

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

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Journal of Thermal Biology

DOI:

[10.1016/j.jtherbio.2023.103683](https://doi.org/10.1016/j.jtherbio.2023.103683)

Published: 01/10/2023

Peer reviewed version

[Cyswllt i'r cyhoeddiad / Link to publication](#)

Dyfyniad o'r fersiwn a gyhoeddwyd / Citation for published version (APA):

Horiuchi, M., & Oliver, S. (2023). Greater post-exercise hypotension in healthy young untrained men after exercising in a hot compared to a temperate environment. *Journal of Thermal Biology*, 117, Article 103683. <https://doi.org/10.1016/j.jtherbio.2023.103683>

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

3

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16 Word count: 4259

17 Number of references: 46

18 Number of tables: 2

19 Number of figures: 3 (all color)

20 **Abstract**

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

39 conductance, and lower HRV.

40 **Keywords:** *cardiac baroreflex sensitivity, cardiac autonomic function, cutaneous vascular*

41 *conductance, hemodynamics, skin blood flow, vasodilation*

42

43 **Abbreviations**

44 ANOVA: Analysis of variance

45 a.u.: arbitrary unit

46 BP: Blood pressure

47 cBRS: Cardiac baroreflex sensitivity

48 CI: Confidential interval

49 CON: Thermoneutral condition

50 CVC: Cutaneous vascular conductance

51 DBP: Diastolic blood pressure

52 η^2 : eta square

53 ES: effect size

54 HF: High frequency

55 HOT: Hot environment

56 HR: Heart rate

57 HRV: Heart rate variability

- 58 ISO: International Society for Organization
- 59 LF: Low frequency
- 60 MAP: Mean arterial pressure
- 61 PEH: Post-exercise hypotension
- 62 RMSSD: Root mean square of the successive differences in R-R interval
- 63 SBP: Systolic blood pressure
- 64 SD: Standard deviation
- 65 SDNN: Standard deviation of the normal-to-normal intervals
- 66 SkBF: Skin blood flow
- 67 T : Temperature
- 68 T_{arm} : Arm temperature
- 69 T_{chest} : Chest temperature
- 70 T_{core} : Sublingual temperature
- 71 T_{sk} : Mean skin temperature
- 72 T_{thigh} : Thigh temperature
- 73 $\dot{V}O_{2\text{max}}$: Maximal oxygen consumption
- 74 W: Watts

75 **Introduction**

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

93 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

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

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

132

133 **Methods**

134 *Ethical approval*

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

138

139 *Participants*

140 Twelve healthy untrained young men volunteered for this study (age 22 ± 3 years, height $173 \pm$
141 6 cm, body mass 65.71 ± 10.50 kg, mean \pm standard deviation). Volunteers were non-smokers,
142 who did not engage in regular physical activity, and had no prior history of orthopedic or
143 cardiovascular diseases, including hypertension, diabetes, and hyperlipidemia. After the study
144 procedures, possible risks, and benefits were explained, written informed consent to participate
145 in this study was obtained from each volunteer.

146

147 *Experimental procedures*

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

151 procedures including wearing devices; Visits 2 and 3 to complete two experimental trials
152 (control [CON] and hot [HOT]). The trial order was randomized and counterbalanced with at
153 least 1 week between trials. Participants' visits 2 and 3 were completed at the same time of day
154 (08:30–11:30 h) to avoid any circadian rhythm effects. On visits 2 and 3, participants drank a
155 bottle of water (300 ml) on arrival to the laboratory to promote hydration. All visits were
156 performed in a temperature and humidity-controlled environmental chamber (40% relative
157 humidity; TBR-4, 5SA2GX, Tabai Espec Co., Ltd., Tokyo, Japan). Participants completed two
158 experimental trials: (a) Temperate control (CON), which involved 15-min supine rest, 40-min
159 upright leg cycling exercise and a 60-min supine recovery at 23°C, and (b) Hot (HOT), which
160 involved 15-min supine rest and a 40-min upright leg cycling exercise at 35°C, followed by a
161 60-min supine recovery at 23°C. After exercise in the HOT, the chamber door was immediately
162 opened, and the thermostat was set to 23°C. The 40 min cycling exercise comprised of a 5 min
163 warm-up and then 35 minutes at a workload equivalent to 75% of individual heart rate (HR)
164 maximum. The warm-up began at 20 W and increased by 20 W every minute up to 100 W at 5
165 minutes. After the warm-up exercise workload was manually adjusted to maintain 75% of
166 individual HR maximum.

167

168 *Measurements*

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

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

174

175 *Central hemodynamics*

176 Throughout the study heart rate (HR) was recorded by a wireless HR monitor (Polar RC800X;
177 Polar Electro Japan, Tokyo, Japan) whilst HR variability (HRV) was assessed by another HR
178 monitoring device (Check-My-Heart, Daily Care BioMedical, Chungli, Taiwan). HRV was
179 measured and analyzed as described previously (Horiuchi and Thijssen, 2020). In brief,
180 electrodes were attached to the lower left rib and right clavicle, and a 5-minute
181 electrocardiogram was collected, and analyzed automatically by the HRV software in the time
182 and frequency domains (Daily Care BioMedical, Chungli, Taiwan). In the time domain, the
183 HRV was determined from the standard deviation of the normal-to-normal intervals (SDNN),
184 and the root mean square of the successive differences in R-R interval (RMSSD). In the
185 frequency domain, low-frequency oscillations (LF: 0.04–0.15 Hz), and high-frequency
186 oscillations (HF: 0.15–0.4 Hz) were quantified using a fast Fourier transformation (Horiuchi
187 and Thijssen, 2020; Malliani et al., 1991). Beat-by-beat BP was measured by applanation
188 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

208 segment at rest and during recovery (Carrington and White, 2001; Ogoh et al., 2005). Linear
209 regression was applied to each individual baroreflex sequence, with only sequences with an
210 $R^2 > 0.85$ accepted (Iellamo et al., 1994). When BP increases (upward slope) or decreases
211 (downward slope) more than 3 consecutive beats, they were defined as spontaneous “cBRS-up”
212 and “cBRS-down”. The overall average slope of the SBP–RR interval was calculated as
213 spontaneous “cBRS-overall”.

214

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

$$216 \quad T_{sk} = 0.25 \cdot T_{arm} + 0.43 \cdot T_{chest} + 0.32 \cdot T_{thigh}$$

217

218 To calculate CVC for each trial (CON and HOT), we used absolute raw values of SkBF
219 ($_{abs.}SkBF$) data represented as mV, and divided $_{abs.}SkBF$ by MAP, ($_{abs.}CVC$ as $mV \cdot mmHg^{-1}$).

220 Additionally, we calculated changes in these variables from pre-exercise resting values and
221 represented them as relative changes in SkBF (%) and CVC (%).

222

223 For the values of HRV, the normality of the data was checked by Bartlett and Levene test.

224 Logarithmic transformation was used for further HRV analyses if equal variances were not
225 found (Horiuchi and Thijssen, 2020).

226

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 \times Time
234 points) repeated measures ANOVA was used to compare all the physiological variables. BP
235 variables, HR, T_{core} , $T_{\text{sk, abs}}$, 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 ($\eta^2=0.01$), medium ($\eta^2=0.06$), and large ($\eta^2=0.14$) 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**

246 All twelve participants completed the study protocols. After the standard 5 min warm-up,
247 manual adjustment of the leg cycling workload (CON 141 ± 16 W, HOT 120 ± 14 W, Mean
248 difference [95% confidence interval], -22 [$-25, -19$] W) maintained exercising HR equivalent
249 to 75% of HR maximum on both CON and HOT (CON 148 ± 3 bpm, HOT 149 ± 3 bpm, 0.3
250 [$-0.6, 1.3$] bpm).

251

252 The time-course changes in central hemodynamics (MAP, SBP, diastolic BP [DBP], and HR)
253 between the two trials are shown in **Figure 2**. A significant interaction and main effects of time
254 and trial were observed in MAP and SBP (**Figures 2A and B**). Post-exercise hypotension was
255 greater after exercising in a hot than temperate environment as indicated by a lower mean
256 arterial pressure (MAP) at 60 min recovery (CON 83 ± 5 mmHg, HOT 78 ± 5 mmHg, -5 [$-8,$
257 -3] mmHg, **Figure 2A**). Compared with the pre-exercise resting values MAP decreased at 60
258 min recovery by 5% in CON (-4 [$-6, -2$] mmHg) and by 10% in HOT (-9 [$-10, -7$] mmHg).
259 Similarly, by 60 min recovery, SBP decreased 3% in CON (-4 [$-7, -2$] mmHg) and 9% in HOT
260 (-11 [$-13, -9$] mmHg, **Figure 2B**). Tukey posthoc test further revealed during recovery that
261 MAP at 10 and 60 min and SBP at 20 min in HOT were lower than in CON (all $P < 0.05$).
262 Significant time and trial main effects, but no interaction, were observed for DBP (**Figure 2C**).
263 Compared with the pre-exercise resting values and by 60 min recovery, DBP decreased 6% in
264 CON (-4 [$-6, -2$] mmHg) and 11% in HOT (-8 [$-10, -5$] mmHg, **Figure 2C**). An interaction

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

267

268 **Figure 3** shows the two trials' time-course changes in the thermoregulatory metrics (T_{core} , T_{sk} ,
269 relative changes in SkBF, and CVC). A significant interaction and main effects of time and trial
270 were observed (all $P < 0.05$) for T_{core} , T_{sk} (**Figure 3A and 3B**), $abs.SkBF$ and $abs.CVC$ (data not
271 shown). Regarding relative changes in SkBF and CVC, significant main effects of time and trial
272 were found (**Figures 3C and D**). During the 60 min recovery period, T_{core} and the cutaneous
273 circulation metrics in HOT were higher than in CON (all $P < 0.05$). A relationship was found
274 between changes in MAP and CVC in HOT ($r = 0.64$, $P = 0.03$) but not CON ($r = -0.46$, $P = 0.14$).
275 Sweat loss was greater in HOT than in CON (HOT -1.1 ± 0.2 kg, CON -0.5 ± 0.1 kg, $\Delta -0.6 [-$
276 $0.7, -0.5]$ kg). A relationship was observed between the changes in MAP and sweat loss in HOT
277 ($r = 0.66$, $P = 0.02$) but not CON ($r = 0.33$, $P = 0.30$).

278

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

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

289

290 **Discussion**

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

301 In the present study, we observed greater PEH after exercising in a hot compared to a
302 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%VO₂max 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% VO₂peak 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

322 status of the participants. In contrast to the endurance-trained participants in the study by Lynn
323 et al. (2009) those in our study and that by Cunha et al. (2020) were inactive and untrained.
324 Based on a comparison of the current study and that by Lynn and colleagues (2009) it is possible
325 to speculate that the untrained experience PEH because their body temperature and CVC
326 remained elevated throughout the 60 min recovery, and this prolonged demand for heat
327 dissipation led a prolonged reduction in peripheral resistance, which is cited as the principal
328 mechanism for PEH in most studies (Brito et al., 2014). In contrast, due to more effective heat
329 dissipation mechanisms developed via regular exercise training (McIntyre et al., 2022;
330 Ravanelli et al., 2021), body temperature and CVC recovered toward pre-exercise values within
331 60 minutes in the trained, and they did not suffer PEH (Lynn et al., 2009). In combination, these
332 studies suggest that after exercising in the heat inactive and untrained men experience greater
333 PEH than endurance-trained individuals. PEH is related to syncope risk, and therefore, inactive
334 and untrained people may be at an increased risk of syncope and falls after exercising in the
335 heat.

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

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

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

360 after exercise in the heat, is to support a higher cardiac output requirement, which is necessary
361 to aid heat dissipation and restore core temperature to resting normothermia (Flouris et al.,
362 2014). The current study also observed significant relationships between changes in MAP and
363 RMSSD and log [HF] after exercising in the heat. However, no relationships were observed
364 between changes in MAP and BRS indices, which indicates that other mechanisms may be
365 responsible for PEH after exercising in the heat. The HRV indices suggest enhanced cardiac
366 sympathetic activity, which may be important to maintain BP in the presence of elevated skin
367 blood flow and dehydration from sweat loss (Crandall et al., 2000; Kinugasa and Hirayanagi,
368 1999), and/or water restriction-induced greater dehydration (Schwabe et al., 2007). Indeed, a
369 recent study found that changes in MAP post-exercise were significantly related to the changes
370 in plasma volume in thermoneutral and hot environments (Cunha et al., 2020).

371

372 *Methodological considerations*

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

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

392

393 *Perspectives*

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

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

411

412 **Conclusion**

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

416 **Statements and Declarations**

417 **Funding**

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

420

421 **Conflicts of interest/Competing interests (include appropriate disclosures)**

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

423

424 **Acknowledgements**

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

427

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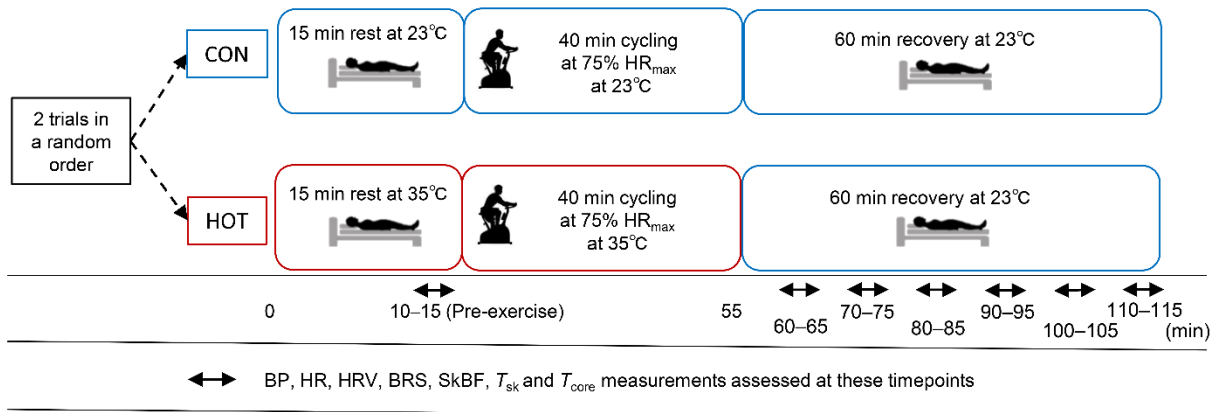
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548

549 **Figure legends**

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

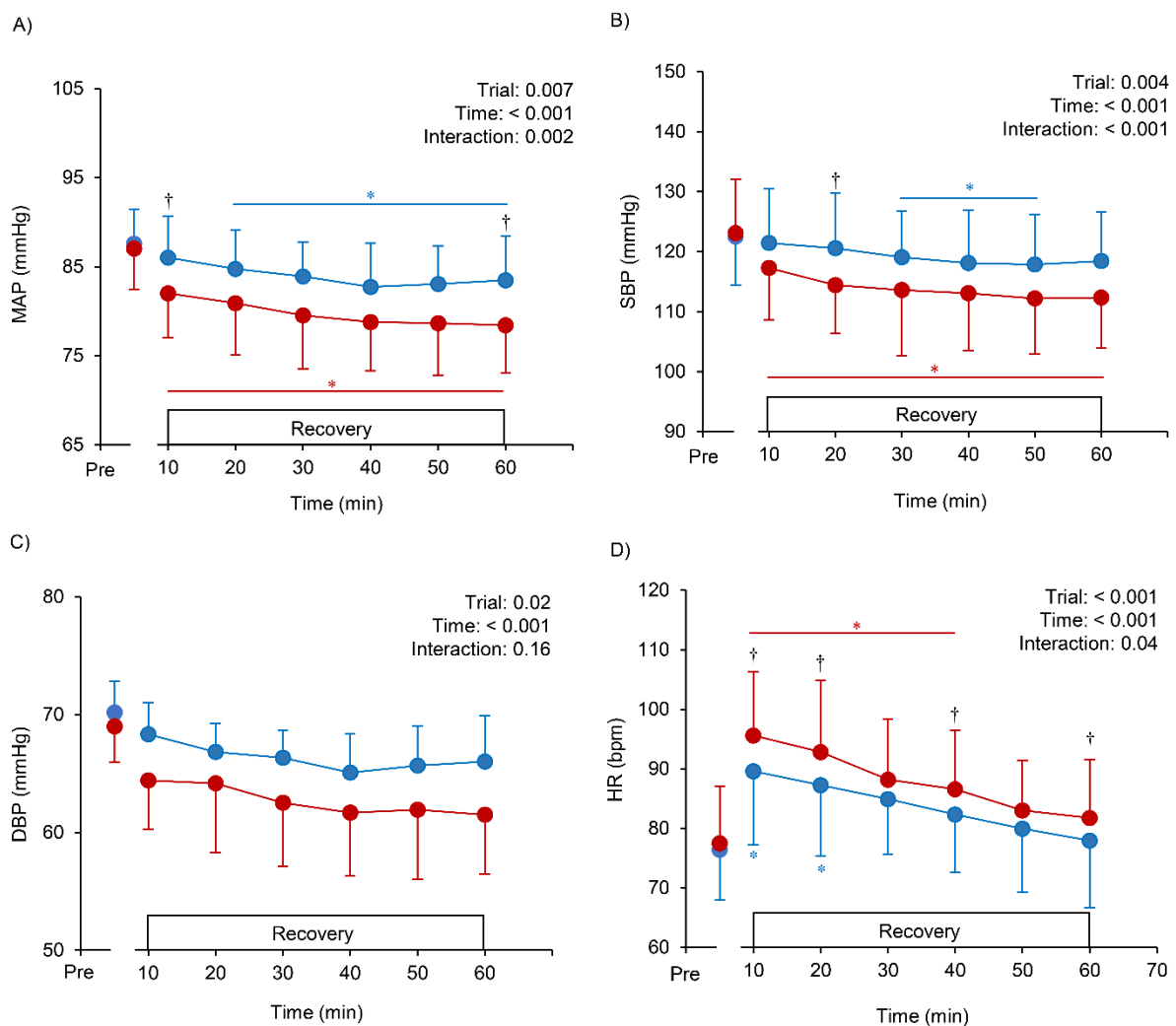


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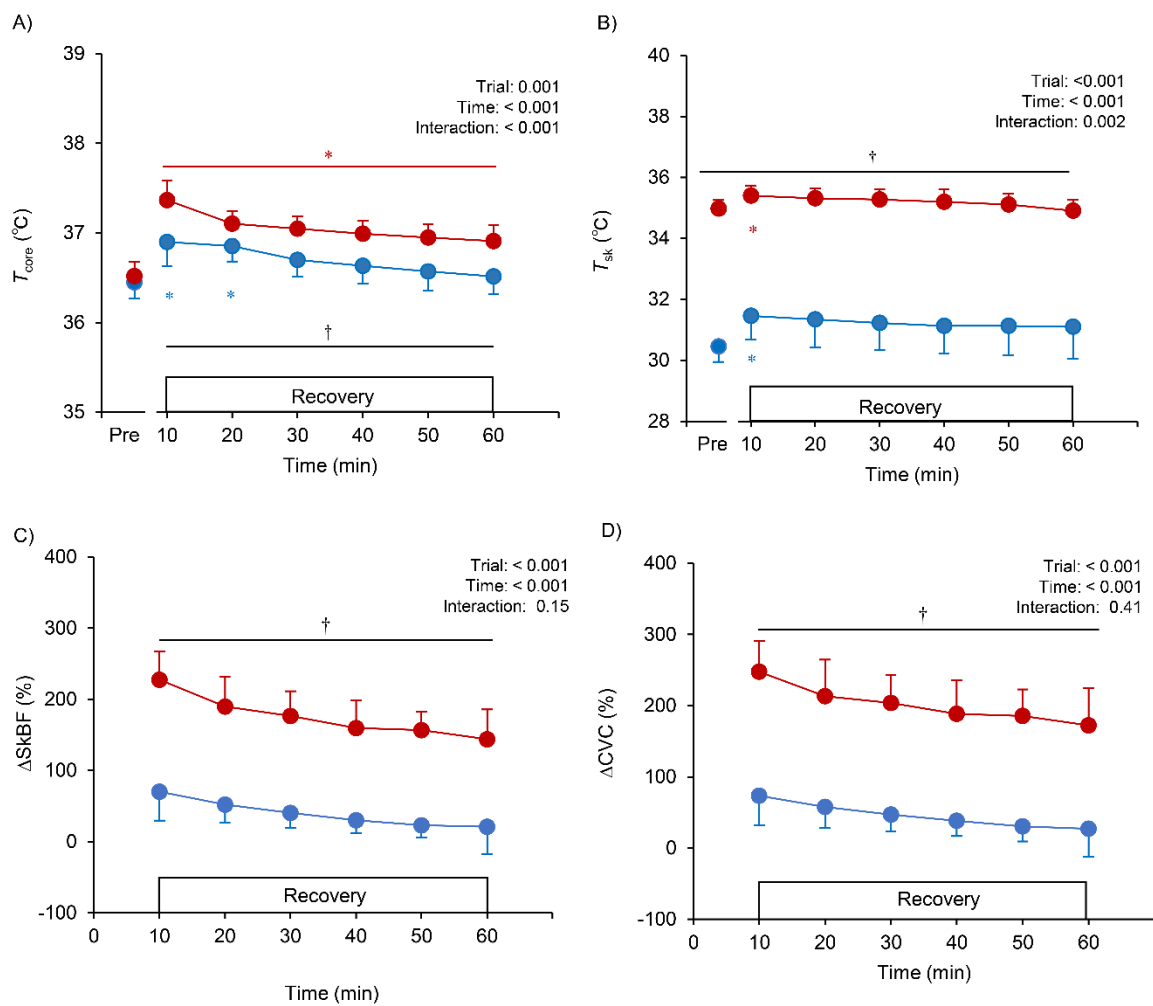
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556 **Figure 2.** Systolic blood pressure (SBP; panel A), diastolic blood pressure (DBP; panel B),
 557 mean arterial pressure (MAP; panel C), and heart rate (HR; panel D) during a 1 h recovery
 558 period after exercising in a hot (35°C, HOT) compared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



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



570

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 (ms)	CON	58 (9)	35 (14)*	31 (7)*	35 (8)*	42 (13)*	46 (12)*	50 (15)*	F	13.5	30.3	2.8
	HOT	54 (13)	22 (7)*†	31 (9)*	36 (11)*	38 (14)*	38 (10)*	38 (13)*†	<i>P</i>	0.004	<0.001	0.02
									η^2	0.04	0.36	0.03
RMSDD (ms)	CON	42 (9)	16 (5)*	19 (4)*	24 (8)*	27 (9)*	29 (10)*	35 (11)*	F	17.9	53.7	2.7
	HOT	36 (11)	15 (6)*	16 (7)*	20 (9)*	22 (10)*	23 (11)*	25 (11)*†	<i>P</i>	0.001	<0.001	0.02
									η^2	0.05	0.41	0.01
Log [HF] (ms ²)	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
	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)*	<i>P</i>	0.04	<0.001	0.01
									η^2	0.06	0.17	0.02
Log [LF/HF] (a.u.)	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
	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)	<i>P</i>	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; RMSDD, 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 (ms·mmHg ⁻¹)	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
	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) *†	<i>P</i>	<0.001	<0.001	0.04
									η^2	0.19	0.38	0.02
cBRS-down (ms·mmHg ⁻¹)	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
	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) *	<i>P</i>	<0.001	<0.001	0.10
									η^2	0.15	0.36	0.01
cBRS-overall (ms·mmHg ⁻¹)	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
	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) *†	<i>P</i>	<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.