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Hornby-Foster, Ian ORCID logo ORCID: <https://orcid.org/0000-0001-5500-3215>, Richards, Cory T ORCID logo ORCID: <https://orcid.org/0000-0003-3605-7044>, Drane, Aimee L. ORCID logo ORCID: <https://orcid.org/0000-0002-5208-917X>, Lodge, Freya M., Stemberge, Michael ORCID logo ORCID: <https://orcid.org/0000-0003-0818-6420>, Lord, Rachel ORCID logo ORCID: <https://orcid.org/0000-0002-5385-7548>, Yousef, Zaheer, Stone, Keeron ORCID logo ORCID: <https://orcid.org/0000-0001-6572-7874>, McDonnell, Barry J. ORCID logo ORCID: <https://orcid.org/0000-0001-5912-1278> and Pugh, Christopher J. A. ORCID logo ORCID: <https://orcid.org/0000-0002-5932-4793> (2026) Resistance-trained young men exhibit enhanced carotid vasoreactivity during the cold pressor test. *Experimental Physiology*. doi:10.1113/EP093966 (In Press)

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Resistance-trained young men exhibit enhanced carotid vasoreactivity during the cold pressor test

Ian Hornby-Foster^{1,2}  | Cory T. Richards^{1,3}  | Aimee L. Drane⁴  | Freya M. Lodge⁵ | Michael Stembridge¹  | Rachel Lord¹  | Zaheer Yousef⁵ | Keeron Stone^{1,6}  | Barry J. McDonnell^{1,6}  | Christopher J. A. Pugh^{1,6,7} 

¹Centre for Cardiovascular Research, Innovation and Development, Cardiff Metropolitan University, Cardiff, UK

²Weston General Hospital, Bristol NHS Foundation Trust, Weston-Super-Mare, UK

³School of Education, Health and Science, University of Gloucestershire, Gloucester, UK

⁴Health and Wellbeing Academy, Faculty of Medicine, Health and Life Sciences, Swansea University, Swansea, UK

⁵Department of Cardiology, University Hospital of Wales, Cardiff, UK

⁶National Cardiovascular Research Network, Wales, UK

⁷School of Life Course and Population Health Sciences, King's College London, London, UK

Correspondence

Christopher J. A. Pugh, School of Life Course & Population Health Sciences, Faculty of Life Sciences & Medicine, King's College London, Guy's Campus, Newcomen Street, London, SE1 1UL, UK.
Email: chris.pugh@kcl.ac.uk

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Abstract

The function of the common carotid artery (CCA) is integral to the regulation of cerebral perfusion. Although endurance training is associated with consistent vascular benefits, the effects of resistance training on CCA function are unclear. The cold pressor test (CPT) assesses CCA vasoreactivity via sympathetic stimulation, with peak CCA vasoreactivity ($CAR\%_{peak}$) reflecting integrated endothelial-autonomic vascular function. In this study, we compared $CAR\%_{peak}$ during the CPT in resistance-trained and untrained young men and examined associations with CCA stiffness and haemodynamics. CCA ultrasound was performed in 10 resistance-trained [24 years (95% confidence interval: 22,26)] and 10 untrained [24 years (22,25)] men at baseline, during a 3-min CPT and after recovery, with simultaneous haemodynamic assessment via finger plethysmography. $CAR\%_{peak}$ was defined as the maximal percentage increase in mean diameter from baseline. Secondary outcomes included CCA stiffness indices and haemodynamics. Both groups exhibited CCA vasodilatation during CPT; however, $CAR\%_{peak}$ was greater in resistance-trained men ($P = 0.046$). The resistance-trained group also displayed greater increases in systolic diameter at end-CPT ($P = 0.033$), alongside attenuated pulse pressure ($P = 0.050$; $\eta^2_p = 0.201$) and β_1 -stiffness ($P = 0.041$) responses during CPT. $CAR\%_{peak}$ was negatively correlated with baseline β_2 -stiffness in resistance-trained men ($r = -0.655$; $P = 0.040$), whereas in untrained control subjects it was positively correlated with peak pulse pressure ($r = 0.677$; $P = 0.031$). In conclusion, resistance-trained young men exhibit enhanced CCA vasoreactivity and attenuated pulse pressure and stiffness responses to sympathetic stimulation, reflecting favourable adaptations in endothelial-autonomic vascular regulation. These findings highlight the potential of resistance training to improve central vascular function in early adulthood.

KEYWORDS

cold pressor test, common carotid artery, peak carotid artery reactivity, resistance training

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1 | INTRODUCTION

The common carotid artery (CCA) is a major conduit responsible for delivering cerebral blood flow and plays a vital role in regulating cerebral perfusion through its dynamic structural and functional characteristics (Tarumi & Zhang, 2018). Physical activity is a primary modifiable risk factor for optimising vascular health, whereas a sedentary lifestyle promotes a shift in the endothelial phenotype of the CCA towards a more vasoconstrictive, proliferative and pro-inflammatory state (Seals et al., 2011). Regular endurance exercise promotes favourable CCA adaptations that support cerebral perfusion at rest and during physiological stress, helping to preserve cerebral blood flow and reduce the risk of extracranial and cerebrovascular disease (Seal et al., 2019). These adaptations are evident in both young and older endurance-trained individuals and include increased CCA diameter, enhanced endothelial function and improved wall compliance, collectively augmenting the capacity of the vessel to buffer pulsatile flow (Seals et al., 2019).

Whilst endurance exercise is consistently associated with favourable central arterial adaptations, whereas the effects of resistance training on central arterial function remain less clear. Although some studies report beneficial reductions in central arterial stiffness (Au et al., 2017; Hornby-Foster et al., 2025), others report increased stiffness (Kawano et al., 2008; Miyachi, 2013). In contrast, resistance training appears to improve peripheral artery endothelial function more consistently, as evidenced by enhanced flow-mediated dilatation (FMD) across young (Beck et al., 2013), middle-aged (Boeno et al., 2020) and adolescent (Yu et al., 2016) populations. However, adaptations in peripheral muscular arteries might not necessarily translate to central elastic arteries, which possess distinct structural and functional characteristics important for buffering pulsatile flow (Leloup et al., 2015). Indeed, although peripheral and central arterial endothelial function are positively correlated (Vallbracht-Israng et al., 2007), peripheral arteries appear to adapt more rapidly to exercise training than central vessels (Sun et al., 2024). Therefore, direct assessment of the CCA might provide a more specific local measure of central arterial function and adaptation to resistance training.

The cold pressor test (CPT) is an established tool for assessing CCA endothelial function by eliciting sympathetic nervous system activation, alongside transient increases in blood pressure and vascular resistance (van Mil et al., 2017). The resulting CCA diameter response provides insight into local vascular tone, endothelial bioavailability and vasoreactivity to sympathoexcitatory stimuli (van Mil et al., 2017), with responses varying according to arterial health and adrenergic sensitivity. During the CPT, individuals with coronary artery disease exhibit CCA vasoconstriction (Rubenfire et al., 2000), whereas those with cardiovascular disease (CVD) risk factors typically show no change in diameter (Rubenfire et al., 2000; van Mil et al., 2017). Healthy older adults also show limited vasodilatory capacity, possibly reflecting altered α/β -adrenergic balance (Flück et al., 2017), and young healthy adults elicit significant CCA vasodilatation consistent with preserved endothelial function (Stone et al., 2019). Evidence

Highlights

- **What is the central question of the study?**

The impact of chronic resistance training on common carotid artery (CCA) vasoreactivity, a key marker of endothelial function, remains poorly defined in young adults. In this study, we investigated whether resistance-trained men exhibit distinct CCA responses during a sympathetic challenge (cold pressor test) compared with untrained age-matched control subjects.

- **What is the main finding and its importance?**

Resistance-trained men demonstrate augmented CCA vasoreactivity during the cold pressor test compared with age-matched untrained control subjects. These findings indicate that chronic resistance training promotes superior large-artery endothelial function, which is likely to optimise the regulation of extracranial and cerebrovascular perfusion during acute sympathetic stress.

on resistance training and CCA vasoreactivity is limited, with cross-sectional (Kawano et al., 2008) and longitudinal (Cortez-Cooper et al., 2008) studies in middle-aged adults reporting no effect, suggesting that age-related CCA remodelling might constrain adaptive responses to sympathetic stimulation. Whether resistance training influences CCA vasoreactivity in young healthy adults, in whom age-related vascular remodelling is less likely to constrain adaptive responses, remains unknown.

Therefore, the aim of this study was to examine CCA vasoreactivity to sympathetic stimulation using the CPT in young resistance-trained men and an age-matched untrained control group. We hypothesised that young resistance-trained males would demonstrate significant CCA vasodilatation in response to the CPT, similar to untrained control subjects.

2 | MATERIALS AND METHODS

2.1 | Ethical approval

Individuals provided written informed consent. Ethical approval was granted by the School of Sport and Health Sciences Research Ethics Committee at Cardiff Metropolitan University (17/3/01S). All procedures were conducted in accordance with the *Declaration of Helsinki* (2008), excluding registration in a public trials database.

2.2 | Participants

Data collection for this study was conducted as part of a broader programme of investigations examining training-specific cardiac and carotid artery adaptations in young men in response to a variety of physiological stimuli (Dawkins et al., 2020, 2022; Hornby-Foster et al., 2025). However, the present study addresses a distinct a priori hypothesis focused explicitly on CPT-derived CCA vasoreactivity in resistance-trained and untrained young men. Although previous work from this programme reported data from 14 resistance-trained participants, only 10 had complete CPT-derived CCA image datasets suitable for analysis across all required time points. The remaining four were not included because CPT testing was either not performed or incomplete or because the acquired image sequences were not analysable across all time points. Accordingly, 20 young males were included in the present analysis, comprising 10 resistance-trained [24 years, 95% confidence interval (CI) (22, 26)] and 10 untrained [24 years, 95% CI (22, 25)] individuals.

Habitual physical activity and training status were assessed during the initial laboratory visit using a structured lifestyle and training questionnaire administered by the investigative team. Participants in the resistance-trained group had engaged exclusively in structured full-body resistance training for a minimum of 2 years consecutively, with a training frequency of at least three sessions per week and no participation in structured aerobic or endurance exercise. Untrained men had not engaged in regular structured exercise during the preceding 2 years and self-reported ≤ 150 min week⁻¹ of structured physical activity (Bull et al., 2020). Exclusion criteria included the use of cardioactive drugs and prescribed medications, history of cardiovascular, musculoskeletal or metabolic disease, history of asthma or smoking or any contraindications to exercise. No participants reported using performance-enhancing drugs.

2.3 | Study overview

The study implemented a cross-sectional design and was conducted in a quiet, temperature-controlled room. Participants attended the laboratory on two occasions. Prior to each visit, they were instructed to abstain from alcohol, caffeine and vigorous exercise for 24 h and to fast for ≥ 6 h. During the first session, participants completed a one-repetition maximum (1RM) seated leg press, followed by an incremental cycling test to determine peak oxygen consumption ($\dot{V}_{O_{2peak}}$), as previously described (Hornby-Foster et al., 2025). In brief, the first attempt at 1RM was performed at $\sim 50\%$ of the participant's weight-predicted 1RM. The load was then progressively increased over three to five attempts until the participant could complete only a single repetition, thereby reflecting their true 1RM. The $\dot{V}_{O_{2peak}}$ was determined using an incremental cycling test, with initial loads of 50 W for both groups, which were subsequently increased by 20 W every minute until volitional exhaustion. Measurements of ventilatory gas exchange were obtained using a breath-by-breath

analyser (Oxycon Pro, Jaeger, Warwick, UK). In the absence of a distinct $\dot{V}_{O_2}$ plateau, $\dot{V}_{O_{2peak}}$ was defined as the highest $\dot{V}_{O_2}$ achieved over a 30 s consecutive period prior to volitional exhaustion. At the second visit, short-axis ultrasound images of the CCA were obtained at baseline, during a CPT and following a recovery period.

2.4 | Experimental procedures

2.4.1 | CPT

Participants completed a 3-min CPT by placing their left hand into ice water (4°C) whilst supine. This is a widely accepted and validated model to stimulate the sympathetic nervous system, and this particular protocol has previously been shown to elicit transient increases in sympathetic activation and evoke haemodynamic and arterial responses (Thijssen et al., 2006; van Mil et al., 2017). Furthermore, 3-min CPT protocols ensure that CCA responses are correlated with the period of maximal sympathetic stimulation, normally present during 90 s into the CPT (van Mil et al., 2017). A hand-immersion protocol was selected because it represents the most widely validated and reproducible model for eliciting an acute, systemic sympathetic pressor response (van Mil et al., 2017). CCA ultrasound measurements were obtained prior to the onset of the CPT (baseline), 90 s following hand immersion (mid), immediately prior to removing the hand (end; 180 s) and 60 s after hand removal (recovery; 240 s). Participants were instructed not to speak and to breathe normally during the CPT and were verbally encouraged throughout. The CPT was performed without prior familiarisation to the ice water stimulus, to preserve the acute cold-induced sympathetic response and avoid habituation that could influence baseline or CPT-evoked measurements. Beat-by-beat blood pressure, stroke volume (SV), cardiac output ($\dot{Q}$) and total peripheral resistance (TPR) were recorded continuously via finger plethysmography (FinometerPro, FMS, Groningen, The Netherlands) on the contralateral hand. These blood pressure data were then calibrated against manual baseline brachial blood pressure measurements taken prior to CPT. All stroke volume data were adjusted to body surface area [stroke volume index (SVI)], which was calculated using the Du Bois formula (Cattermole et al., 2017).

2.4.2 | CCA ultrasound imaging

Short-axis grey-scale cine loops of the right CCA were acquired on a commercially available high-resolution ultrasound system (Vivid q, GE Healthcare, Chalfont, UK), using a 12 MHz multi-frequency linear array probe. Images were recorded ~ 2 cm distal to the carotid bifurcation for six consecutive cardiac cycles. Cardiac cycles were measured continuously using a three-lead ECG connected to the ultrasound machine. Frame rate (92.3 frames s⁻¹), imaging depth and probe orientation were standardised for all participants, and all images were acquired by a single trained technician.

2.4.3 | Common carotid diameter and vasoreactivity

CCA diameters were obtained using M-mode ultrasound. Systolic and diastolic diameters were defined as the maximal and minimal arterial diameters during and following pulsatile ejection, respectively. These were averaged over six cardiac cycles using callipers placed on the inner-to-inner anteroposterior CCA walls. Mean diameter, calculated as the average of systolic and diastolic diameters for each cycle, was recorded at baseline, during both CPT time points and after recovery. Mean diameter was used as a composite measure of vessel calibre reflecting the reactive change in diameter throughout the cardiac cycle in response to sympathetic stimulation. This provides an index of CCA vasoreactivity (van Mil et al., 2017), which is an established marker of local CCA endothelial function (Stone et al., 2019). Peak CCA vasoreactivity ($CAR\%_{peak}$) was defined as the greatest percentage change in mean diameter from baseline, observed at either the mid-point or end of the CPT protocol for each participant.

2.4.4 | CCA stiffness parameters

To characterise local CCA stiffness, the distensibility coefficient (DC) was calculated using the formula: $DC = (2D\Delta D + (\Delta D)^2)/(D^2\Delta P)$ (van Bortel et al., 2002); where ΔD represents distension, D is the diastolic diameter, and ΔP is pulse pressure (PP). This reflects the relative change in CCA diameter during the cardiac cycle for a stroke change in blood pressure and has demonstrated a lower intersession coefficient of variation in comparison to alternative stiffness indices at the CCA (van Bortel et al., 2002). We also used the β_1 -stiffness index [β_1 -stiffness; in arbitrary units (a.u.)] and β_2 -stiffness index (β_2 -stiffness; in arbitrary units) to characterise CCA stiffness, which were calculated using established formulas from our previous investigations (Hornby-Foster et al., 2025; Pugh et al., 2018). β_2 -Stiffness was derived from peak circumferential strain (PCS), assessed via two-dimensional speckle-tracking software (EchoPac v.112, GE Vingmed Ultrasound, Horten, Norway) to quantify global arterial wall deformation across the cardiac cycle, as described in our earlier studies (Hornby-Foster et al., 2025; Pugh et al., 2018; Talbot et al., 2020).

2.5 | Statistical analysis

An a priori sample-size estimation for $CAR\%_{peak}$ was informed by effect size and variance estimates from two previous CPT studies: one examining the effect of sex differences in $CAR\%_{peak}$ in young adults (Stone et al., 2019) and one investigating age-related differences in $CAR\%_{peak}$ (Flück et al., 2017). These studies reported mean absolute between-group differences in $CAR\%_{peak}$ of 5.0% between healthy young and older adults (Cohen's d effect size = 1.66) and ~2.6% between healthy young men and women (d = 1.18). Based on the pooled variance estimate most relevant to the young adult demographic, an independent two-group comparison with 20 participants in total, comprising 10 participants per group, was

estimated to provide 80% power to detect a minimum between-group difference of ~3.0% in $CAR\%_{peak}$ (d = 1.32) using a two-sided α -level of 0.05. All data were checked and confirmed for normality of distribution using a Shapiro–Wilk test. Differences in baseline descriptive characteristics between groups were assessed using independent t -tests. To evaluate responses across the CPT, a two-way repeated-measures ANOVA was conducted to determine the main effects of time, group and any interaction between these factors (group $\times$ time). ANOVAs were conducted using relative change data, expressed as a percentage change from baseline at each protocol time point. When a significant interaction was identified, *post hoc* pairwise comparisons were performed with Bonferroni adjustments. An additional independent t -test was performed to assess the differences in $CAR\%_{peak}$ between groups. Pearson's correlation was used to assess the exploratory relationship between $CAR\%_{peak}$ and baseline haemodynamics and CCA stiffness indices, in addition to the peak percentage changes in these variables during the CPT. Effect sizes were calculated to contextualise the magnitude of the findings close to significance, using partial eta squared (η^2_p) for ANOVA and Cohen's d for pairwise comparisons. Analyses were carried out using SPSS (v.27 for Windows, SPSS Inc., Chicago, IL, USA), and results are reported as mean percentage changes from baseline values [95% confidence intervals]. Statistical significance was set a priori at $P < 0.05$.

3 | RESULTS

3.1 | Descriptive characteristics

Baseline descriptive characteristics, haemodynamics, CCA stiffness indices and diameters are presented in Table 1. In line with our study design, the resistance-trained group demonstrated greater 1RM and training frequency compared with control subjects ($P < 0.001$). They also had a higher body mass, body mass index ($P < 0.001$) and body surface area ($P = 0.003$). Baseline heart rate was lower in the resistance-trained group ($P = 0.001$), and SVI was higher ($P = 0.002$). Finally, baseline systolic blood pressure (SBP) was significantly higher in resistance-trained participants ($P = 0.021$). No other blood pressure variables differed between the groups, nor were there differences in $\dot{V}_{O_{2peak}}$, fat-free mass or body fat percentage ($P > 0.196$). Likewise, CCA diameters ($P > 0.065$) and stiffness indices ($P > 0.120$) did not differ between groups.

3.2 | CCA vasoreactivity during CPT

A significant effect of time was observed for both systolic ($P < 0.001$; Figure 1b) and mean ($P = 0.002$; Figure 1c) diameter. In both groups, values increased at the midpoint and end point of the CPT and returned to baseline during recovery. However, no effect of time was observed for diastolic diameter during the CPT ($P = 0.071$; Figure 1a). No group effects were observed for mean ($P = 0.331$), systolic ($P = 0.106$) or diastolic diameters ($P = 0.808$). A close to significant group $\times$ time

TABLE 1 Baseline descriptive characteristics, haemodynamics, common carotid artery stiffness indices and diameters in resistance-trained and untrained men.

Measured variables	Resistance-trained (n = 10)	Untrained (n = 10)	P-value
Characteristics			
Age, years	24 [22, 26]	24 [22, 25]	0.471
Height, cm	182.4 [178, 186.4]	181 [174, 187.9]	0.703
Body mass, kg	89.7 [84, 95.7]	73.5 [68.3, 78.7]	<0.001
BMI, kg.m ⁻²	27.5 [25.7, 28.3]	22.6 [20.5, 24.7]	<0.001
Body fat, %	13.5 [11, 16.1]	14.7 [9.7, 19.8]	0.208
FFM, kg	69.2 [53.2, 85.2]	62.2 [58, 66.4]	0.482
Body surface area, m ²	2.12 [2.03, 2.21]	1.93 [1.84, 2.02]	0.003
$\dot{V}_{O_{2peak}}$, mL kg ⁻¹ min ⁻¹	39.3 [35.9, 42.7]	41 [38, 44.2]	0.196
1RM, kg	448 [424, 471]	217 [177, 256]	<0.001
Training history, years	6.2 [4.8, 7.5]		
Training frequency, session week ⁻¹	5.1 [4.6, 5.5]	0.7 [0.3, 1.0]	<0.001
Haemodynamics			
SBP, mmHg	127 [123, 131]	121 [116, 126]	0.021
DBP, mmHg	78 [70, 85]	75 [71, 79]	0.422
PP, mmHg	49 [42, 56]	46 [40, 51]	0.156
MAP, mmHg	94 [88, 100]	91 [87, 94]	0.368
SV, mL	141 [129, 153]	112 [106, 117]	<0.001
SVI, mL m ⁻²	67 [51, 72]	57 [55, 60]	0.002
HR, beats min ⁻¹	56 [50, 62]	68 [63, 72]	0.001
$\dot{Q}$, L min ⁻¹	7.9 [6.9, 8.8]	7.6 [7.1, 8]	0.252
TPR, mmHg min mL ⁻¹	12.3 [10.6, 13.9]	12 [11.2, 12.8]	0.362
CCA diameters			
DD, mm	5.72 [5.42, 6]	5.78 [5.65, 5.9]	0.341
SD, mm	6.45 [6.14, 6.76]	6.59 [6.41, 6.76]	0.193
MD, mm	6.1 [5.78, 6.39]	6.18 [6.03, 6.33]	0.255
CCA stiffness indices			
PCS, %	12.1 [10.9, 13.2]	11.5 [10.4, 12.7]	0.249
DC, $\times 10^{-3}$ mmHg ⁻¹	5.88 [4.6, 7.1]	6.8 [5.6, 7.9]	0.121
β_1 , a.u.	3.97 [2.98, 4.97]	3.43 [2.89, 3.97]	0.146
β_2 , a.u.	2.62 [2.5, 2.74]	2.64 [2.53, 2.76]	0.393

Note: Data are presented as absolute means [95% confidence interval]. Abbreviations: 1RM, one-repetition maximum; BMI, body mass index; DBP, diastolic blood pressure; DC, distensibility coefficient; DD, diastolic diameter; FFM, fat free mass; HR, heart rate; MAP, mean arterial pressure; MD, mean diameter; PCS, peak circumferential strain; PP, pulse pressure; $\dot{Q}$, cardiac output; SBP, systolic blood pressure; SD, systolic diameter; SVI, stroke volume index; TPR, total peripheral resistance; $\dot{V}_{O_{2peak}}$, peak oxygen consumption; β_1 , β_1 -stiffness index; β_2 , β_2 -stiffness index.

interaction was observed for systolic diameter ($P = 0.051$; effect size $\eta^2_p = 0.174$), with *post hoc* pairwise comparisons revealing a greater increase at the end point in the resistance-trained group compared with untrained control subjects ($P = 0.033$). However, there were no interaction effects for mean ($P = 0.242$) or diastolic ($P = 0.672$) diameters. Finally, the resistance-trained group exhibited a significantly greater $CAR\%_{peak}$ than untrained control subjects (7.7% [95% CI: 3.5, 11.9] vs. 3.8% [95% CI: 1.7, 5.9]; $P = 0.046$; Cohen's d effect size = 0.84; Figure 2).

3.3 | CCA stiffness and haemodynamic responses during CPT

CCA stiffness and haemodynamics during the CPT are displayed in Table 2. A significant effect of time was observed in DC ($P = 0.013$), PCS ($P = 0.047$; $\eta^2_p = 0.158$), all blood pressure variables ($P < 0.05$) and TPR ($P = 0.006$). In both groups, blood pressures, TPR and DC increased, and PCS decreased, in response to the CPT and returned to baseline values in recovery. There were no time effects observed for β_1 -stiffness, heart

TABLE 2 Haemodynamics and common carotid artery stiffness parameters, expressed as percentage change from baseline values, during a 3 min cold pressor test and recovery period in resistance-trained and untrained young men.

Measured variable (% change)	Resistance trained (<i>n</i> = 10)			Untrained (<i>n</i> = 10)			P-value	
	Midway	End	Recovery	Midway	End	Recovery	Time effect	Group × time interaction
Haemodynamics								
SBP, mmHg	10.5 [5, 16]	6.6 [3, 10.2]	5.8 [2.7, 8.9]	15 [9.4, 20.5]	12.8 [8.4, 17.2]	7 [3.3, 10.7]	<0.001	0.128
DBP, mmHg	13.9 [8.4, 19.3]	9 [5.3, 12.6]	7.2 [3, 11.3]	15.8 [10, 21.5]	13.2 [7.7, 18.7]	6.4 [1.5, 11.3]	<0.001	0.526
PP, mmHg	6.2 [−1.2, 13.7]	3.5 [−2, 9.1]	4.3 [0.3, 8.3]	14.4 [5.4, 23.4]	12.9 [7.2, 18.6]	8.4 [3.6, 13.1]	0.050	0.049
MAP, mmHg	12.3 [7, 17.5]	7.8 [4.5, 11.2]	6.5 [3, 9.9]	15.4 [10, 20.8]	13 [8.1, 17.9]	6.7 [2.4, 10.9]	<0.001	0.282
SVI, mL m ^{−2}	−0.6 [−6.5, 5.3]	2.8 [−3.8, 9.4]	0.9 [−3.4, 5.1]	3.8 [−3.9, 11.6]	6 [−2.4, 14.3]	7.5 [1.7, 13.2]	0.064	0.348
HR, beats min ^{−1}	0 [−10, 10]	−0.6 [−10.7, 9.6]	0.8 [−5.9, 7.5]	8.2 [−0.3, 16.6]	7.9 [−0.5, 16.2]	3.2 [−3.7, 10]	0.588	0.197
Q̇, L min ^{−1}	−1.3 [−9.1, 6.6]	2.2 [−10, 14.8]	1.4 [4.4, 7.3]	11.6 [2.6, 20.7]	13.1 [5, 21.3]	12 [4.7, 19.3]	0.561	0.563
TPR, mmHg min mL ^{−1}	15.3 [2.8, 27.8]	8.2 [5.1, 21.6]	5.7 [−2, 13.7]	3.9 [−4, 11.7]	0.5 [−7.8, 8.8]	−4.9 [−11.7, 1.9]	0.006	0.817
CCA stiffness parameters								
PCS, %	−14 [−22.5, −5.5]	−13.7 [−23.4, −4]	−1.8 [−13, 9.7]	−13.6 [−27, −0.6]	−10 [−23.9, 4]	−12 [−27.3, 3.3]	0.047	0.788
β ₂ , a.u.	4.9 [1.5, 8.2]	5.2 [0.9, 9.5]	0.8 [−4.3, 5.8]	6.2 [0.6, 11.8]	4.9 [1.3, 11.2]	6.4 [−0.4, 13.2]	0.223	0.473
DC, ×10 ^{−3} mmHg ^{−1}	28.5 [−1.3, 58]	21.9 [7.2, 36.6]	−5.8 [−18, 6.3]	−6.9 [−30.8, 17]	−8 [−33.8, 17.8]	−10.7 [−30.5, 9]	0.013	0.063

Note: Data are presented as percentage changes from baseline values [95% confidence interval]. Abbreviations: DBP, diastolic blood pressure; DC, distensibility coefficient; HR, heart rate; MAP, mean arterial pressure; PCS, peak circumferential strain; PP, pulse pressure; Q̇, cardiac output; SBP, systolic blood pressure; SVI, stroke volume index; TPR, total peripheral resistance; β₂, β₂-stiffness index.

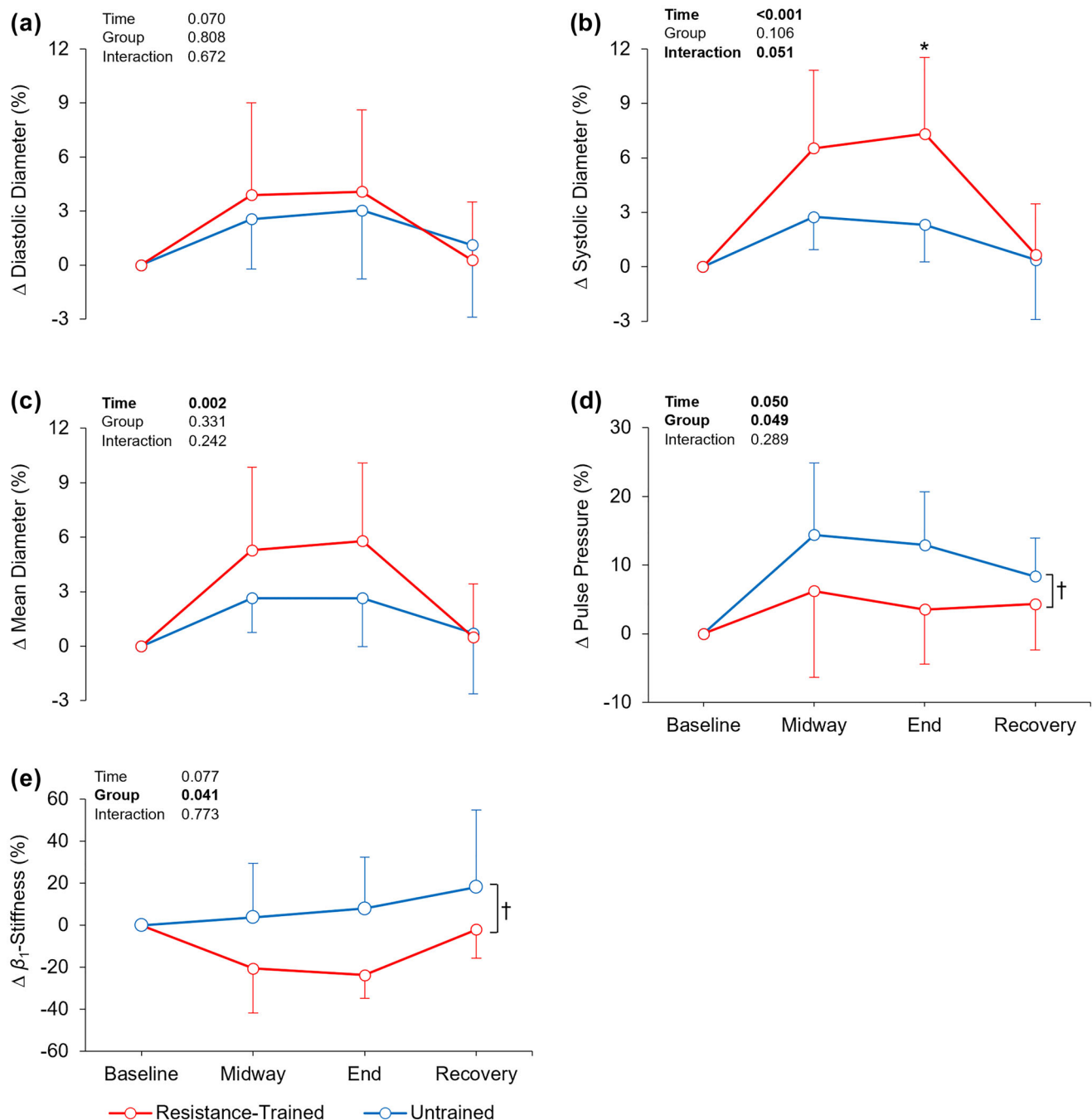


FIGURE 1 Diastolic diameter (a), systolic diameter (b), mean diameter (c), pulse pressure (d) and β_1 -stiffness (e) at baseline, during (mid and end) and after a 3 min cold pressor test in resistance-trained (red, $n = 10$) and untrained (blue, $n = 10$) young men. Data are presented as the mean percentage change from baseline, and error bars represent 95% confidence interval. Individual data points can be found in the [Appendix, Table A1](#). Two-way ANOVA effects for time and group and group $\times$ time interactions are presented. * $P < 0.05$ pairwise difference between resistance-trained vs. untrained groups at the same time point. †Significant between-group difference.

rate, SVI or $\dot{Q}$ ($P > 0.060$). A significant group effect was observed for PP ($P = 0.050$; $\eta^2_p = 0.201$) and β_1 -stiffness ($P = 0.041$; Figure 1e), with the resistance-trained group demonstrating an attenuated rise in PP during the CPT and consistently lower β_1 -stiffness values throughout the stimulus compared with untrained control subjects. A significant group $\times$ time interaction was observed for PCS ($P = 0.041$) and β_2 -

stiffness ($P = 0.040$); however, *post hoc* pairwise comparisons revealed no significant group differences at any time point during the CPT (PCS, $P > 0.269$; β_2 -stiffness, $P > 0.148$). No further effects of time, group or group $\times$ time interactions were detected for any CCA stiffness or haemodynamic variable.

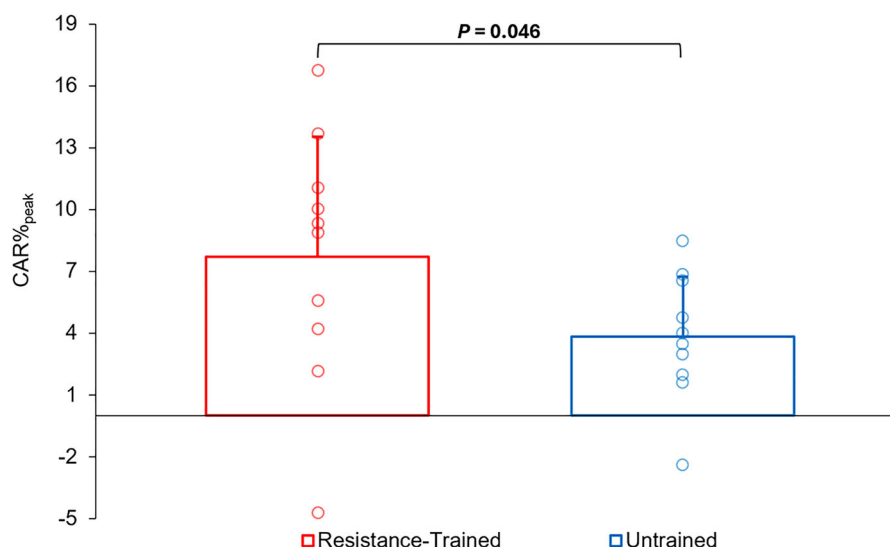


FIGURE 2 Peak common carotid artery vasoreactivity (CAR%_{peak}, presented as the peak percentage change from baseline values) during a 3 min cold pressor test in young resistance-trained (red, $n = 10$) and untrained (blue, $n = 10$) men. Data are presented as means with individual participant data points, and error bars represent the 95% confidence interval. Independent sample t -test: * $P < 0.05$ difference between groups for CAR%_{peak}.

3.4 | Correlates of CAR%_{peak}

No significant correlations were observed when data were pooled across both groups. When conducting subgroup correlation analysis, in the resistance-trained group only, there was a significant negative correlation between CAR%_{peak} and baseline β_2 -stiffness [Figure 3a,b; $r = -0.655$; $P = 0.041$ (vs. untrained group: $r = 0.414$; $P = 0.234$)] and the peak percentage increase in TPR [$r = -0.731$; $P = 0.016$ (vs. untrained group: $r = 0.088$; $P = 0.812$)], whereas, in untrained control subjects only, there was a significant positive correlation between CAR%_{peak} and the peak percentage increase in systolic [$r = 0.722$; $P = 0.018$ (vs. resistance-trained group: $r = -0.397$; $P = 0.256$)] and PPs [Figure 3c,d; $r = 0.677$; $P = 0.031$ (vs. resistance-trained group: $r = -0.216$; $P = 0.549$)]. No other significant correlations were observed between CAR%_{peak} and the remaining pressure variables or CCA stiffness indices at baseline or during peak response to the CPT.

4 | DISCUSSION

The aim of this study was to examine CCA vasoreactivity to sympathetic stimulation, assessed via the CPT, in young resistance-trained men compared with age-matched untrained control subjects. To our knowledge, this is the first study to investigate CCA vasoreactivity to a CPT in this population. In line with our primary hypothesis, untrained men exhibited a normal vasodilatory response; however, resistance-trained men demonstrated a significantly greater CAR%_{peak} during the CPT. These findings indicate that resistance-

training enhances CCA vasoreactivity, reflecting favourable central arterial adaptations and improved endothelial-autonomic regulation.

Our finding of enhanced CAR%_{peak} in young resistance-trained men contrasts with previous reports of no effect of resistance training on CCA vasoreactivity in middle-aged cohorts (Cortez-Cooper et al., 2008; Kawano et al., 2008), suggesting that age might modulate central artery adaptability to resistance training. Consistent with this, CAR%_{peak} in the present cohort (7.7%) exceeded values reported in middle-aged resistance-trained groups (~4%) (Cortez-Cooper et al., 2008; Kawano et al., 2008), young untrained adults (~4%) (Stone et al., 2019; van Mil et al., 2017) and older or CVD populations, in which blunted or constricted responses are common (Flück et al., 2017; Rubenfire et al., 2000; van Mil et al., 2017). This heightened vasoreactivity is likely to reflect greater endothelial nitric oxide bioavailability (Green et al., 2011), shear stress responsiveness (Thijssen et al., 2011), improved local buffering (van Mil et al., 2017) and favourable adrenergic signalling adaptations (Rubenfire et al., 2000) in our cohort. In contrast, age-related stiffening, elevated basal vascular tone and endothelial impairment (Flück et al., 2017) are likely to explain the blunted responses in middle-aged cohorts (Kawano et al., 2008; Cortez-Cooper et al., 2008), whereas young arteries remain fully responsive, enabling unconstrained optimal adaptation to resistance training.

Evidence from studies directly examining CAR%_{peak} in young resistance-trained populations remains limited. However, peripheral vascular studies indicate that resistance training can enhance endothelial function in adolescents and young adults (Beck et al., 2013; Boeno et al., 2020; Yu et al., 2016), suggesting that the present findings might reflect broader endothelial adaptation across peripheral and

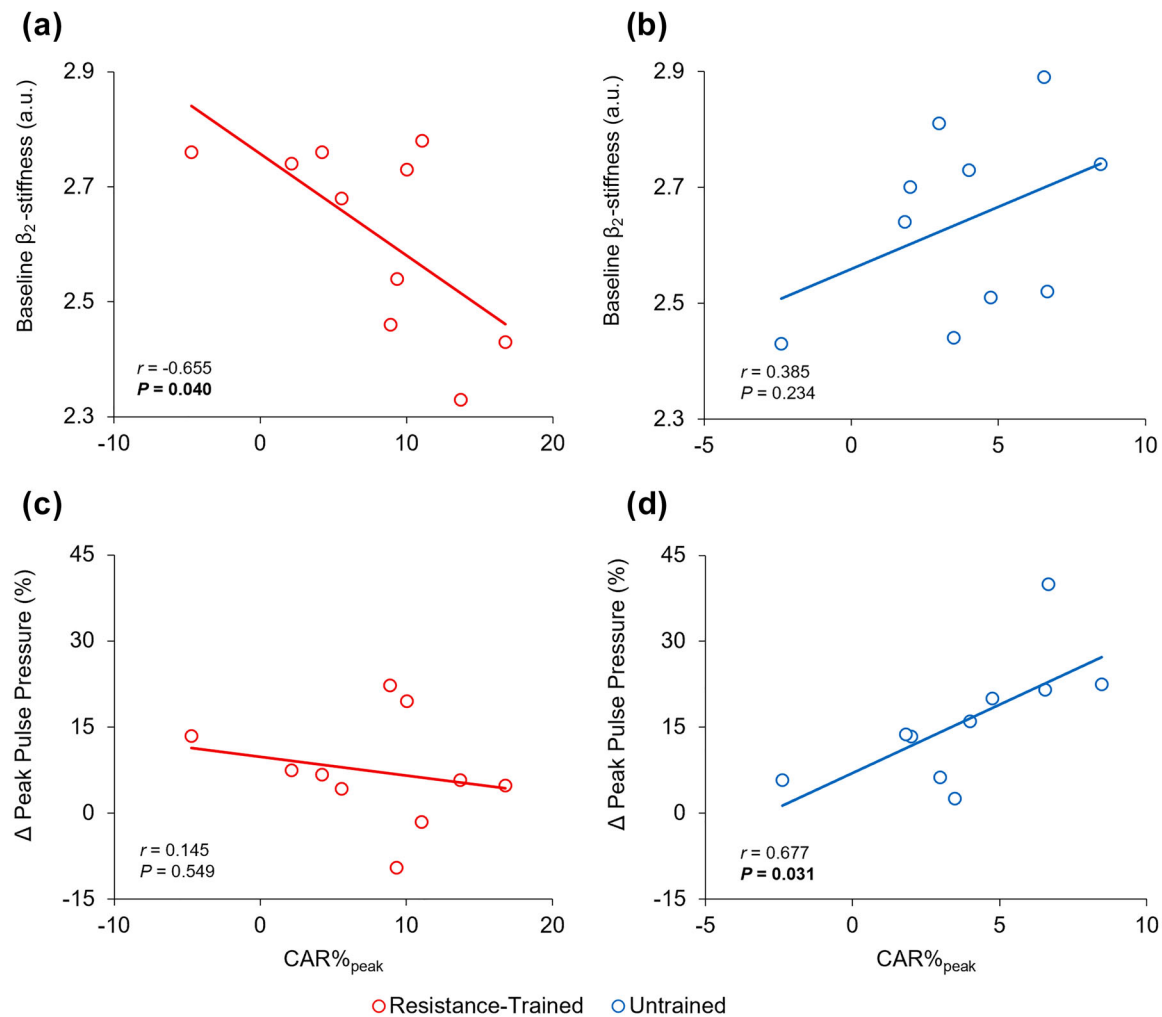


FIGURE 3 Relationship between peak common carotid artery vasoreactivity (CAR%_{peak}) and baseline β_2 -stiffness [(a) resistance trained (red, $n = 10$); and (b) untrained (blue, $n = 10$)], and the change in peak pulse pressure during a 3-min cold pressor test (c, d).

central vascular beds. Although extrapolation from peripheral FMD to the CCA requires caution, endothelial responses across vascular beds are known to be related. For example, brachial FMD is strongly correlated with coronary endothelium-dependent vasodilatation in response to acetylcholine (Broxterman et al., 2019), and CAR%_{peak} has been associated with coronary artery responses during CPT (van Mil et al., 2017). Given that the CAR response to CPT is at least partly endothelium mediated (Tousoulis et al., 1997; van Mil et al., 2017), the elevated CAR%_{peak} observed here might reflect enhanced systemic endothelial function in resistance-trained men.

Although CAR%_{peak} was augmented in the resistance-trained group, mean diameter change did not differ between groups at either fixed time point, consistent with observations in young, healthy adults (Stone et al., 2019). CAR%_{peak} captures the maximal dynamic vasoreactivity of each participant, irrespective of when peak dilatation occurs, whereas mean diameter change measured at a fixed time point might mask group differences attributable to interindividual variability in the timing of peak dilatation during the CPT. Notably, only 30% of resistance-trained and 50% of untrained participants peaked at

90 s, challenging the conventional assumption that maximal dilation occurs at 90 s of CPT (van Mil et al., 2017). Similar interindividual variability has been reported previously (Flück et al., 2017; Tymko et al., 2017), probably reflecting differences in endothelial-autonomic responsiveness. Unlike mean and diastolic diameter, systolic diameter was significantly elevated in the resistance-trained group at the end of the CPT, suggesting enhanced systolic vasodilatory capacity, which might reflect adaptation to the repeated elevated systolic pressures experienced during resistance training.

Basal CCA stiffness appears to influence the CAR%_{peak} response in the resistance-trained group. Baseline β_2 -stiffness was negatively correlated with CAR%_{peak}, indicating that greater arterial compliance is associated with a larger CPT-induced vasodilatory response. The resistance-trained group also exhibited an attenuated rise in β_1 -stiffness during CPT, suggesting preserved buffering of transient pressure loading without excessive local arterial stiffening. This contrasts with findings in middle-aged resistance-trained men, in whom β_1 -stiffness increases more markedly during CPT than in untrained control subjects (Kawano et al., 2008). Notably, the elevated CAR%_{peak}

occurred despite an attenuated increase in PP, suggesting that enhanced vasoreactivity was not driven simply by a larger pressor stimulus. Together, these responses indicate a more compliant vascular phenotype in the resistance-trained cohort, facilitating greater vasodilatory responsiveness during sympathetic stimulation. Although endurance training promotes functional sympatholysis through blunted α_1/α_2 -mediated vasoconstriction and enhanced β -mediated vasodilatation (Mortensen et al., 2014), the effects of resistance training on adrenergic regulation remain less clear. The greater $\text{CAR\%}_{\text{peak}}$ and blunted β_1 -stiffness response observed here might reflect adaptations in local adrenergic responsiveness and/or endothelial nitric oxide bioavailability arising from repeated episodic exposure to elevated pressure, pulsatile flow and shear stress during resistance exercise (Green et al., 2017).

Despite higher resting SBP in the resistance-trained group, the SBP response to CPT was similar between groups, while the PP increase was attenuated. This indicates that elevated resting SBP did not impair CCA buffering of acute pressure loading during sympathetic stress. The enhanced $\text{CAR\%}_{\text{peak}}$ observed alongside an attenuated PP response suggests greater local vasoreactivity, consistent with endothelium-mediated dilatation rather than passive pressure-mediated distension. Correlation analysis further supports this interpretation, because $\text{CAR\%}_{\text{peak}}$ was positively associated with PP in untrained men but not in resistance-trained men, indicating a degree of functional decoupling from haemodynamic loading. This aligns with our previous observations of augmented CCA two-dimensional circumferential strain at rest and in response to isometric hand-grip exercise in resistance-trained men (Hornby-Foster et al., 2025), consistent with enhanced arterial compliance and dynamic responsiveness to acute physiological stress. Together, these findings suggest that resistance-trained men maintain rapid vasodilatory and buffering responses to acute sympathetic pressor stress, which might have favourable implications for carotid-cerebral haemodynamics. Indeed, blunted PP responses and enhanced CCA function have been associated with lower carotid and cerebral pulsatility, reduced plaque formation and lower stroke risk (Lambiase et al., 2014; Matthews et al., 1998; Mitchell et al., 2011; Yang et al., 2018).

This study has limitations that should be considered. First, our findings are limited to young male participants. Given reported sex differences in CCA vasoreactivity during CPT (Stone et al., 2019), resistance-trained females might exhibit distinct baseline characteristics and divergent responses. Future studies should examine the interaction between sex and resistance training on CCA vasoreactivity. Second, this study did not use continuous diameter measurements used in some $\text{CAR\%}$ CPT studies (Flück et al., 2017; Stone et al., 2019; Tymko et al., 2017; van Mil et al., 2017). Instead, cine loops were acquired at preset time points, consistent with previous carotid vasoreactivity studies (Kawano et al., 2008; Londono et al., 2014; Rubenfire et al., 2000), including at 90 s of CPT, a commonly used index of CCA peak dilatation (Thijssen et al., 2011; van Mil et al., 2017). Third, although appropriately powered for the primary $\text{CAR\%}_{\text{peak}}$ analysis, secondary correlations should be interpreted

cautiously given the modest cohort size. Fourth, we did not capture detailed training-intensity history and therefore cannot determine the influence of overall resistance-training load. Finally, concurrent measurements of CCA blood flow and shear rate were not obtained, which might have provided additional mechanistic insight into the divergent vasodilatory responses between groups. However, previous studies in young adults indicate that $\text{CAR\%}_{\text{peak}}$ during the CPT is not directly related to changes in carotid blood flow or shear rate but is more strongly associated with changes in mean arterial pressure (Stone et al., 2019). In the present study, mean arterial pressure responses were similar between groups, whereas the rise in PP was attenuated in resistance-trained men. Given the divergent blood pressure associations, the augmented $\text{CAR\%}_{\text{peak}}$ in the resistance-trained group is likely to reflect intrinsic vascular mechanisms beyond passive local haemodynamics.

5 | CONCLUSION

In conclusion, this study demonstrates, for the first time, that young resistance-trained men exhibit significantly greater $\text{CAR\%}_{\text{peak}}$ during CPT compared with untrained control subjects. They also exhibited blunted PP and β_1 -stiffness rises during CPT, suggesting a functional decoupling from passive pressure-mediated dilatation. Together, these features are likely to reflect integrated adaptations induced by resistance training, culminating in a protective CCA phenotype. Finally, our findings build on a broader conceptual framework that resistance training enhances not only structural integrity (Hornby-Foster et al., 2025), but also functional responsiveness of the CCA during acute sympathetic and haemodynamic stress.

AUTHOR CONTRIBUTIONS

Ian Hornby-Foster and Christopher J. A. Pugh conceived and designed the research; Ian Hornby-Foster, Aimee L. Drane, Rachel Lord, Cory T. Richards, Freya M. Lodge and Michael Stembridge performed experiments; Ian Hornby-Foster analysed data; Ian Hornby-Foster and Christopher J. A. Pugh interpreted results of experiments; Ian Hornby-Foster prepared figures; Ian Hornby-Foster drafted manuscript; Ian Hornby-Foster, Aimee L. Drane, Rachel Lord, Cory T. Richards, Freya M. Lodge, Zaheer Yousef, Michael Stembridge, Keeron Stone, Barry J. McDonnell and Christopher J. A. Pugh edited and revised manuscript. All authors approved the final version of manuscript and agree to be accountable for all aspects of the work in ensuring 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.

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CONFLICT OF INTEREST

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

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DATA AVAILABILITY STATEMENT

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

GENERATIVE AI STATEMENT

No AI tools were used in the conception, study design, data collection, analysis, interpretation of findings or manuscript writing.

ORCID

Ian Hornby-Foster  <https://orcid.org/0000-0001-5500-3215>

Cory T. Richards  <https://orcid.org/0000-0003-3605-7044>

Aimee L. Drane  <https://orcid.org/0000-0002-5208-917X>

Michael Stemberge  <https://orcid.org/0000-0003-0818-6420>

Rachel Lord  <https://orcid.org/0000-0002-5385-7548>

Keeron Stone  <https://orcid.org/0000-0001-6572-7874>

Barry J. McDonnell  <https://orcid.org/0000-0001-5912-1278>

Christopher J. A. Pugh  <https://orcid.org/0000-0002-5932-4793>

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APPENDIX

TABLE A1 Individual data points for the group means in Figure 1.

Subject	Figure 1a: %change in DD			Figure 1b: %change in SD			Figure 1c: %change in MD			Figure 1d: %change in PP			Figure 1e: %change in β_1		
	MID	END	REC	MID	END	REC	MID	END	REC	MID	END	REC	MID	END	REC
Untrained															
1	-0.18	-0.37	1.85	5.82	4.83	4.99	2.97	2.36	3.50	4.17	6.25	4.17	-40.03	-45.10	-26.42
2	-3.39	5.08	6.95	2.22	2.07	3.99	-0.39	3.48	5.37	2.56	2.56	-2.56	25.94	1.44	23.72
3	3.68	10.53	7.89	5.26	6.66	8.05	4.52	8.47	7.98	22.45	14.29	8.16	-13.46	19.63	-0.87
4	5.42	-3.67	-6.64	4.15	-1.38	-5.23	4.75	-2.45	-5.89	18.00	20.00	18.00	11.64	-14.99	-6.06
5	1.02	-3.39	-1.69	2.86	1.65	1.35	1.99	-0.72	-0.08	13.33	8.89	13.33	-16.42	-35.18	-22.48
6	2.36	2.69	5.89	1.03	1.03	0.15	1.65	1.81	2.83	13.79	10.34	3.45	3.81	38.86	81.70
7	-1.66	-2.16	-3.32	-3.17	-2.60	0.00	-2.47	-2.39	-1.54	-5.77	5.77	11.54	-2.39	1.35	-12.18
8	2.95	10.40	0.52	2.73	3.19	0.00	2.83	6.55	0.24	19.61	21.57	5.88	-0.38	69.42	-4.46
9	5.49	4.42	6.37	2.72	2.57	-2.57	3.99	3.42	1.55	16.00	12.00	4.00	32.33	19.82	134.11
10	9.90	6.77	-6.77	3.82	5.19	-7.02	6.66	5.93	-6.90	40.00	27.50	17.50	88.54	24.64	15.48
Resistance trained															
1	11.34	4.09	-1.86	15.75	7.63	1.95	13.69	5.98	0.17	5.71	2.86	0.00	-39.18	-23.05	-24.93
2	4.31	9.69	0.00	10.19	12.26	0.00	7.43	11.05	0.00	-6.15	-1.54	0.00	-44.02	-32.01	-11.15
3	3.16	-3.49	-1.00	7.74	-1.34	-1.64	5.58	-2.36	-1.34	4.26	0.00	0.00	-39.08	-30.82	2.67
4	-3.14	1.57	3.32	2.19	6.56	1.72	-0.33	4.20	2.47	6.67	-1.67	6.67	-45.82	-35.92	20.91
5	-9.52	-8.76	-7.68	-4.82	-0.99	-4.39	-7.07	-4.72	-5.97	13.46	9.62	3.85	-43.80	-53.10	-34.83
6	1.88	7.91	2.64	5.96	10.60	3.48	4.05	9.34	3.08	-11.90	-9.52	-2.38	33.70	-25.84	-9.90
7	15.19	14.20	10.26	14.99	19.05	10.05	15.08	16.76	10.15	4.76	0.00	2.38	18.58	-18.33	21.05
8	5.28	7.84	-2.56	4.01	9.81	-2.23	4.60	8.89	-2.38	22.22	11.11	14.81	10.20	-2.56	-1.15
9	11.82	6.08	2.30	8.46	7.03	0.72	10.03	6.58	1.45	19.51	17.07	12.20	32.32	-5.03	18.18
10	-1.42	1.59	-2.65	0.78	2.64	-2.95	-0.25	2.15	-2.81	3.70	7.41	5.56	-21.23	-10.05	-1.18

Note: Data are presented as percentage changes from baseline values for each individual included within the study sample.

Abbreviations: DD, diastolic diameter; MD, mean diameter; PP, pulse pressure; SD, systolic diameter; β_1 , β_1 -stiffness index.