Greater post-exercise hypotension in healthy young untrained men after exercising in a hot compared to a temperate environment

Horiuchi, Masahiro; Oliver, Sam

Journal of Thermal Biology

DOI:
10.1016/j.jtherbio.2023.103683

Published: 01/10/2023

Peer reviewed version

Greater post-exercise hypotension in healthy young untrained men after exercising in a hot compared to a temperate environment

Masahiro Horiuchi [1,2], Samuel J Oliver [3]

1. Division of Human Environmental Science, Mount Fuji Research Institute

2. Faculty of Sports and Life Science, National Institute of Fitness and Sports in KANOYA

3. Institute for Applied Human Physiology, College of Human Sciences, Bangor University, Bangor, Wales, UK

Corresponding author: Masahiro Horiuchi, PhD

Shiromizu 1, Kanoya City, Kagoshima, 8912393, Japan

mhoriuchi@nifs-k.ac.jp

OCID: 0000-0001-5784-5694 (Masahiro Horiuchi)

ORCID: 0000-0002-9971-9546 (Samuel Oliver)

Word count: 4259

Number of references: 46

Number of tables: 2

Number of figures: 3 (all color)

Abstract

This research examined the effects of exercising in a hot compared to a temperate environment on post-exercise hemodynamics in untrained men. We hypothesized exercise in a hot compared to a temperate environment would elicit greater post-exercise hypotension, and this would be attributable to higher cutaneous vascular conductance and sweat loss, and lower heart rate variability (HRV) and cardiac baroreflex sensitivity (cBRS). In a randomized counterbalanced order, 12 untrained healthy men completed two trials involving 40-min leg-cycling exercise at either 23°C (CON) or 35°C (HOT). Post-exercise participants rested supine for 60 min at 23°C whilst hemodynamic and thermoregulatory measurements were assessed. Post-exercise hypotension was greater after exercising in a hot than a temperate environment as indicated by a lower mean arterial pressure at 60 min recovery (CON 83±5 mmHg, HOT 78±5 mmHg, Mean difference [95% confidence interval], –5 [–8, –3] mmHg). Throughout recovery, cutaneous vascular conductance was higher, and cBRS and HRV were lower after exercising in a hot than in a temperate environment (P<0.05). Sweat loss was greater on HOT than on CON (P < 0.001). Post-exercise hypotension after exercising in the hot environment was associated with sweat loss (r=0.66, P=0.02), and changes in cutaneous vascular conductance (r=0.64, P=0.03), and HRV (RMSDD r=0.75, P=0.01 and log [HF] r=0.66, P=0.02), but not cBRS (all, r≤0.2, P>0.05). Post-exercise hypotension was greater after exercise in a hot compared to a temperate environment and may be partially explained by greater sweat loss and cutaneous vascular

conductance, and lower HRV.

Keywords: *cardiac baroreflex sensitivity, cardiac autonomic function, cutaneous vascular conductance, hemodynamics, skin blood flow, vasodilation*

Abbreviations

ANOVA: Analysis of variance

a.u.: arbitrary unit

BP: Blood pressure

cBRS: Cardiac baroreflex sensitivity

CI: Confidential interval

CON: Thermoneutral condition

CVC: Cutaneous vascular conductance

DBP: Diastolic blood pressure

η^2: eta square

ES: effect size

HF: High frequency

HOT: Hot environment

HR: Heart rate

HRV: Heart rate variability

ISO: International Society for Organization

LF: Low frequency

MAP: Mean arterial pressure

PEH: Post-exercise hypotension

RMSSD: Root mean square of the successive differences in R-R interval

SBP: Systolic blood pressure

SD: Standard deviation

SDNN: Standard deviation of the normal-to-normal intervals

SkBF: Skin blood flow

T: Temperature

T_{arm}: Arm temperature

T_{chest}: Chest temperature

T_{core}: Sublingual temperature

T_{sk}: Mean skin temperature

T_{thigh}: Thigh temperature

$\dot{V}O_{2max}$: Maximal oxygen consumption

W: Watts

Introduction

Mean arterial pressure (MAP) is lowered after physical exercise for up to 24 h; a phenomenon called post-exercise hypotension (PEH) (Halliwill, 2001; Halliwill et al., 2014). The environmental and physiological determinants of PEH after aerobic exercise are incompletely understood with studies to date having largely investigated the effects of exercise intensity, mode, duration, and populations on PEH in a temperate environment (Brito et al., 2014; Gomes Anunciacao and Doederlein Polito, 2011). Most studies indicate that PEH follows a decrease in peripheral vascular resistance, although a reduction in cardiac output may also be an important determinant in some circumstances (Brito et al., 2014; Gomes Anunciacao and Doederlein Polito, 2011). Exercising in a hot environment increases cardiovascular strain (increased cutaneous vasodilation, increased sweat loss, and a reduction in plasma volume), and therefore greater PEH might be expected after exercising in a hot compared to a temperate environment. Determining the effect of exercising in the heat on PEH is important as severe PEH is associated with an increased risk of syncope (Mundel et al., 2015). Further, studies indicate the size of the blood pressure (BP) fall after a single bout of exercise is associated with the chronic reduction in resting BP after exercise training (Hecksteden et al., 2013; Liu et al., 2012), and therefore exercise training performed in the heat may be more effective at lowering BP chronically than exercise training completed in a temperate environment.

Research examining the influence of exercising environmental temperature on PEH

94 reports conflicting findings, with greater PEH observed after exercising in a hot compared to a
95 temperate environment in one study in untrained men (Cunha et al., 2020), but not the other in
96 endurance-trained men (Lynn et al., 2009), despite similar relative exercise intensity, i.e.,
97 55~60% of maximal aerobic capacity. One possible explanation for the contrasting findings is
98 the different training status of the study populations as a previous study in a temperate
99 environment indicated PEH followed a decrease in peripheral vascular resistance in sedentary
100 untrained men and women, and trained women but followed a reduction in cardiac output in
101 trained men (Senitko et al., 2002). These divergent hemodynamic responses occurred despite a
102 similar relative exercise intensity. In addition, trained individuals may experience less
103 thermoregulatory and cardiovascular strain than untrained individuals during exercise recovery
104 owing to more effective heat dissipation mechanisms developed during regular exercise training
105 (McIntyre et al., 2022; Ravanelli et al., 2021).

106 Greater skin blood flow and cutaneous vascular conductance (CVC), and lower plasma
107 volume, after exercising in a hot compared to a temperate environment may lead to larger PEH
108 via greater reductions in peripheral vascular resistance. Whether PEH is attributable in part to
109 higher skin blood flow and CVC after exercising in the heat remains to be determined since the
110 one study in untrained participants to report greater PEH did not evaluate skin blood flow and
111 CVC (Cunha et al., 2020). Other regulatory mechanisms that may contribute to a greater
112 reduction in PEH after exercising in a hot compared to a temperate environment are alterations

in cardiac baroreflex sensitivity (cBRS) (Halliwill, 2001; Halliwill et al., 2014), and cardiac autonomic function, which can be assessed by heart rate variability (HRV) (Cunha et al., 2020; Cunha et al., 2015). cBRS and HRV might be expected to be lower after exercising in a hot environment compared to a temperate environment due to a shift toward greater sympathetic activity to support a reduction in MAP and to compensate for resetting of the baroreflex (Cunha et al., 2020; Halliwill et al., 1996). The one study to have examined cBRS and HRV during PEH reported no differences after exercise in a hot compared to a temperate environment although the authors cautioned against drawing firm conclusions because the small sample size (n = 7) may have led to a type II statistical error (Cunha et al., 2020). Additionally, this study did not discriminate the effects of rising or falling BP on cBRS during PEH and thus, how cBRS -up or -down are related to PEH after exercise in a hot environment has yet to be determined. As the cBRS is less sensitive to falling than to rising changes in BP (Eckberg, 1980; Willie et al., 2011), it is important to investigate, not only overall BRS but also separate BRS-up (upward slope) and BRS-down (downward slope) (Studinger et al., 2007).

This study aimed to examine post-exercise hemodynamics after exercise in a hot compared to a temperate environment in healthy young untrained men. We hypothesized exercise in a hot compared to a temperate environment would elicit greater PEH, and this would be attributable to higher cutaneous vascular conductance (CVC), greater sweat loss, and lower heart rate variability (HRV) and cardiac baroreflex sensitivity (cBRS).

Methods

Ethical approval

Ethical approval was obtained (No. 201201) from the Yamanashi Institute of Environmental Science (Mount Fuji Research Institute after renaming) in Japan and was conducted following the standards of the Declaration of Helsinki.

Participants

Twelve healthy untrained young men volunteered for this study (age 22 ± 3 years, height 173 ± 6 cm, body mass 65.71 ± 10.50 kg, mean ± standard deviation). Volunteers were non-smokers, who did not engage in regular physical activity, and had no prior history of orthopedic or cardiovascular diseases, including hypertension, diabetes, and hyperlipidemia. After the study procedures, possible risks, and benefits were explained, written informed consent to participate in this study was obtained from each volunteer.

Experimental procedures

Before each visit, participants abstained from caffeinated drinks for 12 h and alcohol and arduous exercise for 24 h. They visited the laboratory on three occasions to undertake the following procedures (**Figure 1**): Visit 1, to complete a familiarization of all experimental

procedures including wearing devices; Visits 2 and 3 to complete two experimental trials (control [CON] and hot [HOT]). The trial order was randomized and counterbalanced with at least 1 week between trials. Participants' visits 2 and 3 were completed at the same time of day (08:30–11:30 h) to avoid any circadian rhythm effects. On visits 2 and 3, participants drank a bottle of water (300 ml) on arrival to the laboratory to promote hydration. All visits were performed in a temperature and humidity-controlled environmental chamber (40% relative humidity; TBR-4, 5SA2GX, Tabai Espec Co., Ltd., Tokyo, Japan). Participants completed two experimental trials: (a) Temperate control (CON), which involved 15-min supine rest, 40-min upright leg cycling exercise and a 60-min supine recovery at 23°C, and (b) Hot (HOT), which involved 15-min supine rest and a 40-min upright leg cycling exercise at 35°C, followed by a 60-min supine recovery at 23°C. After exercise in the HOT, the chamber door was immediately opened, and the thermostat was set to 23°C. The 40 min cycling exercise comprised of a 5 min warm-up and then 35 minutes at a workload equivalent to 75% of individual heart rate (HR) maximum. The warm-up began at 20 W and increased by 20 W every minute up to 100 W at 5 minutes. After the warm-up exercise workload was manually adjusted to maintain 75% of individual HR maximum.

Measurements

Before and after each trial nude body weight was assessed by a 50-g resolution scale (UC-321,

A&D Instruments, Tokyo, Japan). Sweat loss was calculated as post-trial body weight minus pre-trial body weight. To obtain precise sweat loss, the participants were not allowed to drink water during the experimental trials and to urinate between pre- and post-exercise body mass measurements.

Central hemodynamics

Throughout the study heart rate (HR) was recorded by a wireless HR monitor (Polar RC800X; Polar Electro Japan, Tokyo, Japan) whilst HR variability (HRV) was assessed by another HR monitoring device (Check-My-Heart, Daily Care BioMedical, Chungli, Taiwan). HRV was measured and analyzed as described previously (Horiuchi and Thijssen, 2020). In brief, electrodes were attached to the lower left rib and right clavicle, and a 5-minute electrocardiogram was collected, and analyzed automatically by the HRV software in the time and frequency domains (Daily Care BioMedical, Chungli, Taiwan). In the time domain, the HRV was determined from the standard deviation of the normal-to-normal intervals (SDNN), and the root mean square of the successive differences in R-R interval (RMSSD). In the frequency domain, low-frequency oscillations (LF: 0.04–0.15 Hz), and high-frequency oscillations (HF: 0.15–0.4 Hz) were quantified using a fast Fourier transformation (Horiuchi and Thijssen, 2020; Malliani et al., 1991). Beat-by-beat BP was measured by applanation tonometry (JENTOW 7700, Colin Medical Technology, Komaki, Japan). This equipment was

189 calibrated from brachial arterial BP. HRV and BP were evaluated at 10–15 min during resting
190 and 5–10, 15–20, 25–30, 35–40, 45–50, and 55–60 min during recovery.
191
192 *Cutaneous circulation*
193 At rest and during recovery core body temperature (T_{core}) was assessed sublingually (LT-8,
194 Gram Co. Ltd., Saitama, Japan) per the International Society for Organization (ISO) 9886
195 guidelines. Using the same device skin temperature was measured at three sites: 1) on the left
196 chest (T_{chest}), the point at 10 cm below the mid-clavicle, 2) on the left upper arm (T_{arm}), the
197 center point of the line between the acromion process and the epicondylitis lateralis humeri; 3)
198 on the left thigh (T_{thigh}), at the point one-third of the length between the patella and the iliac
199 crest. T_{core} and skin temperature were recorded every second. Skin blood flow (SkBF) at the
200 chest was measured by the laser Doppler method (ATBF-LC1; Unique Medical Co., Ltd., Tokyo,
201 Japan) and CVC was calculated as previously described (Horiuchi and Fukuoka, 2019).
202
203 *Data analysis*
204 Beat-by-beat BP was assessed by finger photoplethysmography from the left hand as the time-
205 averaged from the beat-by-beat pressure wave (Horiuchi et al., 2016). To calculate spontaneous
206 cBRS, the beat-to-beat systolic BP (SBP) time series and RR interval were analyzed for more
207 than 3 consecutive beats, with increasing or falling direction from a 5-min steady-state data

segment at rest and during recovery (Carrington and White, 2001; Ogoh et al., 2005). Linear regression was applied to each individual baroreflex sequence, with only sequences with an R^2>0.85 accepted (Iellamo et al., 1994). When BP increases (upward slope) or decreases (downward slope) more than 3 consecutive beats, they were defined as spontaneous "cBRS-up" and "cBRS-down". The overall average slope of the SBP–RR interval was calculated as spontaneous "cBRS-overall".

The following equation was used to calculate mean skin temperature (T_{sk}) (Roberts et al., 1977):

$$T_{sk} = 0.25 \cdot T_{arm} + 0.43 \cdot T_{chest} + 0.32 \cdot T_{thigh}$$

To calculate CVC for each trial (CON and HOT), we used absolute raw values of SkBF ($_{abs.}$SkBF) data represented as mV, and divided $_{abs.}$SkBF by MAP, ($_{abs.}$CVC as mV·mmHg^{-1}). Additionally, we calculated changes in these variables from pre-exercise resting values and represented them as relative changes in SkBF (%) and CVC (%).

For the values of HRV, the normality of the data was checked by Bartlett and Levene test. Logarithmic transformation was used for further HRV analyses if equal variances were not found (Horiuchi and Thijssen, 2020).

227 *Statistical analyses*

228 Using standard alpha (0.05), power (0.8), and a medium effect size (f=0.25), a sample size of
229 12 was calculated (G*Power 3.1), to be required to reject the null hypothesis for a repeated
230 measures two-way analysis of variance (ANOVA) with 2 groups (CON vs. HOT), and 7
231 measurements over time (pre-exercise, 10, 20, 30, 40, 50, and 60 min). Statistical analyses
232 were completed on the commercial-free software Jamovi (ver. 2.2.5). A paired t-test compared
233 exercise mean workload, HR and sweat loss between CON and HOT. Two-way (Trial × Time
234 points) repeated measures ANOVA was used to compare all the physiological variables. BP
235 variables, HR, T_{core}, T_{sk}, $_{abs.}$SkBF, and $_{abs.}$CVC were analyzed including pre-exercise resting
236 values and each time point during the recovery (10, 20, 30, 40, 50, and 60 min). The analysis
237 of percent changes in SkBF and CVC included only recovery time points as these metrics are
238 calculated from pre-exercise resting values. The effect size was calculated as "η^2", defined as
239 small (η^2=0.01), medium (η^2=0.06), and large (η^2=0.14) effects (Lakens, 2013). When *F*
240 values were significant (*P*<0.05), the Tukey post hoc test was used for further comparisons.
241 Pearson correlation coefficient was used to determine PEH (changes in MAP) relationship
242 with sweat loss and changes in cutaneous circulation, HRV, and cBRS metrics during the
243 recovery period. A *P* value <0.05 was considered statistically significant.
244
245 **Results**

All twelve participants completed the study protocols. After the standard 5 min warm-up, manual adjustment of the leg cycling workload (CON 141 ± 16 W, HOT 120 ± 14 W, Mean difference [95% confidence interval], –22 [–25, –19] W) maintained exercising HR equivalent to 75% of HR maximum on both CON and HOT (CON 148 ± 3 bpm, HOT 149 ± 3 bpm, 0.3 [–0.6, 1.3] bpm).

The time-course changes in central hemodynamics (MAP, SBP, diastolic BP [DBP], and HR) between the two trials are shown in **Figure 2**. A significant interaction and main effects of time and trial were observed in MAP and SBP (**Figures 2A and B**). Post-exercise hypotension was greater after exercising in a hot than temperate environment as indicated by a lower mean arterial pressure (MAP) at 60 min recovery (CON 83 ± 5 mmHg, HOT 78 ± 5 mmHg, –5 [–8, –3] mmHg, **Figure 2A**). Compared with the pre-exercise resting values MAP decreased at 60 min recovery by 5% in CON (–4 [–6, –2] mmHg) and by 10% in HOT (–9 [–10, –7] mmHg). Similarly, by 60 min recovery, SBP decreased 3% in CON (–4 [–7, –2] mmHg) and 9% in HOT (–11 [–13, –9] mmHg, **Figure 2B**). Tukey posthoc test further revealed during recovery that MAP at 10 and 60 min and SBP at 20 min in HOT were lower than in CON (all $P<0.05$). Significant time and trial main effects, but no interaction, were observed for DBP (**Figure 2C**). Compared with the pre-exercise resting values and by 60 min recovery, DBP decreased 6% in CON (–4 [–6, –2] mmHg) and 11% in HOT (–8 [–10, –5] mmHg, **Figure 2C**). An interaction

in HR was also observed where HR was higher at 10, 20, 40, and 60 min of recovery in HOT than in CON (**Figure 2D**).

Figure 3 shows the two trials' time-course changes in the thermoregulatory metrics (Tcore, Tsk, relative changes in SkBF, and CVC). A significant interaction and main effects of time and trial were observed (all $P<0.05$) for T_{core}, T_{sk} (**Figure 3A and 3B**), $_{abs.}$SkBF and abs.CVC (data not shown). Regarding relative changes in SkBF and CVC, significant main effects of time and trial were found (**Figures 3C and D**). During the 60 min recovery period, T_{core} and the cutaneous circulation metrics in HOT were higher than in CON (all $P<0.05$). A relationship was found between changes in MAP and CVC in HOT (r=0.64, P=0.03) but not CON (r=–0.46, P=0.14). Sweat loss was greater in HOT than in CON (HOT –1.1 ± 0.2 kg, CON –0.5 ± 0.1 kg, Δ–0.6 [–0.7, –0.5] kg). A relationship was observed between the changes in MAP and sweat loss in HOT (r=0.66, P=0.02) but not CON (r=0.33, P=0.30).

Interaction and main effects of trial were observed for cardiac autonomic function variables (SDNN, RMSSD, log [HF]) where at several time points during recovery these were lower in HOT than in CON (**Table 1**). A relationship was found between changes in MAP and RMSSD in HOT (r=0.75, P=0.01) but not CON (r=0.01, P=0.97). A relationship was also found between changes in MAP and log [HF] in HOT (r=0.66, P=0.02) but not CON (r=0.14, P=0.66). There

were no other significant relationships between changes in MAP and other HRV metrics. Interaction effects were also observed for cBRS-up and cBRS-overall but not cBRS-down (**Table 2**). Moreover, main effects of trial were observed for cBRS-up, cBRS-down and cBRS-overall, with all lower in HOT than in CON**.** There were no relationships between changes in MAP and BRS-up, -down, or -overall in CON and HOT (all, $r \leq 0.2$, $P > 0.05$).

Discussion

The present study examined hemodynamics during supine recovery after exercise in a hot compared to a temperate environment in healthy young untrained men. The major findings were three-fold:(i) post-exercise hypotension was greater after exercising in a hot compared to a temperate environment (ii) sweat loss and CVC were greater, and HRV was lower, after exercising in a hot compared to a temperate environment: greater sweat loss and CVC, and lower HRV, were also associated with greater PEH (iii) BRS-overall and -up were lower after exercising in a hot compared to a temperate environment, however, these BRS metrics were not associated with PEH. In combination, these findings indicate greater PEH observed after exercising in a hot compared to a temperate environment may be partially explained by greater sweat loss, higher CVC, and lower HRV.

In the present study, we observed greater PEH after exercising in a hot compared to a temperate environment (**Figure 2**), which is consistent with the only other study to have

303 examined PEH after exercising in the heat in untrained individuals (Cunha et al., 2020). In these
304 studies, PEH was greater after exercising in the heat, despite the absolute exercise workload
305 being lower than whilst exercising in a temperate environment. Exercise intensity and duration
306 were similar in these studies (75%HRmax ~60%VO_2max and 35–40 min). The exercise
307 completed in the current study has good ecological validity. It is consistent with typical exercise
308 training where most individuals exercise for a set duration at a relative exercise intensity that is
309 perceptually regulated or regulated by HR. In addition, when environmental temperature is
310 increased, perceptual regulation usually leads to a reduction in absolute exercise workload.
311 Currently, it is unknown if exercising at the same absolute workload in a hot and a temperate
312 environment (greater relative exercise intensity in the heat) leads to similar PEH. Based on
313 previous studies to have examined PEH after aerobic exercise at different relative exercise
314 intensities in temperate environments, it might be hypothesized that a similar magnitude of PEH
315 should be expected in untrained persons if the absolute workload elicits a relative exercise
316 intensity of 50% VO_2peak or greater (Forjaz et al., 2004; MacDonald et al., 1999; Rossow et
317 al., 2010).

318 The greater PEH after exercising in a hot compared to a temperate environment
319 observed in the current study is consistent with one previous study (Cunha et al., 2020);
320 however, it contrasts the findings of another that reported no difference (Lynn et al., 2009).
321 One explanation for the opposing findings in these previous studies is the difference in training

status of the participants. In contrast to the endurance-trained participants in the study by Lynn et al. (2009) those in our study and that by Cunha et al. (2020) were inactive and untrained. Based on a comparison of the current study and that by Lynn and colleagues (2009) it is possible to speculate that the untrained experience PEH because their body temperature and CVC remained elevated throughout the 60 min recovery, and this prolonged demand for heat dissipation led a prolonged reduction in peripheral resistance, which is cited as the principal mechanism for PEH in most studies (Brito et al., 2014). In contrast, due to more effective heat dissipation mechanisms developed via regular exercise training (McIntyre et al., 2022; Ravanelli et al., 2021), body temperature and CVC recovered toward pre-exercise values within 60 minutes in the trained, and they did not suffer PEH (Lynn et al., 2009). In combination, these studies suggest that after exercising in the heat inactive and untrained men experience greater PEH than endurance-trained individuals. PEH is related to syncope risk, and therefore, inactive and untrained people may be at an increased risk of syncope and falls after exercising in the heat.

The causes of PEH are multifactorial and may include alterations in thermoregulatory and cardiovascular mechanisms. Skin blood flow, CVC, and sweat loss were greater on HOT than on CON, and sweat loss and CVC changes were associated with PEH after exercising in a hot environment. Greater CVC after exercising in a hot than a temperate environment was necessary to match the greater demands for heat dissipation caused by the greater body

temperature (**Figure 2**). The higher body temperature and greater heat dissipation demands throughout recovery likely led to the greater reduction in peripheral vascular resistance and therefore PEH. Although we did not evaluate total vascular conductance, it is worth considering the influences of vascular conductance in each tissue e.g., brain, internal organs, and skin. Animal research has demonstrated that heating significantly decreases the vascular conductance of the kidney, stomach, small and large intestines, and pancreas (Kenney and Musch, 2004). Regarding vascular responses in the carotid arteries, which inflow blood into the brain and face, hot environments tend to elicit lower vascular conductance for the internal carotid and vertebral arteries but a higher conductance for the external carotid artery, resulting in increased vascular conductance of the facial skin (Sato et al., 2016). These findings suggest that in a hot environment, vascular conductance in the brain and internal organs are unlikely to strongly affect PEH (Kenney and Musch, 2004; Sato et al., 2016), whereas cutaneous vascular conductance does influence PEH (Sato et al., 2016).

During the 1 h recovery period in the current study, HR was higher and several of the HRV and cBRS indices remained lower after exercising in a hot compared to a temperate environment (**Figure 2**, **Tables 1 and 2**). These findings are consistent with previous studies (Cunha et al., 2015; Cunha et al., 2020; Fonseca et al., 2018) that indicate heat stress causes a shift in cardiac autonomic balance, i.e., increased cardiac sympathetic activity and decreased cardiac parasympathetic activity. The most likely reason for the higher HR, and lower HRV,

after exercise in the heat, is to support a higher cardiac output requirement, which is necessary to aid heat dissipation and restore core temperature to resting normothermia (Flouris et al., 2014). The current study also observed significant relationships between changes in MAP and RMSSD and log [HF] after exercising in the heat. However, no relationships were observed between changes in MAP and BRS indices, which indicates that other mechanisms may be responsible for PEH after exercising in the heat. The HRV indices suggest enhanced cardiac sympathetic activity, which may be important to maintain BP in the presence of elevated skin blood flow and dehydration from sweat loss (Crandall et al., 2000; Kinugasa and Hirayanagi, 1999), and/or water restriction-induced greater dehydration (Schwabe et al., 2007). Indeed, a recent study found that changes in MAP post-exercise were significantly related to the changes in plasma volume in thermoneutral and hot environments (Cunha et al., 2020).

Methodological considerations

Some limitations should be considered when interpreting our results. First, our measurement of CVC was limited to one site of non-glabrous skin (chest). While non-glabrous skin comprises 95% of the body surface area and exhibits uniform thermoregulatory responses, we cannot exclude the effects of other sites of both non-glabrous and glabrous skin on PEH (Wilkins et al., 2004). Second, we estimated sympathetic nerve activity using the HRV sequence technique. While this technique is non-invasive and thus has been widely used,

dissociations have been found between HRV indices and sympathetic nerve activity measured directly by microneurography (McGowan et al., 2009; Notarius et al., 1999). Third, only male individuals participated in this and previous studies examining the effect of exercising environmental temperature on PEH (Cunha et al., 2020; Lynn et al., 2009) and thus future studies should be completed to determine the effect of exercising in the heat on PEH in females. This is pertinent as although a similar magnitude of PEH has been typically observed in men and women following aerobic exercise in thermoneutral conditions, the primary physiological determinants of PEH, i.e., reduced peripheral vascular resistance or cardiac output, be different between men and women (Brito et al., 2014; Queiroz et al., 2013; Rossow et al., 2010). Furthermore, alterations in female sex hormones, body temperature and thermoregulation, across the menstrual cycle, pre-and post-menopause, and with hormone-replacement therapy, may consequently influence PEH (Barnes and Charkoudian, 2021; Hutchins et al., 2021).

Perspectives

One cardiovascular health benefit of regular exercise training is to lower BP in those with pre-hypertension and hypertension (Cornelissen and Smart, 2013). The magnitude of the fall in resting BP caused by chronic exercise training is associated with BP reductions with

acute exercise (Hecksteden et al., 2013; Liu et al., 2012). The greater reduction in blood pressure after exercising in a hot compared to a temperate environment highlighted in the current study, and in one previous study that included men with elevated resting blood pressure (Cunha et al., 2020), suggests that chronic exercise training in the heat, even at lower absolute workloads, may lead to greater BP lowering effects than chronic exercise training in temperate conditions. Future studies testing this notion will require careful management of participants as severe PEH is related to the risk of syncope (Mundel et al., 2015), and heat stress predispose individuals to syncope (Crandall and Gonzalez-Alonso, 2010; Pearson et al., 2013). Indeed, our findings, in combination with previous research (Cunha et al., 2020), suggest that inactive and untrained people may be at increased risk of syncope and falls after exercising in the heat. Lastly, our findings that highlight PEH is associated with sweat loss and CVC helps to identify possible countermeasures, such as off-setting sweat loss with adequate fluid intake and cooling the surface of the skin, to reduce PEH, syncope and falls in hot environments (Lynn et al., 2009; Wilson et al., 2002).

Conclusion

Post-exercise hypotension was greater after exercise in a hot compared to a temperate environment in untrained healthy men. This greater PEH may be partially explained by greater sweat loss and CVC, and lower HRV.

Statements and Declarations

Funding

This work was supported in part by the Japan Society for the Promotion of Science (JSPS, KAKENHI, Number; 26440268 to M.H.).

Conflicts of interest/Competing interests (include appropriate disclosures)

No conflict of interest, financial or otherwise, is declared by the authors.

Acknowledgements

We appreciate the time and effort spent by our volunteers. We also thank Ms Junko Endo and Ms Misato Watanabe for their technical assistance.

References

Barnes, J.N., Charkoudian, N., 2021. Integrative cardiovascular control in women: Regulation of blood pressure, body temperature, and cerebrovascular responsiveness. FASEB J 35, e21143.

Brito, L.C., Queiroz, A.C., Forjaz, C.L., 2014. Influence of population and exercise protocol characteristics on hemodynamic determinants of post-aerobic exercise hypotension. Braz J Med Biol Res 47, 626-636.

Carrington, C.A., White, M.J., 2001. Exercise-induced muscle chemoreflex modulation of spontaneous baroreflex sensitivity in man. J Physiol 536, 957-962.

Cornelissen, V.A., Smart, N.A., 2013. Exercise training for blood pressure: a systematic review and meta-analysis. J Am Heart Assoc 2, e004473.

Crandall, C.G., Gonzalez-Alonso, J., 2010. Cardiovascular function in the heat-stressed human. Acta Physiol (Oxf) 199, 407-423.

Crandall, C.G., Zhang, R., Levine, B.D., 2000. Effects of whole body heating on dynamic baroreflex regulation of heart rate in humans. Am J Physiol Heart Circ Physiol 279, H2486-2492.

Cunha, F.A., Farinatti, P., Jones, H., Midgley, A.W., 2020. Postexercise hypotension and related hemodynamic responses to cycling under heat stress in untrained men with elevated blood pressure. Eur J Appl Physiol 120, 1001-1013.

Cunha, F.A., Midgley, A.W., Soares, P.P., Farinatti, P.T., 2015. Postexercise hypotension after maximal short-term incremental exercise depends on exercise modality. Appl Physiol Nutr Metab 40, 605-614.

Eckberg, D.L., 1980. Nonlinearities of the human carotid baroreceptor-cardiac reflex. Circ Res 47, 208-216.

Flouris, A.D., Bravi, A., Wright-Beatty, H.E., Green, G., Seely, A.J., Kenny, G.P., 2014. Heart rate variability during exertional heat stress: effects of heat production and treatment. Eur J Appl Physiol 114, 785-792.

Fonseca, G.F., Farinatti, P.T.V., Midgley, A.W., Ferreira, A., de Paula, T., Monteiro, W.D., Cunha, F.A., 2018. Continuous and Accumulated Bouts of Cycling Matched by Intensity and Energy Expenditure Elicit Similar Acute Blood Pressure Reductions in Prehypertensive Men. J Strength Cond Res 32, 857-866.

Forjaz, C.L., Cardoso, C.G., Jr., Rezk, C.C., Santaella, D.F., Tinucci, T., 2004. Postexercise hypotension and hemodynamics: the role of exercise intensity. J Sports Med Phys Fitness 44, 54-62.

Gomes Anunciacao, P., Doederlein Polito, M., 2011. A review on post-exercise hypotension in hypertensive individuals. Arq Bras Cardiol 96, e100-109.

Halliwill, J.R., 2001. Mechanisms and clinical implications of post-exercise hypotension in humans. Exerc Sport Sci Rev 29, 65-70.

Halliwill, J.R., Sieck, D.C., Romero, S.A., Buck, T.M., Ely, M.R., 2014. Blood pressure regulation X:

what happens when the muscle pump is lost? Post-exercise hypotension and syncope. Eur J Appl Physiol 114, 561-578.

Halliwill, J.R., Taylor, J.A., Eckberg, D.L., 1996. Impaired sympathetic vascular regulation in humans after acute dynamic exercise. J Physiol 495 (Pt 1), 279-288.

Hecksteden, A., Grutters, T., Meyer, T., 2013. Association between postexercise hypotension and long-term training-induced blood pressure reduction: a pilot study. Clin J Sport Med : official journal of the Can Acad Sport Med 23, 58-63.

Horiuchi, M., Endo, J., Dobashi, S., Kiuchi, M., Koyama, K., Subudhi, A.W., 2016. Effect of progressive normobaric hypoxia on dynamic cerebral autoregulation. Exp Physiol 101, 1276-1284.

Horiuchi, M., Fukuoka, Y., 2019. Absence of cardiovascular drift during prolonged arm-crank exercise in individuals with spinal cord injury. Spinal Cord 57, 942-952.

Horiuchi, M., Thijssen, D.H.J., 2020. Ischemic preconditioning prevents impact of prolonged sitting on glucose tolerance and markers of cardiovascular health but not cerebrovascular responses. Am J Physiol Endocrinol Metab 319, E821-E826.

Hutchins, K.P., Borg, D.N., Bach, A.J.E., Bon, J.J., Minett, G.M., Stewart, I.B., 2021. Female (Under) Representation in Exercise Thermoregulation Research. Sports Med Open 7, 43.

Iellamo, F., Hughson, R.L., Castrucci, F., Legramante, J.M., Raimondi, G., Peruzzi, G., Tallarida, G., 1994. Evaluation of spontaneous baroreflex modulation of sinus node during isometric exercise in healthy humans. Am J Physiol 267, H994-1001.

Kenney, M.J., Musch, T.I., 2004. Senescence alters blood flow responses to acute heat stress. Am J Physiol Heart Circ Physiol 286, H1480-1485.

Kinugasa, H., Hirayanagi, K., 1999. Effects of skin surface cooling and heating on autonomic nervous activity and baroreflex sensitivity in humans. Exp Physiol 84, 369-377.

Lakens, D., 2013. Calculating and reporting effect sizes to facilitate cumulative science: a practical primer for t-tests and ANOVAs. Front Psychol 4, 863.

Liu, S., Goodman, J., Nolan, R., Lacombe, S., Thomas, S.G., 2012. Blood pressure responses to acute and chronic exercise are related in prehypertension. Med Sci Sports Exerc 44, 1644-1652.

Lynn, B.M., Minson, C.T., Halliwill, J.R., 2009. Fluid replacement and heat stress during exercise alter post-exercise cardiac haemodynamics in endurance exercise-trained men. J Physiol 587, 3605-3617.

MacDonald, J., MacDougall, J., Hogben, C., 1999. The effects of exercise intensity on post exercise hypotension. J Hum Hypertens 13, 527-531.

Malliani, A., Pagani, M., Lombardi, F., Cerutti, S., 1991. Cardiovascular neural regulation explored in the frequency domain. Circulation 84, 482-492.

McGowan, C.L., Swiston, J.S., Notarius, C.F., Mak, S., Morris, B.L., Picton, P.E., Granton, J.T., Floras, J.S., 2009. Discordance between microneurographic and heart-rate spectral indices of sympathetic activity in pulmonary arterial hypertension. Heart 95, 754-758.

McIntyre, R.D., Zurawlew, M.J., Mee, J.A., Walsh, N.P., Oliver, S.J., 2022. A comparison of medium-term heat acclimation by post-exercise hot water immersion or exercise in the heat: adaptations, overreaching, and thyroid hormones. Am J Physiol. Reg Integr Comp Physiol 323, R601-R615.

Mundel, T., Perry, B.G., Ainslie, P.N., Thomas, K.N., Sikken, E.L., Cotter, J.D., Lucas, S.J., 2015. Postexercise orthostatic intolerance: influence of exercise intensity. Exp Physiol 100, 915-925.

Notarius, C.F., Butler, G.C., Ando, S., Pollard, M.J., Senn, B.L., Floras, J.S., 1999. Dissociation between microneurographic and heart rate variability estimates of sympathetic tone in normal subjects and patients with heart failure. Clin Sci (Lond) 96, 557-565.

Ogoh, S., Fisher, J.P., Dawson, E.A., White, M.J., Secher, N.H., Raven, P.B., 2005. Autonomic nervous system influence on arterial baroreflex control of heart rate during exercise in humans. J Physiol 566, 599-611.

Pearson, J., Lucas, R.A., Crandall, C.G., 2013. Elevated local skin temperature impairs cutaneous vasoconstrictor responses to a simulated haemorrhagic challenge while heat stressed. Exp Physiol 98, 444-450.

Queiroz, A.C., Rezk, C.C., Teixeira, L., Tinucci, T., Mion, D., Forjaz, C.L., 2013. Gender influence on post-resistance exercise hypotension and hemodynamics. Int J Sports Med 34, 939-944.

Ravanelli, N., Gagnon, D., Imbeault, P., Jay, O., 2021. A retrospective analysis to determine if exercise training-induced thermoregulatory adaptations are mediated by increased fitness or heat acclimation. Exp Physiol 106, 282-289.

Roberts, M.F., Wenger, C.B., Stolwijk, J.A., Nadel, E.R., 1977. Skin blood flow and sweating changes following exercise training and heat acclimation. J Appl Physiol Respir Environ Exerc Physiol 43, 133-137.

Rossow, L., Yan, H., Fahs, C.A., Ranadive, S.M., Agiovlasitis, S., Wilund, K.R., Baynard, T., Fernhall, B., 2010. Postexercise hypotension in an endurance-trained population of men and women following high-intensity interval and steady-state cycling. Am J Hypertens 23, 358-367.

Sato, K., Oue, A., Yoneya, M., Sadamoto, T., Ogoh, S., 2016. Heat stress redistributes blood flow in arteries of the brain during dynamic exercise. J Appl Physiol 120, 766-773.

Schwabe, L., Szinnai, G., Keller, U., Schachinger, H., 2007. Dehydration does not influence cardiovascular reactivity to behavioural stress in young healthy humans. Clin Physiol Funct Imaging 27, 291-297.

Senitko, A.N., Charkoudian, N., Halliwill, J.R., 2002. Influence of endurance exercise training status and gender on postexercise hypotension. J Appl Physiol 92, 2368-2374.

Studinger, P., Goldstein, R., Taylor, J.A., 2007. Mechanical and neural contributions to hysteresis in the cardiac vagal limb of the arterial baroreflex. J Physiol 583, 1041-1048.

Wilkins, B.W., Minson, C.T., Halliwill, J.R., 2004. Regional hemodynamics during postexercise

hypotension. II. Cutaneous circulation. J Appl Physiol 97, 2071-2076.
Willie, C.K., Ainslie, P.N., Taylor, C.E., Jones, H., Sin, P.Y., Tzeng, Y.C., 2011. Neuromechanical features of the cardiac baroreflex after exercise. Hypertension 57, 927-933.
Wilson, T.E., Cui, J., Zhang, R., Witkowski, S., Crandall, C.G., 2002. Skin cooling maintains cerebral blood flow velocity and orthostatic tolerance during tilting in heated humans. J Appl Physiology 93, 85-91.

Figure legends

Figure 1. Experimental protocol. CON, control trial; HOT, hot trial; BP, blood pressure; HR, heart rate; HRV, heart rate variability; BRS, baroreflex sensitivity; SkBF, skin blood flow; T_{sk}, skin temperature; T_{core}, core temperature.

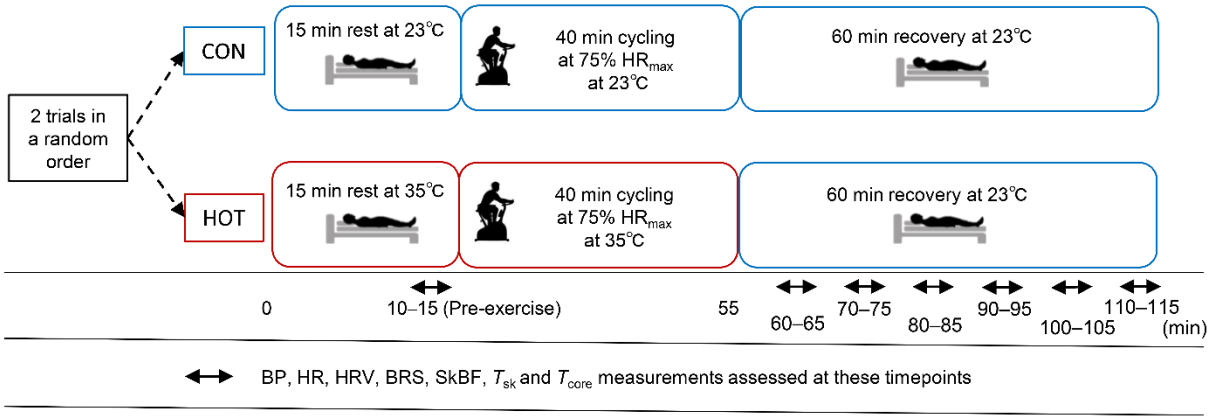

Figure 2. Systolic blood pressure (SBP; panel A), diastolic blood pressure (DBP; panel B), mean arterial pressure (MAP; panel C), and heart rate (HR; panel D) during a 1 h recovery period after exercising in a hot (35°C, HOT) compared to a temperate environment (23°C, CON). The blue and red circles indicate mean values in CON and HOT. Values are mean ± standard deviation (SD). * Significant difference compared with the baseline in each condition (blue or red marks). † Significant differences between CON and HOT within the same period.

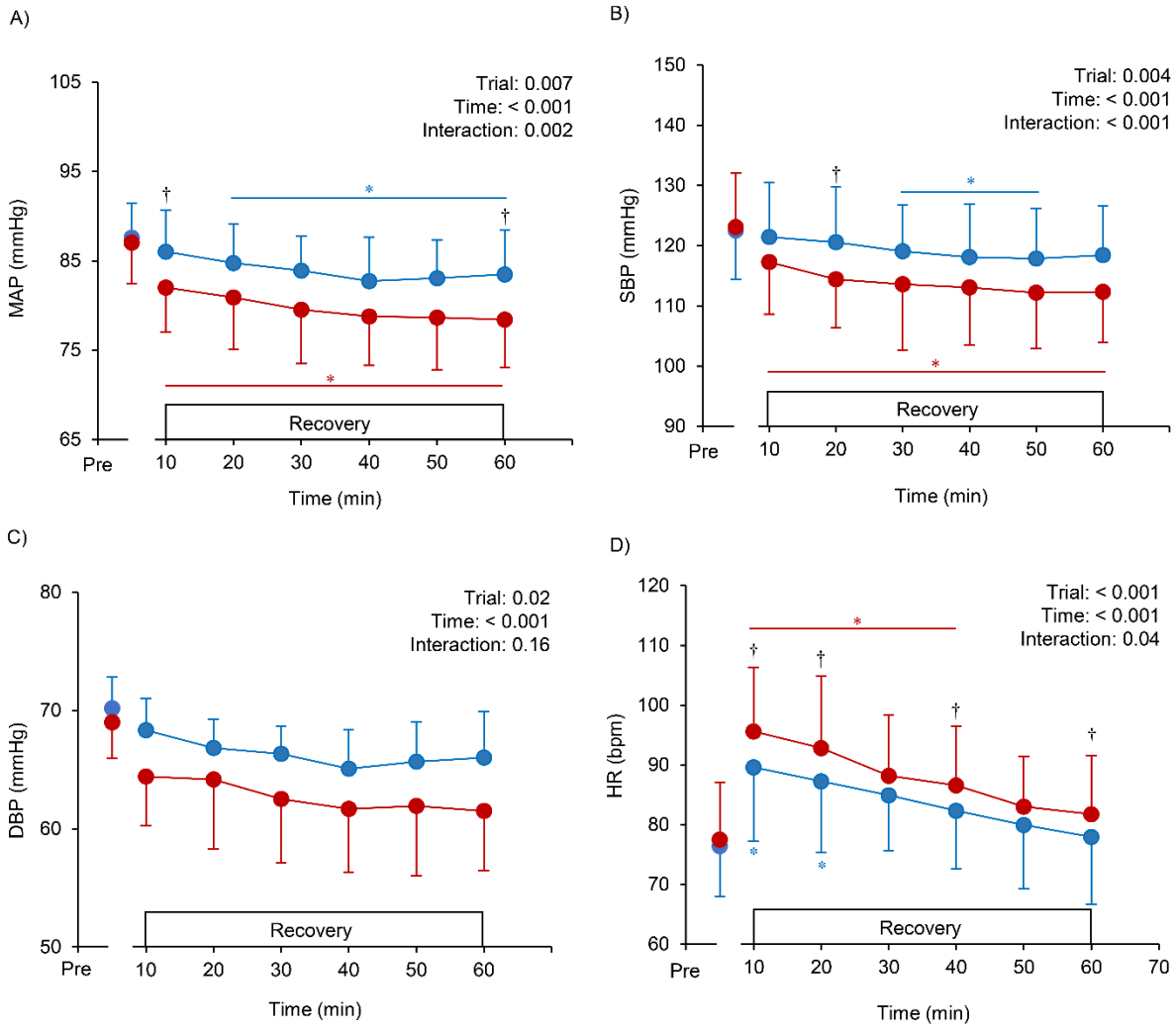

Figure 3. Core temperature (T_{core}; panel A), mean skin temperature (T_{sk}; panel B), relative changes from resting baseline in skin blood flow (SkBF; panel C) and cutaneous vascular conductance (CVC; panel D) during a 1 h recovery period after exercising in a hot (35°C, HOT) compared to a temperate environment (23°C, CON). The blue and red circles indicate mean values in CON and HOT. Values are mean ± standard deviation (SD). * Significant difference compared with the baseline in each condition (blue or red marks). † Significant differences between CON and HOT within the same period.

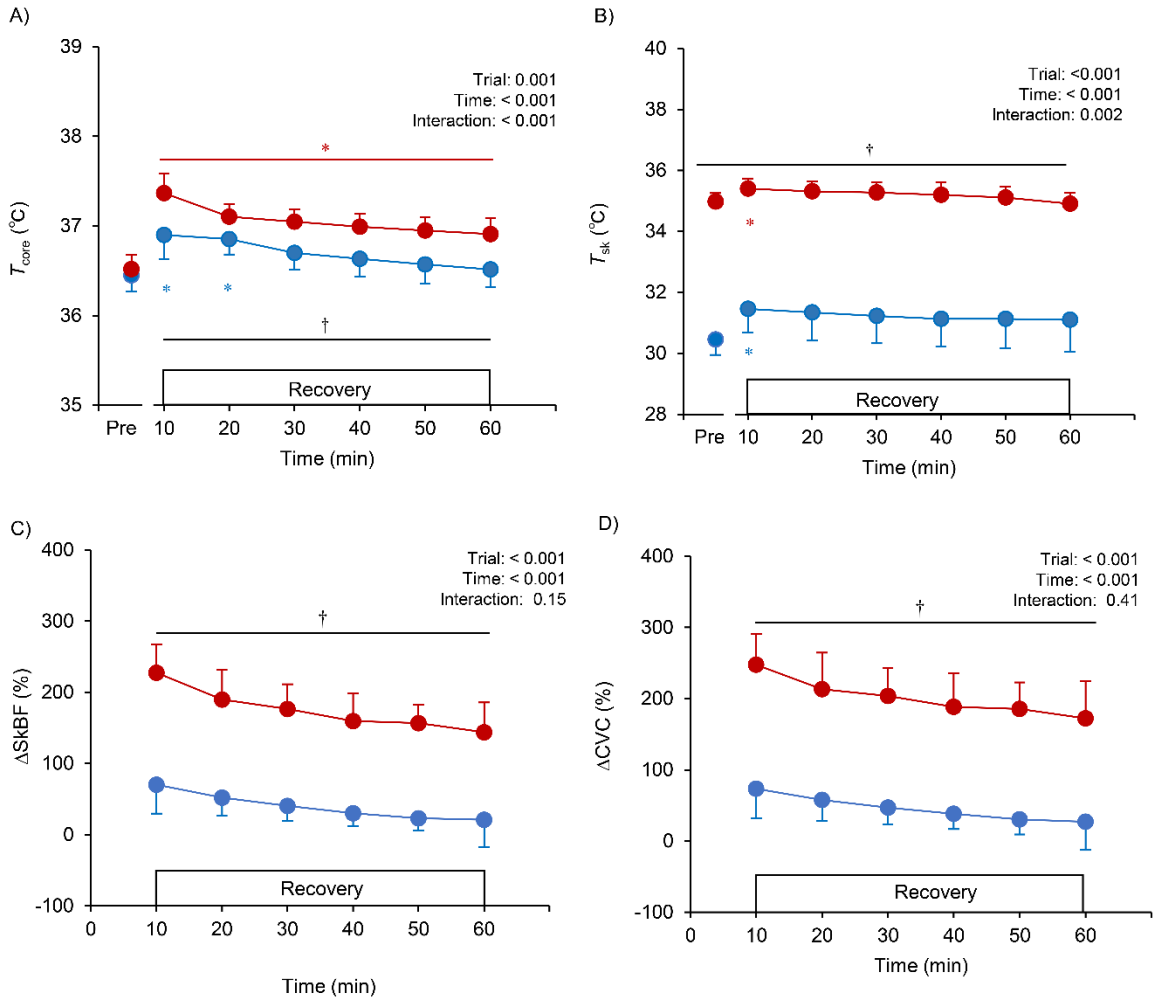

Table 1. Summarized results of cardiac autonomic function between the control (CON) and hot (HOT) trials.

		Recovery period							ANOVA results		
		Pre-exercise	10 min	20 min	30 min	40 min	50 min	60 min		Condition	Time	Interaction
SDNN	CON	58 (9)	35 (14) *	31 (7) *	35 (8) *	42 (13) *	46 (12) *	50 (15) *	F	13.5	30.3	2.8
(ms)	HOT	54 (13)	22 (7) *†	31 (9) *	36 (11) *	38 (14) *	38 (10) *	38 (13) *†	*P*	0.004	<0.001	0.02
									η^2	0.04	0.36	0.03
RMSDD	CON	42 (9)	16 (5) *	19 (4) *	24 (8) *	27 (9) *	29 (10) *	35 (11) *	F	17.9	53.7	2.7
(ms)	HOT	36 (11)	15 (6) *	16 (7) *	20 (9) *	22 (10) *	23 (11) *	25 (11) *†	*P*	0.001	<0.001	0.02
									η^2	0.05	0.41	0.01
Log [HF]	CON	2.50 (0.14)	1.82 (0.24) *	2.00 (0.26) *	2.05 (0.27) *	2.19 (0.30) *	2.19 (0.33) *	2.34 (0.32) *	F	5.2	24.3	2.9
(ms^2)	HOT	2.30 (0.35)	1.82 (0.37) *	1.82 (0.49) *	1.85 (0.51) *	1.96 (0.43) *	1.87 (0.62) *	1.96 (0.52) *	*P*	0.04	<0.001	0.01
									η^2	0.06	0.17	0.02
Log [LF/HF]	CON	0.22 (0.15)	0.53 (0.13) *	0.53 (0.14) *	0.50 (0.18) *	0.41 (0.20) *	0.41 (0.18) *	0.36 (0.17) *	F	19.2	26.8	1.2
(a.u.)	HOT	0.36 (0.23)	0.70 (0.16) *	0.67 (0.16) *†	0.61 (0.20) *	0.63 (0.26) *	0.64 (0.27) *†	0.55 (0.25)	*P*	0.001	<0.001	0.31
									η^2	0.14	0.20	0.008

Values are mean (SD). SDNN, standard deviation of the normal-to-normal intervals; RMSSD, root-mean-square of successive differences in R-R interval; HF, high frequency; LF, low frequency; a.u., arbitrary unit; ES, effect size. Note that *$P < 0.05$ vs. pre-exercise within the same trial, †$P < 0.05$ between CON and HOT at the same time point.

Table 2. Summarized results of cardiac baroreflex sensitivity (cBRS) between the control (CON) and hot (HOT) trials.

		Recovery period								ANOVA results		
		Pre-exercise	10 min	20 min	30 min	40 min	50 min	60 min		Condition	Time	Interaction
cBRS-up	CON	17.9 (3.2)	8.3 (2.1)*	11. 0 (4.9)*	13.0 (3.3)*	13.4 (4.5)*	12.1 (3.5)*	13.4 (3.1)*	F	48.4	32.0	2.3
(ms·mmHg^{-1})	HOT	16.1 (2.4)	6.1 (2.7)*	6.6 (1.9)*†	8.0 (2.0)*†	8.1 (2.6)*†	8.0 (1.5)*†	8.0 (3.5)*†	*P*	<0.001	<0.001	0.04
									η^2	0.19	0.38	0.02
cBRS-down	CON	16.4 (3.4)	8.8 (3.8)*	8.8 (4.8)*	11.1 (5.0)	9.6 (3.4)*	10.7 (4.2)*	10.6 (5.0)	F	40.9	34.5	1.8
(ms·mmHg^{-1})	HOT	15.0 (2.5)	4.6 (1.8)*	4.6 (2.1)*	6.6 (2.7)*	6.2 (2.8)*	7.3 (2.8)*	5.2 (2.2)*	*P*	<0.001	<0.001	0.10
									η^2	0.15	0.36	0.01
cBRS-overall	CON	17.4 (2.9)	8.9 (2.3)*	9.5 (3.9)*	12.0 (3.4)*	11.4 (3.2)*	10.8 (2.2)*	11.8 (4.3)*	F	105.6	54.0	2.6
(ms·mmHg^{-1})	HOT	15.5 (2.4)	5.4 (2.0)*†	5.2 (1.8)*†	7.1 (1.9)*†	6.9 (2.5)*†	7.6 (2.3)*†	6.6 (2.5)*†	*P*	<0.001	<0.001	0.02
									η^2	0.20	0.43	0.02

Values are mean (SD). Note that *$P < 0.05$ vs. pre-exercise within the same trial, †$P < 0.05$ between CON and HOT at the same time point.