water content measured before laying supine for at least 10 min prior to a single venipuncture for the collection of whole venous blood. This was followed by an additional 10-min supine rest prior to vascular tests, which included flow-mediated dilation (FMD) and carotid-femoral pulse wave velocity (cfPWV). Participants then transitioned into a seated position and sat quietly for 10 min prior to having their resting blood pressure collected and their beat-by-beat blood pressure and heart rate recorded for 5 min to determine both cardiovagal baroreflex sensitivity (BRS) and heart rate variability (HRV). During only T1, participants had their body composition assessed at the beginning of the visit and engaged in a single RT session targeting all major muscle groups at the end of the visit. Participants then returned to the laboratory 20–24 (T2) and 72 h (T3) following the end of the single RT session to repeat the same experimental protocol as T1, with the exclusion of the RT session at the end of the visit. There were two premenopausal participants in each group, two of whom had an IUD (TR n = 1; UT n = 1), while the other two completed their visits during the follicular phase (TR n = 1; UT n = 1) to control for the change in circulating sex hormones.

Participants

A screening visit took place 10 weeks prior to the current study, just before the initiation of the 9-week RT program (Fig. 1). Thirty physically inactive male (n = 10) and female (n = 15) MA/O adults (aged 45–64 y) completed a screening visit and were determined to be eligible for the current study (Table 1). A total of five participants dropped out of the study. Following screening but prior to the first experimental visit, four participants dropped out of the study for the following reasons: scheduling conflicts (n = 2), non-study-related injury (n = 1), and

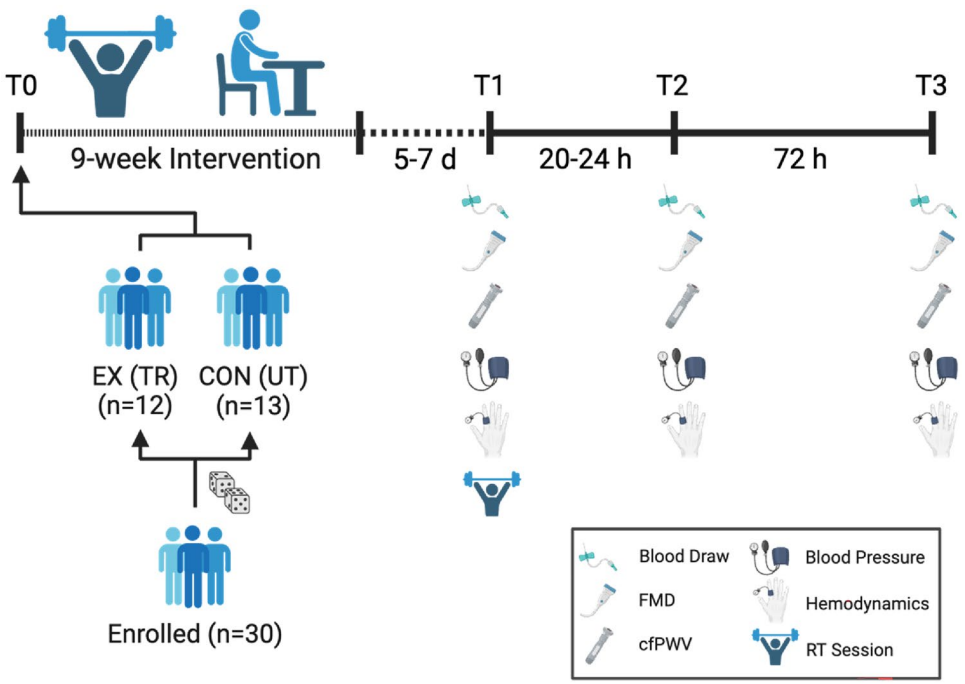

Fig. 1 Overview of the experimental design. Subjects assigned to the resistance training (RT) group (EX) were considered to be trained (TR), and those in the non-exercise control group were considered to be untrained (UT) for the current study period (T1–T3). *cfPWV* carotid-femoral pulse wave velocity, *FMD* flow-mediated dilation. Created in https://BioRender.com

Table 1 Baseline participant characteristics

	TR (n = 12; 5 M/7F)	UT (n = 13; 5 M/8F)
Age (y)	51.4 (5.3)	55.0 (5.6)
Height (cm)	170 (11.5)	172 (7.3)
Weight (kg)	85.9 (9.2)	81.2 (15.2)
BMI (kg/m^2)	29.8 (3.1)	27.5 (4.0)
Post-Menopausal (n)	6	7
Years Post-Menopause (years)	5.9 (7.4)	8.2 (4.3)
T0 Elevated Blood Pressure (n)	3	4
T0 Stage 1 Hypertension (n)	9	9
T1 Elevated Blood Pressure (n)	2	6
T1 Stage 1 Hypertension (n)	4	7

All data are displayed as mean (SD)

BMI body mass index, *TR* trained group, *UT* untrained group, *T0* represents values at the initial screening visit prior to the 9-week intervention period, *T1* represents values immediately prior to the single resistance training session

being unresponsive to study-related communication (n = 1). One additional participant in TR dropped out following T1 due to a non-study-related injury, resulting in 25 participants completing the study. Additionally, one participant was unable to attend the final experimental visit. To determine eligibility, participants arrived at the laboratory for a screening visit after a 6-h fast and 12 h of abstinence from caffeine. During the screening visit, participants completed an informed consent form, health history questionnaire, and the Physical Activity Readiness Questionnaire (PAR-Q +), had their menopause status determined via STRAW-10 principal criteria (Harlow et al. 2012), and had a resting blood pressure collected following a 5-min seated rest. To be eligible, participants must have been 45–64 years old, had a body mass index of 18.5–39.9 kg/m^2, and have been determined to have no known cardiovascular, metabolic, or musculoskeletal disease (excluding hypertension) according to self-reported health history, determined ready to begin an exercise program according to the PAR-Q +, and had a SBP between 120 and 139 mmHg or DBP between 80 and 89 mmHg. While all participants met these criteria prior to the initiation of the 9-week RT program, no additional physiological data was collected until T1. Participants were recruited using IRB-approved emails and the university mass email system, as well as by word of mouth. All study procedures and documents complied with the Declaration of Helsinki and were approved by the University's Institutional Review Board for the protection of human subjects (IRB Approval #: 202,201,319). All participants consented to participate by signing an informed consent form that explained the nature, benefits, and risks of the study prior to participation.

Resistance exercise training session

At the end of T1, all participants engaged in a ~ 40-min RT session targeting all the major muscle groups. Specifically, participants completed a ~ 5-min dynamic warmup followed by three sets of 10 reps of bench press, hack squat, latissimus dorsi pulldown, leg extension, seated row, and leg curl in that order. All exercises were performed using cable machines except for the hack squat, which was plate-loaded. All exercises were well tolerated throughout the testing period. Set 1 served as a warmup, with weight set at 50% of the estimated 10RM, whereas during sets 2 and 3, weight was set at a 10RM load. The initial weight was estimated based on strength testing during the final week of training for RT and the pre-testing strength values from the chronic portion of the study for UT. At the end of the session, participants were asked for their rating of perceived exertion (RPE) between 0 and 10, with 10 representing maximal effort.

Body composition

At T1, participants' body composition was assessed via BodPod (COSMED, USA) at T1 to assess body fat percent (BF%), fat mass (FM), and fat-free mass (FFM).

Resting blood pressure

Resting blood pressure was collected in a seated position following a 10-min resting period in accordance with the American Heart Association guidelines (Muntner et al. 2019). SBP, DBP, mean arterial pressure (MAP), central

SBP (cSBP), and central DBP (cDBP) were collected during each recording using a SphygmoCor device (SphygmoCor XCEL, AtCor Medical, Inc. USA).

Vascular endothelial function

The FMD technique was used on the brachial artery to assess vascular endothelial function and RH in accordance with the most recent guidelines (Thijssen et al. 2019). Prior to data collection, participants were in a supine position in a dark, temperature-controlled room for 10 min. With the participant's left arm laterally extended, a segmental cuff (TMC7, Hokanson, USA) was placed just distal to the medial epicondyle of the humerus and a 12-MHz ultrasound probe (12L-RS, General Electric, USA) was used to visualize the brachial artery and measure blood flow, while a screen capture device (AV.io HD, Epiphan Systems, USA) was used to record the ultrasound screen. Blood flow velocity was collected using an insonation angle of 60° to the axis of the vessel and a sample volume that encompassed the entire width of the artery (Limberg et al. 2020). FMD testing included a 2-min baseline period, a 5-min cuff occlusion period at 240 mmHg using a rapid cuff inflation system (E20, Hokanson, USA), and a 3-min post-occlusive period. Continuous, semiautomated edge detection software (FMD Studio, Quipu srl, Italy) was utilized to continuously measure brachial artery diameter and blood velocity throughout the protocol, which was used to calculate shear rate (4• blood velocity (cm/s) / brachial diameter (mm)). Baseline diameter (D_{base}) and shear rate (SR_{base}) were calculated as the average value during the 2-min baseline period, while peak diameter (D_{max}), shear rate (SR_{peak}), and shear rate area under the curve (SR_{AUC}) were calculated following cuff release up until peak diameter was reached. Percent FMD was calculated as the percent increase from D_{base} to D_{max}, whereas FMD normalized to SR (FMD_{SR}) was calculated as FMD/SR_{AUC}. RH was calculated by taking the difference in the AUC of blood flow between the baseline (BF_{base}) and the post-occlusive period. Probe location was measured from the crease of the elbow during pre-testing to ensure similar placement of the probe during post-testing.

Carotid-femoral pulse wave velocity

Central arterial stiffness was assessed using cfPWV (SphygmoCor XCEL, AtCor Medical, Inc. USA). While remaining in a supine position following the FMD test, participants had their carotid pulse palpated and marked on the left side of the neck and had a blood pressure cuff placed on their upper left thigh to measure the femoral pulse wave. The pulse of the carotid and femoral arteries was then collected simultaneously by a tonometer and the femoral cuff, respectively. The distance between the site of the carotid and femoral pulse was then divided by the difference in pulse wave transit time between the two arteries (e.g., distance/time) to determine cfPWV.

Hemodynamic monitoring

While seated, participants had a finger photoplethysmograph placed on the middle finger of the right hand, which was utilized to collect beat-by-beat blood pressure (NOVA Finometer, Finapres Medical Systems, The Netherlands). The participants held their right hand over their hearts during all hemodynamic testing, with their arms supported. Modelflow technology was used to calculate cardiac output (CO) and total peripheral resistance (TPR). Additionally, heart rate was collected using a 3-lead electrocardiogram, and respiratory rate was collected using a respiratory belt with participants instructed to breathe at a normal rate during all testing (TN1132/ST; ADInstruments). Data were collected at 1000 Hz using a data acquisition system (Powerlab Series 26; ADInstruments, USA) and stored offline.

Cardiovagal baroreflex sensitivity and heart rate variability

Following the transition into a seated position, participants rested quietly for 10 min before having both cardiovagal BRS and HRV collected during a 5-min period. Raw beat-by-beat blood pressure waveforms and ECG data were uploaded to Ensemble-R software, and the sequence method was utilized to assess BRS and HRV. BRSpooled was assessed by averaging the slopes between three sequences of either increasing (BRSup) or decreasing (BRSdown) pulse waveform peak pressures with subsequent decreases or increases in R-R interval length, respectively, with a minimum correlation of $r = 0.8$, increase in SBP of 1 mmHg, and R-R interval length of 4 ms. The log-transformed root mean square of successive differences (lnRMSSD), high-frequency power (lnHF), and low-frequency power (lnLF) were calculated to represent both time and frequency domain HRV.

C-reactive protein

Whole venous blood was taken from the antecubital vein and collected in a lithium heparin plasma separator tube (BD Vacutainer, Becton Dickinson, USA) before being spun for 15 min at 1000 g. Plasma was then transferred to 1.7-mL microcentrifuge tubes for storage at – 80 °C. Samples were later thawed, and high-sensitivity CRP was assessed using a commercially available enzyme-linked immunosorbent assay (CRP ELISA, Immundiagnostik AG, Germany). The detection range of the CRP ELISA kit was 1.8–150 ng/mL, with a sensitivity of 0.124 ng/ml and an inter-assay coefficient of variation of < 10%. All assays were performed in

accordance with the manufacturer's instructions and read using a microplate photometer (Multiskan™ FC Microplate Photometer, ThermoFisher Scientific™, USA).

Lifestyle controls

All participants were asked to refrain from any other forms of exercise outside of the study starting 5–7 days prior to T1. Participants filled out dietary food logs every day during the testing period starting one day prior to T1. Prior to T3, participants completed the International Physical Activity Questionnaire (IPAQ) to provide physical activity data via self-report during the post-testing period.

Statistics

Multiple independent two-way repeated mixed linear models (group*visit) were run to determine the impact of the single RT session on training modality on all resting blood pressure, vascular endothelial function, CRP, HRV, and BRS variables. Prior data suggest that sex differences likely exist when examining the relationship between exercise training and the primary outcome variables; therefore, sex was included as a covariate in the analyses (Moreau et al. 2013; DuPont et al. 2019). A two-way repeated mixed linear model with sex and mean arterial pressure (MAP) as covariates was run to determine the impact of training status and the single RT session on cfPWV. Mixed linear models were utilized to allow the inclusion of participants in the analysis who had data missing at random (e.g., CRP, RH, and BF_{base}). Independent samples t-tests were used to compare groups for baseline characteristics, physical activity and dietary data during the testing period, and the weight lifted and perceived exertion during the single RT session. Lower-order linear mixed models with Tukey- or Sidak-adjusted post hoc analyses were performed to investigate any variables with a significant interaction effect further. Effect sizes were determined using Cohen's d. Pearson's product correlations were used to assess the relationship between all primary variables. Data are reported as mean (±95% CI) unless denoted otherwise, and significance was set at $p \leq 0.05$. Statistical analyses were performed using R (v. 2022.12.0 + 353), and figures were created using GraphPad Prism (v. 9.5.1).

Results

Training session

There was a difference for combined weight ($p < 0.001$), where TR (mean: 379.4 ± SD: 112.9 kg) lifted significantly more weight during the RT session compared to UT (236.4 ± 73.1 kg), reflecting differences in training status. There were no differences in session RPE ($p = 0.49$) between TR (7.9 ± 1.1 au) and UT (7.7 ± 0.6 au).

Blood pressure

There were no significant group*visit interactions for SBP, cSBP, DBP, cDBP, or MAP ($p = 0.26$; $\eta_p^2 = 0.06$). There was a significant group main effect for cSBP, where cSBP was significantly lower in TR versus UT (− 6.98 ± 6.54 mmHg; $p = 0.038$; d = 1.70). There were significant visit main effects for both DBP and cDBP, with both groups experiencing increased DBP (+ 3.19 ± 1.59 mmHg; $p = 0.004$; d = 0.99) and cDBP (+ 2.84 ± 1.38 mmHg; $p = 0.013$; d = 0.85) at T3 compared to T1. There were no significant group main effects for SBP, DBP, cDBP, or MAP, and no visit main effects for SBP or MAP (Fig. 2).

Resting hemodynamics

There were no significant interactions, group main effects, or visit main effects for RHR or CO (Table 2). While there was no interaction or visit main effect for TPR, there was a significant main effect for the group, where TPR was significantly lower in TR compared to UT across the entire study period (+ 306 ± 284.7 mmHg·s/L; $p = 0.036$; d = 0.90).

Central arterial stiffness

There was no significant interaction, group main effect, or visit main effect for cfPWV (Table 2).

Vascular function

Due to technical errors, two values were missing at the T3 visit for RH and BF_{base}. There were significant group*visit interactions for FMD and $cFMD_{SR}$ but not for RH or BF_{base} (Fig. 3). Specifically, TR experienced a reduction in FMD from T1 to T2 (− 3.22 ± 1.65%; $p < 0.001$; d = − 1.94) and an increase back to baseline levels from T2 to T3 (+ 2.18 ± 1.64%; $p = 0.016$; d = 1.31), whereas FMD did not change in UT ($p \geq 0.88$). Additionally, while FMD tended to be greater at T1 in TR than UT ($p = 0.056$), FMD was not different in TR versus UT at either T2 ($p = 0.58$) or T3 ($p = 0.13$). $cFMD_{SR}$ was significantly lower at both T2 (− 2.12 ± 1.15%; $p < 0.001$; d = − 1.83) and T3 (− 1.42 ± 1.15%; $p = 0.012$; d = − 1.23) compared to T1 in TR, with no changes observed in UT ($p \geq 0.85$). Consequently, $cFMD_{SR}$ was significantly greater in TR at T1 (+ 1.95 ± 0.44%; $p = 0.001$; d = 1.51) but not at T2 or T3 ($p \geq 0.77$) compared to UT. There were significant group main effects for both RH and BF_{base} (Fig. 3). Both RH (+ 53 ± 40 mL/min·10^{-2}; $p = 0.012$; d = 1.18) and BF_{base}

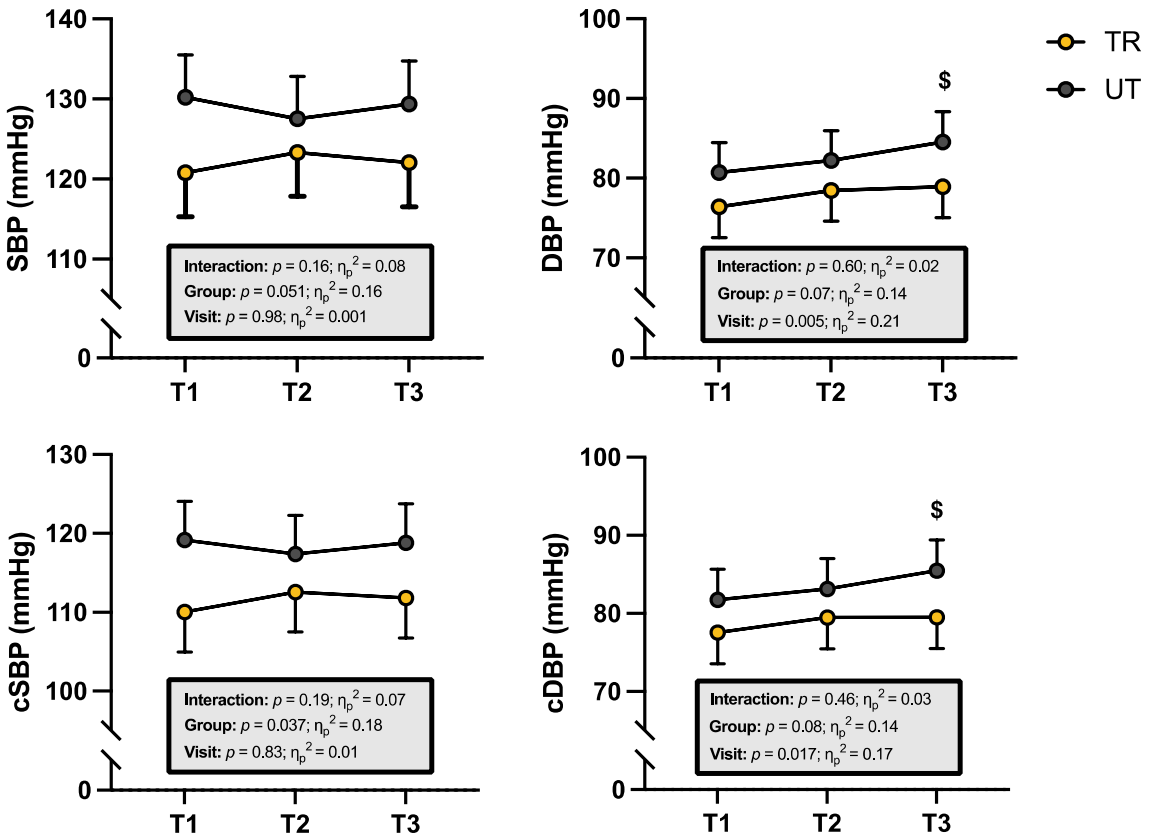

Fig. 2 The impact of a single RT session using the same relative load in trained (TR) and untrained (UT) middle-aged to older adults with untreated elevated blood pressure or stage 1 hypertension. T1 represents baseline measurements before the lift, whereas T2 was held 20–24 h, and T3 was held 72 h following the single RT session. All data are displayed as the estimated marginal mean (±model 95% CI); $ = significant differences at that timepoint compared to T1 in all groups ($p < 0.05$); *cDBP* central diastolic blood pressure, cSBP = central systolic blood pressure, *DBP* diastolic blood pressure, *SBP* systolic blood pressure

(+35 ± 29.5 mL/min; $p = 0.022$; d = 1.35) were significantly greater in TR versus UT across the entire testing period.

Cardiovagal baroreflex sensitivity

Participants had an average of 27.8 ± 13.1 valid sequences acquired at each visit. There was a significant group*visit interaction for BRSpooled, BRSup, but not BRSdown (Fig. 4). Specifically, UT had a significantly lower BRSpooled (− 1.76 ± 1.47 ms/mmHg; p = 0.019; d = − 1.46) and BRSup (− 1.97 ± 1.72 ms/mmHg; p = 0.026; d = − 1.36) compared to TR at T2, with no differences at any other timepoint. While BRSpooled did not change in TR ($p \geq 0.23$), it significantly decreased from T1 to T2 in UT (− 1.16 ± 1.15 ms/mmHg; p = 0.026; d = −0.96) and then significantly increased back to baseline levels from T2 to T3 (+1.62 ± 1.18 ms/mmHg; p = 0.005; d = 1.34). BRSup significantly increased from T2 to T3 in UT (+1.54 ± 1.41 ms/mmHg; $p = 0.029$; d = 1.07) but did not change significantly at any other timepoint in either TR or UT ($p \geq 0.12$).

Heart rate variability

There were no significant group*visit interactions or group main effects for lnRMSSD, lnLF, or lnHF. There was a significant visit main effect for lnLF, with significant reductions from T1 to T2 (− 0.51 ± 0.30 ms^2; p = 0.018; d = − 0.81) but not T2 to T3 (+0.44 ± 0.85 ms^2; p = 0.05; d = 0.69) irrespective of group. There was also a significant visit main effect for lnRMSSD, with values decreasing from T1 to T3 (− 0.29 ± 0.07 ms; p = 0.015; d = − 0.83), but no differences between T1 and T2 (− 0.17 ± 0.25 ms; p = 0.19; d = −0.50), or T2 and T3 (− 0.11 ± 0.36 ms; p = 0.48; d = − 0.33). There was no visit main effect for lnHF (Fig. 4).

C-reactive protein

Three CRP values were outside of the detectable range, with two missing data points at T1 and one at T2. There was no significant group*visit interaction or group main effect for CRP. There was a significant visit main effect, with significant increases from T1 to T2 (+0.61 ± 0.29

Table 2 The acute impact of a single rt session on vascular function and hemodynamics

	T1		T2		T3		Group*Visit		Group		Visit	
	TR	UT	TR	UT	TR	UT	*p*	η_p^2	*p*	η_p^2	*p*	η_p^2
cfPWV (m/s)	6.80 (0.5)	7.19 (0.5)	6.93 (0.5)	7.41 (0.5)	6.91 (0.5)	7.32 (0.5)	0.90	0.14	0.21	0.07	0.23	0.06
MAP (mmHg)	89.9 (4.4)	95.9 (4.2)	92.1 (4.4)	95.4 (4.2)	92.0 (4.4)	98.2 (4.3)	0.26	0.06	0.07	0.14	0.10	0.10
CO (L/min)	5.61 (0.9)	4.94 (0.9)	5.91 (0.9)	4.85 (0.9)	5.73 (0.9)	5.48 (0.9)	0.21	0.07	0.23	0.07	0.33	0.05
TPR (mmHg·s/L)	960 (260)	1,290 (250)	980 (260)	1,430 (250)	1,010 (260)	1,140 (260)	0.52	0.03	0.033*	0.19	0.63	0.02
D_{base} (mm)	3.64 (0.23)	3.73 (0.22)	3.78 (0.23)	3.76 (0.22)	3.69 (0.23)	3.77 (0.23)	0.53	0.03	0.53	0.73	0.26	0.06
D_{max} (mm)	3.98 (0.23)	3.94 (0.23)	4.00 (0.23)	3.95 (0.23)	3.99 (0.23)	3.96 (0.23)	0.96	0.002	0.78	0.004	0.94	0.003
SR_{base} (s^{-1})	204.9 (61.9)	191.3 (59.8)	193.0 (61.9)	173.7 (59.8)	213.0 (62.0)	188.2 (61.4)	0.96	0.002	0.59	0.01	0.65	0.019
SR_{peak} (s^{-1})	1037.6 (190)	995.4 (185)	1042.7 (191)	957.7 (184)	1154.8 (191)	960.2 (188)	0.33	0.05	0.36	0.04	0.53	0.03
SR_{AUC} ($au \cdot 10^{-3}$)	2.17 (0.52)	2.08 (0.50)	2.46 (0.52)	2.01 (0.50)	2.74 (0.52)	1.68 (0.52)	0.06	0.11	0.07	0.15	0.86	0.007

The impact of a single RT session using the same relative load in trained (TR) and untrained (UT) middle-aged to older adults with untreated, elevated blood pressure or stage 1 hypertension. T1 represents baseline measurements before the lift, whereas T2 was held 20–24 h, and T3 was held 72 h following the single RT session. All data are displayed as estimated mean (± model 95% CI)

AUC area under the curve, *cfPWV* carotid-femoral pulse wave velocity, *CO* cardiac output, D_{base} baseline diameter, D_{max} maximal diameter after cuff release, *SR* shear rate, *TPR* total peripheral resistance

* = $p \leq 0.05$

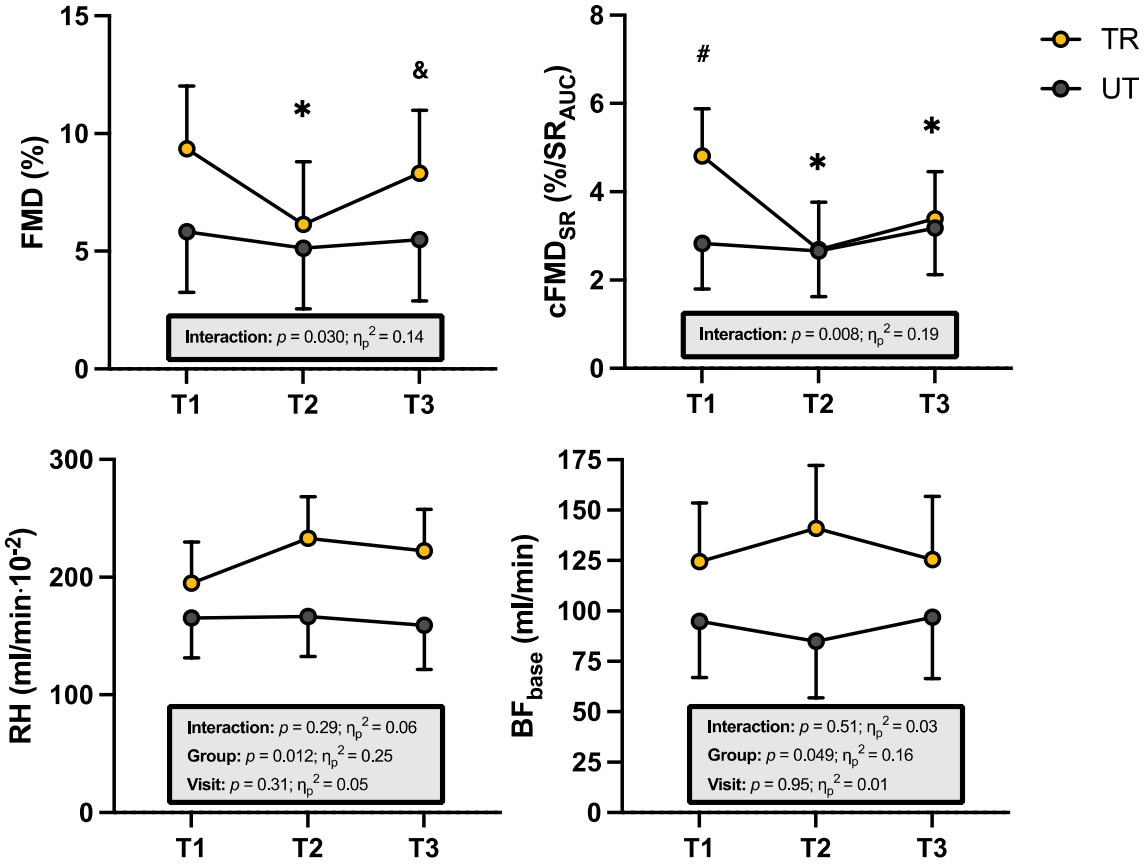

Fig. 3 The impact of a single RT session using the same relative load in trained (TR) and untrained (UT) middle-aged to older adults with untreated elevated blood pressure or stage 1 hypertension. T1 represents baseline measurements before the lift, whereas T2 was held 20–24 h, and T3 was held 72 h following the single RT session. All data are displayed as the estimated marginal mean (±model 95% CI); *=significant difference in TR between that timepoint and T1 ($p<0.05$), &=significant difference in TR between T3 and T2 ($p<0.05$); #=significant between-group differences at that timepoint ($p<0.05$); *FMD* flow-mediated dilation, $cFMD_{SR}$ FMD corrected for shear rate, *RH* reactive hyperemia

mg/L; p = 0.037; d = 0.75) but no changes from T2 to T3 (− 0.57 ± 4.31 mg/L; p = 0.51; d = 0.33) irrespective of group (Fig. 5).

Lifestyle controls

TR reported engaging in 390.5 ± 156.0 MET min/week, consuming 2095 ± 452 cal, 94.5 ± 25.3 g of fat, 215.7 ± 52.4 g of carbohydrates, and 95.9 ± 22.3 g of protein. UT reported engaging in 392.1 ± 158.4 MET min/week, consuming 1917 ± 417 cal, 89.5 ± 22.8 g of fat, 186.3 ± 50.4 g of carbohydrates, and 98.9 ± 19.1 g of protein. There were no significant differences between TR and UT during the study period for MET min/week ($p=0.99$), or the consumption of calories ($p=0.53$), fat ($p=0.75$), carbohydrates ($p=0.39$), or protein ($p=0.82$).

Correlations

ΔFMD was significantly correlated with ΔCRP (r = − 0.380; $p=0.011$) and $\Delta cFMD_{SR}$ was significantly correlated with ΔcfPWV (r = − 0.287; $p=0.046$). ΔD_{base} was significantly correlated with ΔCRP (r = 0.475; $p=0.001$), ΔlnLF (r = − 0.472; $p<0.001$), ΔlnHF (r = − 0.507; $p<0.001$), and ΔlnRMSSD (r = − 0.298; $p=0.038$), but not significantly with ΔCO (r = 0.282; $p=0.05$) and ΔTPR (r = − 0.236; $p=0.10$). Additionally, ΔBF_{base} was significantly correlated with ΔRH (r = 0.421; $p=0.003$), ΔFMD (r = − 0.294; $p=0.045$), ΔlnLF (r = − 0.329; $p=0.024$), ΔlnHF (r = −0.401; $p=0.005$), and ΔlnRMSSD (r = − 0.321; $p=0.028$) (Fig. 6). Combined weight lifted was significantly correlated changes from T1 to T2 with ΔRH (r = 0.404; $p=0.045$), ΔBF_{base}

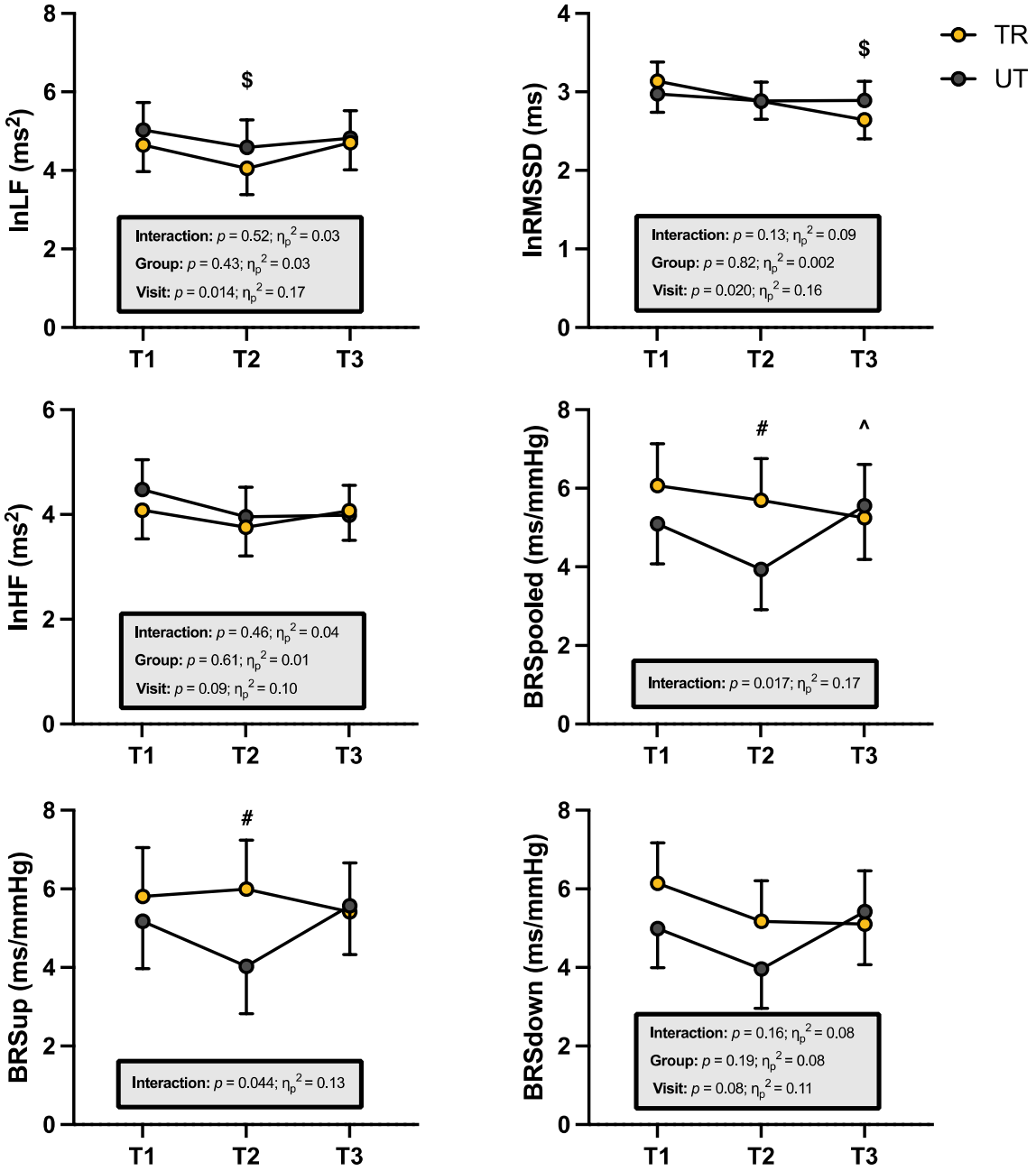

Fig. 4 The impact of a single RT session using the same relative load in trained (TR) and untrained (UT) middle-aged to older adults with untreated elevated blood pressure or stage 1 hypertension. T1 represents baseline measurements before the lift, whereas T2 was held 20–24 h, and T3 was held 72 h following the single RT session. All data are displayed as the estimated marginal mean (±model 95% CI); ^ = significant difference in UT between T3 and T2 ($p < 0.05$); $ = significant differences at that timepoint compared to T1 in all groups ($p < 0.05$); # = significant between-group difference at that timepoint (UT < TR, $p < 0.05$); *BRS* baroreflex sensitivity, *lnHF* log-transformed high-frequency power, *lnLF* log-transformed low-frequency power, *lnRMSSD* log-transformed root mean square of successive difference

(r = 0.406; $p = 0.042$), $\Delta cFMD_{SR}$ (r = − 0.523; $p = 0.007$), and moderately correlated with ΔFMD (r = − 0.367; $p = 0.07$), ΔCRP (r = 0.392; $p = 0.07$), ΔlnHF (r = − 0.302; $p = 0.14$) but not ΔlnLF (r = − 0.01; $p = 96$).

Discussion

This was the first study to investigate the acute impact of a single RT session in both trained and untrained MA/O

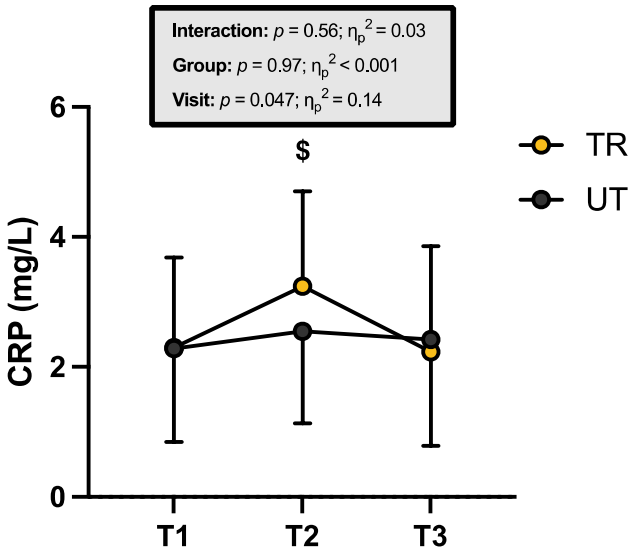

Fig. 5 The impact of a single RT session using the same relative load on c-reactive protein (CRP) in trained (TR) and untrained (UT) middle-aged to older adults with untreated elevated blood pressure or stage 1 hypertension. T1 represents baseline measurements before the lift, whereas T2 was held 20–24 h, and T3 was held 72 h following the single RT session. All data are displayed as the estimated marginal mean (± model 95% CI). $ = significant differences at that timepoint compared to T1 in all groups

adults with untreated ES1H. The main finding of the current study was that a single RT session caused significant reductions in vascular endothelial function in TR, significant reductions in BRS in UT, and decreases in HRV and increases in CRP and DBP in both TR and UT. While trained individuals were relatively protected against reductions in BRS, they had significant reductions in vascular endothelial function 20–24 h following the session, although FMD never decreased lower than that of UT. The single RT session did not alter cfPWV, SBP, TPR, or CO in either group. Lastly, while most variables returned to baseline values by the 72-h timepoint, $cFMD_{SR}$ was still reduced in TR, potentially indicating that a longer recovery period may be necessary to fully recover endothelial function in TR individuals, which also has important implications for study design.

Contrary to our hypothesis, we observed no changes in cfPWV in either TR or UT during the days following the RT session. Authors who have collected measures of central arterial stiffness 20–24 h following the final training session of a RT intervention have consistently reported elevated stiffness in the exercise group (Cortez-Cooper et al. 2005; Kawano et al. 2006; Miyachi et al. 2004; Okamoto et al. 2013, 2006, 2009a, 2009b). Additionally, Barnes et al. reported that cfPWV was elevated for up to 72 h following a single eccentric RT session (Barnes et al. 2010). Among the aforementioned studies that have reported an increase in central arterial stiffness following a RT intervention, multiple reported that stiffness returned to pre-training levels following a detraining period (Kawano et al. 2006; Miyachi et al. 2004; Okamoto et al. 2008). Unfortunately, these data were collected months following the initial post-testing visit, and it is therefore unclear how transient post-intervention changes in stiffness are. While we report no changes following the training session in either group, acute changes in cfPWV were negatively correlated with $cFMD_{SR,}$ suggesting that acute changes are likely at least partially attributable to impaired endothelial function. Ultimately, it is still unclear why cfPWV is mostly unaltered in most studies implementing RT interventions, while cross-sectional data and a handful of RCTs suggest a worsening of stiffness associated with or caused by RT. Both the volume and intensity of the RT session likely influence the acute responses that follow, and while participants in the current study rated the session at a ~ 7.8/10 RPE, it is possible that greater training loads (and an increase in the associated intra-exercise hemodynamic response) are necessary to cause changes in cfPWV acutely. Further, the protocol used in the current study, which utilized cable- and plate-loaded machines rather than free weights and prevented the use of the Valsalva maneuver, may have resulted in a lesser degree of exertional hypertension compared to other protocols. Still, the loads and volume of resistance exercise prescribed were in accordance with current recommendations for individuals with ES1H and thus suggest that this prescription does not cause transient increases in central arterial stiffness.

One of the primary mechanisms proposed for the ability of RT to reduce cardiovascular disease is through improvements in vascular endothelial function. The vascular endothelium is able to regulate vascular tone, and thus blood pressure, through the production of vasodilatory molecules like nitric oxide (NO) and vasoconstricting molecules, including endothelin-1 and reactive oxygen species (ROS) (Silva et al. 2021). Vascular endothelial function is heavily dependent on the bioavailability of NO, a molecule that actively protects against the development and progression of atherosclerosis (Widlansky et al. 2003). Prior data indicate that training status protects against impairments in FMD immediately (< 1 h) following a single RT session, but it was unknown if this protection extends beyond 1 h to the days after the exercise session (Jurva et al. 2006; Phillips et al. 2011). It is also unclear if this protection extends to individuals with high blood pressure who may have elevated sympathetic nervous system activity and inflammatory responses to exercise (Kim and Ha 2016). In the present study, both FMD and $cFMD_{SR}$ were significantly reduced 20–24 h following the single RT session in TR, but not UT. Importantly, neither FMD nor $cFMD_{SR}$ decreased to a level lower than that observed in UT at any point, suggesting that the TR group was still better protected overall than UT.

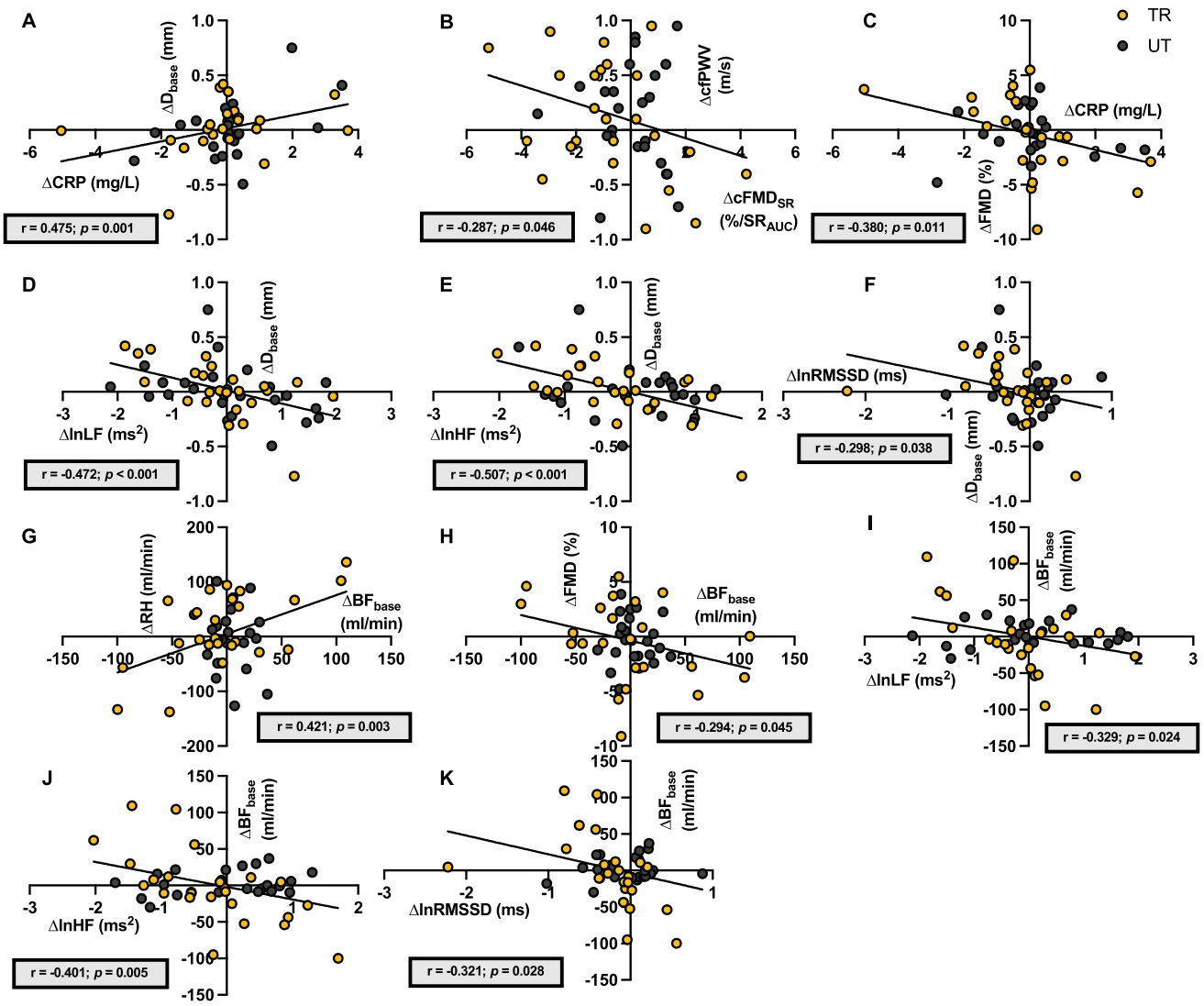

Fig. 6 Correlations between the change (Δ) in **A** baseline brachial artery diameter (D_{base}) and c-reactive protein (CRP); **B** carotid-femoral pulse wave velocity (cfPWV) and flow-mediated dilation (FMD) corrected to shear rate ($cFMD_{SR}$); **C** FMD and CRP; **D** D_{base} and log-transformed low-frequency power (lnLF); **E** D_{base} and ln high-frequency power (lnHF); **F** D_{base} and ln root mean square of successive differences (lnRMSSD); **G** baseline blood flow (BF_{base}) and reactive hyperemia (RH); **H** BF_{base} and FMD; **I** BF_{base} and lnLF; **J** BF_{base} and lnHF; **K** BF_{base} lnRMSSD from baseline to 20–24 h and 20–24 h to 72 h following a single RT session

The reduced FMD in TR is in agreement with prior literature indicating that acute eccentric RT causes a reduction in FMD from 9.4% at baseline to 5.4–6.3% in the 24–72 h following exercise (Stacy et al. 2013). These decrements in FMD were accompanied by reductions in SR_{AUC} (Stacy et al. 2013), although the normalization of FMD to SR_{AUC} did not completely abolish the exercise-related decreases in FMD (Stacy et al. 2013). Moreover, in the current study, RH was not significantly altered by the session, and normalization of FMD for SR accentuated the changes observed in TR. Interestingly, ΔFMD was most strongly correlated with ΔCRP, indicating that the transient inflammatory response, which led to elevations in CRP following exercise, may have impacted peripheral vascular function. It is well established that inflammation impairs FMD, and the inflammatory response caused by the RT session in the current study may have increased the production of ROS during the days following the training session (Prasad 2004). Additionally, while CRP was not significantly greater in TR compared to UT, TR had increases of 1 mg/L compared to the 0.25 mg/L increase seen in UT from T1 to T2 and was moderately correlated with the combined weight lifted during the single RT session (r = 0.392). When interpreted alongside the significant correlation between ΔFMD and ΔCRP in the recovery period, these data support that increased inflammation secondary to increased training intensity may have been driving

the reductions in FMD observed in TR but not UT. It is also possible that UT experienced a floor effect, which limited the degree to which FMD decreased beyond baseline levels. Additionally, other inflammatory markers not represented through a circulating plasma CRP test may have influenced the FMD response to training.

The current study indicates that a single RT session causes alterations in autonomic function that persist up to three days into the recovery period in a manner that is partially dependent upon training status. We observed significantly lower BRSpooled and BRSup levels in UT 20–24 h following training compared to TR, with no significant differences in BRSdown. RT has been consistently shown to acutely reduce BRS within the 30 min following a training session, likely driven by decreased vagal modulation (Mayo et al. 2016; Heffernan et al. 2008, 2007), but we show for the first time that this effect is likely dependent on training status. We also show significant reductions in both lnLF and lnHF, respectively, with lnRMSSD decreasing throughout the three-day recovery period independent of training status. Prior literature has shown that a single RT session impairs both time- and frequency-domain HRV, with decreases in RMSSD and HF and increases in LF power reported for up to 24 h (Vale et al. 2018; Garber et al. 2011). The reduction in HRV total spectral power is in agreement with data published by Heffernan et al., who reported similar findings 30 min after a single RT session (Heffernan et al. 2006). Therefore, acutely, RT likely reduces HRV in both trained and untrained individuals, while trained individuals are protected from the decrements in BRS seen 20–24 h following a single RT session.

In summary, we report that FMD was reduced in the 20–24-h period following a single RT session in TR only, cardiovagal BRS was reduced in UT only, while CRP increased, and cfPWV remained unchanged in both groups. Notably, these responses were observed in response to a resistance exercise session that was performed in accordance with the RT guidelines for individuals with high blood pressure (Medicine ACoS. 2018). Moreover, whereas prior studies have used cross-sectional designs and grouped participants by training status based on self-report, here we randomized untrained participants and implemented a resistance training intervention and thus tightly controlled training status. If resistance exercise acutely suppresses vascular endothelial function for 20–24 h each time a trained individual engages in RT, an individual engaging in RT three times per week would spend roughly half of their week in a state of suppressed vascular endothelial function compared to their baseline state. Since the cardioprotective effects of vascular endothelial function are in part due to its ability to slow the progression and development of atherosclerosis, determining how to reduce acute decrements in vascular endothelial function following RT will be significant. However, it is also still important to note that FMD was greater in TR than UT at baseline and 72 h, and even when suppressed at 20–24 h, it was not lower than observed in UT individuals, indicating that RT has a net positive effect provided the prescription is appropriate. Future studies may consider how changes to RT programming via manipulation of load or volume may differentially impact the acute effects of resistance exercise on FMD or whether the concurrent completion of different exercise modalities (e.g., aerobic exercise) may protect against acute impairments in FMD. The findings that cardiovagal BRS was reduced only in UT are also notable, and training status should be strongly considered in studies investigating the acute impact of RT on this variable. Finally, the reduction in cardiovagal BRS could be potentially avoided by performing lower volume and intensities of RT during the initial sessions upon beginning RT, but this needs to be explored further.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00421-025-05754-w.

Author contribution The study was conceptualized and planned by NFB, AES, KMW, and NDMJ. NFB and EMR completed data collection and entry. NFB, EMR, and NDMJ performed data analysis. NFB was the primary creator of all figures and tables. NFB and NDMJ completed the original draft, and all authors contributed to the review and editing. NFB was responsible for project administration, and NDMJ provided resources to complete the project. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work to ensure that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

Data availability Raw data supporting this study's findings are available from the corresponding author upon reasonable request.

Declarations

Conflict of interest No conflicts of interest, financial or otherwise, are declared by the authors.

References

Banks NF, Rogers EM, Berry AC, Jenkins NDM (2024) Progressive iso-inertial resistance exercise promotes more favorable cardiovascular adaptations than traditional resistance exercise in young adults. Am J Physiol Heart Circ Physiol 326(1):H32–H43. https://doi.org/10.1152/ajpheart.00402.2023. (**PubMed PMID: 37889252**)

Barnes JN, Trombold JR, Dhindsa M, Lin HF, Tanaka H (2010) Arterial stiffening following eccentric exercise-induced muscle damage. J Appl Physiol 109(4):1102–1108. https://doi.org/10.1152/japplphysiol.00548.2010. (**PubMed PMID: 20671032**)

Bertovic DA, Waddell TK, Gatzka CD, Cameron JD, Dart AM, Kingwell BA (1999) Muscular strength training is associated with low arterial compliance and high pulse pressure. Hypertension 33(6):1385–1391. https://doi.org/10.1161/01.hyp.33.6.1385. (**PubMed PMID: 10373221**)

Bruno RM, Cartoni G, Stea F, Armenia S, Bianchini E, Buralli S et al (2017) Carotid and aortic stiffness in essential hypertension and their relation with target organ damage: the CATOD study. J Hypertens 35(2):310–318. https://doi.org/10.1097/HJH.0000000000001167

Casey DP, Beck DT, Braith RW (2007) Progressive resistance training without volume increases does not alter arterial stiffness and aortic wave reflection. Exp Biol Med (Maywood) 232(9):1228–1235. https://doi.org/10.3181/0703-RM-65. (**PubMed PMID: 17895531**)

Chen JL, Yeh DP, Lee JP, Chen CY, Huang CY, Lee SD et al (2011) Parasympathetic nervous activity mirrors recovery status in weightlifting performance after training. J Strength Cond Res 25(6):1546–1552. https://doi.org/10.1519/JSC.0b013e3181da7858. (**PubMed PMID: 21273908**)

Cortez-Cooper MY, DeVan AE, Anton MM, Farrar RP, Beckwith KA, Todd JS et al (2005) Effects of high intensity resistance training on arterial stiffness and wave reflection in women. Am J Hypertens 18(7):930–934. https://doi.org/10.1016/j.amjhyper.2005.01.008. (**PubMed PMID: 16053989**)

Croymans DM, Krell SL, Oh CS, Katiraie M, Lam CY, Harris RA et al (2014) Effects of resistance training on central blood pressure in obese young men. J Hum Hypertens 28(3):157–164. https://doi.org/10.1038/jhh.2013.81.PubMedPMID:24005959; PubMedCentralPMCID:PMCPMC4119468

da Silva GM, da Silva MC, Nascimento DVG, Lima Silva EM, Gouvea FFF, de Franca Lopes LG et al (2021) Nitric oxide as a central molecule in hypertension: focus on the vasorelaxant activity of new nitric oxide donors. Biology (Basel). https://doi.org/10.3390/biology10101041

DuPont JJ, Kenney RM, Patel AR, Jaffe IZ (2019) Sex differences in mechanisms of arterial stiffness. Br J Pharmacol 176(21):4208–4225. https://doi.org/10.1111/bph.14624

Fecchio RY, Brito LC, Pecanha T, de Moraes Forjaz CL (2021) Potential mechanisms behind the blood pressure-lowering effect of dynamic resistance training. Curr Hypertens Rep 23(6):35. https://doi.org/10.1007/s11906-021-01154-5. (**PubMed PMID: 34152491**)

Garber CE, Blissmer B, Deschenes MR, Franklin BA, Lamonte MJ, Lee IM et al (2011) American college of sports medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Med Sci Sports Exerc 43(7):1334–59. https://doi.org/10.1249/MSS.0b013e318213fefb

Gokce N, Holbrook M, Duffy SJ, Demissie S, Cupples LA, Biegelsen E et al (2001) Effects of race and hypertension on flow-mediated and nitroglycerin-mediated dilation of the brachial artery. Hypertension 38(6):1349–1354. https://doi.org/10.1161/hy1201.096575. (**PubMed PMID: 11751716**)

Hage FG (2014) C-reactive protein and hypertension. J Hum Hypertens 28(7):410–415. https://doi.org/10.1038/jhh.2013.111. (**PubMed PMID: 24226100**)

Harlow SD, Gass M, Hall JE, Lobo R, Maki P, Rebar RW et al (2012) Executive summary of the Stages of Reproductive Aging Workshop + 10: addressing the unfinished agenda of staging reproductive aging. Menopause 19(4):387–395. https://doi.org/10.1097/gme.0b013e31824d8f40

Heffernan KS, Kelly EE, Collier SR, Fernhall B (2006) Cardiac autonomic modulation during recovery from acute endurance versus resistance exercise. Eur J Cardiovasc Prev Rehabil 13(1):80–86. https://doi.org/10.1097/01.hjr.0000197470.74070.46. (**PubMed PMID: 16449868**)

Heffernan KS, Collier SR, Kelly EE, Jae SY, Fernhall B (2007) Arterial stiffness and baroreflex sensitivity following bouts of aerobic and resistance exercise. Int J Sports Med 28(3):197–203. https://doi.org/10.1055/s-2006-924290. (**PubMed PMID: 17024636**)

Heffernan KS, Sosnoff JJ, Jae SY, Gates GJ, Fernhall B (2008) Acute resistance exercise reduces heart rate complexity and increases QTc interval. Int J Sports Med 29(4):289–293. https://doi.org/10.1055/s-2007-965363. (**PubMed PMID: 17990212**)

Ihalainen JK, Ahtiainen JP, Walker S, Paulsen G, Selanne H, Hamalainen M et al (2017) Resistance training status modifies inflammatory response to explosive and hypertrophic resistance exercise bouts. J Physiol Biochem 73(4):595–604. https://doi.org/10.1007/s13105-017-0590-0. (**PubMed PMID: 28914426**)

Iiyama K, Nagano M, Yo Y, Nagano N, Kamide K, Higaki J et al (1996) Impaired endothelial function with essential hypertension assessed by ultrasonography. Am Heart J 132(4):779–782. https://doi.org/10.1016/s0002-8703(96)90311-7. (**PubMed PMID: 8831366**)

Izquierdo M, Ibanez J, Calbet JA, Navarro-Amezqueta I, Gonzalez-Izal M, Idoate F et al (2009) Cytokine and hormone responses to resistance training. Eur J Appl Physiol 107(4):397–409. https://doi.org/10.1007/s00421-009-1139-x. (**PubMed PMID: 19649649**)

Jurva JW, Phillips SA, Syed AQ, Syed AY, Pitt S, Weaver A et al (2006) The effect of exertional hypertension evoked by weight lifting on vascular endothelial function. J Am Coll Cardiol 48(3):588–589. https://doi.org/10.1016/j.jacc.2006.05.004. (**PubMed PMID: 16875990**)

Kawano H, Tanaka H, Miyachi M (2006) Resistance training and arterial compliance: keeping the benefits while minimizing the stiffening. J Hypertens 24(9):1753–1759. https://doi.org/10.1097/01.hjh.0000242399.60838.14. (**PubMed PMID: 16915024**)

Kim D, Ha JW (2016) Hypertensive response to exercise: mechanisms and clinical implication. Clin Hypertens 22:17. https://doi.org/10.1186/s40885-016-0052-y

Lakatta EG, Levy D (2003) Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a "set up" for vascular disease. Circulation 107(1):139–146. https://doi.org/10.1161/01.cir.0000048892.83521.58. (**PubMed PMID: 12515756**)

Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L et al (2001) Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. Hypertension 37(5):1236–1241. https://doi.org/10.1161/01.hyp.37.5.1236. (**PubMed PMID: 11358934**)

Limberg JK, Casey DP, Trinity JD, Nicholson WT, Wray DW, Tschakovsky ME et al (2020) Assessment of resistance vessel function in human skeletal muscle: guidelines for experimental design, doppler ultrasound, and pharmacology. Am J Physiol Heart Circ Physiol 318(2):H301–H325. https://doi.org/10.1152/ajpheart.00649.2019

Markworth JF, Vella LD, Figueiredo VC, Cameron-Smith D (2014) Ibuprofen treatment blunts early translational signaling responses in human skeletal muscle following resistance exercise. J Appl Physiol 117(1):20–28. https://doi.org/10.1152/japplphysiol.01299.2013. (**PubMed PMID: 24833778**)

Mayo X, Iglesias-Soler E, Carballeira-Fernandez E, Fernandez-Del-Olmo M (2016) A shorter set reduces the loss of cardiac autonomic and baroreflex control after resistance exercise. Eur J Sport Sci 16(8):996–1004. https://doi.org/10.1080/17461391.2015.1108367. (**PubMed PMID: 26568203**)

Medicine ACoS. ACSM's Guidelines For Exercise Testing And Prescription. Tenth edition ed. Philadelphia : Wolters Kluwer2018.

Miyachi M, Donato AJ, Yamamoto K, Takahashi K, Gates PE, Moreau KL et al (2003) Greater age-related reductions in central arterial compliance in resistance-trained men. Hypertension 41(1):130–135. https://doi.org/10.1161/01.hyp.0000047649.62181.88. (**PubMed PMID: 12511542**)

Miyachi M, Kawano H, Sugawara J, Takahashi K, Hayashi K, Yamazaki K et al (2004) Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. Circulation 110(18):2858–2863. https://doi.org/10.1161/01.CIR.0000146380.08401.99. (**PubMed PMID: 15492301**)

Moreau KL, Stauffer BL, Kohrt WM, Seals DR (2013) Essential role of estrogen for improvements in vascular endothelial function with endurance exercise in postmenopausal women. J Clin Endocrinol Metab 98(11):4507–4515. https://doi.org/10.1210/jc.2013-2183. PubMedPMID:24092827;PubMedCentralPMCID:PMCPMC3816259

Muiesan ML, Salvetti M, Paini A, Monteduro C, Galbassini G, Poisa P et al (2008) Prognostic role of flow-mediated dilatation of the brachial artery in hypertensive patients. J Hypertens 26(8):1612–1618. https://doi.org/10.1097/HJH.0b013e328304b083. (**PubMed PMID: 18622240**)

Muntner P, Shimbo D, Carey RM, Charleston JB, Gaillard T, Misra S et al (2019) Measurement of blood pressure in humans: a scientific statement from the american heart association. Hypertension 73(5):e35–e66. https://doi.org/10.1161/HYP.0000000000000087. (**PubMed PMID: 30827125**)

Murton AJ, Billeter R, Stephens FB, Des Etages SG, Graber F, Hill RJ et al (2014) Transient transcriptional events in human skeletal muscle at the outset of concentric resistance exercise training. J Appl Physiol 116(1):113–125. https://doi.org/10.1152/japplphysiol.00426.2013. (**PubMed PMID: 24265280**)

Nakamura N, Muraoka I (2021) Effects of greater central arterial stiffness on cardiovagal baroreflex sensitivity in resistance-trained men. Sports Med Open 7(1):77. https://doi.org/10.1186/s40798-021-00367-x. (**PubMed PMID: 34698951**)

Nardone M, Floras JS, Millar PJ (2020) Sympathetic neural modulation of arterial stiffness in humans. Am J Physiol Heart Circ Physiol 319(6):H1338–H1346. https://doi.org/10.1152/ajpheart.00734.2020. (**PubMed PMID: 33035441**)

Okamoto T, Masuhara M, Ikuta K (2006) Effects of eccentric and concentric resistance training on arterial stiffness. J Hum Hypertens 20(5):348–354. https://doi.org/10.1038/sj.jhh.1001979. (**PubMed PMID: 16496019**)

Okamoto T, Masuhara M, Ikuta K (2008) Effects of low-intensity resistance training with slow lifting and lowering on vascular function. J Hum Hypertens 22(7):509–511. https://doi.org/10.1038/jhh.2008.12. (**PubMed PMID: 18337757**)

Okamoto T, Masuhara M, Ikuta K (2009a) Effects of muscle contraction timing during resistance training on vascular function. J Hum Hypertens 23(7):470–478. https://doi.org/10.1038/jhh.2008.152. (**PubMed PMID: 19092847**)

Okamoto T, Masuhara M, Ikuta K (2009b) Upper but not lower limb resistance training increases arterial stiffness in humans. Eur J Appl Physiol 107(2):127–134. https://doi.org/10.1007/s00421-009-1110-x. (**PubMed PMID: 19533164**)

Okamoto T, Masuhara M, Ikuta K (2013) Low-intensity resistance training after high-intensity resistance training can prevent the increase of central arterial stiffness. Int J Sports Med 34(5):385–390. https://doi.org/10.1055/s-0032-1312604. (**PubMed PMID: 23041961**)

Otsuki T, Maeda S, Iemitsu M, Saito Y, Tanimura Y, Ajisaka R et al (2007) Vascular endothelium-derived factors and arterial stiffness in strength- and endurance-trained men. Am J Physiol Heart Circ Physiol 292(2):H786–H791. https://doi.org/10.1152/ajpheart.00678.2006. (**PubMed PMID: 16997889**)

Phillips SA, Das E, Wang J, Pritchard K, Gutterman DD (2011) Resistance and aerobic exercise protects against acute endothelial impairment induced by a single exposure to hypertension during exertion. J Appl Physiol 110(4):1013–1020. https://doi.org/10.1152/japplphysiol.00438.2010

Prasad K (2004) C-reactive protein increases oxygen radical generation by neutrophils. J Cardiovasc Pharmacol Ther 9(3):203–209. https://doi.org/10.1177/107424840400900308. (**PubMed PMID: 15378141**)

Rossow LM, Fahs CA, Thiebaud RS, Loenneke JP, Kim D, Mouser JG et al (2014) Arterial stiffness and blood flow adaptations following eight weeks of resistance exercise training in young and older women. Exp Gerontol 53:48–56. https://doi.org/10.1016/j.exger.2014.02.010. (**PubMed PMID: 24566193**)

Saito M, Iwase S, Hachiya T (2009) Resistance exercise training enhances sympathetic nerve activity during fatigue-inducing isometric handgrip trials. Eur J Appl Physiol 105(2):225–234. https://doi.org/10.1007/s00421-008-0893-5. (**PubMed PMID: 18941773**)

Smith LL, Anwar A, Fragen M, Rananto C, Johnson R, Holbert D (2000) Cytokines and cell adhesion molecules associated with high-intensity eccentric exercise. Eur J Appl Physiol 82(1–2):61–67. https://doi.org/10.1007/s004210050652. (**PubMed PMID: 10879444**)

Stacy MR, Bladon KJ, Lawrence JL, McGlinchy SA, Scheuermann BW (2013) Serial assessment of local peripheral vascular function after eccentric exercise. Appl Physiol Nutr Metab 38(12):1181–1186. https://doi.org/10.1139/apnm-2012-0448. (**PubMed PMID: 24195617**)

Thijssen DHJ, Bruno RM, van Mil A, Holder SM, Faita F, Greyling A et al (2019) Expert consensus and evidence-based recommendations for the assessment of flow-mediated dilation in humans. Eur Heart J 40(30):2534–2547. https://doi.org/10.1093/eurheartj/ehz350. (**PubMed PMID: 31211361**)

Tieland M, Trouwborst I, Clark BC (2018) Skeletal muscle performance and ageing. J Cachexia Sarcopenia Muscle 9(1):3–19. https://doi.org/10.1002/jcsm.12238

Vale AF, Carneiro JA, Jardim PCV, Jardim TV, Steele J, Fisher JP et al (2018) Acute effects of different resistance training loads on cardiac autonomic modulation in hypertensive postmenopausal women. J Transl Med 16(1):240. https://doi.org/10.1186/s12967-018-1615-3.PubMedPMID:30165858;PubMedCentralPMCID:PMCPMC6117915

Vlachopoulos C, Dima I, Aznaouridis K, Vasiliadou C, Ioakeimidis N, Aggeli C et al (2005) Acute systemic inflammation increases arterial stiffness and decreases wave reflections in healthy individuals. Circulation 112(14):2193–2200. https://doi.org/10.1161/CIRCULATIONAHA.105.535435. (**PubMed PMID: 16186422**)

Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C et al (2018) 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: executive summary: a report of the American college of cardiology/American heart association task force on clinical practice guidelines. Hypertension 71(6):1269–1324. https://doi.org/10.1161/HYP.0000000000000066. (**PubMed PMID: 29133354**)

Widlansky ME, Gokce N, Keaney JF Jr, Vita JA (2003) The clinical implications of endothelial dysfunction. J Am Coll Cardiol 42(7):1149–1160. https://doi.org/10.1016/s0735-1097(03)00994-x. (**PubMed PMID: 14522472**)

Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M et al (2018) 2018 ESC/ESH Guidelines for the management of arterial hypertension: the task force for the management of arterial hypertension of the European society of cardiology and the European society of hypertension: the task force for the management of arterial hypertension of the European society of cardiology and the European society of hypertension. J Hypertens 36(10):1953–2041. https://doi.org/10.1097/HJH.0000000000001940. (**PubMed PMID: 30234752**)

Yoshizawa M, Maeda S, Miyaki A, Misono M, Saito Y, Tanabe K et al (2009) Effect of 12 weeks of moderate-intensity resistance training on arterial stiffness: a randomised controlled trial in women aged 32–59 years. Br J Sports Med 43(8):615–618. https://doi.org/10.1136/bjsm.2008.052126. (**PubMed PMID: 18927168**)