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Effects of HIIT in Cool and Hot on Temperate Performance and Physiological Response in Trained Cyclists

Jason R. Boynton,¹ Jeremiah J. Peiffer,² and Chris R. Abbiss¹

¹School of Medical and Health Sciences, Edith Cowan University, Joondalup, Western Australia, Australia; and ²Centre for Healthy Ageing, Health Futures Institute, Murdoch University, Murdoch, Western Australia, Australia

Abstract

Boynton, JR, Peiffer, JJ, and Abbiss, CR. Effects of HIIT in cool and hot on temperate performance and physiological response in trained cyclists. *J Strength Cond Res* 39(3): e485–e495, 2025—This study investigated cardiopulmonary responses in hot and cool high-intensity interval training (HIIT) and the subsequent effects on time-trial (TT) performance and physiological responses in temperate conditions. Twenty trained cyclists were separated into 2 groups and completed a 4-week HIIT intervention (8 sessions, 5 × 4 minute) at an environmental temperature (T_A) of either 13° C (HIIT₁₃) or 35° C (HIIT₃₅). Cardiorespiratory data (e.g., heart rate [HR] and time above 90% $\dot{V}O_{2\max}$ [$T > 90\% \dot{V}O_{2\max}$]) were assessed for the first and last interval sessions. Subjects completed 20 km TTs in temperate conditions (22° C) before (TT1) and after (TT2) HIIT training, during which power output, HR, and thermoregulatory measures were recorded. $T > 90\% \dot{V}O_{2\max}$ was greater in HIIT₁₃ (875 ± 295 seconds; $p = 0.007$), compared with HIIT₃₅ (420 ± 395 seconds). Average power output during the TT increased from TT1 to TT2 in both groups (HIIT₁₃ $p = 0.023$, 3.3 ± 3.4%; HIIT₃₅ $p = 0.003$, 7.3 ± 6.3%) but with no significant interactions or differences between groups ($p = 0.115$; $p = 0.421$, respectively). Within-subject increases for HR and core temperature were observed during TT2 for HIIT₁₃ (2.7 ± 2.1%, 0.5 ± 0.6%) but not HIIT₃₅ (1.5 ± 4.9%, 0.0 ± 0.7%). After 4 weeks of HIIT, the improvement in temperate 20-km TT performance was not greater for HIIT₁₃ than HIIT₃₅, despite greater $T > 90\% \dot{V}O_{2\max}$ during 13° C HIIT vs. 35° C HIIT. Physiological responses (e.g., HR and thermoregulation) during the TT differed between HIIT₁₃ and HIIT₃₅, indicating varying adaptive responses.

Key Words: time-trial, cycling, gross mechanical efficiency, heat stress, high-intensity interval training, submaximal exercise

Introduction

High-intensity interval training (HIIT) stresses the oxygen transport and utilization systems (5), as a result of high cardiac load (1) and large motor unit recruitment (13). This stress stimulates adaptations important for maximizing endurance performance (e.g., increased maximal oxygen consumption [$\dot{V}O_{2\max}$]), ventilatory thresholds, anaerobic capacity (21,23), and gross efficiency (15)). It has been suggested that aerobic adaptations during HIIT are stimulated most effectively when time spent at or near $\dot{V}O_{2\max}$ is maximized (20,28,44). Indeed, given the potent stimulus and important benefits of HIIT for endurance athletes (21) and the relatively short duration of time high-intensity exercise is observed during training of these athletes (42), optimizing the conditions under which HIIT is performed could potentially enhance adaptation and endurance performance in a practical and efficient manner.

During exercise in hot environmental temperature (T_A), an increase in skin blood flow occurs (33) to increase dissipation of metabolic heat to the environment. It is believed that this shunting of blood to the skin reduces muscle blood flow causing an increase

in the heart rate (HR) for a given workload (33,48). Conversely, performing high-intensity intervals in cooler conditions increases the core temperature (T_C) to skin temperature (T_{sk}) gradient (4), reducing skin blood flow (40,48), increasing venous return, and therefore potentially enhancing the proportion of cardiac output delivered to the working muscles (40) and time spent at or near $\dot{V}O_{2\max}$. In addition, performing high-intensity intervals in cooler conditions would potentially result in improved locomotor muscle recruitment compared with intervals performed in hot conditions (43). High-intensity interval training in the heat may therefore induce central adaptations but compromise peripheral adaptations compared with HIIT performed in cool T_A . Indeed, evidence for symmorphosis between central and peripheral components of $\dot{V}O_{2\max}$ in trained endurance athletes (and humans in general) (50) implies that key peripheral aerobic adaptations stimulated by HIIT could be potentially improved by maximizing blood flow to the working muscles and (or) locomotor muscle recruitment. However, importantly, it is possible that attenuated peripheral stimulation during hot HIIT could be negated by the peripheral stimulation provided during high-volume low-intensity training characteristically performed by endurance athletes (2,42). To date, it is not well understood if differences in peripheral oxygen transport and utilization associated with hot and cool HIIT would affect subsequent endurance performance outcomes.

Endurance training performed in hot conditions has been demonstrated to increase performance in temperate conditions (9). This performance benefit is believed to be from heat

Address correspondence to Jason R. Boynton, jason@boyntoncoaching.com.

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acclimation (HA) adaptations (e.g., increases in plasma and improved sweat response) that attenuate thermal limiters and their negative effects on performance (9,22). Lorenzo et al. (22) observed greater increases in power output and $\dot{V}O_2\text{max}$ under temperate conditions with highly trained endurance athletes after 10 days in 40° C conditions at a submaximal intensity (2×45 minutes at 50% $\dot{V}O_2\text{max}$), compared with a group performing the same intervention in 13° C. Conversely, a study of similar design conducted by Keiser et al. (19) demonstrated no change in time-trial (TT) performance or $\dot{V}O_2\text{max}$ when cardiovascular strain during training was similar between groups (18° C vs. 38° C). Collectively, these results provide evidence that additional cardiovascular load caused by training in the heat is potentially a key component for stimulating adaptations that increase endurance performance in temperate conditions. It is plausible that the incorporation of heat stress with HIIT could induce greater cardiovascular adaptations than HIIT in cooler conditions, leading to improved performance in temperate conditions. Previous studies have investigated the effects of T_A on HIIT in healthy adults (41) and training interventions in endurance athletes (and consequently, the integrated HIIT sessions) (25–27). In addition, previous studies have investigated high-intensity exercise incorporated into HA protocols in endurance (36,39) and team-sport (34) athletes. Correspondingly, results for cardiovascular adaptations and performance outcomes in this body of research have been mixed. However, importantly, to our knowledge, previous research has not investigated T_A as an effect isolated to the HIIT component of endurance athlete training. Nor, to our knowledge, has previous research investigated the effects of T_A on HIIT sessions performed maximally and selected specifically for their elevated time at or near $\dot{V}O_2\text{max}$.

Several contradictory and complementary physiological factors can potentially affect endurance performance outcomes after HIIT in either hot or cool conditions. Therefore, the overall purpose of this study was to determine the effects 4 weeks of HIIT performed at 13° C or 35° C, incorporated into high-volume endurance training, would have on the performance and physiological responses of trained cyclists in temperate conditions (22° C). It was postulated that more time would be spent at or near $\dot{V}O_2\text{max}$ during HIIT at 13° C and this response would exceed the potential benefits of HA during HIIT at 35° C. Subsequently, it was hypothesized that HIIT performed in 13° C would result in an increase in power output during a simulated 20-km TT and gross efficiency during a submaximal test that would exceed the outcomes of HIIT performed in 35° C.

Methods

Experimental Approach to the Problem

The 2 groups of subjects partook in a 4-week HIIT intervention (8 sessions) at a T_A of either 13° C (HIIT₁₃) or 35° C (HIIT₃₅; Figure 1A), which was incorporated into their normal training routine (i.e., maintaining volume and frequency) and replaced any regular structured HIIT they may have performed. 13° C was selected as an intervention T_A for its likelihood to provide maximal power output and time at or near $\dot{V}O_2\text{max}$ during individual interval sessions (4,12). 35° C was selected as an intervention T_A for its likelihood to provide a thermal impulse high enough to stimulate HA. Performance and physiological data (described below) were collected during the first and last interval sessions (INT1 & INT8, respectively). On 3 separate days before the intervention, subjects completed a graded exercise test (GXT),

a familiarization TT, and baseline TT (TT1). The familiarization and TT1 were separated from each other and the first interval session by at least 48 hours. A final TT (TT2) was conducted ~7 days after the last interval session. Environmental conditions for the GXT and TT sessions were set at 22° C and 40% relative humidity. 22° C was selected as the TT test T_A because it was independent from the intervention temperatures (i.e., did not share the qualities noted above for 13° C and 35° C), is known for its qualities as an ambient temperature (i.e., room temperature), and is a T_A likely to be experienced frequently during endurance competition.

Subjects

Twenty-one male and female trained cyclists and triathletes (19–55 years) (11) were recruited from the Perth, Western Australia region for this study. Twenty were included for the final analysis as a single subject did not complete the study protocol due to schedule conflicts. Subjects were assigned to 1 of 2 groups that were matched for $\dot{V}O_2\text{max}$, peak power output (PPO), 20-km TT performance, and biological sex, and a 2-tailed *t* test was used to determine if there were any significant differences in the characteristics between the 2 experimental groups (Table 1). Seven males and 3 females were included in each group. All subjects were informed of the benefits and risks of the investigation and signed a written informed consent before data collection. The Edith Cowan University Human Research Ethics Committee approved the study prior to its commencement.

Procedures

All cycling test sessions (GXT, INT1, INT8, familiarization TT, TT1, and TT2) were performed on an electromagnetically-braked cycling ergometer (Velotron, Racermate, Redmond, WA). Before all testing sessions, subjects were asked to refrain from strenuous exercise in the 36 hours prior, eating large meals in the 3 hours prior, and consuming nonhabitual caffeine in the 24 hours prior. Subjects were instructed to eat and drink as they would before a competitive event and record their consumption in a log before the first testing session to replicate their dietary consumption 24 hours before subsequent sessions. Subjects were also instructed to wear the same athletic clothing to all experimental and training sessions (excluding the GXT), and cycling jerseys remained zipped up in all T_A conditions. A 75-cm diameter fan was placed ~75 cm from the subject's face as they sat on the cycle ergometer during all testing and intervention sessions. Subject's training volume regarding duration (hours) of aerobic exercise (e.g., cycling, running, and swimming) completed 6 weeks before and during the interval intervention was acquired through the subject's personal training logs (Table 1). Previous HIIT history and years as a cyclist were reported by subjects through a questionnaire (Table 1).

Subjects were instructed not to perform any structured HIIT outside of the laboratory during the course of the 4-week intervention. Interval training sessions 2 to 7 (of 8) were performed at least 36 hours apart on a WattBike (WattBike Pro, WattBike, West Bridgford, Nottingham, United Kingdom) adjusted to mimic the subject's own bike. During these interval training sessions, a simulated wind at a speed of ~17 km·h⁻¹ (measured at the face; LM-8000 Anemometer, Lutron Electronic Enterprise CO., LTD, Taipei, Taiwan) was provided by a fan for the duration of the work intervals. This windspeed was lower than what

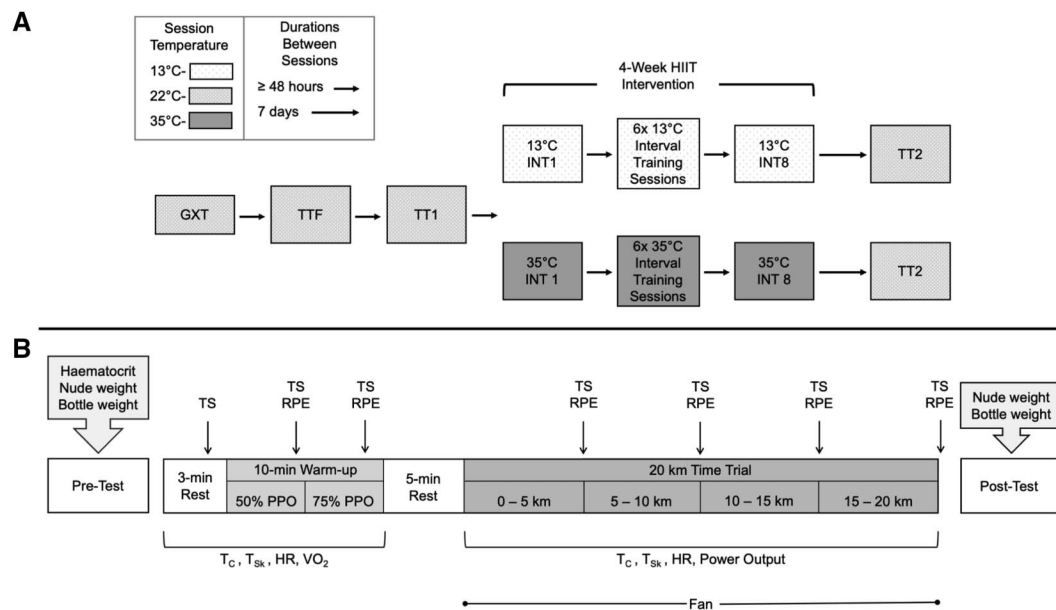


Figure 1. A) Experimental design. GXT indicates graded exercise test; TTF = familiarization time-trial; TT1 = pre-intervention time-trial; INT1 = first interval testing session; INT8 = last interval session and second interval testing session; TT2 = post-intervention time-trial. B) Time-trial testing protocol. PPO indicates peak power output; TS = thermal sensation; RPE = rate of perceived exertion; T_c = core temperature; T_{sk} = skin temperature; HR = heart rate; $\dot{V}O_2$ = oxygen consumption.

was provided during testing sessions due to limited equipment availability.

Graded Exercise Test. Subjects began cycling at 100 W (male) or 50 W (female) for 3 minutes, after which power increased 20 W

Table 1

Baseline physical, performance, and training characteristics of subjects in 13° C (HIIT₁₃) and 35° C (HIIT₃₅) high-intensity interval training groups.*†

	HIIT ₁₃ n = 10 m = 7, f = 3 cy = 7, tri = 3	HIIT ₃₅ n = 10 m = 7, f = 3 cy = 5, tri = 4, du = 1	p
Age (y)	33 ± 6	36 ± 12	0.526
Body mass (kg)	71.5 ± 13.5	70.1 ± 11.4	0.800
Height (cm)	177 ± 10	175 ± 8	0.610
Sum of 8 (mm)	92 ± 46	85 ± 36	0.725
Hematocrit	42 ± 3%	42 ± 3%	0.999
PPO (W)	352 ± 59	346 ± 63	0.810
PPO (W·kg ⁻¹)	4.97 ± 0.64	4.94 ± 0.74	0.915
$\dot{V}O_{2max}$ (L·min ⁻¹)	4.06 ± 1.00	4.01 ± 0.77	0.710
$\dot{V}O_{2max}$ (ml·kg ⁻¹ ·min ⁻¹)	56.9 ± 11.2	57.3 ± 9.5	0.921
Peak HR	177 ± 9	178 ± 17‡	0.919
Resting HR	61 ± 8‡	59 ± 11	0.543
20 km TT PO (W)	268 ± 49	246 ± 49	0.343
Time as a cyclist (y)	10.2 ± 7.3	14.4 ± 16.9	0.505
Use HIIT in program (n =)	6	6	NA
Training hours per week (h)	10.93 ± 4.25	10.28 ± 2.95‡	0.706

*Data presented as group mean ± SD.

†Sum of 8 = total 8 sites of skinfolds; PPO = peak power output; $\dot{V}O_{2max}$ = maximal oxygen consumption; HR = heart rate; 20 km TT PO = baseline 20 km time-trial power output; Time as cyclist = time subject reported that they had been riding recreationally (i.e., total time cycling); Use HIIT in program = number of subjects per group that had previous high-intensity interval training experience; Training hours per week = average hours per week subjects trained per week (6 wk before intervention start + training during the intervention); m = male; f = female; cy = cyclists; tri = triathletes; du = duathletes.

‡n = 9.

per minute until volitional fatigue. Expired gases were collected continuously and averaged over 5-second intervals (Parvo Medics, Sandy, UT). $\dot{V}O_{2max}$ was determined as the average of the 6 highest consecutive 5-second intervals (30 seconds) and was used to calculate time at or above 90% $\dot{V}O_{2max}$ ($T > 90\% \dot{V}O_{2max}$) during INT1 and INT8 (described below). Peak power output was determined using the power value corresponding with the time of volition (cadence below 60 rpm) during the GXT (17). Overall, 50% PPO and 75% PPO were subsequently calculated using this value. Peak HR was determined as the highest 5-second average.

Intervention and High-Intensity Interval Testing. The high-intensity interval session protocol was previously used by Boynton et al. (4) and chosen for its demonstrated time at or above $\dot{V}O_{2max}$ (28). Sessions consisted of a 10-minute warm-up at 50% PPO at room temperature (~24° C). After the warm-up and a 5-minute period allowed for transition to the environmental chamber, subjects performed 5 × 4-minute intervals with 5 minutes of recovery between each interval at either 13° C or 35° C (~45 minutes of chamber time per session). The recovery period consisted of 1 minute at rest, 3 minutes pedaling at volition (where subjects could drink water ad libitum), followed by 1 minute at 50% PPO. Athletes were instructed to pace the intervals to achieve the highest achievable combined intensity (i.e., average power output) for the 5 efforts while being provided lapsed time and being blinded to their power output, speed, and physiological data (e.g., HR). During the last interval, subjects were verbally encouraged to produce a maximal effort. Water for consumption during the test was from a tap, stored at room temperature before testing, and brought into the environmental chamber with the athlete to reduce any potential effects of water temperature differences between the interventions.

During interval testing sessions (INT1 & INT8), HR, power output, and T_c were monitored throughout the trials and averaged over each work interval. $\dot{V}O_2$ was continuously collected

except when athletes were free to drink ad libitum during the 3-minute period of pedaling at volition in the rest interval. $T > 90\%$ $\dot{V}O_{2\max}$ was calculated by analyzing $\dot{V}O_2$ collected for the entire session. A fan provided a simulated wind at a speed of $\sim 28 \text{ km}\cdot\text{h}^{-1}$ during each work interval to better simulate the convection experienced in situ.

Temperate Time-Trial Sessions. During the TT sessions, subjects performed a 3-minute resting baseline, a 10-minute standardized submaximal warm-up, 5 minutes of rest, followed by a flat simulated 20-km TT (Figure 1B). T_C , T_{Sk} , (Squirrel SQ2020 Data Logger, Grant Instruments, Shepreth Cambridgeshire, United Kingdom), and HR were measured continuously throughout TT1 and TT2 but not during the familiarization TT. Respiration measures were recorded continuously during the pretest resting baseline and warm-up. Resting HR was determined as the lowest 30-second average during the 3-minute rest. Thermal sensation (TS) (49) was recorded during the rest before warm-up. During the warm-up, subjects cycled at 50% PPO, followed by 75% PPO for 5 minutes. Gross mechanical efficiency (GME) was calculated (17) for the last 30 seconds of the 50% PPO segment of the submaximal warm-up, while all other measures were analyzed for the last 30 seconds of both stages. Whole-body rate of perceived exertion (RPE) (3) and TS were recorded at 4.5 and 9.5 minutes into the submaximal warm-up. During the 20-km TT, a fan provided a simulated wind at a speed of $\sim 28 \text{ km}\cdot\text{h}^{-1}$. Subjects started the 20-km TT in a virtual gearing of 56 x 17 but could self-select cadence and gearing once the TT began. Subjects were instructed to pace the 20-km TT at the highest average power output possible with the goal of traveling the virtual distance in the least amount of time. Subjects were blinded to their power output, HR, speed, and time lapsed while completed distance was displayed. Subjects were verbally encouraged to perform at their best and allowed to consume water ad libitum during the 20-km TT. Power output, HR, TS, T_C , and T_{Sk} were analyzed for every 5 km of the 20-km TT. Time-trial sweat loss was calculated by subtracting the nude bodyweight after exercise from the nude bodyweight before exercise and weight of water consumed during exercise. Water consumed during the TT was determined by measuring pre- and post-water bottle weights.

Experimental Measures. Height was measured by a stadiometer, and weight was measured on a calibrated scale (GWB Mettler ID1 Multi-Range, Toledo, Columbus, OH). Body composition regarding sum of skinfolds (Harpender skinfold caliper, Baly International, West Sussex, United Kingdom.) was assessed through an 8-site skinfold test (24). Rate of perceived exertion and TS were recorded using visual numerical scales. Heart rate was recorded (0.2 Hz) and averaged over 5-second intervals. To minimize data loss, HR was measured through Polar (primary method, Polar Precision Performance SW5.20, Polar Electro, Kempele, Finland), Parvo Medics (6 session), and Garmin (3 sessions; Edge 810, Garmin Ltd., Olathe, KS) systems. Linear modelling was used to replace 36 HR data points lost (180 seconds) during the steady state of one 20-km TT. Average power output (Velotron software; recorded at 1 Hz) for the work intervals and 20-km TT was determined using cycling power analytics software (GoldenCheetah, version 3.4, 2016).

T_C was measured using a disposable rectal thermometer (Mon-a-therm, Mallinckrodt Medical, St. Louis, MO) inserted $\sim 10 \text{ cm}$ past the anal sphincter. T_{Sk} was measured through 4 thermistors (YTS Temperature, 400 Series, Dayton, OH) placed on the chest

(T_{chest}), bicep (T_{arm}), thigh (T_{thigh}), and calf (T_{leg}) and mean T_{Sk} and was calculated (35) using the following formula:

$$T_{Sk} = 0.3 (T_{\text{chest}} + T_{\text{arm}}) + 0.2 (T_{\text{thigh}} + T_{\text{leg}})$$

T_C and T_{Sk} measurements were used to calculate mean body temperature (T_B) and the $T_C:T_{Sk}$ gradient. T_B was calculated using the formulas from (8) for neutral (13°C & 22°C) and hot (35°C) conditions, respectively:

$$T_B = 0.66 \times T_C + 0.34 \times T_{Sk}$$

$$T_B = 0.79 \times T_C + 0.21 \times T_{Sk}$$

Test sessions measuring T_C and T_{Sk} were performed at the same time of day for each subject to avoid circadian body temperature variations (37). Fluctuations in temperature data greater than 0.10°C/s were deemed physiologically improbable and likely the result of faulty temperature probes and thus were removed from analysis.

Hematocrit measurements were taken before exercise testing, as a double baseline before the intervention (separated by a minimum of 48 hours and averaged together for analysis) and at TT2. Finger prick blood for hematocrits was collected through capillary (100 Capillette, 955 05 32 02, Selzer Labor Technik, Mannheim-Heidelberg-Karlsruhe, Germany), spun at 3,500 rpm for 5 minutes (Centrifuge MPW-212, MPW Medical Instruments, Thebarton, South Australia), and assessed by the same researcher for all subjects.

Statistical Analyses

Values are reported as mean $\pm$ SD. Sample number is indicated where a full sample ($n = 10$) was not obtained for a group due to sampling error. A 2-way repeated measure ANOVA was used to compare all between group measures and within-subject measures recorded during the TTs. A 2-tailed paired t test was used to compare within-subject measures recorded at rest and during the warm-up. Significance was determined at a confidence level of 95% ($p \leq 0.050$). When sphericity was violated, a Greenhouse-Geisser correction was used. Where significant differences were observed, Bonferroni corrections for multiple comparisons were used to locate where differences existed. A Hedges' g correction (g) and partial eta squared (η_p^2) were reported as uninterpreted effect sizes. Hedges' g was calculated using a statistical spreadsheet (Jared DeFife, PhD, Emory University, 2009). SPSS software version 25.0 (IBM, Armonk, NY) was used to calculate η_p^2 and analyze data.

Results

Anthropometric and physiological characteristics were not significantly different between experimental groups (Table 1). During the GXT, 18 of 20 subjects experienced a "plateau" in $\dot{V}O_2$ ($\leq 200 \text{ ml}\cdot\text{min}^{-1}$ difference as a response to an increase in the work rate). The 2 subjects outside of that range ($230 \text{ ml}\cdot\text{min}^{-1}$ and $210 \text{ ml}\cdot\text{min}^{-1}$) had RERs over 1.2 (1.24 and 1.28, respectively). All subjects reached an RER over 1.00 (range: 1.03 to 1.38) during the GXT.

Interval Testing

Averaged environmental conditions during INT1 and INT8 for HIIT₁₃ and HIIT₃₅ were $12.9 \pm 0.3^\circ \text{C}$, $63 \pm 5\% \text{ RH}$ and $34.9 \pm$

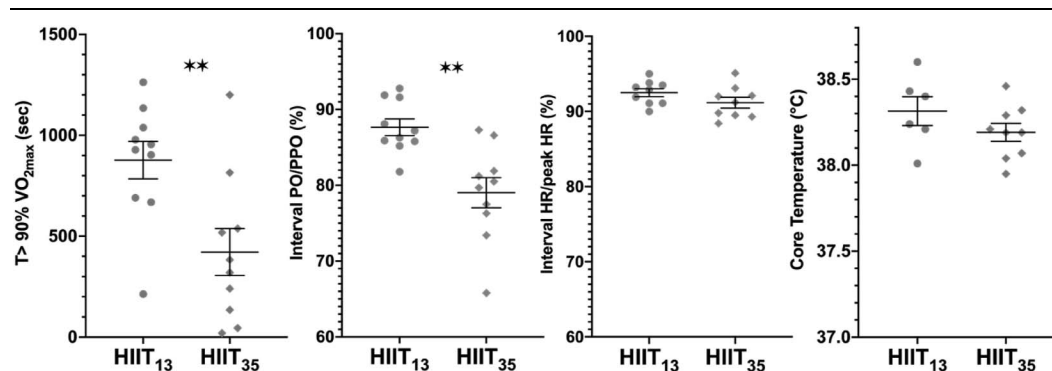


Figure 2. Mean $\pm$ SEM and individual measures for time above 90% $\dot{V}O_{2\max}$ ($T > 90\% \dot{V}O_{2\max}$), percentage interval power output (PO) of peak power output (PPO), percentage interval heart rate (HR) of peak HR, and core temperature. Values were averaged for the first (INT1) and last (INT8) session of a 4-week (8 sessions) high-intensity interval training intervention performed by 2 matched groups of trained cyclists in either 13° C (HIIT₁₃) or 35° C (HIIT₃₅). **Significance between groups.

0.5° C, $47 \pm 4\%$ RH (RH $p < 0.001$), respectively. $T > 90\% \dot{V}O_{2\max}$ during INT1 and INT8 was greater in HIIT₁₃ (875 ± 295 seconds; $p = 0.007$, $g = 1.278$), compared with HIIT₃₅ (420 ± 365 seconds; Figure 2). However, work interval power output, HR (HIIT₁₃ $n = 9$), T_C (HIIT₁₃ $n = 6$, HIIT₃₅ $n = 9$; Figure 2), and RPE during INT1 and INT8 were not different ($p = 0.146$, $g = 0.644$; $p = 0.989$, $g = -0.006$; $p = 0.229$, $g = 0.676$; $p = 0.328$, respectively) between HIIT₁₃ (309 ± 55 W, 164 ± 9 bpm, $38.32 \pm 0.21^\circ$ C, 17 ± 1 AU) and HIIT₃₅ (273 ± 52 W, 164 ± 13 bpm, $38.19 \pm 0.16^\circ$ C, 17 ± 1 AU). Work interval power output as a percentage of PPO was lower in HIIT₃₅ ($79 \pm 6\%$, $p < 0.001$, $g = 1.635$) vs. HIIT₁₃ ($88 \pm 4\%$; Figure 2), but interval HR as a percentage of peak HR was similar between groups ($91 \pm 2\%$ & $93 \pm 2\%$, respectively; $p = 0.149$, $g = 0.711$; Figure 2). Training hours for HIIT₁₃ and HIIT₃₅ during the 4-week HIIT intervention were 12.73 ± 4.96 h·wk⁻¹ and 11.10 ± 4.83 h·wk⁻¹ ($n = 9$), respectively ($p = 0.479$, $g = 0.318$).

Temperate Time-Trial Testing

Time-trial testing sessions (20-km TT, submaximal warm-up, and resting measures) were conducted at $22.3 \pm 0.3^\circ$ C, $56 \pm 7\%$ RH.

Twenty Kilometers Time-Trial. No differences between groups or interactions were noted for 20-km TT power output ($p = 0.421$; $p = 0.115$, $\eta_p^2 = 0.132$, respectively). However, power output significantly increased from TT1 to TT2 in both groups (HIIT₁₃, $3.3 \pm 3.4\%$, $p = 0.023$, $g = 0.163$; HIIT₃₅, $7.3 \pm 6.3\%$, $p = 0.003$, $g = 0.336$; Figure 3). Significant interactions for power output between TT1 (HIIT₁₃, 268 ± 49 W; HIIT₃₅, 246 ± 49 W) and TT2 (HIIT₁₃, 276 ± 46 W; HIIT₃₅, 263 ± 44 W) and 5-km TT split were also noted in both HIIT₁₃ ($p = 0.005$) and HIIT₃₅ ($p = 0.050$; Figure 4). No significant differences between groups or interactions were noted for HR (HIIT₁₃ $n = 9$, $p = 0.535$; $p = 0.747$, $\eta_p^2 = 0.006$) or RPE ($p = 0.658$; $p = 0.126$, $\eta_p^2 = 0.126$). Heart rate and RPE increased from TT1 to TT2 in HIIT₁₃ ($p = 0.025$, $g = 0.398$; $p = 0.013$; respectively) but not in HIIT₃₅ ($p = 0.342$, $g = 0.183$; $p = 0.405$; Figure 4).

Core temperature (HIIT₃₅ $n = 8$), T_{sk} (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$), and T_C : T_{sk} gradient (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 7$) were not significantly different between HIIT₁₃ and HIIT₃₅ during the 20-km TT nor were any significant interactions noted ($p = 0.127$, $\eta_p^2 = 0.139$; $p = 0.241$, $\eta_p^2 = 0.090$; $p = 0.369$, $\eta_p^2 = 0.058$, respectively). A significant interaction in T_B (HIIT₁₃ $n = 9$,

HIIT₃₅ $n = 7$) was observed between groups and TT1 and TT2 ($p = 0.042$, $\eta_p^2 = 0.263$; Figure 5). Thermal sensation was higher in both TT1 and TT2 for HIIT₃₅, compared with HIIT₁₃ ($p = 0.027$), while no significant interaction was noted ($p = 0.401$).

Submaximal Warm-Up. No significant interactions or main effects were observed for cardiorespiratory, thermoregulatory, and subjective measures during the 75% PPO of the warm-up (Figure 6). Gross mechanical efficiency was not different between HIIT₁₃ and HIIT₃₅ during 50% PPO ($p = 0.922$), and there was

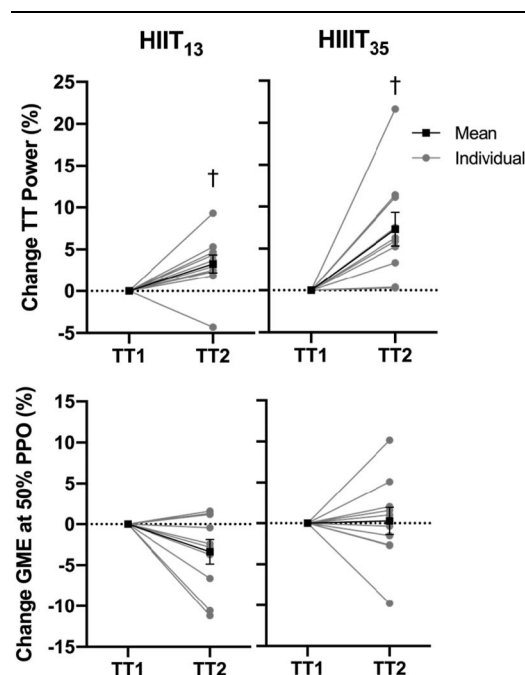


Figure 3. Percent change for power output during a 20-km time-trial (TT) and gross mechanical efficiency (GME) during a 50% peak power output (PPO) warm-up pre (TT1) and post (TT2) a 4-week (8 sessions) high-intensity interval training intervention performed in either 13° C (HIIT₁₃) or 35° C (HIIT₃₅). TT conditions were set to 22° C and 40% relative humidity with a fan providing a simulated wind at a speed of ~ 28 km·h⁻¹. The fan was not used during the warm-up. Mean values are mean $\pm$ SEM, $n = 10$ per group. †Significant within-subject change. Percent change for GME within HIIT₁₃ was noteworthy in magnitude but not significant ($p = 0.051$, $g = -0.744$).

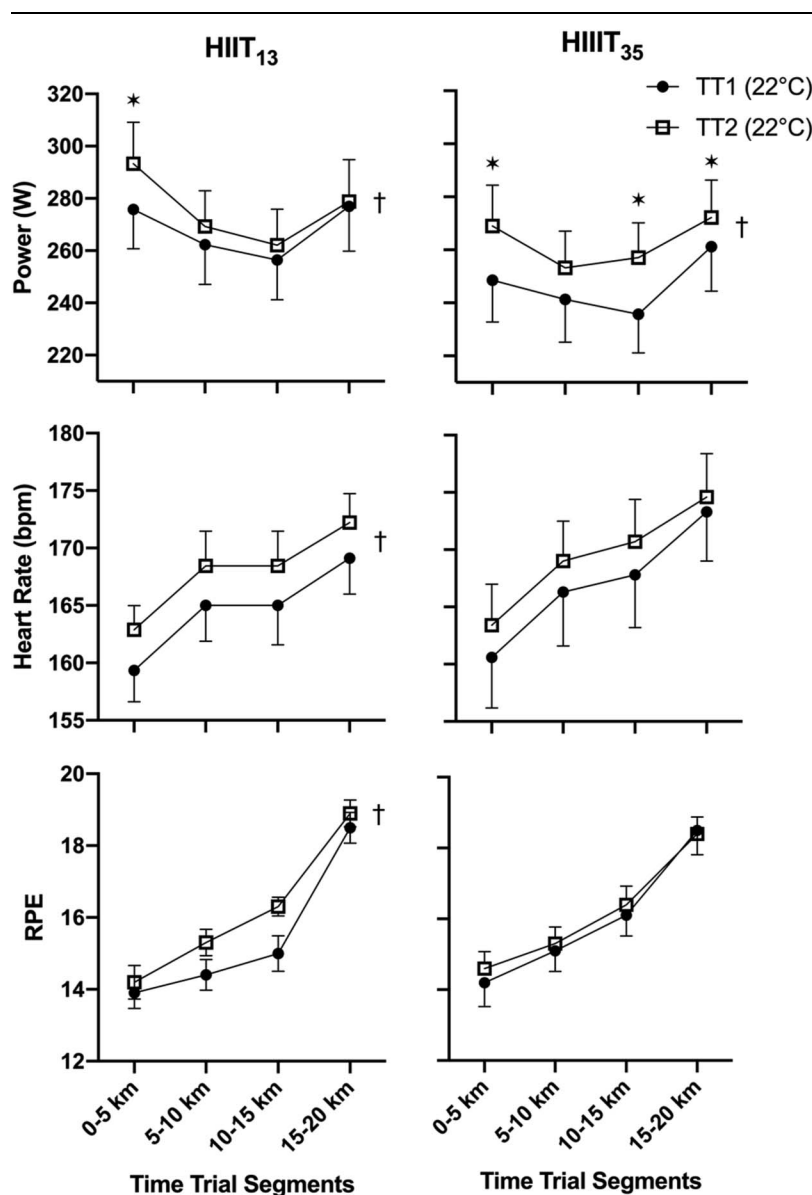


Figure 4. Power output, heart rate, and rate of perceived exertion (RPE) during 20-km time-trials (at 22° C) pre (TT1) and post (TT2) 4 weeks (8 sessions) of high-intensity interval training performed by 2 matched groups of trained cyclists in either 13° C (HIIT₁₃) or 35° C (HIIT₃₅). Testing conditions were set to 22° C and 40% relative humidity with a fan providing a simulated wind at a speed of ~28 km·h⁻¹. bpm indicates beats per minute. Values are mean ± SEM, *n* = 10 per group except within heart rate in HIIT₁₃ (*n* = 9). *Significant differences between time-trial segments in TT1 vs. TT2. †Significant within-subject change.

an absence of interaction between the groups and TT1 and TT2 ($p = 0.125$, $\eta_p^2 = 0.126$). The change in GME for HIIT₁₃ between TT1 and TT2 ($p = 0.051$) was however noteworthy in magnitude ($g = -0.744$; Figure 3). Related, $\dot{V}O_2$ significantly increased in HIIT₁₃ ($p = 0.041$, $g = 0.333$) between TT1 (2.43 ± 0.36 L·min⁻¹) and TT2 (2.52 ± 0.43 L·min⁻¹) but not in HIIT₃₅ ($p = 0.876$, $g = 0.020$). Cadence during 50% PPO did not change between TT1 and TT2 for HIIT₁₃ ($p = 0.326$, $g = 0.404$) or HIIT₃₅ ($p = 0.062$, $g = 0.777$).

Significant interactions were observed between groups and TTs during 50% PPO for T_{sk} (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$; $p = 0.003$, $\eta_p^2 = 0.462$), T_B (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$; $p = 0.008$, $\eta_p^2 = 0.382$), T_C : T_{sk} gradient (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$; $p = 0.045$, $\eta_p^2 = 0.242$), and TS ($p = 0.009$, $\eta_p^2 = 0.323$; Figure 6).

The interaction for T_C was $p = 0.062$ ($\eta_p^2 = 0.181$), where the temperature value was lower for HIIT₃₅ relative to HIIT₁₃ in TT2. Within-subject temperature measures for the warm-up are displayed in Figure 6.

Resting Measures. Resting HR (Table 1) recorded before the submaximal warm-up was not significantly different between groups ($p = 0.353$) nor was a significant interaction noted ($p = 0.843$, $\eta_p^2 = 0.002$; $p = 0.318$, $\eta_p^2 = 0.062$). Effects for resting HR between TT1 and TT2 in HIIT₁₃ and HIIT₃₅ were $g = 0.350$ and $g = -0.028$, respectively.

Resting T_C was not significantly different between HIIT₁₃ and HIIT₃₅ ($p = 0.113$) nor was a significant interaction noted ($p = 0.140$, $\eta_p^2 = 0.117$). Effects sizes for resting T_C between TT1 and

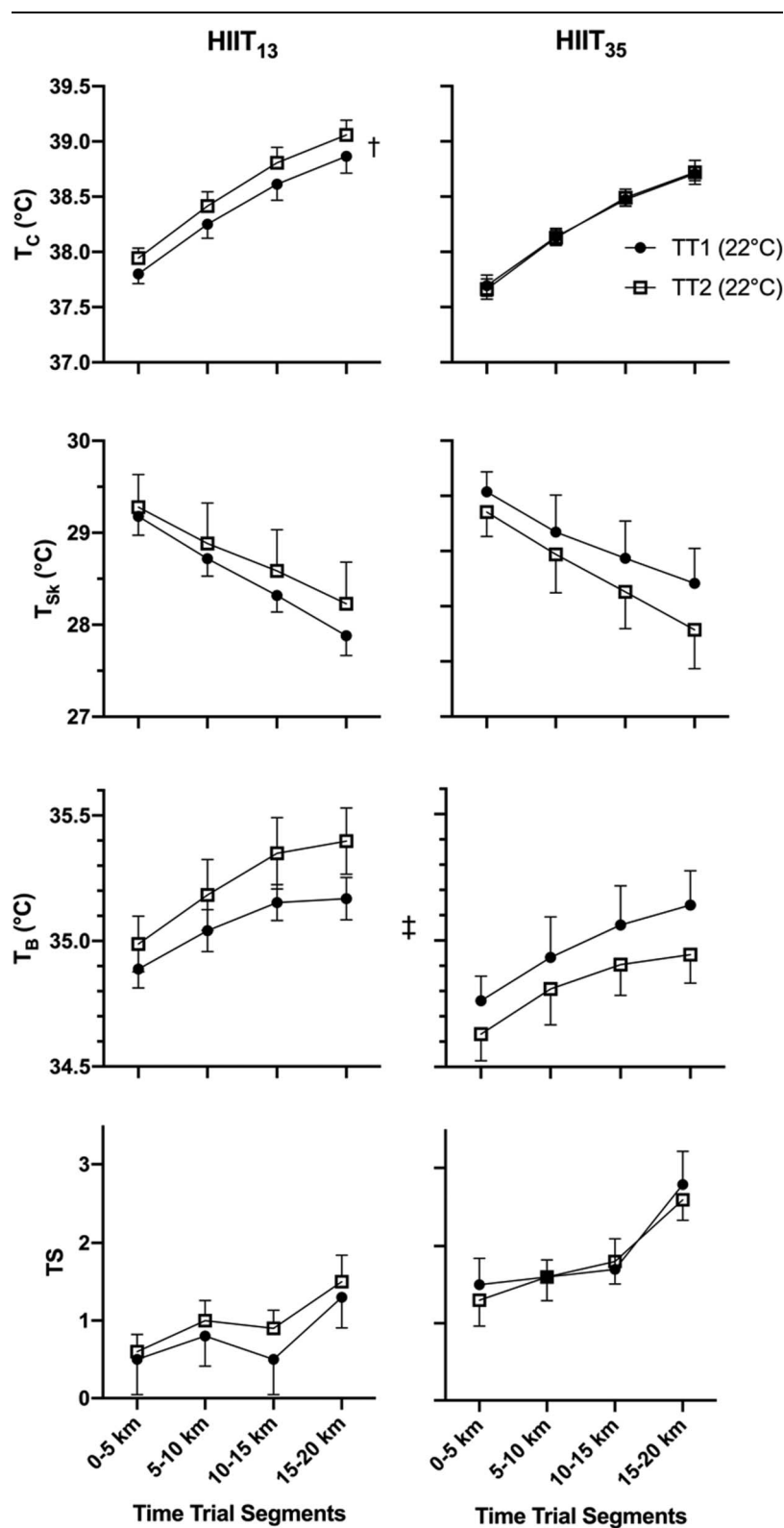


Figure 5. Thermoregulatory measures during 20-km time-trials pre (TT1) and post (TT2) 4 weeks (8 sessions) of high-intensity interval training performed by 2 matched groups of trained cyclists in either 13° C (HIIT₁₃) or 35° C (HIIT₃₅). Testing conditions were set to 22° C and 40% relative humidity with a fan providing a simulated wind at a speed of ~28 km·h⁻¹. Values are mean ± SEM, *n* = 10 per group unless otherwise noted. Core temperature (*T_c*), HIIT₃₅ *n* = 8; skin temperature (*T_{sk}*), HIIT₁₃ *n* = 9, HIIT₃₅ *n* = 8; body temperature (*T_b*), HIIT₁₃ *n* = 9, HIIT₃₅ *n* = 7. TS indicates thermal sensation. No significant differences between time-trial segments in TT1 vs. TT2 were noted for thermoregulatory measures. †Significant within-subject change. ‡Significant interaction between groups and TT1 vs. TT2.

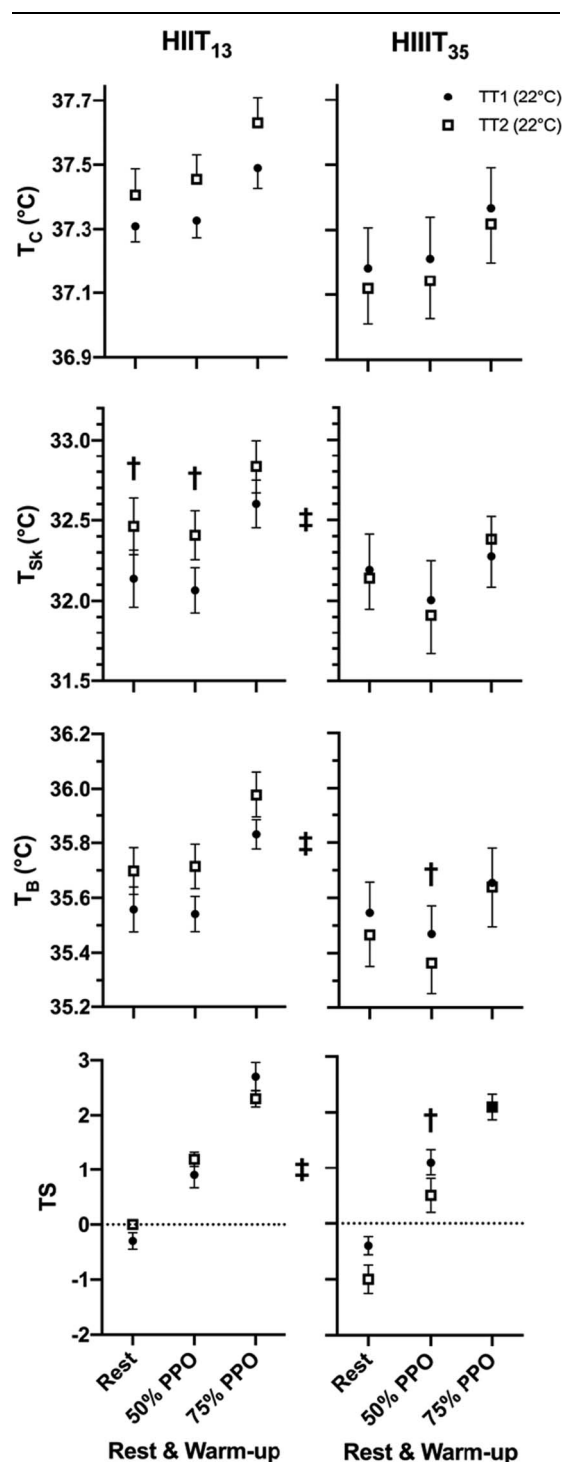


Figure 6. Thermoregulatory measures during rest and a sub-maximal warm-up pre (TT1) and post (TT2) 4 weeks (8 sessions) of high-intensity interval training performed by 2 matched groups of trained cyclists in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). The warm-up consisted of 5 minutes at 50% peak power output (PPO), followed by 5 minutes at 75% PPO. Testing conditions were set to 22°C and 40% relative humidity with no fan operating. Values are mean $\pm$ SEM, $n = 10$ per group unless otherwise noted. T_c indicates core temperature. TS indicates thermal sensation. Skin temperature (T_{sk}) and body temperature (T_b): Rest HIIT₁₃ $n = 9$, HIIT₃₅ $n = 9$; 50% PPO HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$; 75% PPO HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$. †Significant within-subject differences between TT1 vs. TT2. ‡Significant interaction between groups and TT1 vs. TT2 for rest and 50% PPO.

TT2 in HIIT₁₃ and HIIT₃₅ were $g = 0.439$ and $g = -0.159$, respectively. Resting T_{sk} , T_b , and $T_c:T_{sk}$ ($n = 9$ for all respective measures in HIIT₁₃ & HIIT₃₅) were not different between groups ($p = 0.619$, $p = 0.373$, $p = 0.932$, respectively). Significant interactions were observed for resting TS, T_{sk} , T_b , and $T_c:T_{sk}$ gradient ($p = 0.017$, $\eta_p^2 = 0.278$; $p = 0.008$, $\eta_p^2 = 0.362$; $p = 0.027$, $\eta_p^2 = 0.270$; $p = 0.049$, $\eta_p^2 = 0.220$, respectively; Figure 6). T_{sk} and T_b were higher in TT2 for HIIT₁₃ than HIIT₃₅ (Figure 6). Resting T_{sk} increased (Figure 6) and $T_c:T_{sk}$ decreased ($p = 0.005$, $g = -0.518$) in HIIT₁₃ significantly between TT1 and TT2.

Body Mass, Fluids, and Hematocrit. Baseline body mass for HIIT₁₃ and HIIT₃₅ is reported in Table 1. Body mass measured before exercise was not significantly different between the TT1 and TT2 ($p = 0.194$), HIIT₁₃ and HIIT₃₅ ($p = 0.914$), nor was there a significant interaction ($p = 0.991$, $\eta_p^2 < 0.001$). There was an absence of significant differences for all water consumption (TT1: HIIT₁₃, 0.13 ± 0.10 L; HIIT₃₅, 0.23 ± 0.12 L) and sweat loss analyses (TT1: HIIT₁₃, 0.91 ± 0.19 kg; HIIT₃₅, 0.85 ± 0.17 kg). Hematocrit was not different between HIIT₁₃ and HIIT₃₅ (Table 1), and an interaction was absent between groups and TT1 and TT2. However, hematocrit levels for HIIT₁₃ significantly increased from $42 \pm 3\%$ to $44 \pm 3\%$ ($p = 0.008$, $g = 0.655$).

Discussion

The purpose of this study was to determine the effect of 4 weeks of HIIT performed at 13°C or 35°C on endurance performance and physiological responses of trained cyclists in temperate conditions (22°C). In support of our hypothesis, $T > 90\%$ $\dot{V}O_{2max}$ was higher during 13°C HIIT vs. 35°C HIIT. However, contrary to our hypothesis, the observed improvement in 20-km TT performance was not greater after 13°C HIIT vs. 35°C HIIT, and the effect size for HIIT₃₅ was greater than twice that of HIIT₁₃. In addition, adaptations in cardiorespiratory (e.g., increased HR for HIIT₁₃), thermoregulatory (e.g., increased T_c for HIIT₁₃ and decreased T_b for HIIT₃₅), and subjective responses (e.g., increased RPE for HIIT₁₃ and decreased TS for HIIT₃₅) observed during temperate submaximal exercise and TTs appear to be dependent on the T_A HIIT is performed in.

This study raises uncertainty in the suggestion that performance improvements from aerobic HIIT are most effective when time at or near $\dot{V}O_{2max}$ is maximized (20,28,44), especially when considering HIIT in different environmental conditions. The lower $T > 90\%$ $\dot{V}O_{2max}$ and interval power output relative to PPO observed during HIIT in 35°C (Figure 2) are consistent with previous research demonstrating reduced $\dot{V}O_{2max}$ and work capacity while performing high-intensity exercise during severe heat stress (14,29). However, despite lower $T > 90\%$ $\dot{V}O_{2max}$ in 35°C the improvement in TT power output was not greater in HIIT₁₃ compared with HIIT₃₅ (Figure 3). It is therefore plausible that central stresses to cardiovascular function and (or) T_c during HIIT provided the necessary stimulation to increase TT performance, in lieu of a similar time at or near $\dot{V}O_{2max}$. Indeed, HR and T_c values during 35°C HIIT were similar to (Figure 2; or potentially higher than (4)) values noted in 13°C HIIT. In addition, lower power output during HIIT at 35°C (4) (Figure 2) and presumably lower muscle oxygen delivery (14) may indicate a more nuanced role for the peripheral stimulation associated with time at or near $\dot{V}O_{2max}$ in trained endurance athletes.

Indeed, it has been previously noted that a long time at or near $\dot{V}O_{2\max}$ may not optimally stimulate physiological components limiting $\dot{V}O_{2\max}$ in all cases (5,28). However, it is also important to note that the difference in time spent at or near $\dot{V}O_{2\max}$ observed between conditions (52%) in this study may not be physiologically meaningful. For example, training at a running velocity of 90% $\dot{V}O_{2\max}$, compared with 100% $\dot{V}O_{2\max}$ has demonstrated no differences in subsequent performance outcomes (31). Similarly, there is a potential that there is a duration of time at or near $\dot{V}O_{2\max}$ where its benefits to performance begin to diminish. Indeed, despite intervals in 35° C having a lower $T > 90\%$ $\dot{V}O_{2\max}$ than those in 13° C, the $T > 90\%$ $\dot{V}O_{2\max}$ for 35° C HIIT was comparable to values observed in other HIIT studies (5). Currently, further investigation into time at or near $\dot{V}O_{2\max}$, its mechanisms, and its association with physiological and performance outcomes after HIIT is warranted.

Findings from this study potentially provide additional evidence that greater performance gains observed in temperate conditions after training in the heat (i.e., HA) compared with training in cooler T_A are not exclusively an outcome of increased thermal stress but potentially the product of corresponding increases in cardiovascular load (30). However, no significant differences in average HR were observed between 13° C and 35° C HIIT (Figure 2) and subsequent improvements in temperate 20-km TT performance were not significantly different between intervention groups (Figure 3). This is in accordance with previous studies where cardiovascular strain was similar between hot and cool training conditions, and no differences in performance outcomes in temperate T_A were observed (18,19). Conversely, matched group design studies that have demonstrated improvements in temperate endurance performance after training in the heat had external loads that were uniform between experimental groups (22,26,46). Under these conditions, cardiovascular strain would have been higher for subjects training in the heat compared with subjects training in cooler conditions and consequently could have been the reason for the superior performance improvements observed after training in the heat (30). However, a recent study by Maunder et al. (25) observed improved temperate TT performance and citrate synthase activity in endurance-trained male athletes after performing a 3-week training intervention in 33° C matched for relative cardiovascular demand vs. a control group in 18° C. In contrast to the HA studies with similar cardiovascular strain cited above (18,19), the Maunder et al. (25) training intervention included 2 HIIT sessions per week (with 1 session paced as a “best session effort” similar to the HIIT sessions of this study). This distinction potentially indicates the importance of considering the type of training performed in the heat when seeking ergonomic benefits in temperate conditions.

Studies observing ergogenic benefits from HA have suggested physiological responses to heat exposure (e.g., increased plasma volume and improved sweat response) as possible mechanisms for the increased performance (9,22,26). However, in this study, the degree to which adaptations to heat exposure may have enhanced performance for HIIT₃₅ is unclear. For example, although plasma volume was not measured within this study, neither a decrease in hematocrit nor an increase in subject mass (i.e., indicating a potential increase in plasma volume or total body water) was observed between TT1 and TT2 in HIIT₃₅. Nonetheless, if an undetected increase in plasma volume occurred in this study, the claim it could have

increased performance in trained cyclists is questionable (47). Similarly, other measures that provide evidence of HA, including a decline in resting HR and T_C , did not change within the 35° C HIIT group. Indeed, the effect sizes for resting HR and T_C (Figure 6) changes were smaller than the range presented in a recent meta-analysis of HA adaptations (45). However, this is not to imply hot and cool HIIT increase endurance performance with the same physiological adaptations. While HR may be similar (Figure 2) or higher (4,14) in hot vs. cool conditions during high-intensity exercise, the underlying cardiophysiology (e.g., stroke volume) between the 2 conditions is likely different (14) and potentially stimulates different cardiovascular adaptations (28). Currently, more research is needed examining specific physiological adaptations to hot and cool HIIT.

It is possible that thermoregulatory adaptations after cool HIIT impaired endurance performance. In this study, significant increases in T_{sk} (Figure 6) and decreased $T_C:T_{sk}$ gradient occurred during the 50% PPO portion of the warm-up (22° C with no fan) after 13° C HIIT that were not observed after 35° C HIIT. Concurrent with these thermoregulatory changes during the warm-up, was a significant increase in $\dot{V}O_2$ and a noteworthy magnitude for the change in GME ($p = 0.051$, $g = -0.744$; Figure 3) for HIIT₁₃. Indeed, it has been suggested that submaximal aerobic performance in the heat is impaired by high T_{sk} (38) and low $T_C:T_{sk}$ gradient (10). In addition, it has been demonstrated that increasing T_{sk} during submaximal steady-state exercise subsequently increases HR and T_C and reduces stroke volume (6,7). However, while increased T_{sk} implies an influence on cardiovascular responses, it is also important to consider the potential effects cool HIIT could have on skeletomuscular adaptations, which could affect $\dot{V}O_2$ and subsequently influence GME (16). In this study, T_C , HR, and RPE (i.e., cardiovascular, thermal, and subjective stresses) increased during the temperate 20 km after 13° C HIIT but not after 35° C HIIT (Figures 4 and 5). In previous research, increased T_{sk} , T_C , and HR (i.e., cardiovascular strain) have been associated with decreases in TT performance (32,33). While no difference in performance improvements was noted between intervention conditions, power output increased during the last 2 5-km sections of TT2 for HIIT₃₅ but not for HIIT₁₃ (Figure 4). In addition, the effect size and percentage for increased TT power output for HIIT₃₅ was larger than what was observed for HIIT₁₃ (Figure 3). However, more research investigating the effects a range of T_A have on thermoregulation and performance after repeated exposures during HIIT is needed before concluding that cool HIIT is detrimental to performance.

Contrary to our initial hypothesis, the data in this article indicate that 4 weeks of HIIT in 13° C do not improve 20-km TT performance in 22° C to a greater extent than HIIT in 35° C. This outcome was unexpected considering less time was spent at or near $\dot{V}O_{2\max}$ during HIIT in the heat. In addition, little evidence was found for thermoregulatory adaptations in temperate T_A after hot HIIT that could potentially provide a mechanism for the greater effect size post 35° C and lack of significant difference in performance gains between groups. These findings highlight the possibility that cardiovascular strain during HIIT, which was similar between intervention groups, is more important to performance outcomes than the time spent at or near $\dot{V}O_{2\max}$ in trained endurance athletes. Thermoregulatory changes in HIIT₁₃ were more apparent than those in HIIT₃₅ and associated with a near-significant decrease in GME for HIIT₁₃.

Practical Applications

This study demonstrated that endurance performance improvements in temperate conditions from hot HIIT were similar to those of cool HIIT. These findings can potentially provide valuable assurance for athletes who may have to conduct HIIT in hot environmental conditions due to unavoidable circumstances. These study findings could also be useful when considering hot HIIT in conjunction with a HA strategy (e.g., stimulating HA, attenuating HA decay). Hot HIIT as an independent HA modality needs to be further explored. However, given acute thermoregulatory data from this study and previous research in our lab, the thermal impulse provided during a hot HIIT session should be sufficient enough to stimulate HA, especially when incorporated along with more established methods of HA in endurance athletes (e.g., sauna sessions). In a case where this hybrid approach to HA is used, practitioners should consider the additional stress athletes would experience during hot HIIT and the subsequent adjustments to training needed to ensure that athletes could recover effectively. In addition, after training in 13° C, combined thermoregulatory, $\dot{V}O_2$, GME, HR, and RPE findings suggest a caveat when considering cool T_A (i.e., T_A in the “optimal” performance range (12)) for training interventions in controlled environments (e.g., indoor training, exercise research).

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