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The acute and chronic effects of high-intensity exercise in hypoxia on blood pressure and post-exercise hypotension

A randomized cross-over trial

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Abstract

Background: Acute exercise leads to an immediate drop in blood pressure (BP), also called post-exercise hypotension (PEH). Exercise in hypoxia is related to additional vasodilation, potentially contributing to more profound PEH. Therefore, we investigated the impact of hypoxia versus normoxia on the magnitude of PEH. Second, we examined whether these changes in PEH relate to the BP-lowering effects of 12-week exercise training under hypoxia.

Methods: In this prospective study, 21 healthy individuals (age 22.2 ± 3.0 years, 14 male) performed a 45-minute high-intensity running exercise on 2 different days in a random order, under hypoxia (fraction of inspired oxygen 14.5%) and normoxia (fraction of inspired oxygen 20.9%). BP was examined pre-exercise ($t=0$) and at $t=15$, $t=30$, $t=45$, and $t=60$ minutes post-exercise. Afterward, subjects took part in a 12-week hypoxic running exercise training program. Resting BP was measured before and after the 12-week training program.

Results: Acute exercise induced a significant decrease in systolic BP (systolic blood pressure [SBP], $P=.001$), but not in diastolic BP (diastolic blood pressure [DBP], $P=.113$). No significant differences were observed in post-exercise BP between hypoxic and normoxic conditions (SBP, $P=.324$ and DBP, $P=.204$). Post-exercise changes in SBP, DBP, and mean arterial pressure significantly correlated to the 12-week exercise training-induced changes in SBP ($r=0.557$, $P=.001$), DBP ($r=0.615$, $P<.001$), and mean arterial pressure ($r=0.458$, $P=.011$).

Conclusion: Our findings show that hypoxia does not alter the magnitude of PEH in healthy individuals, whilst PEH relates to the BP-lowering effects of exercise. These data highlight the strong link between acute and chronic changes in BP.

Abbreviations: AMS = acute mountain sickness, BMI = body mass index, BP = blood pressure, CO = cardiac output, CO_2 = carbon dioxide, CPET = cardiopulmonary exercise test, DBP = Diastolic blood pressure, FiO_2 = fraction of inspired oxygen, HR = heart rate, LLS = Lake Louis score, MAP = mean arterial pressure, O_2 = oxygen, PEH = post-exercise hypotension, RPE = rate of perceived exertion, SBP = systolic blood pressure, SpO_2 = oxygen consumption, SV = Stroke volume, TPR = total peripheral resistance.

Keywords: antihypertensive treatment, blood pressure, cardiovascular disease, exercise training, hypoxia, post-exercise hypotension

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The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

None of the content has been published or presented somewhere.

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1. Introduction

Post-exercise hypotension (PEH) is a reduction in systolic and/or diastolic arterial blood pressure (BP) below resting BP levels following a single bout of exercise and is usually observed minutes to hours after exercise.[1–3] The decline in BP after exercise relates to a marked decrease in total peripheral resistance (TPR),[3,4] due to sustained post-exercise local vasodilator mechanisms,[5,6] with unmatched elevations in cardiac output (CO).[7] The potential clinical relevance of PEH is that the magnitude of PEH relates to the BP-lowering effect of exercise training.[8–10] Enhancing the magnitude of PEH may, therefore, have the potential to increase the anti-hypertensive effects of exercise training.

Previous research revealed that several factors, including exercise intensity,[11–13] duration,[13,14] mode (interval or continuous[15]), time of day[16] and body position,[17] may influence the magnitude of PEH. Relatively little work has examined the impact of hypoxia on PEH. This is relevant since previous work revealed that hypoxia contributes to a higher decrease in TPR post-exercise[18–20] potentially contributing to a larger PEH. Indeed, 1 previous study found a more profound PEH in response to resistance exercise under hypoxia versus normoxia.[21] Therefore, hypoxia may elicit a larger magnitude of PEH compared to normoxia. This is potentially relevant, since acute changes in BP with (hypoxic) exercise may relate to long-term changes in resting BP after regular exercise training.[8–10] A larger PEH in hypoxia may therefore, translate into a larger decrease in resting BP, as previously suggested for normoxic exercise.[9] The aim of this study was to investigate the influence of hypoxia versus normoxia on the PEH magnitude of post-endurance exercise (high-intensity) in healthy individuals, and whether the magnitude of PEH relates to the reduction in BP after a 12-week hypoxic endurance exercise-training program.

According to previous research, we hypothesized that high-intensity endurance exercise under hypoxia would elicit greater reductions in post-exercise BP compared to normoxia, and that the magnitude of PEH would relate to the training-induced BP reduction.

2. Methods

2.1. Study population

Twenty-one healthy normotensive individuals (14 males) were recruited for the study. Participants were eligible to take part in this study if they were able to run on a treadmill and did not train for more than 2 hours a week at moderate-to-high intensity for the last 6 months. Exclusion criteria were a body mass index <18 or $>30\,kg/m^2$, a possibility of pregnancy, personal history of cardiovascular disease, positive family history of cardiovascular death (<55 years), exercise-limiting respiratory disease and physical (i.e. musculoskeletal) complaints making completion of the 12-week training program impossible.

The procedures were in accordance with institutional guidelines and conformed to the Declaration of Helsinki. The study was approved by the Ethics Research Committee of the Liverpool John Moores University (18/SPS/065). Participants gave full written and verbal informed consent before participation.

2.2. Study design

In this prospective randomized cross-over study, participants attended the laboratory on 36 separate occasions divided into 4 parts, see Figure 1. During the first visit, baseline measurements were performed. Visits 2 and 3 included the actual test days to study the acute effects of hypoxia versus normoxia on PEH. Visits 4 to 35 (training program) and visit 36 (follow-up measurements) comprised the chronic part to study; the relationship between PEH during the first visit versus the long-term changes in BP.

2.2.1. Baseline and follow-up measurements. The measurements included determination of height (SECA stadiometer, SECA GmbH, Germany), weight (SECA scale, SECA GmbH), oxygen saturation (SpO_2, pulse oximetry; Ana Pulse 100, Ana Wiz Ltd., UK) and maximal oxygen consumption (VO_2max). Resting heart rate (HR, Polar, Kempele, Finland) and resting BP (Dinamap V100, GE Medical, Norway) were determined at the end of 10 minutes of quiet rest in a supine position. Resting HR was averaged over 1 minute of continuous recording. Resting BP determination involved 3 serial measurements from the right arm taken 30 seconds apart. Cuff size was adjusted to arm circumference. A standardized maximal cardiopulmonary exercise test for VO_2max assessment was conducted on a motorized treadmill (HP Cosmos, Nussdorf, Germany) after a 10-minutes warm-up and familiarisation. The test started at a speed of 7 km/h for 3 minutes followed by speed increments of 1 km/h every minute until subjects' volitional exhaustion. Careful calibration of flow sensors and gas analysers was performed before each measurement according to the manufacturer's instructions (Oxycon pro, CareFusion, VS). VO_2max was defined as the highest value of a 30-s average,[22] and attainment was verified according to previous recommend criteria.[23]

2.2.2. Test days. Figure 1 gives an overview of the test days described below. Participants were randomly allocated to 1 of 2 groups in a counterbalanced design and blinded for the order of testing days. One test day was performed at normoxia (sea level, equivalent to fraction of inspired oxygen [FiO_2] 20.9%)) and the other test day at normobaric hypoxia (3000 m simulated altitude, equivalent to FiO_2 14.5%), separated by at least 48 hours and maximal 72 hours of rest. Participants were subjected to 30 minutes of acclimation in seated position followed by 45-minute of high-intensity endurance running exercise on a motorized treadmill (HP Cosmos) and 60 minutes of recovery in seated position. Exercise intensity was set by using 85% of maximal HR for both hypoxia and normoxia sessions. HR, SpO_2, and BP measurements were performed at the end of acclimation (baseline) and at 15, 30, 45, and 60 minutes during post-exercise recovery in the seated position. HR was averaged over 1 minute of continuous recording. BP determination involved 3 serial measurements from the right arm taken 30 seconds apart. To assess PEH, post-exercise BP measurements were averaged to calculate the decline in BP from baseline. Participants remained in a seated upright position with back support and BP measurements were obtained using an appropriately sized cuff. HR was measured continuously throughout (Polar), and rate of perceived exertion (RPE) was monitored during exercise.[24] Echocardiography (Vivid E9; GE Medical; Horten, Norway) was performed at baseline and at 60 minutes of recovery to obtain cardiac hemodynamic parameters (stroke volume, CO). Estimated TPR (TPR_{est}) was calculated from the echocardiography-derived estimate of CO and mean arterial pressure (MAP) at baseline and at 60 minutes of recovery ($TPR_{est} = MAP/CO$). Measurements were performed at the same time at both days to control for diurnal variation, and fluid intake was controlled by providing the same amount of water to participants during both testing days.

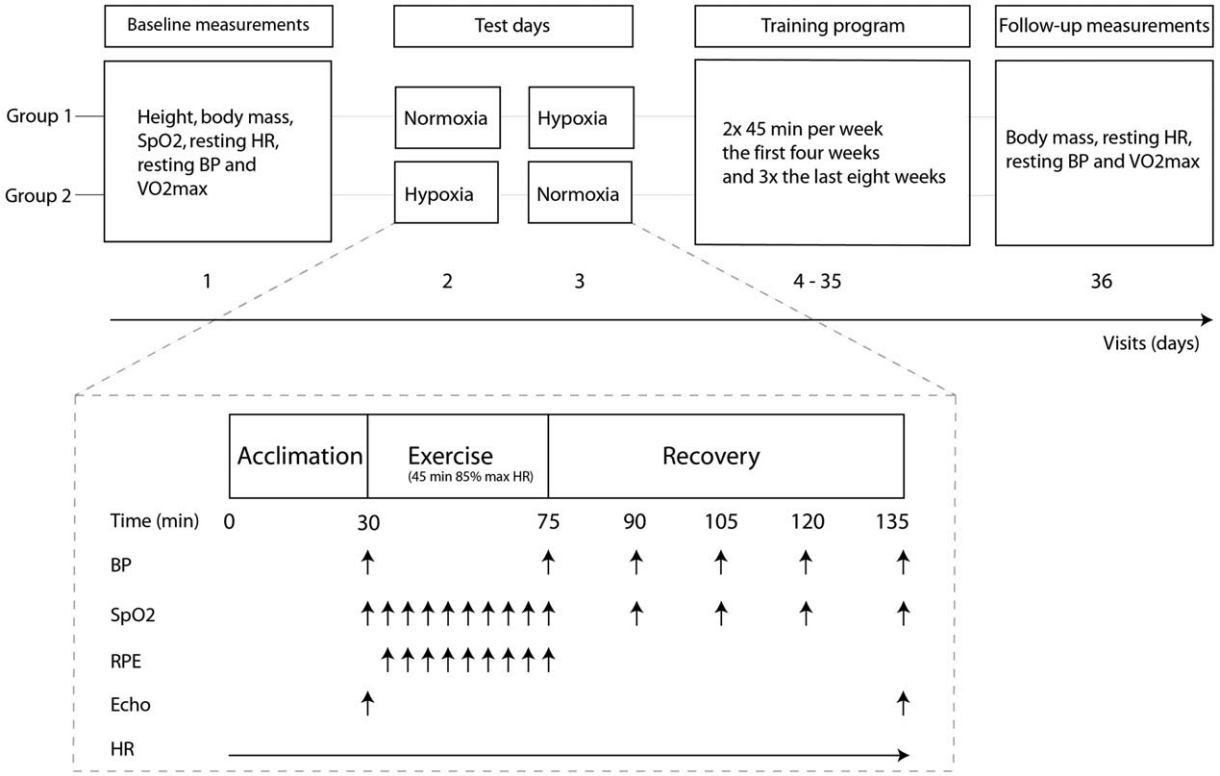

Figure 1. Overview of study design, where the dotted panel is highlighting visit 2 and 3 (test days).

2.2.3. Training program. Following the test days, subjects took part in a 12-week normobaric hypoxic exercise endurance-training program consisting of 2 × 45 minute sessions a week in the first 4 weeks and 3 × 45 minute sessions in the last 8 weeks. This running exercise was performed on a motorized treadmill at 3000 m simulated altitude (equivalent to FiO_2 14.5%) at high-intensity (85% of maximal HR).

2.2.4. Environmental chamber and safety. All exercise tests and training sessions were conducted in an environmental chamber (TISS, Alton, UK; Sportingedge, Bastingstoke, UK), which was set-up by a qualified technician. Normobaric hypoxia was achieved by a nitrogen dilution technique. Ambient temperature, carbon dioxide (CO_2), and oxygen (O_2) levels were controlled in all sessions (20°C; FiO_2 14.5%; CO_2 0.03%), whilst a Servomex gas analysis system (Servomex MiniMP 5200, Servomex Group Ltd., UK) was used inside the chamber to provide the researcher continuous information regarding O_2 and CO_2 levels. Acute mountain sickness symptoms (AMS, measured by Lake Louise score[2,5]) were monitored during testing and training sessions every 20 minutes. The subject was removed from the environmental chamber if oxygen saturation levels dropped below 80% or severe acute mountain sickness was suspected (Lake Louis score ≥6).

2.3. Statistical analysis

Statistical analysis was performed using SPSS Statistics 24 (SPSS Inc., Chicago, IL, VS). All parameters were visually inspected for normality and tested with Shapiro–Wilk normality tests. Categorical variables were presented as proportions and continuous variables were reported as mean ± standard deviation, unless indicated otherwise. A 2-way repeated measures analysis of variance was conducted to compare

(1) pre- and post-exercise training data and
(2) conditions.

A Greenhouse-Geisser correction was used for estimating *P*-values if the sphericity assumption was violated ($P<0.05$, tested with Mauchly test). A Sidak post-hoc correction was used to account for multiple testing.

Associations between acute PEH and chronic BP lowering effects were analysed by Pearson correlation coefficient and compared using Fisher *Z*-transform, in which acute is defined as the BP response to a single bout of high intensity hypoxic exercise and chronic as the change in post-acclimated resting BP following a 12-week training program. An alpha level of $P \leq 0.05$ was accepted a priori for significance.

3. Results

Participants were aged 22.2 ± 3.0 years, had a body mass of 69.5 ± 10.7 kg, a VO_2max/kg of 52.4 ± 8.1 mL/min/kg, and were all normotensive (<140/90 mm Hg). All participants were non-smokers. Baseline characteristics are shown in Table 1. Fifteen of the 21 included participants completed the chronic part of the study (Table 2).

3.1. Post-exercise BP response in normoxia and hypoxia (acute study)

HR during exercise was matched in exercise sessions in normoxia and in hypoxia (173 ± 7 bpm, 172 ± 7 bpm respectively, $P=.23$).

Table 1

Subject characteristics: baseline.

Sex (m/f)	14/7
Age (yr)	22.2 ± 3.0
Height (cm)	170.3 ± 10.4
Body mass (kg)	69.5 ± 10.7
BMI (kg/m^2)	24.0 ± 2.7
BSA (kg)	1.81 ± 0.18
Resting HR (bpm)	65 ± 8
Resting SBP (mm Hg)	119 ± 5
Resting DBP (mm Hg)	69 ± 8
Resting MAP (mm Hg)	85 ± 6
SpO2 (%)	98.4 ± 1.2
VO_2max (L/min)	3.6 ± 0.7
VO2max/kg (mL/min/kg)	52.4 ± 8.1
VE (L/min)	138 ± 28
Hrmax (bpm)	199 ± 8

Data are expressed as means ± SD.

BMI = body mass index, BSA = body surface area, DBP = diastolic blood pressure, f = female, HR = heart rate, m = male, MAP = mean arterial pressure, SBP = systolic blood pressure, SpO2 = oxygen saturation, VE = ventilation, VO_2max = maximal oxygen uptake.

Table 2

Subject characteristics: baseline and post-training program.

	Pre	Post	*P*-value
Sex (m/f)	10/5		
Age (yr)	22.0 ± 2.4		
Height (cm)	172 ± 11		
Body mass (kg)	71.2 ± 11.7	70.3 ± 12.3	.17
BMI (kg/m^2)	24.0 ± 3.0	23.6 ± 2.7	.14
BSA (kg)	1.84 ± 0.19	1.83 ± 0.20	.18
Resting HR (bpm)	77 ± 10	66 ± 6	**<.001**
Resting SBP (mm Hg)	118 ± 4	113 ± 9	**.02**
Resting DBP (mm Hg)	67 ± 8	63 ± 5	.07
Resting MAP (mm Hg)	84 ± 6	80 ± 6	**.03**
VO2max (L/min)	3.7 ± 0.7	3.9 ± 0.8	**<.001**
VO2max/kg (mL/min/kg)	52.1 ± 7.1	55.7 ± 7.3	**<.001**
VE (L/min)	138 ± 29	145 ± 34	**.002**
Hrmax (bpm)	199 ± 8	195 ± 6	**.008**

Data are expressed as means ± SD.

BMI = body mass index, BSA = body surface area, DBP = diastolic blood pressure, f = female, HR = heart rate, m = male, MAP = mean arterial pressure, SBP = systolic blood pressure, VE = ventilation, VO_2max = maximal oxygen uptake.

Body mass loss (hypoxia –410 ± 320 g vs normoxia –410 ± 199 g, $P = .99$) and water intake (hypoxia 373 ± 228 mL vs normoxia 336 ± 196 mL, $P = .24$) during exercise did not differ between both testing sessions. Mean distance covered during exercise was significantly higher in normoxia (6655 ± 1266 m) compared to hypoxia (5797 ± 1112 m, $P < .001$), whilst there was no significant difference in subjective ratings of perceived exertion (RPE normoxia 12.5 ± 1.3, RPE hypoxia 13.3 ± 1.5; $P = .07$). Stroke volume was significantly decreased during recovery ($P < .01$), whilst this decline did not differ between hypoxia and normoxia ($P = .54$) (Table 3). Echocardiography showed a significantly higher CO during hypoxia compared to normoxia ($P < .01$), whilst no differences were found between rest and post-exercise ($P = .09$) (Table 3). TPR_{est} was significantly lower during hypoxia compared to normoxia ($P < .01$), whilst no difference was found between baseline and recovery ($P = .83$).

SBP and MAP significantly decreased over time during recovery ($P < .01$), while DBP did not change ($P = .11$) (Fig. 2 and Table 3). The mean PEH response for SBP, DBP, and MAP in normoxia were –2.6 ± 8.5, –2.5 ± 5.0, and –2.6 ± 4.9 mm Hg respectively, and in hypoxia –6.2 ± 8.4, –1.9 ± 5.4, and –3.4 ± 5.4, respectively. SBP, DBP, and MAP did not differ between conditions at any time point (all $P > .05$) (Fig. 2 and Table 3). For all BP responses, there were no significant interactions between condition and time (all $P > .05$). Similar findings were observed when post-exercise BP responses were presented as relative changes (data not shown).

3.2. Correlation of acute and chronic BP response (chronic study)

During the prospective intervention part of our study, 6 participants dropped-out (motivational issues n = 4; health problems unrelated to the study n = 2). Participants completed on average 30 ± 2 training sessions (94% adherence) at an average 83.5% of their maximum HR. These 15 participants showed a significant increase in VO_2max/kg (52.1–55.7 mL/min/kg, $P < .001$) (Table 2). Resting SBP, MAP, and resting HR significantly decreased (118–113 mm Hg, 84–80 mm Hg, and 78–66 bpm, respectively, $P < .05$) (Table 2). Resting DBP did not significantly change (67–63 mm Hg, $P = .067$) (Table 2). Pooled data derived from the experiments under normoxia and hypoxia indicate that the magnitude of PEH significantly correlated with the decrease in BP after 12-week of exercise training for DBP, SBP, and MAP (Fig. 3). When comparing data derived under normoxia versus hypoxia, no significant differences were observed in the correlation between PEH and resting BP (Fisher *Z*: SBP, $P = .22$; DBP, $P = .35$; MAP, $P = .65$).

Table 3

Pre- and post-exercise values of physiological parameters.

	Pre		15 min		30 min		45 min		60 min		*P*-value		
	Normoxia	Hypoxia	Normoxia	Hypoxia	Normoxia	Hypoxia	Normoxia	Hypoxia	Normoxia	Hypoxia	C	T	C*T
HR (bpm)	65 ± 8	69 ± 6	98 ± 12	101 ± 9	90 ± 113	95 ± 9	86 ± 12	92 ± 9	80 ± 12	87 ± 9	.002	<.001	.08
SpO2 (%)	98 ± 1	89 ± 2	97 ± 1	89 ± 3	97 ± 1	89 ± 3	97 ± 1	90 ± 3	98 ± 2	90 ± 3	<.001	.001	.55
SBP (mm Hg)	121 ± 10	124 ± 10	121 ± 10	120 ± 9	119 ± 9	118 ± 9	117 ± 9	117 ± 8	118 ± 9	118 ± 10	.93	<.001	.32
DBP (mm Hg)	70 ± 8	70 ± 8	66 ± 8	68 ± 8	67 ± 9	69 ± 7	67 ± 9	68 ± 7	69 ± 9	67 ± 7	.53	.11	.20
MAP (mm Hg)	87 ± 6	88 ± 7	85 ± 8	85 ± 7	85 ± 8	85 ± 7	84 ± 8	85 ± 7	85 ± 8	84 ± 7	.61	.004	.41
LVSV (mL)	70 ± 20	73 ± 18							64 ± 18	64 ± 18	.33	<.001	.54
LVCO (L/min)	4.6 ± 1.5	5.1 ± 1.3							4.4 ± 1.3	4.8 ± 1.4	.002	.09	.89
TPR_{est}	20.9 ± 6.7	18.3 ± 4.5							21.0 ± 6.7	18.5 ± 4.7	.003	.83	.96

Data are expressed as means ± SD.

C = condition, CO = cardiac output, DBP = diastolic blood pressure, HR = heart rate, MAP = mean arterial pressure, SBP = systolic blood pressure, SpO2 = oxygen saturation, SV = stroke volume, T = time, TPR_{est} = estimated total peripheral resistance.

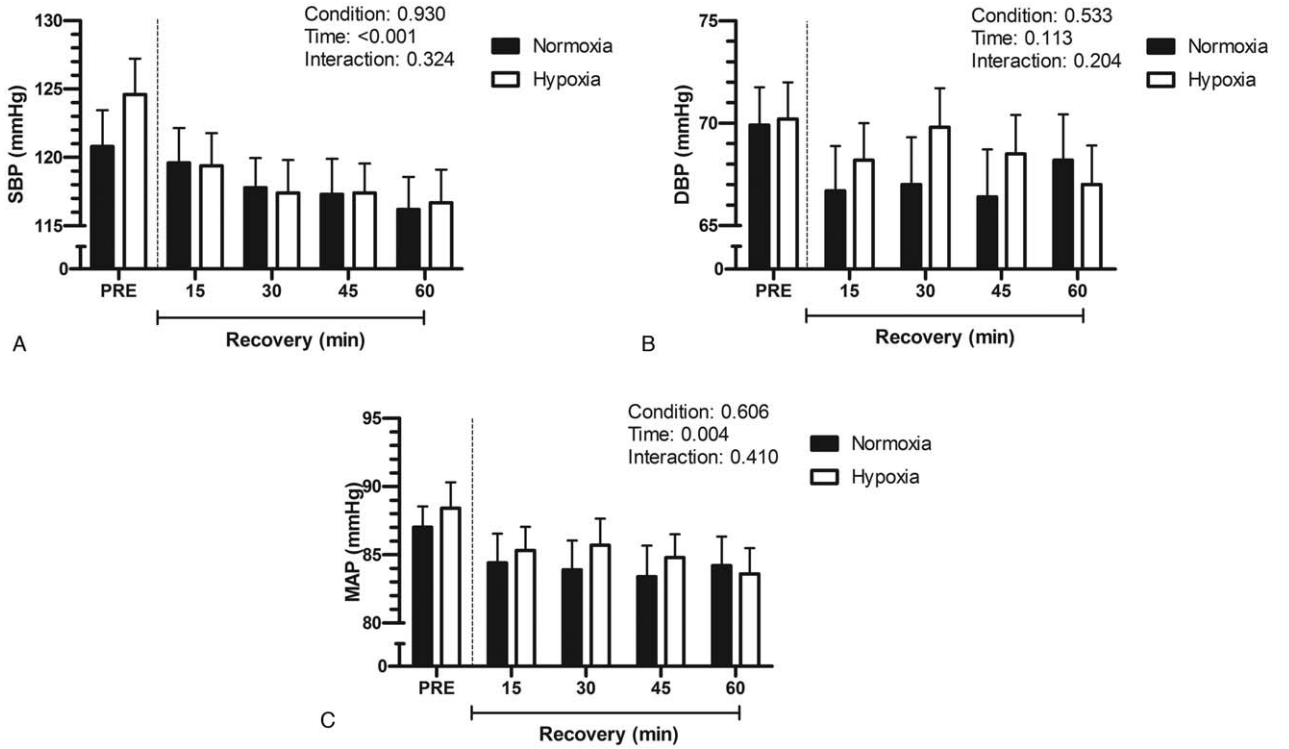

Figure 2. Post-exercise systolic blood pressure (panel A), diastolic blood pressure (panel B), and mean arterial pressure (panel C) response under normoxia (black bars) and hypoxia (white bars). The dotted line indicates 45 min high-intensity exercise and the error bars reflect the standard error of the mean.

4. Discussion

The aim of this study was to investigate the impact of hypoxia on PEH, and whether the magnitude of PEH relates to the BP-lowering effect of 12-week hypoxic endurance exercise-training. We present the following findings. First, the magnitude of PEH does not differ when exercise, matched at relative intensity, is performed under hypoxia or normoxia. Second, the magnitude of PEH during the first exercise bout was positively related to the magnitude of the BP-lowering effect of 12-weeks high-intensity running exercise training under hypoxia. Taken together, our results demonstrate that hypoxia does not alter the PEH response, whilst we reveal the close relationship between acute and chronic changes in BP in response to high-intensity running exercise in healthy individuals.

4.1. PEH in normoxia and hypoxia

Our study showed that a 45-minute high intensity running exercise bout leads to a decrease in mean arterial BP of ~3 mm Hg after exercise in healthy individuals, supporting the presence of PEH. This observation confirms findings from several previous studies that demonstrated the presence of PEH after a variety of types, durations, and intensities of endurance exercise.[11,12,15,26] However, in contrast to our hypothesis, the magnitude of PEH was not altered by hypoxia (FiO_2 14.5%). Under physiological conditions, changes in CO and TPR lead to alterations in BP.[27] After exercise, PEH seems to be largely explained by a decrease in TPR, likely due to a combination of centrally (ie, arterial baroreflex resetting with inhibition of sympathetic outflow) and locally mediated vasodilator mechanisms, which is not compensated by adequate elevations in CO.[3] Several previous studies have shown that hypoxia represents a powerful vasodilator signal for cerebral and peripheral arteries, subsequently leading to a decrease in TPR.[18–20] Despite the decrease in TPR under hypoxia, BP and PEH did not differ between normoxia and hypoxia, possibly because of a compensatory increase in HR and CO under hypoxia. The elevated HR and CO under hypoxia may be explained by a preserved and well-functioning baroreflex sensitivity in healthy young individuals under hypoxia,[28] or the hypoxia-induced chemoreceptors stimulation promoting greater sympathetic activation.[29,30] Interestingly, HR recovery tend to be slower under hypoxia (Table 3), while total work done was lower under hypoxia. These differences in HR recovery and total work may also have contributed to the preserved PEH response under normoxia versus hypoxia.[13,31,32]

Our finding contrasts with a previous study that investigated PEH in hypoxia following resistance exercise.[21] In this study, healthy young males showed significantly lower SBP and DBP levels 10, 20, and 30 minutes post-resistance exercise in hypoxia (FiO2 13.0%) compared to normoxia.[21] A key difference with our study is that they examined resistance exercise, compared to endurance exercise in our study. Whilst this difference in exercise mode may explain cross-study findings, former within-subject comparisons support the hypothesis that the mode of exercise (resistance vs endurance) does not alter the magnitude of PEH.[33,34] However, none of these previous comparisons have taken hypoxia into account. In addition, in the study of Horiuchi et al post-exercise recovery was performed under normoxia, making any comparisons with the previous investigations difficult, due to the persistent influence of hypoxic stress on autonomic and hemodynamic post-exercise responses. Future work is required to better understand the potential difference in

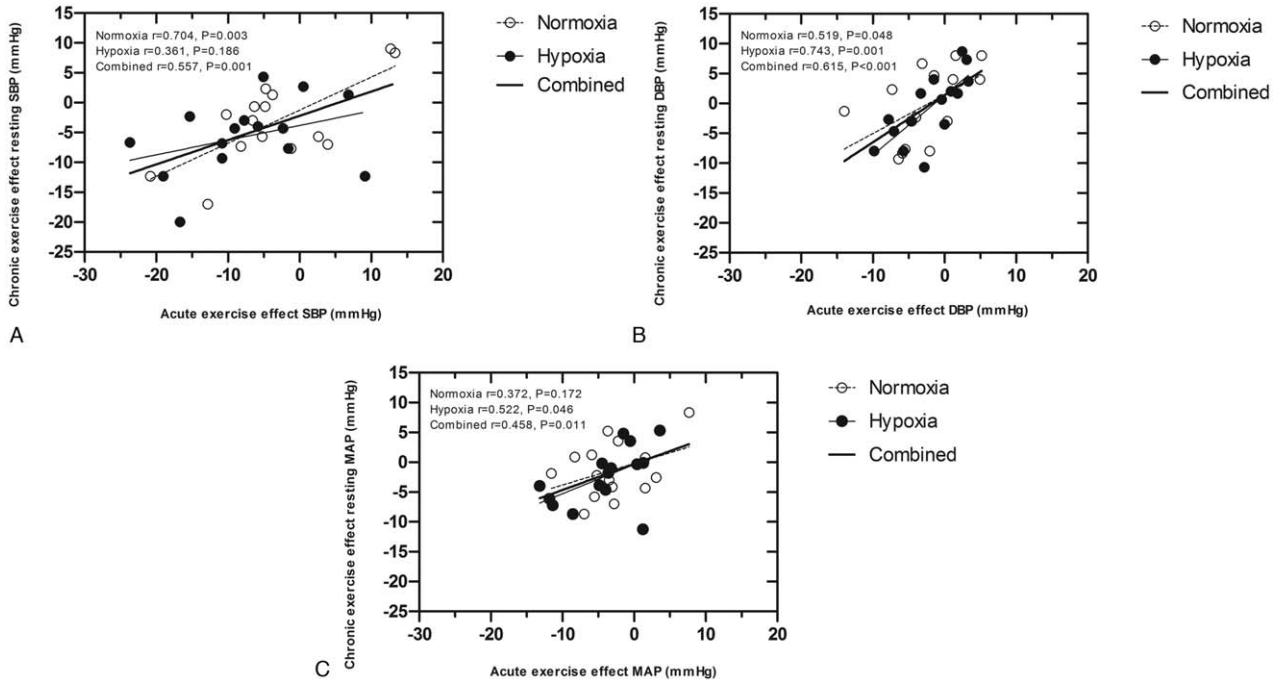

Figure 3. Correlations between acute exercise effect and chronic blood pressure lowering effect of 12-wk hypoxic training intervention (SBP, panel A), diastolic blood pressure (DBP, panel B), and mean arterial pressure (MAP, panel C). Error bars are omitted for clarity.

effect size of PEH between the different modes of exercise under hypoxia and recovery modalities.

4.2. Correlation PEH versus BP changes to training

The anti-hypertensive effects of regular exercise training for the general population are well known. This study further explored the relation between PEH and the long-term benefits of regular exercise training. The decrease of ~5 mm Hg in mean BP after 12-weeks of exercise training may seem marginal, but actually exceeds that of most previous studies examining the benefits of exercise training on BP in healthy individuals.[35] Within this context, it is important to realize that larger anti-hypertensive effects may be observed in those with (borderline) hypertension.[35] Importantly, we were able to link PEH, observed during the first session of high-intensity running exercise, to changes in resting BP after 12-weeks of exercise training. This observation provides further support that acute changes in BP after exercise ultimately relate to long-term changes.[8–10] An important addition to this knowledge, is that the correlation disappeared when we related PEH (taken after hypoxic exercise) to post-training BP assessed under normoxia. This suggests that the BP responses to acute and chronic exercise training, despite the similar magnitude of PEH, are related through distinct pathways. From a personalized exercise perspective, this observation means that those with the largest decline in PEH under normoxia, even when exercise training is performed under hypoxia, can expect the largest decline in resting BP (under normoxia). This may contribute to further personalize strategies to lower BP.

4.2.1. Limitations. A limitation is that we did not include a control group who either did not perform exercise or performed exercise under normoxic conditions across a 12-week period. Whilst this may have provided additional insight, this does not impact our primary finding of our study, in that PEH is strongly related to long-term declines in resting BP. In addition, our findings on PEH in hypoxia relate to a group of healthy young normotensive individuals and cannot be directly extrapolated to pre-hypertensive and hypertensive individuals, where PEH magnitude may be different.[36]

4.3. Perspectives

Hypoxia represents a relatively common stimulus that importantly alters the physiological demands of the cardiovascular system during exercise compared to normoxia. Nonetheless, we found that acute, high-intensity exercise under normoxia and hypoxia leads to a comparable post-exercise decline in BP in healthy volunteers (ie, PEH), whilst the magnitude of PEH strongly relates to the anti-hypertensive effects of exercise training. Whilst this provides novel insight into the acute and chronic regulation of BP, the comparable effects of hypoxic and normoxic exercise may have potential clinical relevance. Whilst both types of exercise are linked to a similar subjective level of effort, absolute workloads with hypoxic endurance exercise are significantly lower. This makes hypoxic exercise a suitable alternative for sedentary and frail individuals as a non-drug antihypertensive treatment, since lower workloads will be linked to fewer injuries and risks of exercise.[31,37,38] Hypoxic exercise has already been used effectively to enhance vascular structure and function,[39,40] adaptive responses in metabolic capacity[41] and glucose tolerance,[42] highlighting the potential of hypoxic exercise for health improvement.

5. Conclusion

Our findings show that hypoxia does not alter the magnitude of PEH in healthy individuals, whilst PEH relates to the BP-lowering effects of exercise. These data highlight the strong link between acute and chronic changes in BP.

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Author contributions

Geert Kleinnibbelink: Concept and design, data collection, data analysis and interpretation, drafting the manuscript and final approval of the manuscript. Niels A. Stens: Data collection, data analysis and interpretation, drafting the manuscript and final approval of the manuscript. Alessandro Fornasiero: Concept and design, data collection, data analysis and interpretation, revising the manuscript and final approval of the manuscript. Guilherme F. Speretta: Data collection, revising the manuscript and final approval of the manuscript. Arie P.J. van Dijk: Concept and design, revising the manuscript and final approval of the manuscript. David A. Low: Concept and design, revising the manuscript and final approval of the manuscript. David L. Oxborough: Concept and design, data collection, data analysis and interpretation, revising the manuscript and final approval of the manuscript. Dick H.J. Thijssen: Concept and design, data analysis and interpretation, revising the manuscript and final approval of the manuscript.

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