Effects of moderate-intensity aerobic cycling and swim exercise on post-exertional blood pressure in healthy young untrained and triathlon-trained men and women

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Abstract
Aerobic exercises such as running, walking and cycling are known to elicit a PEH (post-exercise hypotensive) response in both trained and UT (untrained) subjects. However, it is not known whether swim exercise produces a similar effect in normotensive individuals. The complex acute physiological responses to water immersion suggest swimming may affect BP (blood pressure) differently than other forms of aerobic exercises. We tested the hypothesis that an acute bout of swimming would fail to elicit a PEH BP response compared with an equivalent bout of stationary cycling, regardless of training state. We studied 11 UT and ten triathlon-trained young healthy normotensive [SBP/DBP (systolic BP/diastolic BP) <120/80 mmHg] men and women (age 23 ± 1 years) who underwent 30 min of intensity-matched cycling and swimming sessions to assess changes in BP during a 75-min seated recovery. CO (cardiac output), SV (stroke volume), TPR (total peripheral resistance), HR (heart rate), HRV (HR variability) and core and skin temperature were also assessed. In UT subjects, PEH was similar between cycling (−3.1 ± 1 mmHg) and swimming (−5.8 ± 1 mmHg), with the greater magnitude of PEH following swimming, reflecting a significant fall in SV between modalities (P < 0.05). Trained individuals did not exhibit a PEH response following swimming (0.3 ± 1 mmHg), yet had a significant fall in SBP at 50 min post-cycling exercise (−3.7 ± 1 mmHg) (P < 0.05). The absence of PEH after swimming in the trained group may reflect a higher cardiac sympathetic outflow [as indicated by the LF (low-frequency) spectral component of HRV) (25 and 50 min) (P < 0.05)] and a slower return of vagal tone, consistent with a significant increase in HR between modalities at all time points (P < 0.05). These results suggest that training may limit the potential for an effective post-exertional hypotensive response to aerobic swimming.

Key words: acute aerobic exercise, autonomic nervous system, blood pressure, haemodynamics, post-exercise hypotension (PEH), swimming

INTRODUCTION
Regular physical exercise is known to reduce cardiovascular risk in both men [1] and women [2]. Various exercise modalities have been shown to improve aerobic fitness and cardiovascular function. Benefits include reductions in BP (blood pressure), HR (heart rate), TPR (total peripheral resistance) and regional sympathetic outflow [3]. In particular, aerobic exercise can elicit chronic reductions in arterial BP in individuals who are hypertensive (>140 mmHg SBP (systolic BP) and >90 mmHg DBP (diastolic BP)], pre-hypertensive (120–139 mmHg SBP or 80–89 mmHg DBP) or normotensive (<120 mmHg SBP or <80 mmHg DBP) [4].

It is well-established that aerobic exercise elicits an acute increase in HR and arterial SBP, followed by a sustained PEH (post-exercise hypotensive) response in both male and female participants [5]. PEH has been shown to be present, regardless of endurance training status [5]. The mechanisms underlying PEH remain poorly understood but probably involve alterations in one or more components of cardiovascular control including

Abbreviations: ANS, autonomic nervous system; BP, blood pressure; CO, cardiac output; DBP, diastolic BP; HF, high-frequency; HR, heart rate; HRR, HR reserve; HRV, HR variability; LF, low-frequency; MAP, mean arterial pressure; MSNA, muscle sympathetic nerve activity; n.u., normalized units; PEH, post-exercise hypotensive; RPE, ratings of perceived exertion; RRI, RR-interval; SBP, systolic BP; SI, stroke index; V02, oxygen consumption; V02max, maximal V02.

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peripheral vascular resistance, CO (cardiac output), as well as autonomic and baroreflex function [6]. Furthermore, chronic exercise training has the potential to impact these putative mechanisms [7,8]. However, very few studies have examined the BP-lowering effects of regular swim exercise.

Recent studies have reported that swim training may actually increase resting BP in young [9] and older [10] normotensive individuals, raising concern over its efficacy as a suitable intervention in hypertension [11]. Furthermore, the complex acute physiological responses to water immersion suggest swimming exercise may affect BP differently than other forms of exercise [12].

Our group has shown previously that the acute PEH response may be linked to the long-term reduction in BP after exercise training [13]. However, there are no reports describing the acute PEH response to swimming. Therefore the purpose of our study was to compare the effects of intensity- and duration-matched cycling and swimming exercise on the post-exertional BP response in young, healthy endurance-trained and UT (untrained) individuals. Furthermore, we sought to characterize the hemodynamic, autonomic and thermoregulatory responses in the post-exercise period. We hypothesized that swimming would fail to elicit a PEH BP response of the same magnitude as land-based cycling, irrespective of training status.

**Subjects**
A total of 21 healthy, young males (n = 11) and females (n = 10) aged 18–30 years were recruited into two groups: UT capable of completing 30 min of each exercise modality but involved in less than 1 h per week of physical activity; and triathlon-trained subjects who were established (>2 years of training and competitive experience), recreational triathletes who performed a mixture of running, cycling and swimming training 4–6 days/week (3–4 h/week per modality) with exercise sessions ranging from 45 to 90 min in duration. All subjects were normotensive, free of cardiovascular or other chronic diseases, were not taking any medications for the management of BP, lipids, diabetes or any cardiovascular-related conditions. Females using oral contraceptives were studied only during the luteal phase. Written informed consent was obtained and a PAR-Q (Physical Activity Readiness Questionnaire) was completed by all subjects. This study was approved by the institutional ethics board and conformed to the Helsinki Declaration (2008) on the use of human subjects in research.

Participants were instructed to avoid exercise for 48 h and caffeine-containing beverages for 8 h prior to any measurements. Four separate assessments were conducted: (1) resting haemodynamic, ANS (autonomic nervous system) assessment and graded exercise testing; (2) swim fitness testing to establish swim velocity; and (3 & 4) randomly assigned 30 min bout of swimming or cycling exercise, 48 h apart, at an intensity equivalent to 60–70% of HRR (HR reserve), with exercise performed at the same time of day. HRR was calculated based on maximal HR during the graded exercise test and resting HR on the study day. Within 30 s of each exercise session, multiple assessments of BP, skin and core body temperature were made over a 6-min period and recovery data recorded for 70 min, following the transition from the pool area (27–28 °C ambient temperature), or time-matched walking, to the laboratory (22–23 °C). The experimental assessment timeline is outlined in Figure 1.

**Swim testing**
To assess swimming proficiency and establish an appropriate relative intensity and swim velocity, a 12-min Swim-Cooper-Test [14] was performed. A wireless monitor (Polar T31) was used to display and record HR during exercise. The pool temperature was maintained at 26 °C during the study.

**Acute cycling and swimming exercise intervention**
Exercise sessions consisted of 5 min warm up including stretching and low-intensity (10–15% HRR) exercise, immediately followed by 30 min of swimming or cycling at a moderate intensity (60–70% HRR) based upon prior exercise testing. The predicted maximal HR during swimming was adjusted downward by 10 beats/min to match the HR/\(\dot{V}O_2\) responses observed during cycling, [15] to ensure equivalent relative intensity. The Borg scale was used to determine RPE (ratings of perceived exertion) [16]. Water was provided ad libitum. Participants were instructed to exercise continuously, limiting breaks to fewer than 30 s and only if necessary. In both cases, exercise intensity was monitored by wireless HR recordings described above, with adjustments in swim pace made accordingly.

**Recovery measurements**
Following the immediate post-exercise measurements and transition to the laboratory, participants were dried and changed into pre-exercise clothing prior to body mass and post-exercise recovery data recordings in the seated position, which were obtained every 20 min during early (25–45 min), mid- (50–70 min) and late- (75–95 min) recovery with 5 min of rest between collection periods.
**Exercise and post-exercise blood pressure**

**Figure 1** Exercise session protocol
Timeline of pre- and post-exercise measurements during the acute cycling and swim exercise protocol. (1) Beat-to-beat BP and haemodynamic variables (CO, SV, TPR and HR) were measured continuously (averaged over 1-min interval) along with an automated sphygmomanometer (2-min intervals) during the pre- and post-exercise periods; (2) continuous skin and triplicate core body temperature (1 min); and (3) HRV (5.5 min).

**BP**
Beat-to-beat arterial BP was obtained continuously at heart level (using a sling) from the third digit of the right hand, using a photoplethysmographic technique (Finometer MIDI-2; Finapres Medical Systems). A custom macro was incorporated to average the Finometer recordings over 1-min intervals. In addition, brachial BP was recorded at 2-min intervals by automated sphygmomanometry (BPTru Medical Devices) on the left arm to allow for calibration and correction of any upward drift in Finometer measures associated with long-duration recordings or fluctuations related to reaplication of the Finometer measurement cuff [17]. Recovery BP measurements were based on 5-min rolling averages of the initial 9 min of each collection phase (to eliminate short-term fluctuations in BP) from beat-to-beat BP values and were reported as the mean change from pre-exercise values.

**CO, SV (stroke volume) and TPR**
SV and CO were calculated using a three-element model of aortic input impedance for arterial BP (Finometer MIDI) based on continuously recorded data averaged every minute, using BeatScope pulse contour analysis software (TNO Biomedical Instrumentation) as previously validated [18,19]. MAP (mean arterial pressure) was calculated as \((\frac{(SBP-DBP) \times 0.33 + DBP}{80})\) and was used to determine TPR as follows: TPR (dyn·s·cm⁻⁵) = \(80 \times (MAP/CO)\).

**Skin and core body temperature**
To determine the potential influence of thermoregulatory mechanisms on post-exercise BP and haemodynamic responses, skin temperature was measured using hard-wired thermistors from four sites (left forearm, chest, left thigh and left calf). A data logger (ACR Systems) recorded surface (skin) temperature (Trendreader 2; ACR Systems) with data sampled every 8 s, averaged over 1-min periods as described previously [20]. Core body temperature was estimated using tympanic membrane thermometry (Braun Ear Thermometer; Kaz Canada), which has been validated previously [21]. Temperature in the tympanic ear canal was sampled every 20 s concurrent with skin temperature recordings to determine the core-to-skin temperature gradient within the body over a 1-min interval [22].

**Cardiac autonomic function**
To account for the influence of cardiac autonomic balance on post-exercise BP responses, HRV (HR variability) was used to assess cardiac autonomic responses. HR was recorded using a three-lead ECG sampled at 1000 Hz and stored for analysis using LabView (2010 version; National Instruments). Kubios HRV data analyses was used to analyse the 5-min windows of RRs (R-R intervals; Biosignal Analysis and Medical Imaging Group), in accordance with recommendations by the Task Force of the European Society of Cardiology and North American Society of Pacing and Electrophysiology [23] with ectopic beats (defined as beats > or <30 % of the average of the previous four RRs) excluded. Fast Fourier transformation was applied to the RRI data to obtain mean RRI, STDRR (S.D. of RRRs), LF (low-frequency) spectral power (0.04–0.15 Hz), LF n.u. (normalized units), which represents the relative value of each power component in proportion to the total power minus the VLF (very LF) component, HF (high-frequency) spectral power (0.15–0.50 Hz), HF n.u. and the ratio of LF/HF spectral components. LF/HF ratio was used as an indicator of cardiac sympathovagal balance, HF n.u. used as an index of cardiac vagal activity and LF n.u. used as an index of cardiac sympathetic activity [23].

**Respiratory frequency**
To account for the potential influence of respiratory variations on HRV [23], particularly at a low breathing frequency that would prevent separation of the vagal component of HRV [24], breathing frequency was recorded using a Biopac Respiration Module (Biopac Systems Canada) from measurements of tidal volume. Participants were instructed to maintain a regular breathing frequency to minimize inter-subject differences.

**Statistical analysis**
The primary outcome variable was SBP after acute cycling and swimming exercise. Secondary outcomes included cardiovascular haemodynamic (CO, SV and HR), autonomic and thermoregulatory indices. A two-way repeated measures ANOVA was used to examine the main effects of each exercise modality on post-exercise changes in BP and haemodynamic variables, with post-hoc analysis performed using paired Student’s t tests with Bonferroni correction to isolate significant main effects. In the
absence of a significant post-hoc interaction, simple main effects pairwise comparison tests were performed to assess mean differences between the pre-exercise and recovery. A mixed-model repeated measures ANOVA was used to assess whether exercise modality had a differential effect on endpoints by group. Pearson correlation coefficients were used to determine the relationship between predictor variables and the SBP response following each exercise modality. All analyses were performed using statistical software (SPSS for Windows, version 19.0). Statistical significance was set at $P \leq 0.05$ and all values are reported as means ± S.E.M.

## RESULTS

### Subject characteristics

All subjects ($n = 21$) completed the protocol without adverse responses to exercise testing or acute exercise sessions. Subject characteristics are presented in Table 1. For UT subjects, the mean $\dot{V}_{O_2,\text{max}}$ corresponded to the 20th and 50th percentiles for males and females, respectively, compared with the mean $\dot{V}_{O_2,\text{max}}$ for trained subjects which corresponded to the 80th and 90th percentiles for males and females, respectively ($P < 0.01$) [25]. Resting HR was significantly lower in the trained compared with UT group ($P < 0.01$). There was no significant difference in resting BP measurements within groups before the acute cycling and swimming sessions.

### Exercise stress

Subjects performed exercise in both conditions within the prescribed intensity and no significant differences were observed between exercise modalities within or between groups. In the UT group, exercise HRs were $145 \pm 3$ beats/min ($66.7 \pm 0.8 \% \text{ HRR}$; $12.7 \pm 0.3 \text{ RPE}$) and $147 \pm 3$ beats/min ($69.8 \pm 1.4 \% \text{ HRR}$; $12.6 \pm 0.2 \text{ RPE}$) throughout cycling and swimming, respectively. In the trained group, average exercise HRs were $141 \pm 3$ beats/min ($66.4 \pm 0.6 \% \text{ HRR}$; $13.7 \pm 0.3 \text{ RPE}$) and $140 \pm 3$ beats/min ($68.1 \pm 0.7 \% \text{ HRR}$; $13.4 \pm 0.5 \text{ RPE}$) for cycling and swimming sessions, respectively.

### Body mass and fluid consumption

Body mass was unchanged after cycling exercise in UT ($-0.09 \pm 0.1 \text{ kg}$) and trained ($-0.1 \pm 0.06 \text{ kg}$) groups, but was significantly reduced after swimming exercise in both UT ($-0.2 \pm 0.1 \text{ kg}$) ($P < 0.05$) and trained ($-0.3 \pm 0.1 \text{ kg}$) ($P < 0.01$) groups. In the UT group, fluid consumption was significantly greater during cycling ($277 \pm 48 \text{ ml}$) compared with swimming ($159 \pm 30 \text{ ml}$) ($P < 0.05$). In the trained group, fluid consumption was significantly greater during cycling ($286 \pm 43 \text{ ml}$) compared with swimming ($120 \pm 43 \text{ ml}$) ($P < 0.05$). No significant difference between groups was observed.

### Post-exercise BP response

Post-exercise BPs (1, 3 and 5 min post-exercise) are presented in Table 2. In the UT group, SBP was significantly increased 1 min post-exercise following cycling compared with swimming ($P < 0.01$). DBP following cycling exercise was significantly increased compared with swimming exercise ($P < 0.01$) at all time points. In the trained group, SBP and DBP were not significantly different at any time point immediately post-exercise between modalities.

### Recovery BP and haemodynamic responses

BP and haemodynamic responses throughout the recovery period for UT subjects are presented in Figure 2. The SBP was significantly reduced compared with baseline at early- and mid-recovery following cycling exercise. Furthermore, SBP was significantly reduced compared with pre-exercise at early-, mid- and late-recovery (Figure 2). In addition, DBP was significantly reduced compared with baseline at all time points following swimming exercise only. The greater magnitude of SBP reduction following swimming was reflective of a significantly reduced SV and a trend toward a lower CO following swimming relative to cycling at all time periods during recovery. No significant changes in CO, TPR or HR were observed between exercise modalities.

In trained individuals (Figure 3), a significant decrease in SBP was observed compared with baseline after cycling exercise only (mid-recovery) which was reflective of a significant reduction in SV compared with baseline at early- and mid-recovery. However, no significant change in BP was observed following swimming exercise. The absence of a PEH response was reflective of a significantly reduced SV and significantly increased HR at all time points, resulting in a well-compensated CO. No significant changes in TPR were observed following either exercise modality.
While both UT and trained groups demonstrated similar SBP and DBP responses following cycling exercise, SBP in the UT group was significantly reduced at all recovery time points \((P < 0.01)\), in addition to DBP during early recovery \((P < 0.01)\), following swimming. A significant correlation was observed between change in SBP following swimming and \(VO_{2\text{max}}\) during early- \((r = 0.65, P < 0.01)\), mid- \((r = 0.52, P < 0.05)\) and late-recovery \((r = 0.46, P < 0.05)\), respectively, suggesting the PEH response following swimming is dependent on training status. No significant correlation was observed after cycling.

### HRV

Changes in cardiac autonomic function are presented in Table 3. In the UT group, no significant differences in indices of cardiac autonomic function were observed between cycling and swim exercise. The trained group demonstrated a significant increase in LF n.u. and reduction in HF n.u. at early- and mid-recovery compared with pre-exercise following swimming. In addition, LF/HF ratio was significantly increased compared with baseline (early- and mid-recovery) and cycling exercise (all time points) after swimming, indicating a slower recovery of these indices following swimming.

In comparing the groups, no significant differences in indices of cardiac autonomic function were observed following cycling exercise. However, following swimming, judging from the HF and LF components, respectively, vagal activity was significantly reduced and cardiac sympathetic activity was significantly increased during early- and mid-recovery in the trained compared with UT group, suggesting the response is dependent on both training status and exercise modality. Furthermore, significant correlations between change in LF n.u. and change in SBP during early- \((r = 0.59, P < 0.01)\) and mid- \((r = 0.54, P < 0.05)\) recovery were observed. In addition, change in LF/HF was significantly correlated with change in SBP during mid-recovery \((r = 0.48, P < 0.05)\).

Similar respiratory frequencies were observed during all pre- and post-exercise assessments within the UT (17 ± 1 compared with 18 ± 1 breaths/min) and trained (15 ± 1 compared with 16 ± 1 breaths/min) groups, regardless of exercise modality.

### Core and skin temperature

Core, skin and temperature gradient data are presented in Table 4. Following swimming in the UT group, core temperature was significantly decreased at 2, 4 and 6 min immediately after exercise and throughout recovery compared with cycling exercise. In trained individuals, core temperature was significantly lower immediately post-exercise (2, 4 and 6 min) following swimming compared with cycling, but not during the rest of recovery.

### DISCUSSION

To our knowledge, this is the first study to compare the effects of intensity- and duration-equivalent bouts of acute cycling and swimming exercise on the PEH response in young, healthy UT and trained normotensives. Contrary to our hypothesis, we found a similar degree of PEH following swim and cycle exercise in UT subjects. However, trained individuals failed to exhibit a PEH response following swimming. This response was associated with a slower return of vagal tone and a prolonged elevation in sympathetic activity, resulting in a significant elevation in HR during recovery from swimming compared with cycling exercise. These data suggest that training may limit or abolish the PEH response to swim exercise.

### PEH response to exercise in UT subjects

Our observation of a significant PEH response to cycling exercise in UT individuals is consistent with prior studies [26,27]. The time course of the PEH effect was comparable with that observed in sedentary, healthy individuals following cycling, walking or running exercise [6], remaining below pre-exercise values during late-recovery [28,29].

The mechanisms involved in the PEH response remain elusive [6,28]. PEH is thought to be achieved through a sustained decrease in vascular resistance that is not completely offset by elevations in CO [6,28]. While our results suggest a similar mechanism is operative following cycling exercise in UT individuals, with the reduction in TPR failing to reach significance due to the wide degree of variation in the response, CO was unchanged, linked to a persistent fall in SV that contributed to the observed PEH. The magnitude of the PEH response was greater following swimming, which is in contrast to our hypothesis. A larger PEH response after swimming was associated with a significant reduction in SV at all time points compared with cycling, despite similar HR and TPR responses post-exercise. It is possible that this response may have been secondary to a reduction in plasma volume and central venous pressure, which lowered left ventricular preload [30]. This is supported indirectly through a greater reduction in body mass and less fluid consumption observed with swim exercise. Alternatively, the fall in SV may reflect a reduction in venous return due to an absence of the skeletal muscle

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**Table 2** Immediate post-exercise SBP and DBP

<table>
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<th>Untrained ((n = 11))</th>
<th>Trained ((n = 10))</th>
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<td>Pre-exercise</td>
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<td>3 min</td>
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<td>Cycling</td>
<td>SBP (mmHg)</td>
<td>108 ± 2</td>
<td>125 ± 3**††</td>
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<td></td>
<td>DBP (mmHg)</td>
<td>70 ± 2</td>
<td>78 ± 2**††</td>
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<tr>
<td>Swimming</td>
<td>SBP (mmHg)</td>
<td>108 ± 2</td>
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<td>DBP (mmHg)</td>
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Figure 2  Recovery BP and haemodynamic responses in UT individuals
Change in BP and haemodynamics from absolute values measured pre-exercise and 25, 50 and 75 min post-acute cycling (dashed line) and swimming (solid line) exercise. A significant decrease in SBP compared with pre-exercise was observed following both cycling and swim exercise, with no significant differences observed between modalities. Following swimming, SV was significantly reduced at all time points compared with cycling. Values are expressed as the means ± S.E.M. Baseline values were normalized to zero. *P < 0.05 and **P < 0.01 compared with pre-exercise within group. †P < 0.05 and ††P < 0.01 between exercise modalities within group.

pump and redistribution of blood to more compliant vascular beds during inactive sitting recovery [31]. This may have produced an orthostatic stress, lowering CO, while enhancing peripheral vasoconstriction [32].

We anticipated that swim exercise would lower the core-to-skin temperature gradient, linking thermoregulatory-induced cutaneous blood flow [22] to the PEH response, following exercise [33]. A reduction in skin temperature following swim exercise would theoretically limit the extent of PEH by enhancing peripheral vascular resistance. However, as reported by others [6], our data do not support a cutaneous thermoregulatory influence on the post-exercise BP response in either condition regardless of training. In fact, the temporal pattern of heat loss and PEH were not correlated, as PEH persisted after cutaneous blood flow was likely to have returned to baseline [34]. Furthermore, our data indicates that the magnitude of PEH was greater after swimming compared with cycling exercise in UT subjects despite the significant reduction in core temperature.

Influence of exercise training on PEH responses
A key finding in our study is that a different haemodynamic profile contributes to PEH between trained and UT subjects after cycling exercise despite the magnitude of PEH being similar. Consistent with previous studies [5,35,36], endurance trained individuals exhibited reductions in SV and a trend for a fall in CO with no change in TPR during PEH. Although inadequate fluid replacement may contribute to this differential response to cycling exercise in trained individuals, it has been previously shown that a fall in CO drives this response even when plasma volume is maintained [36]. This suggests that the mechanisms
and degree of PEH may depend on training status, which in turn influences the redistribution of CO at a given plasma volume and filling pressure. In support of this, a greater fall in SV has been observed in endurance-trained individuals for a given filling pressure, reflecting the shifting of the Frank–Starling pressure–volume curve [37]. However, the explanation for this divergent haemodynamic response to cycling exercise between trained and UT individuals remains unclear.

In the present study, power spectral analysis of HRV was used to assess cardiac autonomic regulation, as described previously [23]. While the cardiac autonomic response was similar between exercise modalities in the UT group, trained individuals exhibited a differential response. The significant decrease in HF n.u. and increased LF n.u. observed until the mid-recovery period in the trained group as compared with the UT group following swimming, as well as the prolonged increase in LF/HF ratio into late-recovery compared with cycling exercise in the trained group, suggests that the post-exercise pattern of cardiac autonomic regulation is dependent on both training status and exercise modality. This response is in contrast to cycling exercise, in which changes in autonomic regulation and the pattern of recovery were similar between groups. A reduction in the inhibitory influences of vagal outflow on HR, concomitant with the enhancement of cardiac sympathetic outflow, may be a training-specific adaptation from swim exercise [38]. In support of this, BP was unchanged following swim exercise in the trained group, reflective of a well-regulated CO. Specifically, a significant increase in HR offset the fall in SV after swimming, but not cycling exercise. This response is consistent with a greater fall in SV post-swim exercise, which would accentuate the deactivation of cardiopulmonary receptors, thus increasing sympathetic activation to the heart as found in our study [39]. Unfortunately, HRV data does not reflect peripheral sympathetic activity, confining our interpretation to cardiac autonomic regulation [23]. PEH has been linked to a reduction in MSNA (muscle sympathetic nerve activity) and a decreased responsiveness to sympathetic outflow in the periphery [28], although there is wide individual variability in SNS (sympathetic nervous system) activity and BP [40]. Nevertheless, our
findings suggest the pattern of cardiac ANS regulation of BP is dependent on both training status and exercise modality.

Swim exercise may lead to training-induced adaptations that may, in part, account for the observed differential response between groups. A dominance of arm exercise may produce a disproportionate rise in HR, peripheral vascular resistance and BP compared with lower limb exercise when matched for workload [41]. Furthermore, swim-trained individuals have been shown to have higher \( \alpha_2 \)-receptor and lower \( \beta \)-receptor densities and higher resting vasopressin levels, thereby increasing vasoconstrictor responsiveness to resting sympathetic tone and increased sympathetic activity [42]. These factors could limit the PEH in trained subjects.

Respiratory frequency during the recovery period remained consistent between groups, at a rate to ensure separation of the HF and LF domains. However, respiratory patterns during swimming may induce irregular, intermittent breathing patterns that may be exacerbated with training [43]. Swim training is associated with a reduction in breathing frequency, consequently increasing its apnoic nature. In addition, highly-trained swimmers may exhibit arterial hypoxaemia [44] and hypoxia [45] during exercise, which is known to increase sympathetic activation for a period beyond the stimulus [46]. Furthermore, hypercapnia has also been observed following swimming [44,47], which is associated with an increase in LF n.u., decrease in HF n.u. and elevations in BP [48]. It is possible that this contributed to increased sympathetic outflow during recovery, but the absence of corroborating evidence including exercise respiratory and blood and ventilatory gas data limits our interpretation.

It is possible that repeated exposure to cold water in the trained group may have enhanced sympathetic activation and the vasoconstrictor response [49] such that thermoregulatory mechanisms make a larger contribution to BP regulation. Although the temperature of the pool in our study (26°C) was higher than that reported by O’Brien et al. [49] (20°C) and there was no difference in the extent of the decrease in skin and core temperature between groups, previous repeated exposure to cold stimuli in the trained group may have contributed to the greater cardiac sympathetic activity that occurred in the trained as compared with the UT group.

### Limitations

Our study has some limitations. The trained group had regular experience with swim and cycling exercise, which may have influenced the post-exercise BP responses to cycling and swimming respectively. In addition, it is unknown how training for running may have influenced the response to either exercise modality.

The inability to transfer equipment between our laboratory and the pool deck resulted in a gap of data between the immediate post-exercise and recovery periods. However, as the PEH response typically persists for hours following an acute bout of exercise [6], we suggest the impact of this is minimal.

We also acknowledge there are limitations to the measurement of CO by the Modelflow technique [50], which would also affect the calculation of TPR. However, this technique may be more robust than pulse Doppler echocardiography [51], and any error would be consistent between study groups.

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</table>
Exercise and post-exercise blood pressure

Finally, it was not feasible to measure core temperature directly. Although tympanic membrane thermometry has been validated [21] and ear plugs were used to limit water exposure, local tympanic cooling may have underestimated the core temperature measures. Furthermore, measurements of regional blood flow and MSNA would have enhanced our interpretation of the BP changes observed.

Conclusions
Cycle exercise elicits a significant PEH response, regardless of training state. However, unlike UT individuals, the PEH response following swim exercise is absent in triathlon-trained athletes. Although SV was significantly reduced following both cycling and swim exercise, vagal inhibition and a significant, prolonged elevation in sympathetic outflow and HR during recovery from swimming contributed to the absence of PEH in the trained athletes. As the magnitude of the PEH response may predict the chronic BP lowering effect of chronic-exercise, these observations may have implications for those seeking a long-term BP-lowering effect from swim exercise, as the PEH effect from swim exercise may gradually be attenuated or abolished in the transition from an UT to a trained state.

**CLINICAL PERSPECTIVES**
- Current evidence suggests that all forms of regular aerobic exercise are equally effective at lowering BP following an acute bout of exercise; however, whether swimming elicits the same response remains unknown.
- The results of the present study suggest that BP and haemodynamic responses following an acute bout of exercise are dependent on both exercise modality and training status. This finding may have significant implications for the use of chronic swim exercise as an intervention to lower BP, as exercise regimens may not be interchangeable in terms of their BP-lowering capacity.
- This is of clinical relevance in the prescription of exercise for the treatment of hypertension, as improving fitness levels over time may be linked to an attenuation of the BP lowering effect of acute exercise.

**REFERENCES**

**AUTHOR CONTRIBUTION**
All authors were involved in the study conception, design and management, data interpretation and drafting of the paper. Robert Lakin was involved in the recruitment of the participants, data collection and analysis, and paper preparation.

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Table 4  Changes in core, skin and core-to-skin gradient temperature from pre-exercise following acute exercise

<table>
<thead>
<tr>
<th>Exercise modality</th>
<th>Time (min)</th>
<th>Untrained (n = 11)</th>
<th>Trained (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Core (°C)</td>
<td>Skin (°C)</td>
</tr>
<tr>
<td>Cycling</td>
<td>2</td>
<td>0.07 ± 0.09</td>
<td>-1.75 ± 0.20**</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>-0.06 ± 0.08</td>
<td>-1.07 ± 0.21**</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>-0.08 ± 0.07</td>
<td>-0.82 ± 0.18*</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>-0.09 ± 0.06</td>
<td>-0.13 ± 0.14</td>
</tr>
<tr>
<td></td>
<td>55</td>
<td>-0.09 ± 0.06</td>
<td>-0.23 ± 0.18</td>
</tr>
<tr>
<td></td>
<td>80</td>
<td>-0.13 ± 0.06</td>
<td>-0.48 ± 0.17</td>
</tr>
<tr>
<td>Swimming</td>
<td>2</td>
<td>-1.60 ± 0.19***†</td>
<td>-2.50 ± 0.33**</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>-1.26 ± 0.16***†</td>
<td>-1.52 ± 0.36*</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>-1.08 ± 0.17***†</td>
<td>-1.14 ± 0.34</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>-0.52 ± 0.10***†</td>
<td>-0.31 ± 0.10</td>
</tr>
<tr>
<td></td>
<td>55</td>
<td>-0.34 ± 0.06***†</td>
<td>-0.42 ± 0.15</td>
</tr>
<tr>
<td></td>
<td>80</td>
<td>-0.29 ± 0.06***†</td>
<td>-0.62 ± 0.15</td>
</tr>
</tbody>
</table>


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