European Journal of Applied Physiology
https://doi.org/10.1007/s00421-025-05948-2

ORIGINAL ARTICLE

Vascular function responses to resistance exercise followed by intermittent hypoxic exposure in untrained males

Witid Mitranun[1] · Chaiyawat Namboonlue[2] · Nattha Muangritdech[3] · Kampanart Paditsaeree[4]

Received: 5 May 2025 / Accepted: 15 August 2025

Abstract

Purpose This study investigated whether intermittent hypoxic exposure following resistance exercise mitigates acute vascular dysfunction. The main objective was to assess the effects of post-exercise hypoxia on flow-mediated dilation (FMD), blood pressure, and brachial-ankle pulse wave velocity (baPWV) in untrained males.

Methods Thirteen untrained male university students (age: 20.46 ± 0.87 years; body mass index: 23.6 ± 0.67 kg/m^2) participated in a crossover trial involving three experimental conditions: (1) resistance exercise followed by normoxia (RE, $FIO_2 = 21\%$), (2) resistance exercise followed by intermittent hypoxia (RE-H1, alternating $FIO_2 = 13.6\%$ and 21%), and (3) resistance exercise followed by mild intermittent hypoxia (RE-H2, alternating $FIO_2 = 15.8\%$ and 21%). Participants performed five sets of 10 leg extensions at 70% of their one-repetition maximum. FMD, blood pressure, and baPWV were measured at baseline and 0, 10, 20, 30, and 60 min post-exercise.

Results Post-exercise systolic blood pressure decreased in all conditions ($p < 0.05$), with no significant differences between conditions, while diastolic blood pressure showed no significant changes ($p > 0.05$). baPWV decreased in RE and RE-H2 ($p < 0.05$) but not in RE-H1, with RE showing greater reductions than RE-H1 ($p < 0.05$). Blood flow and shear rates increased in all conditions ($p < 0.05$), with greater responses in RE-H1 and RE-H2 ($p < 0.05$). FMD decreased after RE ($p < 0.05$) but was maintained in RE-H1 and RE-H2 for 60 min post-exercise. The %FMD/Δ shear rate remained consistently higher in RE-H1 and RE-H2 than in RE throughout the post-exercise period ($p < 0.05$).

Conclusion Intermittent hypoxia following resistance exercise appears to preserve endothelial function and prevent transient vascular impairment, suggesting its potential as a recovery-enhancing strategy.

Keywords Flow-mediated dilation · Arterial stiffness · Leg extension · Blood pressure · Intermittent hypoxia · Resistance exercise

Abbreviations

baPWV	Brachial-ankle pulse wave velocity
DBP	Diastolic blood pressure
FMD	Flow-mediated dilation
HIF	Hypoxia-inducible factor
IH	Intermittent hypoxia
IHT	Intermittent hypoxia training
NO	Nitric oxide
RE	Resistance exercise
RE-H1	Resistance exercise followed by intermittent hypoxia
RE-H2	Resistance exercise followed by mild intermittent hypoxia
1RM	One-repetition maximum
SBP	Systolic blood pressure

Communicated by Stephen Ives.

✉ Witid Mitranun
mitranunwitid@hotmail.com

[1] Department of Sports Science, Faculty of Physical Education, Sports, and Health, Srinakharinwirot University, Ongkharak, Nakhon Nayok 26120, Thailand

[2] Program of Sports and Exercise Science, Faculty of Science, Ubon Ratchathani Rajabhat University, 2 Ratchathani Road, Nai-Muang, Muang District, Ubon Ratchathani 34000, Thailand

[3] Cholangiocarcinoma Research Institute, Khon Kaen University, Khon Kaen 40002, Thailand

[4] Department of Physical Education, Faculty of Education, Phuket Rajabhat University, Phuket 83000, Thailand

Published online: 29 August 2025

Introduction

Endothelial dysfunction, a key marker of cardiovascular disease, is commonly assessed using flow-mediated dilation (FMD)—a noninvasive technique that measures nitric oxide (NO)-mediated vasodilation to evaluate endothelial function. FMD is an important predictor of cardiovascular risk. The acute effects of exercise on vascular function, particularly on FMD, have been extensively studied. Several factors influence FMD, including the type, intensity, and duration of exercise, as well as the individual's fitness level (Dawson et al. 2013). While resistance exercise is widely recommended for improving muscular strength and overall health, some studies have reported that a single session of resistance exercise can acutely reduce FMD, with reported reductions ranging from 1 to 5 percentage points, or approximately 10% to over 60% relative to baseline levels, especially in male individuals who are not regularly active (Buchanan et al. 2017; Mitranun 2016; Mitranun and Phongsri 2015; Morishima et al. 2018). Additionally, resistance exercise may acutely increase peripheral arterial stiffness, as assessed by brachial-ankle pulse wave velocity (baPWV) (Rangabprai et al. 2024). Persistently elevated baPWV values over the long term may indicate an increased risk of cardiovascular disease (Saz-Lara et al. 2021). A previous study reported that an immediate impairment in function was linked to a reduction in baseline vascular function following training, suggesting that transient vascular responses may play a pivotal role in initiating vascular adaptations (Dawson et al. 2018). This raises concerns about how to mitigate the acute endothelial dysfunction induced by resistance exercise.

Various strategies have been investigated to prevent acute vascular dysfunction following resistance exercise. Morishima et al. (2018) found that adopting a high-intensity, low-repetition training approach could counteract this effect. Similarly, Paditsaeree and Mitranun reported that combining elastic bands with weights helped prevent the acute decline in FMD and the acute elevation in baPWV, compared to using weights alone (Paditsaeree and Mitranun 2018, 2024). When the exercise program was not altered, Morishima et al. (2019) showed that 10 min of post-exercise cycling effectively preserved FMD. Notably, the decline in FMD is especially pronounced in overweight, physically inactive men (Harris et al. 2008). However, limited research has examined how to prevent acute endothelial dysfunction caused by resistance exercise without modifying the training program or incorporating additional exercise.

Hypoxia is known to induce acute arterial vasodilation (Rowell and Blackmon 1987). When aerobic exercise is performed under hypoxic conditions, NO production increases, leading to enhanced vasodilation (Casey et al. 2010). This suggests that hypoxia may improve FMD. Katayama et al. (2013) investigated 30 min of arm cycling at 60% VO_2 max under both normoxic and hypoxic conditions ($FIO_2 = 12–13\%$) and found that hypoxia attenuated the reduction in FMD. However, Katayama et al. (2016) reported conflicting results, showing no significant difference in FMD when leg cycling was performed under similar conditions. In the context of resistance exercise and hypoxic exposure, research suggests that combining hypoxia with low-load resistance training may promote muscle hypertrophy (Namboonlue et al. 2020). Clinically, intermittent hypoxia applied for a specific duration has been shown to enhance exercise performance and reduce arrhythmias in individuals with coronary artery disease and pulmonary conditions (Mallet et al. 2018). Despite these promising findings, the acute effects of resistance exercise followed by hypoxic exposure on vascular function remain unclear.

This study, therefore, aimed to investigate the effects of resistance exercise followed by intermittent hypoxic exposure on vascular function, addressing the key research question: Can post-exercise intermittent hypoxia mitigate acute vascular impairment induced by resistance training? This question was further guided by the dose–response concept proposed by Navarrete-Opazo and Mitchell (2014), who emphasized that an FIO_2 range of 9–16% represents a critical therapeutic window in which intermittent hypoxia is most likely to elicit beneficial effects. Accordingly, we investigated two hypoxic intensities within this range to explore potential differences in post-exercise vascular responses. Hypoxic conditions have been shown to lower long-term blood pressure in individuals with hypertension (Mukharliamov et al. 2006). Given that the acute decline in FMD following resistance exercise is closely linked to transient blood pressure elevation (Buchanan et al. 2017), it is plausible that intermittent hypoxia, when applied post-exercise, may counteract these adverse vascular effects. This study offers valuable insights into the potential vascular benefits of hypoxic exposure following resistance exercise.

Methods

Study design and population

This study employed a randomized crossover design. The sample size was based on previous studies that included 10 participants, which was sufficient to detect a 2% difference in FMD between exercise conditions (Atkinson et al. 2015; Birk et al. 2013). Based on an assumed standard deviation of 2% and a statistical power of 80%, 13 participants were recruited

to allow for potential dropouts. Thirteen male university students from Ubon Ratchathani Rajabhat University, aged 19–24 years with a body mass index of 23.0–24.9 kg/m^2, were enrolled in this study (Fig. 1). All participants resided in Ubon Ratchathani, Thailand (approximately 123 m above sea level), and had no exposure to chronic hypoxia. Strict inclusion criteria were applied to ensure both participant safety and study validity. Participants had to be in good health, non-smokers, and free from anabolic steroids, creatine, sympathoadrenal drugs, and dietary supplements. They also must not have stayed at altitudes above 1000 m within the past 3 months. Eligibility was assessed using the Physical Activity Readiness Questionnaire Plus, and only individuals who answered "No" to all questions were included. Participants were also required to be sedentary, which means engaging in structured exercise no more than once per week over the past 6 months. Further exclusion criteria included any history of cardiovascular, vascular, or respiratory disease; musculoskeletal, joint, or neurological disorders; recent bone surgery or fractures (within the past 3 months); and a history of seizures or fainting. All participants provided informed consent and voluntarily agreed to participate. Individuals who experienced symptoms such as dizziness, lightheadedness, chest pain, or tightness during exercise were excluded from the study, as these conditions could interfere with safe participation.

Study approval

This study was approved by the Human Research Ethics Committee of Srinakharinwirot University. It was conducted in accordance with established ethical guidelines, including the Declaration of Helsinki, the Belmont Report, and the International Conference on Harmonisation for Good Clinical Practice, and complied with relevant Thai laws and regulations. All participants provided informed consent prior to participation in the study.

Screening, initial instruction, and randomization

Participant enrollment took place at the Sports Science Laboratory of Ubon Ratchathani Rajabhat University. Prior to the intervention, all participants received a detailed explanation of the study's purpose, procedures, potential benefits, and associated risks. To ensure familiarity with the exercise protocol, participants attended two preliminary sessions. In the first session, participants practiced using the exercise equipment (Nautilus One™ S6LE, USA) until they were able to perform the bilateral leg extension correctly with a full range of motion—from 90° of knee flexion (starting position) to 0° of knee extension (end position)—across a range of light to heavier loads. Additionally, they became familiar with the testing environment, including the hypoxicator device, FMD measurement, and blood pressure and baPWV assessments. During the second session, the study protocol was repeatedly explained, and baseline assessments, including body mass and height, were conducted. A standardized warm-up was performed, consisting of 5 min of cycling at 80 W. This was followed by maximal strength testing to determine the one-repetition maximum (1RM) for the leg extension exercise. The 1RM was estimated using submaximal loads, with participants performing repetitions to failure using weights that allow for no more than 10 repetitions. To determine the appropriate load, participants performed 1–3 attempts, with 3–5 min of rest between attempts to ensure adequate recovery and maintain performance quality. The estimated 1RM was then calculated using the Brzycki equation (Brzycki 1988). Thereafter, participants became familiar with performing leg extensions at 70% of their 1RM, completing five sets of 10 repetitions with a 1-min rest between sets. The intraclass correlation coefficient (ICC) for the 1RM test ranged from 0.85 to 0.90. To reduce potential confounding variables, a Latin square design (LSD 3 × 3) was used to randomly assign the 13 participants to the three experimental conditions.

Experimental visit

After 1 week of familiarization, the third, fourth, and fifth experimental visits were conducted at the Sports Science Laboratory, where participants underwent a series of physiological assessments and exercise protocols. Blood pressure, baPWV (~ 2–3 min), and FMD (~ 7–8 min) were measured under consistent environmental conditions (temperature: ~ 23–25 °C). Participants were instructed to avoid physical exercise for 24 h before each session, refrain from eating for at least 2 h prior to testing, abstain from caffeine for 8 h, and ensure adequate sleep. To maintain consistency and ensure safety, all testing procedures were supervised by an exercise physiologist. A warm-up protocol was implemented, consisting of 15 repetitions of leg extensions at 20% of 1RM. Following this, participants performed resistance exercises under pre-assigned conditions.

- Resistance Exercise (RE) Condition: Participants performed bilateral leg extensions at 70% of their 1RM, completing five sets of 10 repetitions with a 1-min rest between sets. This exercise was conducted under normoxic conditions ($FIO_2 = 21\%$), equivalent to sea-level altitude.
- Resistance Exercise Followed by Hypoxic Condition 1 (RE-H1): Participants first completed the RE protocol, followed by intermittent hypoxic exposure using a hypoxicator device (Altitude Training Systems Hypoxic Unit, Australia). Exposure alternated between

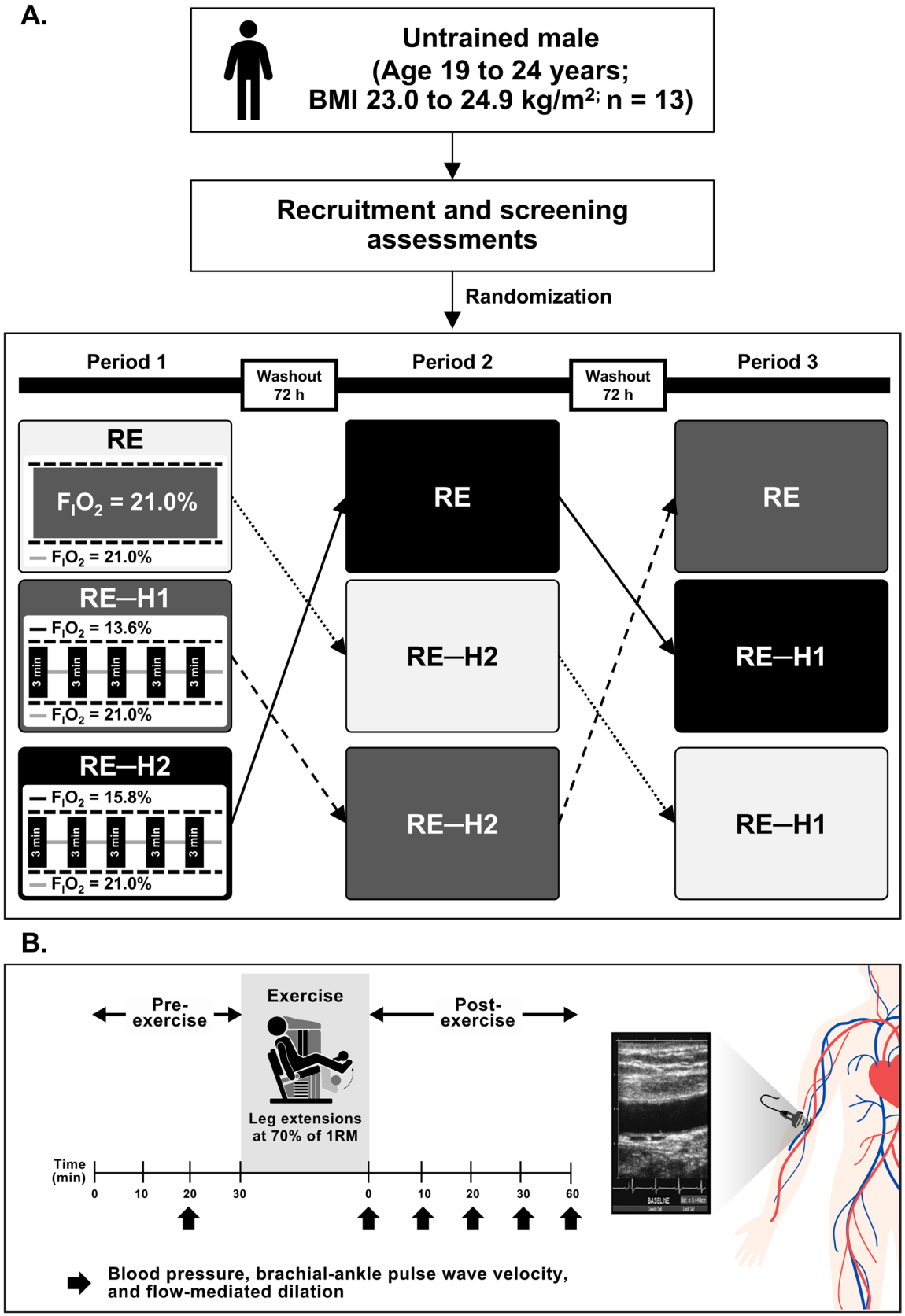

Fig. 1 Schematic overview of the crossover study design (**A**) and timeline for measurements of flow-mediated dilation, blood pressure, and brachial-ankle pulse wave velocity (**B**)

$FIO_2 = 13.6\%$ (~3,400 m above sea level) for 3 min and $FIO_2 = 21\%$ for 3 min.

- Resistance Exercise Followed by Hypoxic Condition 2 (RE-H2): This condition followed the RE-H1 protocol but used a slightly higher oxygen concentration ($FIO_2 = 15.8\%$), corresponding to an altitude of ~2240 m above sea level.

To maintain blinding, the hypoxicator device was worn by participants after all three exercise conditions. Oxygen saturation and heart rate were continuously monitored using a finger pulse oximeter (Beurer model: PO30) and recorded to ensure participant safety.

Post-exercise physiological assessments were conducted at 0, 10, 20, 30, and 60 min after resistance exercise, as illustrated in Fig. 1. To minimize potential bias, the outcome assessor—an exercise physiologist (Witid Mitranun), independent from the one managing the exercise sessions and hypoxic conditions (Chaiyawat Namboonlue)—was blinded to the experimental conditions. Additionally, to mitigate carry-over effects, a washout period of at least 3 days (72 h) was implemented between testing sessions before participants completed the remaining assigned conditions (Paditsaeree and Mitranun 2018, 2024). Before each subsequent condition, muscle soreness was assessed using a visual analog scale (VAS), and no participant reported moderate or severe discomfort (VAS < 3/10).

FMD measurement

FMD was assessed using previously established techniques (Corretti et al. 2002). After a 20-min rest period to establish baseline measurements, longitudinal images of the brachial artery at the antecubital fossa were obtained using ultrasound imaging (Vivid i-GE Healthcare, Cardiovascular Ultrasound System; GE Medical Systems). Following 1 min of baseline recording, a cuff was placed around the right forearm and inflated to 50 mmHg above the participant's resting systolic blood pressure. All participants were right-handed to minimize variability related to limb dominance. This pressure was maintained for 5 min before being released over the next 5 min, during which continuous data collection occurred. The recorded data were analyzed using the Brachial Analyzer Program (Medical Imaging Applications, Coralville, IA, USA). Shear rate was estimated as four times the mean blood velocity divided by the arterial diameter, excluding the influence of blood viscosity (Atkinson et al. 2013). Blood flow (mL/min) was calculated using the formula: $3.14 \times (\text{Diameter}/2)^2 \times \text{mean velocity} \times 60$, providing an estimate in milliliters per minute. FMD was calculated as the percentage change in arterial diameter from baseline to peak dilation. Owing to technical limitations of the ultrasound system, arterial diameter and blood velocity could not be measured simultaneously. Therefore, measurements were obtained sequentially within the same hyperemic period. In our laboratory setting, peak diameter was not consistently observed at 10–12 s post cuff release. Accordingly, normalization of FMD was performed using the relative change in shear rate, calculated as the difference between peak and basal shear rate (i.e., FMD divided by the change in shear rate [Δ shear rate]). All FMD measurements were conducted by an experienced operator with a record of collecting > 500 samples. In our laboratory, the ICC for FMD exceeded 0.90, indicating high measurement reliability.

Blood pressure and baPWV measurement

This assessment utilized a noninvasive vascular screening device (VP-1000 Plus; Omron Healthcare, Kyoto, Japan). Participants were positioned in a supine posture, while electrocardiogram and phonocardiogram recordings were obtained either along the left sternal border or near the second intercostal space. Cuffs with integrated electrocardiography electrodes were placed on both wrists, upper arms, and ankles. These cuffs were connected to a plethysmographic sensor to capture volume pulse waveforms and an oscillometric sensor for blood pressure measurements. Data collection spanned approximately 10 s of cardiac cycles, and the average baPWV from the right and left sides were analyzed. The ICC for baPWV measurements in our laboratory ranges from 0.88 to 0.90, indicating high reliability.

Statistical analysis

Each dependent variable under all conditions is expressed as the mean ± standard deviation (SD). The Shapiro–Wilk test was used to assess data normality. Two-way repeated measures ANOVA was used to assess the interaction and main effects of time (baseline, post-exercise [0 min], post-exercise [10 min], post-exercise [20 min], post-exercise [30 min], and post-exercise [60 min]) and condition (RE, RE-H1, and RE-H2), with corresponding F- and *p*-values reported. When necessary, the Bonferroni post hoc test was applied to identify significant pairwise differences. Mean differences and 95% confidence intervals (CI) were determined across conditions and at each time point. Partial eta-squared (ηp^2) was used to ascertain the effect size (ES) of the overall treatment. All statistical analyses were performed using IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, NY, USA), with significance set at $p < 0.05$.

Table 1 Demographic and physiological characteristics of study participants at baseline

Characteristics (n = 13, all male)	Mean	Standard deviation
Age, years	20.46	0.87
Body weight, kg	69.36	5.47
Height, m	1.71	0.06
Body mass index, kg/m^2	23.6	0.67
Resting heart rate, beats/min	72	18.72
Systolic blood pressure (SBP), mmHg	132.31	11.35
Diastolic blood pressure (DBP), mmHg	75.46	7.16
One-repetition maximum (1RM) – leg extension, kg	65.38	10.66

Results

This study included 13 participants; their characteristics and biological data are summarized in Table 1. Significant main effects of condition were observed for several vascular and hemodynamic parameters. Particularly, differences between groups were found in baPWV ($p = 0.011$, partial $\eta^2 = 0.222$), blood flow ($p < 0.001$, partial $\eta^2 = 0.835$), basal shear rate ($p < 0.001$, partial $\eta^2 = 0.874$), Δ shear rate ($p < 0.001$, partial $\eta^2 = 0.530$), and %FMD normalized to Δ shear rate ($p = 0.003$, partial $\eta^2 = 0.283$). These results indicate that vascular responses during the recovery period were significantly modulated by the specific post-exercise intervention applied in each condition (Not demonstrated in table/figure).

Systolic blood pressure (SBP) significantly decreased at 10, 20, 30, and 60 min post-exercise in the RE condition ($p < 0.05$), at 20 and 60 min in the RE-H1 condition ($p < 0.05$), and at 20, 30, and 60 min in the RE-H2 condition ($p < 0.05$), as shown in Fig. 2 and Table 2. No significant differences in SBP were observed between conditions at any time point ($p > 0.05$), as presented in Fig. 2 and Table 3. No significant changes in diastolic blood pressure were observed in any of the three conditions ($p > 0.05$) (Fig. 3, Tables 2, 3).

A reduction in baPWV was observed at 10, 20, 30, and 60 min post-exercise compared to baseline in the RE condition ($p < 0.05$), whereas the RE-H2 condition showed a significant decrease at 30 and 60 min post-exercise. No significant changes in baPWV were observed in the RE-H1 group ($p > 0.05$) (Fig. 4, Table 2). Significant differences between conditions were found at 0, 10, 20, 30, and 60 min post-exercise, with baPWV consistently lower in RE than in RE-H1 ($p < 0.05$) (Fig. 4, Table 3).

Basal diameter significantly increased at 10 and 20 min post-exercise in the RE condition ($p < 0.05$), at 0, 10, and 20 min in the RE-H1 condition ($p < 0.05$), and at 0, 10, 20, and 30 min post-exercise in the RE-H2 condition ($p < 0.05$). These time-dependent changes are presented in Table 2.

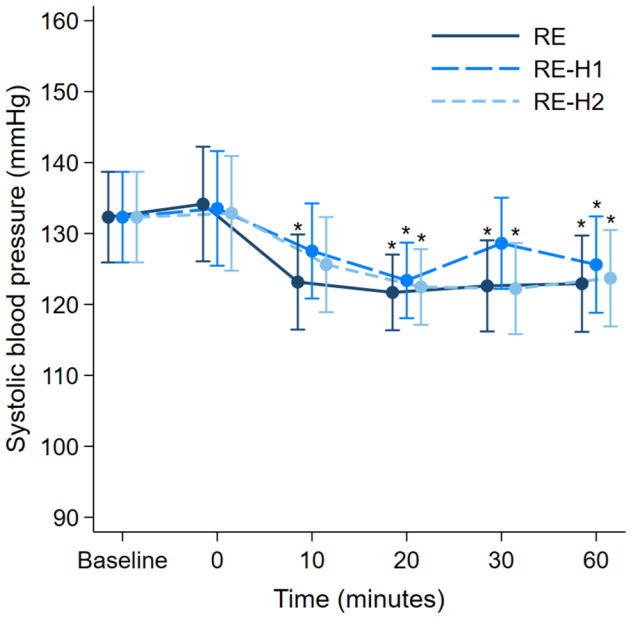

Fig. 2 Systolic blood pressure in the RE, RE-H1, and RE-H2 conditions. Data are presented as mean ± standard deviation. * Significant difference from baseline. † Significant difference between RE-H1 and RE. ‡ Significant difference between RE-H2 and RE

However, no significant differences were found among the three conditions ($p > 0.05$), as shown in Table 3.

Blood flow significantly increased at 0, 10, 20, and 30 min post-exercise in the RE condition ($p < 0.05$), and at 0, 10, 20, 30, and 60 min post-exercise in both the RE-H1 and RE-H2 conditions ($p < 0.05$). These time-dependent changes are presented in Table 2. Furthermore, blood flow was significantly higher in RE-H1 and RE-H2 compared to RE at 10, 20, 30, and 60 min post-exercise ($p < 0.05$), as shown in Table 3.

Basal shear rate significantly increased at 0, 10, and 20 min post-exercise in the RE condition ($p < 0.05$), and at 0, 10, 20, 30, and 60 min in both the RE-H1 and RE-H2 conditions ($p < 0.05$). These within-condition changes are presented in Table 2. Additionally, RE-H1 and RE-H2 demonstrated significantly higher basal shear rate compared to RE at 10, 20, 30, and 60 min post-exercise ($p < 0.05$), as shown in Table 3.

The Δ shear rate significantly increased at 0 and 10 min post-exercise in the RE condition ($p < 0.05$), and at 0, 10, 20, 30, and 60 min in the RE-H1 and RE-H2 conditions ($p < 0.05$), as presented in Table 2. Between-condition comparisons revealed that RE-H1 had significantly higher Δ shear rate than RE at 20, 30, and 60 min post-exercise ($p < 0.05$), and RE-H2 had significantly higher values than RE at 0, 20, and 30 min ($p < 0.05$), as shown in Table 3.

A significant reduction in FMD was observed at 0, 10, 20, and 60 min post-exercise in the RE condition. No

Table 2 Time-course changes in blood pressure and vascular responses within each condition

Outcomes	RE-H1			RE-H2			RE		
	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value
Systolic blood pressure (mmHg)									
Baseline	132.31 ± 11.35	Reference		132.31 ± 11.35	Reference		132.31 ± 11.35	Reference	
0 min	133.54 ± 17.00	1.23 (− 7.33, 9.79)	1.000	132.85 ± 9.82	0.54 (− 8.02, 9.10)	1.000	134.15 ± 15.30	1.85 (− 6.72, 10.41)	1.000
10 min	127.54 ± 15.00	− 4.77 (− 12.65, 3.11)	0.975	125.62 ± 8.57	− 6.69 (− 14.57, 1.19)	0.169	123.15 ± 11.31	− 9.15 (− 17.03, − 1.28)	0.012
20 min	123.38 ± 9.71	− 8.92 (− 15.27, − 2.57)	0.001	122.46 ± 7.45	− 9.85 (− 16.20, − 3.50)	< 0.001	121.69 ± 10.95	− 10.62 (− 16.97, − 4.27)	< 0.001
30 min	128.62 ± 12.51	− 3.69 (− 9.91, 2.53)	1.000	122.23 ± 8.99	− 10.08 (− 16.29, − 3.86)	< 0.001	122.62 ± 12.41	− 9.69 (− 15.91, − 3.48)	< 0.001
60 min	125.62 ± 12.90	− 6.69 (− 13.1, − 0.29)	0.034	123.69 ± 8.34	− 8.62 (− 15.02, − 2.21)	0.002	122.92 ± 14.20	− 9.39 (− 15.79, − 2.98)	0.001
Diastolic blood pressure (mmHg)									
Baseline	75.46 ± 7.16	Reference		75.46 ± 7.16	Reference		75.46 ± 7.16	Reference	
0 min	75.62 ± 11.15	0.15 (− 8.39, 8.70)	1.000	75.23 ± 6.58	− 0.23 (− 8.78, 8.32)	1.000	72.92 ± 10.36	− 2.54 (− 11.09, 6.01)	1.000
10 min	72.15 ± 7.58	− 3.31 (− 10.62, 4.00)	1.000	72.31 ± 6.68	− 3.15 (− 10.47, 4.16)	1.000	71.92 ± 8.53	− 3.54 (− 10.85, 3.77)	1.000
20 min	70.23 ± 7.44	− 5.23 (− 12.96, 2.50)	0.604	71.46 ± 5.36	− 4.00 (− 11.73, 3.73)	1.000	70.08 ± 8.66	− 5.39 (− 13.11, 2.34)	0.525
30 min	71.38 ± 8.16	− 4.08 (− 10.70, 2.55)	0.912	69.62 ± 7.26	− 5.85 (− 12.47, 0.78)	0.130	69.31 ± 7.12	− 6.15 (− 12.78, 0.47)	0.090
60 min	72.46 ± 8.65	− 3.00 (− 9.95, 3.95)	1.000	69.54 ± 7.53	− 5.92 (− 12.88, 1.03)	0.166	70.62 ± 10.24	− 4.85 (− 11.80, 2.11)	0.525
baPWV (cm/s)									
Baseline	1212.19 ± 94.25	Reference		1212.19 ± 94.25	Reference		1212.19 ± 94.25	Reference	
0 min	1265.27 ± 110.13	53.08 (− 50.48, 156.63)	1.000	1209.12 ± 92.28	− 3.08 (− 106.63, 100.48)	1.000	1141.08 ± 108.72	− 71.12 (− 174.67, 32.44)	0.564
10 min	1199.15 ± 112.68	− 13.04 (− 110.25, 84.17)	1.000	1136.31 ± 91.37	− 75.89 (− 173.09, 21.32)	0.286	1073.85 ± 79.08	− 138.35 (− 235.55, − 41.14)	0.001
20 min	1180.00 ± 87.39	− 32.19 (− 126.42, 62.03)	1.000	1119.73 ± 67.01	− 92.46 (− 186.69, 1.76)	0.058	1078.88 ± 81.50	− 133.31 (− 227.53, − 39.08)	0.001
30 min	1174.35 ± 89.77	− 37.85 (− 122.54, 46.85)	1.000	1111.12 ± 75.30	− 101.08 (− 185.77, − 16.38)	0.009	1062.46 ± 87.95	− 149.73 (− 234.42, − 65.04)	< 0.001
60 min	1168.23 ± 90.40	− 43.96 (− 135.19, 47.27)	1.000	1087.77 ± 99.74	− 124.42 (− 215.65, − 33.19)	0.002	1068.19 ± 91.18	− 144.00 (− 235.23, − 52.77)	< 0.001
Basal diameter (mm)									
Baseline	3.78 ± 0.13	Reference		3.78 ± 0.13	Reference		3.78 ± 0.13	Reference	
0 min	3.84 ± 0.14	0.06 (0.02, 0.10)	0.003	3.86 ± 0.13	0.08 (0.03, 0.12)	< 0.001	3.82 ± 0.13	0.04 (0.00, 0.09)	0.089
10 min	3.90 ± 0.11	0.12 (0.06, 0.18)	< 0.001	3.91 ± 0.09	0.13 (0.07, 0.19)	< 0.001	3.89 ± 0.12	0.11 (0.05, 0.17)	< 0.001
20 min	3.95 ± 0.10	0.17 (0.10, 0.24)	< 0.001	3.98 ± 0.10	0.20 (0.13, 0.27)	< 0.001	3.95 ± 0.10	0.17 (0.09, 0.24)	< 0.001

Table 2 (continued)

Outcomes	RE-H1			RE-H2			RE		
	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value
30 min	3.85 ± 0.06	0.07 (− 0.01, 0.15)	0.166	3.86 ± 0.06	0.08 (0.00, 0.16)	0.036	3.86 ± 0.10	0.08 (0.00, 0.16)	0.060
60 min	3.79 ± 0.10	0.01 (− 0.06, 0.08)	1.000	3.82 ± 0.07	0.04 (− 0.04, 0.10)	1.000	3.80 ± 0.10	0.02 (− 0.05, 0.09)	1.000
Blood flow (ml/min)									
Baseline	48.96 ± 3.79	Reference		48.96 ± 3.79	Reference		48.96 ± 3.79	Reference	
0 min	129.97 ± 11.9	81.01 (71.24, 90.78)	< 0.001	128.47 ± 12.89	79.51 (69.74, 89.28)	< 0.001	124.2 ± 10.62	75.23 (65.46, 85.00)	< 0.001
10 min	139.67 ± 14.16	90.71 (79.08, 102.33)	< 0.001	134.11 ± 18.28	85.15 (73.52, 96.77)	< 0.001	108.45 ± 8.99	59.49 (47.86, 71.11)	< 0.001
20 min	138.74 ± 12.03	89.78 (77.79, 101.77)	< 0.001	138.06 ± 20.89	89.09 (77.10, 101.09)	< 0.001	78.54 ± 7.66	29.58 (17.59, 41.57)	< 0.001
30 min	111.53 ± 19.27	62.57 (52.63, 72.51)	< 0.001	105.32 ± 5.17	56.36 (46.42, 66.29)	< 0.001	61.19 ± 5.16	12.22 (2.29, 22.16)	0.007
60 min	99.50 ± 15.49	50.54 (40.54, 60.54)	< 0.001	98.89 ± 8.21	49.93 (39.92, 59.93)	< 0.001	50.57 ± 4.01	1.60 (− 8.40, 11.61)	1.000
Basal shear rate (s^{-1})									
Baseline	77.02 ± 5.10	Reference		77.02 ± 5.10	Reference		77.02 ± 5.10	Reference	
0 min	195.53 ± 19.70	118.51 (103.34, 133.68)	< 0.001	190.35 ± 16.51	113.33 (98.16, 128.5)	< 0.001	189.5 ± 18.72	112.48 (97.32, 127.65)	< 0.001
10 min	199.88 ± 19.63	122.86 (105.42, 140.30)	< 0.001	189.96 ± 24.21	112.94 (95.50, 130.38)	< 0.001	156.66 ± 11.4	79.64 (62.20, 97.08)	< 0.001
20 min	190.61 ± 14.04	113.59 (98.97, 128.22)	< 0.001	185.50 ± 22.35	108.48 (93.86, 123.11)	< 0.001	108.75 ± 10.89	31.74 (17.11, 46.36)	< 0.001
30 min	166.05 ± 26.72	89.03 (75.09, 102.98)	< 0.001	155.22 ± 5.38	78.20 (64.26, 92.15)	< 0.001	90.62 ± 8.11	13.60 (− 0.35, 27.54)	0.062
60 min	155.37 ± 23.21	78.35 (64.54, 92.16)	< 0.001	151.29 ± 12.65	74.27 (60.46, 88.08)	< 0.001	78.14 ± 4.83	1.13 (− 12.68, 14.94)	1.000
Δ Shear rate (s^{-1})									
Baseline	432.06 ± 38.43	Reference		432.06 ± 38.43	Reference		432.06 ± 38.43	Reference	
0 min	347.67 ± 33.62	− 84.39 (− 132.16, − 36.62)	< 0.001	339.14 ± 52.91	− 92.93 (− 140.70, − 45.16)	< 0.001	382.25 ± 11.67	− 49.82 (− 97.59, − 2.05)	0.035
10 min	328.48 ± 35.29	− 103.59 (− 145.95, − 61.22)	< 0.001	338.80 ± 44.85	− 93.26 (− 135.63, − 50.90)	< 0.001	355.55 ± 36.12	− 76.52 (− 118.88, − 34.15)	< 0.001
20 min	301.15 ± 40.02	− 130.92 (− 182.95, − 78.89)	< 0.001	309.19 ± 55.37	− 122.87 (− 174.91, − 70.84)	< 0.001	391.83 ± 54.79	− 40.23 (− 92.26, 11.80)	0.302
30 min	325.57 ± 37.55	− 106.50 (− 148.73, − 64.26)	< 0.001	333.22 ± 47.97	− 98.84 (− 141.08, − 56.61)	< 0.001	393.71 ± 28.09	− 38.35 (− 80.59, 3.88)	0.107
60 min	345.98 ± 53.84	− 86.08 (− 129.58, − 42.58)	< 0.001	360.68 ± 34.87	− 71.39 (− 114.89, − 27.89)	< 0.001	420.31 ± 38.05	− 11.75 (− 55.25, 31.75)	1.000
Brachial artery FMD (%)									
Baseline	10.55 ± 2.04	Reference		10.55 ± 2.04	Reference		10.55 ± 2.04	Reference	
0 min	10.78 ± 2.23	0.23 (− 0.48, 0.93)	1.000	10.64 ± 2.10	0.09 (− 0.62, 0.79)	1.000	8.67 ± 1.39	− 1.88 (− 2.58, − 1.18)	< 0.001
10 min	11.00 ± 2.47	0.45 (− 0.31, 1.21)	1.000	10.73 ± 2.18	0.18 (− 0.58, 0.95)	1.000	8.94 ± 1.25	− 1.61 (− 2.37, − 0.85)	< 0.001

Table 2 (continued)

Outcomes	RE-H1			RE-H2			RE		
	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value	Mean ± SD	Mean difference from baseline (95% CI)	*p*-value
20 min	10.77 ± 2.42	0.22 (− 0.49, 0.93)	1.000	10.85 ± 2.08	0.30 (− 0.41, 1.01)	1.000	9.63 ± 1.96	− 0.92 (− 1.63, − 0.21)	0.004
30 min	10.69 ± 2.49	0.14 (− 0.62, 0.90)	1.000	10.95 ± 1.85	0.40 (− 0.36, 1.16)	1.000	9.79 ± 2.11	− 0.76 (− 1.52, 0.00)	0.051
60 min	10.8 ± 2.24	0.25 (− 0.47, 0.97)	1.000	10.96 ± 1.91	0.41 (− 0.31, 1.13)	1.000	9.78 ± 1.92	− 0.77 (− 1.49, − 0.05)	0.028
% FMD / Δ Shear rate (%·s)									
Baseline	0.025 ± 0.005	Reference		0.025 ± 0.005	Reference		0.025 ± 0.005	Reference	
0 min	0.031 ± 0.007	0.01 (0.003, 0.011)	< 0.001	0.032 ± 0.007	0.01 (0.003, 0.011)	< 0.001	0.023 ± 0.003	0.00 (− 0.006, 0.002)	1.000
10 min	0.034 ± 0.008	0.01 (0.005, 0.013)	< 0.001	0.032 ± 0.006	0.01 (0.003, 0.011)	< 0.001	0.025 ± 0.004	0.00 (− 0.003, 0.005)	1.000
20 min	0.036 ± 0.010	0.01 (0.006, 0.017)	< 0.001	0.036 ± 0.008	0.01 (0.006, 0.017)	< 0.001	0.025 ± 0.006	0.00 (− 0.005, 0.006)	1.000
30 min	0.034 ± 0.010	0.01 (0.004, 0.014)	< 0.001	0.034 ± 0.008	0.01 (0.004, 0.014)	< 0.001	0.025 ± 0.006	0.00 (− 0.004, 0.005)	1.000
60 min	0.032 ± 0.008	0.01 (0.003, 0.011)	< 0.001	0.031 ± 0.005	0.01 (0.002, 0.010)	0.001	0.023 ± 0.005	0.00 (− 0.005, 0.003)	1.000

baPWV, brachial-ankle pulse wave velocity; *FMD*, flow-mediated dilation; *CI*, confidence interval; *SD*, standard deviation

Table 3 Between-condition comparisons of blood pressure and vascular variables

Outcomes	RE-H1 vs RE			RE-H2 vs RE			RE-H1 vs RE-H2		
	Mean difference (95%CI)		*p*-value	Mean difference (95%CI)		*p*-value	Mean difference (95%CI)		*p*-value
Systolic blood pressure (mmHg)									
Baseline	0.00	(− 11.18, 11.18)	1.000	0.00	(− 11.18, 11.18)	1.000	0.00	(− 11.18, 11.18)	1.000
0 min	− 0.62	(− 14.77, 13.54)	1.000	− 1.31	(− 15.46, 12.85)	1.000	0.69	(− 13.46, 14.85)	1.000
10 min	4.38	(− 7.36, 16.13)	1.000	2.46	(− 9.28, 14.20)	1.000	1.92	(− 9.82, 13.66)	1.000
20 min	1.69	(− 7.64, 11.03)	1.000	0.77	(− 8.57, 10.11)	1.000	0.92	(− 8.41, 10.26)	1.000
30 min	6.00	(− 5.25, 17.25)	0.566	− 0.38	(− 11.63, 10.86)	1.000	6.38	(− 4.86, 17.63)	0.488
60 min	2.69	(− 9.20, 14.59)	1.000	0.77	(− 11.13, 12.66)	1.000	1.92	(− 9.97, 13.82)	1.000
Diastolic blood pressure (mmHg)									
Baseline	0.00	(− 7.05, 7.05)	1.000	0.00	(− 7.05, 7.05)	1.000	0.00	(− 7.05, 7.05)	1.000
0 min	2.69	(− 6.74, 12.12)	1.000	2.31	(− 7.12, 11.74)	1.000	0.38	(− 9.05, 9.81)	1.000
10 min	0.23	(− 7.29, 7.75)	1.000	0.38	(− 7.13, 7.90)	1.000	− 0.15	(− 7.67, 7.36)	1.000
20 min	0.15	(− 7.02, 7.33)	1.000	1.38	(− 5.79, 8.56)	1.000	− 1.23	(− 8.41, 5.94)	1.000
30 min	2.08	(− 5.34, 9.49)	1.000	0.31	(− 7.11, 7.72)	1.000	1.77	(− 5.65, 9.19)	1.000
60 min	1.85	(− 6.90, 10.59)	1.000	− 1.08	(− 9.82, 7.67)	1.000	2.92	(− 5.82, 11.67)	1.000
baPWV (cm/s)									
Baseline	0.00	(− 92.83, 92.83)	1.000	0.00	(− 92.83, 92.83)	1.000	0.00	(− 92.83, 92.83)	1.000
0 min	124.19	(21.73, 226.65)	0.013	68.04	(− 34.42, 170.50)	0.312	56.15	(− 46.30, 158.61)	0.532
10 min	125.31	(31.35, 219.26)	0.006	62.46	(− 31.49, 156.42)	0.311	62.85	(− 31.11, 156.80)	0.305
20 min	101.12	(23.21, 179.02)	0.007	40.85	(− 37.06, 118.75)	0.589	60.27	(− 17.64, 138.17)	0.180
30 min	111.89	(28.58, 195.19)	0.005	48.65	(− 34.65, 131.96)	0.454	63.23	(− 20.08, 146.54)	0.194
60 min	100.04	(7.59, 192.49)	0.030	19.58	(− 72.87, 112.03)	1.000	80.46	(− 11.99, 172.91)	0.106
Basal diameter (mm)									
Baseline	0.00	(− 0.13, 0.13)	1.000	0.00	(− 0.13, 0.13)	1.000	0.00	(− 0.13, 0.13)	1.000
0 min	0.02	(− 0.11, 0.15)	1.000	0.03	(− 0.09, 0.16)	1.000	− 0.02	(− 0.15, 0.11)	1.000
10 min	0.01	(− 0.09, 0.12)	1.000	0.02	(− 0.08, 0.13)	1.000	− 0.01	(− 0.12, 0.09)	1.000
20 min	0.01	(− 0.09, 0.11)	1.000	0.03	(− 0.07, 0.14)	1.000	− 0.03	(− 0.13, 0.07)	1.000
30 min	− 0.01	(− 0.08, 0.06)	1.000	0.00	(− 0.07, 0.08)	1.000	− 0.01	(− 0.09, 0.06)	1.000
60 min	− 0.01	(− 0.10, 0.08)	1.000	0.01	(− 0.08, 0.10)	1.000	− 0.03	(− 0.12, 0.06)	1.000
Blood flow (ml/min)									
Baseline	0.00	(− 3.73, 3.73)	1.000	0.00	(− 3.73, 3.73)	1.000	0.00	(− 3.73, 3.73)	1.000
0 min	5.78	(− 5.88, 17.43)	0.665	4.28	(− 7.38, 15.94)	1.000	1.50	(− 10.16, 13.16)	1.000
10 min	31.22	(17.11, 45.33)	< 0.001	25.66	(11.55, 39.77)	< 0.001	5.56	(− 8.55, 19.67)	0.987
20 min	60.20	(45.82, 74.59)	< 0.001	59.52	(45.13, 73.9)	< 0.001	0.69	(− 13.70, 15.07)	1.000
30 min	50.35	(38.63, 62.06)	< 0.001	44.13	(32.41, 55.85)	< 0.001	6.21	(− 5.50, 17.93)	0.574
60 min	48.94	(38.72, 59.16)	< 0.001	48.32	(38.10, 58.55)	< 0.001	0.61	(− 9.61, 10.84)	1.000
Basal shear rate (s^{-1})									
Baseline	0.00	(− 5.02, 5.02)	1.000	0.00	(− 5.02, 5.02)	1.000	0.00	(− 5.02, 5.02)	1.000
0 min	6.02	(− 12.06, 24.11)	1.000	0.85	(− 17.24, 18.93)	1.000	5.18	(− 12.91, 23.26)	1.000
10 min	43.22	(24.34, 62.09)	< 0.001	33.30	(14.42, 52.17)	< 0.001	9.92	(− 8.95, 28.79)	0.586
20 min	81.86	(65.62, 98.10)	< 0.001	76.75	(60.51, 92.98)	< 0.001	5.11	(− 11.12, 21.35)	1.000
30 min	75.43	(59.26, 91.60)	< 0.001	64.60	(48.43, 80.77)	< 0.001	10.83	(− 5.34, 27.00)	0.304
60 min	77.22	(61.94, 92.50)	< 0.001	73.14	(57.86, 88.42)	< 0.001	4.08	(− 11.20, 19.36)	1.000
Δ Shear rate (s^{-1})									
Baseline	0.00	(− 37.85, 37.85)	1.000	0.00	(− 37.85, 37.85)	1.000	0.00	(− 37.85, 37.85)	1.000
0 min	− 34.57	(− 70.83, 1.69)	0.066	− 43.11	(− 79.37, − 6.85)	0.015	8.54	(− 27.72, 44.80)	1.000
10 min	− 27.07	(− 65.47, 11.34)	0.256	− 16.75	(− 55.15, 21.66)	0.842	− 10.32	(− 48.72, 28.08)	1.000
20 min	− 90.69	(− 140.48, − 40.89)	< 0.001	− 82.64	(− 132.44, − 32.84)	< 0.001	− 8.04	(− 57.84, 41.75)	1.000
30 min	− 68.15	(− 106.29, − 30.00)	< 0.001	− 60.49	(− 98.64, − 22.35)	< 0.001	− 7.65	(− 45.80, 30.49)	1.000

Table 3 (continued)

Outcomes	RE-H1 vs RE		RE-H2 vs RE		RE-H1 vs RE-H2	
	Mean difference (95%CI)	*p*-value	Mean difference (95%CI)	*p*-value	Mean difference (95%CI)	*p*-value
60 min	− 74.33 (− 116.74, − − 31.92)	< 0.001	− 59.63 (− 102.04, − 17.22)	0.003	− 14.69 (− 57.10, 27.72)	1.000
Brachial artery FMD (%)						
Baseline	0.00 (− 2.01, 2.01)	1.000	0.00 (− 2.01, 2.01)	1.000	0.00 (− 2.01, 2.01)	1.000
0 min	2.11 (0.19, 4.02)	0.027	1.97 (0.05, 3.88)	0.042	0.14 (− 1.77, 2.05)	1.000
10 min	2.06 (0.05, 4.06)	0.042	1.79 (− 0.21, 3.79)	0.092	0.26 (− 1.74, 2.26)	1.000
20 min	1.14 (− 0.99, 3.27)	0.558	1.22 (− 0.91, 3.35)	0.477	− 0.08 (− 2.21, 2.05)	1.000
30 min	0.90 (− 1.23, 3.03)	0.888	1.16 (− 0.98, 3.29)	0.545	− 0.26 (− 2.39, 1.88)	1.000
60 min	1.02 (− 0.98, 3.02)	0.627	1.18 (− 0.82, 3.18)	0.441	− 0.16 (− 2.16, 1.84)	1.000
% FMD/Δ Shear rate (%·s)						
Baseline	0.000 (− 0.005, 0.005)	1.000	0.000 (− 0.005, 0.005)	1.000	0.000 (− 0.005, 0.005)	1.000
0 min	0.009 (0.003, 0.015)	0.002	0.009 (0.003, 0.015)	0.001	− 0.001 (− 0.006, 0.005)	1.000
10 min	0.008 (0.002, 0.014)	0.004	0.006 (0.001, 0.012)	0.033	0.002 (− 0.004, 0.008)	1.000
20 min	0.012 (0.003, 0.020)	0.004	0.011 (0.003, 0.019)	0.007	0.001 (− 0.008, 0.009)	1.000
30 min	0.009 (0.001, 0.017)	0.038	0.008 (0.001, 0.017)	0.043	0.000 (− 0.008, 0.008)	1.000
60 min	0.008 (0.002, 0.014)	0.003	0.007 (0.001, 0.013)	0.013	0.001 (− 0.005, 0.007)	1.000

baPWV, Brachial-ankle pulse wave velocity; *FMD*, Flow-mediated dilation; *CI*, confidence interval; *SD*, standard deviation

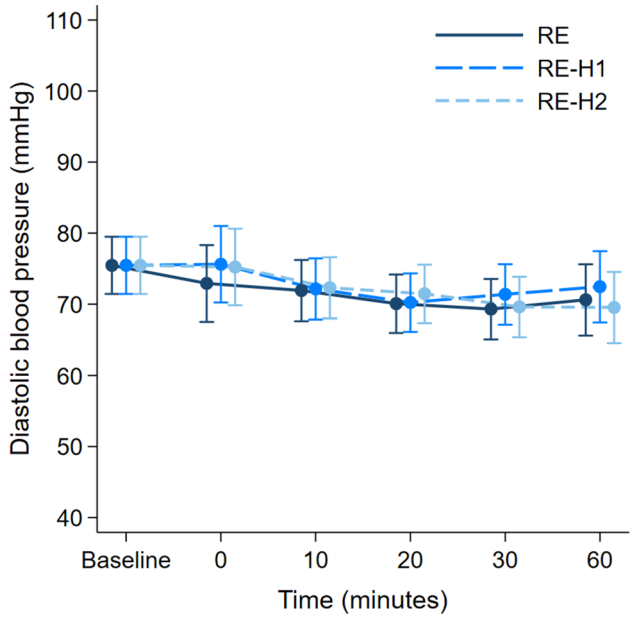

Fig. 3 Diastolic blood pressure in the RE, RE-H1, and RE-H2 conditions. Data are presented as mean ± standard deviation

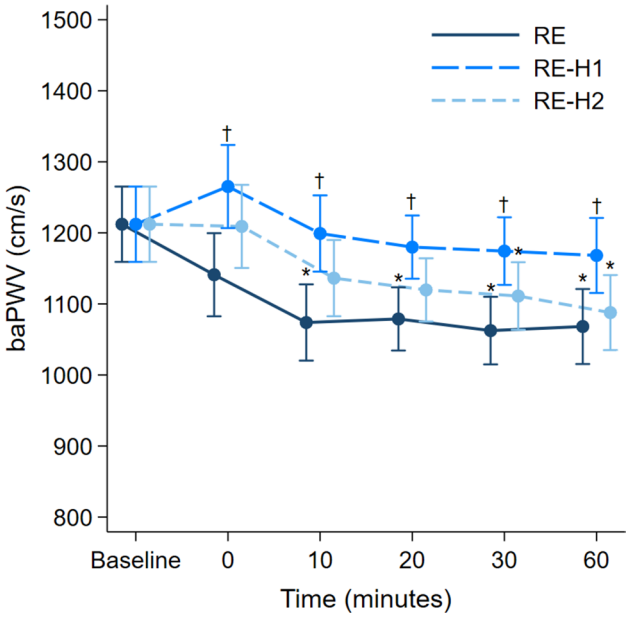

Fig. 4 Brachial-ankle pulse wave velocity (baPWV) in the RE, RE-H1, and RE-H2 conditions. Data are presented as mean ± standard deviation. * Significant difference from baseline. † Significant difference between RE-H1 and RE

significant changes in FMD were observed in either the RE-H1 or RE-H2 conditions ($p > 0.05$) (Fig. 5, Table 2). Between conditions, RE-H1 showed significantly higher values than RE at 0 and 10 min post-exercise, while RE-H2 was significantly higher than RE at 0 min post-exercise ($p < 0.05$), as presented in Fig. 5 and Table 3.

A significant increment in FMD/ Δ shear rate was observed from 0, 10, 20, 30, and 60 min post-exercise in the RE-H1 and RE-H2 conditions. No significant changes were observed in RE ($p > 0.05$) (Fig. 6, Table 2). Significant differences between conditions were noted throughout the 0–60 min post-exercise period, with FMD/ Δ shear rate consistently higher in RE-H1 and RE-H2 than RE ($p < 0.05$) (Fig. 6, Table 3).

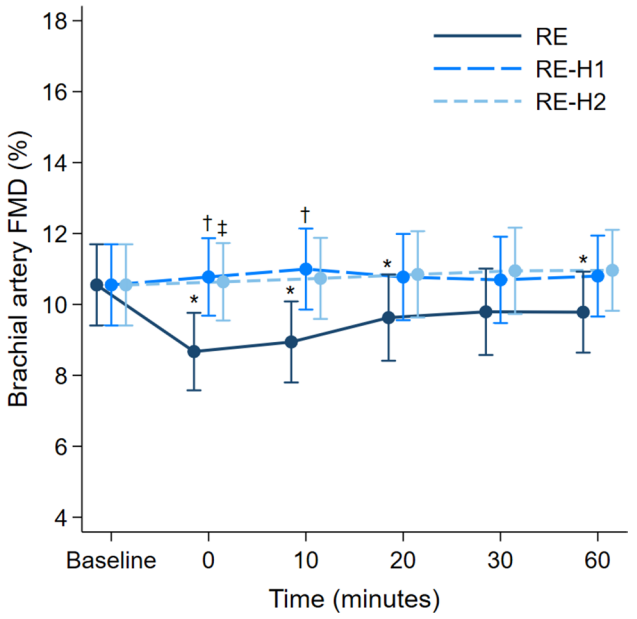

Fig. 5 Flow-mediated dilation in the RE, RE-H1, and RE-H2 conditions. Data are presented as mean ± standard deviation. * Significant difference from baseline. † Significant difference between RE-H1 and RE. ‡ Significant difference between RE-H2 and RE

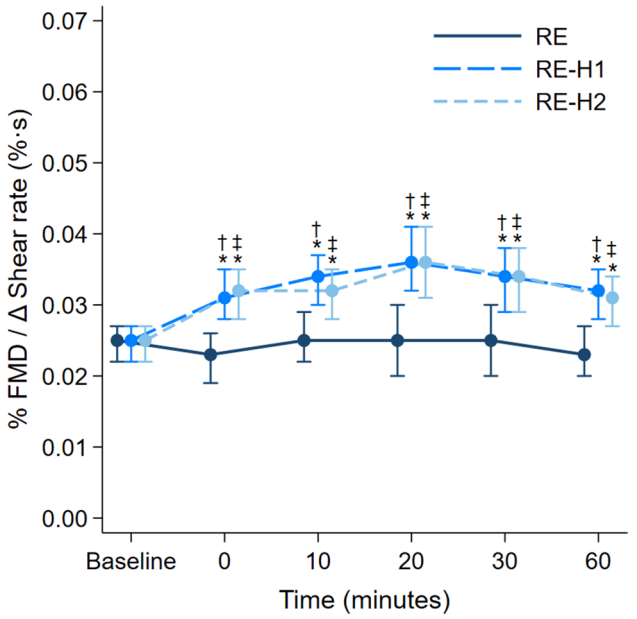

Fig. 6 Percentage flow-mediated dilation/Δ shear rate in the RE, RE-H1, and RE-H2 conditions. Data are presented as mean ± standard deviation. * Significant difference from baseline. † Significant difference between RE-H1 and RE. ‡ Significant difference between RE-H2 and RE

Discussion

The primary findings of this study indicate that resistance exercise acutely impairs brachial FMD, resulting in a temporary reduction in vascular function lasting up to 60 min post-exercise. However, exposure to intermittent hypoxia following exercise—alternating between reduced oxygen levels ($FIO_2 = 13.6\%$ or 15.8%) and normoxia ($FIO_2 = 21\%$)—appears to mitigate this impairment throughout the recovery period. These results suggest that intermittent hypoxia may serve as a protective strategy for maintaining vascular function after resistance exercise.

Previous studies have reported that resistance exercise may acutely reduce FMD at 0 min post-exercise (Mitranun 2016; Mitranun and Phongsri 2015; Paditsaeree and Mitranun 2018). This temporary vascular dysfunction may stem from exercise-induced elevations in blood pressure, which alter vascular tone and activate the sympathetic nervous system, ultimately promoting vasoconstriction (Pratley et al. 1994). Sustained increases in blood pressure have been linked to reduced arterial elasticity, decreased elastin levels, and suppressed NO release (Bilfinger and Stefano 2000; London and Guerin 1999). Furthermore, acute blood pressure spikes have been shown to impair endothelium-dependent vasodilation in both normotensive individuals and those with hypertension (Millgård and Lind 1998). However, some studies have found no change or improvements in FMD following resistance exercise (Mitranun 2016; Mitranun and Phongsri 2015), potentially due to variations in exercise intensity. In this study, participants performed five sets of 10 repetitions of leg extensions at 70% of their 1RM—an intensity likely sufficient to induce a sustained reduction in FMD up to 60 min post-exercise.

Intermittent hypoxia training (IHT) protocols may significantly influence endothelial function. Katuntsev et al. (2021) demonstrated that a 3-week daily IHT regimen enhanced endothelium-dependent vasodilation in muscular-type arteries among healthy individuals. This improvement was accompanied by elevated erythropoietin levels, stimulated erythropoiesis, and increased red blood cell and hemoglobin concentrations. The observed improvements in endothelial function during adaptation to intermittent hypoxia are likely mediated by the activation of hypoxia-inducible transcription factors—particularly HIF-1α—which help establish a broad molecular framework that enhances endothelial cell function and promotes NO production (Burtscher et al. 2024; Muangritdech et al. 2020).

When combined with exercise, hypoxic conditions may further enhance vascular adaptations. A meta-analysis by Montero and Lundby (2016) examined cycling endurance

training performed under normobaric hypoxia or normoxia over 3–10 weeks. Despite similar training intensities, exercise under hypoxic conditions yielded additional benefits, such as increased muscle capillarization and improved vascular function, without adversely affecting arterial stiffness. In terms of acute effects, a single session of exercise performed under hypoxia has been shown to result in greater FMD compared to the same exercise under normoxia (Katayama et al. 2013). Our study demonstrated that intermittent hypoxic exposure following resistance exercise in both RE-H1 and RE-H2 may counteract the FMD impairment observed from 0 to 60 min post-exercise after a single bout of resistance training. Previous research suggests that the reduction in FMD following resistance exercise may be partially attributed to elevated SBP (Buchanan et al. 2017). However, in our study, SBP remained lower than baseline across all conditions (RE, RE-H1, and RE-H2). This suggests that the protective effect of hypoxic conditions against FMD impairment is unlikely to be mediated solely by reductions in SBP. Instead, the preservation of endothelial function may be linked to increased NO production. Previous research has suggested that hypoxic exposure can attenuate reductions in brachial artery FMD following acute inactivity, potentially via increased total shear rate stimulus from baseline (Hanson et al. 2022). In our study, basal shear rate following resistance exercise was elevated under hypoxic conditions (RE-H1 and RE-H2) compared to normoxia. This increased shear rate may have contributed to the preservation of endothelial function post-exercise. Moreover, when FMD was normalized to shear rate, values remained higher in the hypoxic conditions, suggesting that acute hypoxic exposure may enhance endothelial responsiveness. This effect may be mediated, at least in part, by increased NO bioavailability. These findings underscore the potential benefits of hypoxic exposure for enhancing endothelium-dependent vasodilation.

baPWV is a well-established indicator of arterial stiffness, with elevated values potentially signifying an increased risk of cardiovascular disease (Saz-Lara et al. 2021). While long-term exercise has been shown to reduce baPWV, the acute effects of resistance exercise on this measure remain inconclusive. Resistance exercise at 75% of 1RM to failure has been reported to cause an immediate reduction in baPWV, with effects lasting up to 30 min post-exercise in young adults (DeVan et al. 2005). In contrast, a recent study in older adults observed an increase in baPWV at 0 min post-exercise compared to baseline (Rangabprai et al. 2024). These discrepancies may be influenced by age-related vascular changes, which aligns with the findings of the present study. In our study involving young adults, baPWV decreased relative to baseline following resistance exercise in the RE condition. However, baPWV remained relatively unchanged in both RE-H1 and RE-H2 conditions, with a modest reduction at 30 min post-exercise in RE-H2. Although resistance exercise reduced arterial stiffness as indicated by baPWV, hypoxic conditions in both RE-H1 and RE-H2 did not acutely increase vascular stiffness. Instead, intermittent hypoxia appeared to preserve baPWV during the recovery period. Interestingly, our findings show a divergence between baPWV and FMD responses; while intermittent hypoxia mitigated the FMD impairment, it did not significantly alter arterial stiffness. This pattern is consistent with previous research on Pilates training under hypoxic conditions, which reported greater acute improvements in FMD compared to normoxic conditions, with no change in baPWV (Jung et al. 2020). These findings underscore the importance of further investigation into the chronic effects of hypoxic exposure on arterial stiffness following resistance exercise.

A key strength of our study lies in the repeated measurement of both FMD and baPWV at multiple time points, providing a comprehensive view of vascular responses. However, several limitations must be acknowledged. First, the sample size was relatively small, which may limit the generalizability of the results. Second, the absence of blood chemistry data, such as NO concentrations and markers of smooth muscle function, restricts our ability to explore the underlying mechanisms in greater detail.

Conclusion

This study demonstrated that resistance exercise acutely impairs brachial FMD, resulting in a temporary decline in vascular function lasting up to 60 min post-exercise. However, intermittent hypoxic exposure following resistance training effectively attenuated this impairment throughout the recovery phase. Incorporating intermittent hypoxia into post-resistance exercise protocols may optimize recovery strategies in untrained individuals by mitigating post-exercise vascular dysfunction—an adaptation that could prove beneficial for both short-term recovery and long-term vascular health. Nevertheless, long-term interventional studies are necessary to validate these findings and explore their applicability to broader populations.

Author contribution Witid Mitranun, Chaiyawat Namboonlue, and Nattha Muangritdech developed the study concept and design, analyzed the data, and wrote and edited the article. Kampanart Paditsaeree analyzed the data and wrote and edited the article.

Funding This study was financially supported by the Faculty of Physical Education, Sports, and Health at Srinakharinwirot University, Thailand, under Grant Number 230/2565.

Data availability The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Conflict of interest The authors have no competing interests to declare that are relevant to the content of this article.

Ethical approval This study was approved by the Human Research Ethics Committee of Srinakharinwirot University. It was conducted in accordance with established ethical guidelines, including the Declaration of Helsinki, the Belmont Report, and the International Conference on Harmonisation for Good Clinical Practice, and complied with relevant Thai laws and regulations.

Informed consent All participants provided informed consent prior to participation in the study.

References

Atkinson CL, Carter HH, Dawson EA, Naylor LH, Thijssen DHJ, Green DJ (2015) Impact of handgrip exercise intensity on brachial artery flow-mediated dilation. Eur J Appl Physiol 115:1705–1713. https://doi.org/10.1007/s00421-015-3157-1

Atkinson G, Batterham AM, Thijssen DH, Green DJ (2013) A new approach to improve the specificity of flow-mediated dilation for indicating endothelial function in cardiovascular research. J Hypertens 31:287–291. https://doi.org/10.1097/hjh.0b013e32835b8164

Bilfinger TV, Stefano GB (2000) Human aortocoronary grafts and nitric oxide release: relationship to pulsatile pressure. Ann Thorac Surg 69:480–485. https://doi.org/10.1016/s0003-4975(99)01083-8

Birk GK, Dawson EA, Batterham AM, Atkinson G, Cable T, Thijssen DHJ, Green DJ (2013) Effects of exercise intensity on flow mediated dilation in healthy humans. Int J Sports Med 34:409–414. https://doi.org/10.1055/s-0032-1323829

Brzycki M (1988) A practical approach to strength training. Master Press, Illinois

Buchanan CE, Kadlec AO, Hoch AZ, Gutterman DD, Durand MJ (2017) Hypertension during weight lifting reduces flow-mediated dilation in nonathletes. Med Sci Sports Exerc 49:669–675. https://doi.org/10.1249/MSS.0000000000001150

Burtscher J, Citherlet T, Camacho-Cardenosa A, Camacho-Cardenosa M, Raberin A, Krumm B, Hohenauer E, Egg M, Lichtblau M, Müller J, Rybnikova EA, Gatterer H, Debevec T, Baillieul S, Manferdelli G, Behrendt T, Schega L, Ehrenreich H, Millet GP, Gassmann M, Schwarzer C, Glazachev O, Girard O, Lalande S, Hamlin M, Samaja M, Hüfner K, Burtscher M, Panza G, Mallet RT (2024) Mechanisms underlying the health benefits of intermittent hypoxia conditioning. J Physiol 602:5757–5783. https://doi.org/10.1113/JP285230

Casey DP, Madery BD, Curry TB, Eisenach JH, Wilkins BW, Joyner MJ (2010) Nitric oxide contributes to the augmented vasodilatation during hypoxic exercise. J Physiol 588:373–385. https://doi.org/10.1113/jphysiol.2009.180489

Corretti MC, Anderson TJ, Benjamin EJ, Celermajer D, Charbonneau F, Creager MA, Deanfield J, Drexler H, Gerhard-Herman M, Herrington D, Vallance P, Vita J, Vogel R, International Brachial Artery Reactivity Task Force (2002) Guidelines for the ultrasound assessment of endothelial-dependent flow-mediated vasodilation of the brachial artery: a report of the International Brachial Artery Reactivity Task Force. J Am Coll Cardiol 39:257–265. https://doi.org/10.1016/s0735-1097(01)01746-6

Dawson EA, Cable NT, Green DJ, Thijssen DHJ (2018) Do acute effects of exercise on vascular function predict adaptation to training? Eur J Appl Physiol 118:523–530. https://doi.org/10.1007/s00421-017-3724-8

Dawson EA, Green DJ, Cable NT, Thijssen DHJ (2013) Effects of acute exercise on flow-mediated dilatation in healthy humans. J Appl Physiol (1985) 115:1589–1598. https://doi.org/10.1152/japplphysiol.00450.2013

DeVan AE, Anton MM, Cook JN, Neidre DB, Cortez-Cooper MY, Tanaka H (2005) Acute effects of resistance exercise on arterial compliance. J Appl Physiol 98(6):2287–2291. https://doi.org/10.1152/japplphysiol.00002.2005

Hanson BE, Iwamoto E, Mouser BL, Miller KA, Casey DP (2022) Hypoxia offsets the decline in brachial artery flow-mediated dilation after acute inactivity. Am J Physiol Regul Integr Comp Physiol 323:R787–R796. https://doi.org/10.1152/ajpregu.00161.2022

Harris RA, Padilla J, Hanlon KP, Rink LD, Wallace JP (2008) The flow-mediated dilation response to acute exercise in overweight active and inactive men. Obesity (Silver Spring) 16:578–584. https://doi.org/10.1038/oby.2007.87

Jung K, Seo J, Jung WS, Kim J, Park HY, Lim K (2020) Effects of an acute Pilates program under hypoxic conditions on vascular endothelial function in Pilates participants: a randomized crossover trial. Int J Environ Res Public Health 17:2584. https://doi.org/10.3390/ijerph17072584

Katayama K, Fujita O, Iemitsu M, Kawano H, Iwamoto E, Saito M, Ishida K (2013) The effect of acute exercise in hypoxia on flow-mediated vasodilation. Eur J Appl Physiol 113:349–357. https://doi.org/10.1007/s00421-012-2442-5

Katayama K, Yamashita S, Iwamoto E, Ishida K (2016) Flow-mediated dilation in the inactive limb following acute hypoxic exercise. Clin Physiol Funct Imaging 36:60–69. https://doi.org/10.1111/cpf.12194

Katuntsev VP, Baranov MV, Zakharov SY, Suchostavtseva TV, Puchkova AA, Stavrovskaya DM (2021) Adaptation to intermittent hypoxia: influence on the state of endothelial function. Hum Physiol 47:306–312. https://doi.org/10.1134/S0362119721030063

London GM, Guerin AP (1999) Influence of arterial pulse and reflected waves on blood pressure and cardiac function. Am Heart J 138:220–224. https://doi.org/10.1016/s0002-8703(99)70313-3

Mallet RT, Manukhina EB, Ruelas SS, Caffrey JL, Downey HF (2018) Cardioprotection by intermittent hypoxia conditioning: evidence, mechanisms, and therapeutic potential. Am J Physiol Heart Circ Physiol 315:H216–H232. https://doi.org/10.1152/ajpheart.00060.2018

Millgård J, Lind L (1998) Acute hypertension impairs endothelium-dependent vasodilatation. Clin Sci (Lond) 94:601–607. https://doi.org/10.1042/cs0940601

Mitranun W (2016) The acute effects of short and long durations of plank training on endothelial function. Songklanakarin J Sci Technol 38:691–697

Mitranun W, Phongsri K (2015) The acute effects on endothelial function in the different abdominal training postures. Songklanakarin J Sci Technol 37:545–551

Montero D, Lundby C (2016) Effects of exercise training in hypoxia versus normoxia on vascular health. Sports Med 46:1725–1736. https://doi.org/10.1007/s40279-016-0570-5

Morishima T, Iemitsu M, Ochi E (2019) Short-term cycling restores endothelial dysfunction after resistance exercise. Scand J Med Sci Sports 29:1115–1120. https://doi.org/10.1111/sms.13434

Morishima T, Tsuchiya Y, Iemitsu M, Ochi E (2018) High-intensity resistance exercise with low repetitions maintains endothelial function. Am J Physiol Heart Circ Physiol 315:H681–H686. https://doi.org/10.1152/ajpheart.00281.2018

Muangritdech N, Hamlin MJ, Sawanyawisuth K, Prajumwongs P, Saengjan W, Wonnabussapawich P, Manimmanakorn N, Manimmanakorn A (2020) Hypoxic training improves blood pressure, nitric oxide and hypoxia-inducible factor-1 alpha in hypertensive patients. Eur J Appl Physiol 120:1815–1826. https://doi.org/10.1007/s00421-020-04410-9

Mukharliamov FI, Smirnova MI, Bedritskiĭ SA, Liadov KV (2006) Interval hypoxic training in arterial hypertension. Vopr Kurortol Fizioter Lech Fiz Kult 2:5–6

Namboonlue C, Hamlin MJ, Sirasaporn P, Manimmanakorn N, Wonnabussapawich P, Thuwakum W, Sumethanurakkhakun W, Manimmanakorn A (2020) Optimal degree of hypoxia combined with low-load resistance training for muscle strength and thickness in athletes. J Phys Educ Sport 20:828–838. https://doi.org/10.7752/jpes.2020.02119

Navarrete-Opazo A, Mitchell GS (2014) Therapeutic potential of intermittent hypoxia: a matter of dose. Am J Physiol Regul Integr Comp Physiol 307:R1181–R1197. https://doi.org/10.1152/ajpregu.00208.2014

Paditsaeree K, Mitranun W (2018) Does combining elastic and weight resistance acutely protect against the impairment of flow-mediated dilatation in untrained men? Artery Res 23:1–8. https://doi.org/10.1016/j.artres.2018.05.003

Paditsaeree K, Mitranun W (2024) Acute effects of combining weight and elastic resistance exercise on vascular function in older adults. Geriatrics 9:56. https://doi.org/10.3390/geriatrics9030056

Pratley R, Nicklas B, Rubin M, Miller J, Smith A, Smith M, Hurley B, Goldberg A (1994) Strength training increases resting metabolic rate and norepinephrine levels in healthy 50- to 65-yr-old men. J Appl Physiol (1985) 76:133–137. https://doi.org/10.1152/jappl.1994.76.1.133

Rangabprai Y, Mitranun W, Mitarnun W (2024) Effect of 60-min single bout of resistance exercise, reformer Pilates, on vascular function parameters in older adults: a randomized crossover study. Gerontology 70:764–775. https://doi.org/10.1159/000539144

Rowell LB, Blackmon JR (1987) Human cardiovascular adjustments to acute hypoxaemia. Clin Physiol 7:349–376. https://doi.org/10.1111/j.1475-097x.1987.tb00179.x

Saz-Lara A, Cavero-Redondo I, Álvarez-Bueno C, Notario-Pacheco B, Ruiz-Grao MC, Martínez-Vizcaíno V (2021) The acute effect of exercise on arterial stiffness in healthy subjects: a meta-analysis. J Clin Med 10:291. https://doi.org/10.3390/jcm10020291