

Self-paced exercise in hot and cool conditions is associated with the maintenance of $\% \dot{V}O_{2\text{peak}}$ within a narrow range

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Périard JD, Racinais S. Self-paced exercise in hot and cool conditions is associated with the maintenance of $\% \dot{V}O_{2\text{peak}}$ within a narrow range. *J Appl Physiol* 118: 1258–1265, 2015. First published March 26, 2015; doi:10.1152/jappphysiol.00084.2015.—This study examined the time course and extent of decrease in peak oxygen uptake ($\dot{V}O_{2\text{peak}}$) during self-paced exercise in HOT (35°C and 60% relative humidity) and COOL (18°C and 40% relative humidity) laboratory conditions. Ten well-trained cyclists completed four consecutive 16.5-min time trials (15-min self-paced effort with 1.5-min maximal end-spurt to determine $\dot{V}O_{2\text{peak}}$) interspersed by 5 min of recovery on a cycle ergometer in each condition. Rectal temperature increased significantly more in HOT ($39.4 \pm 0.7^\circ\text{C}$) than COOL ($38.6 \pm 0.3^\circ\text{C}$; $P < 0.001$). Power output was lower throughout HOT compared with COOL ($P < 0.001$). The decrease in power output from trial 1 to 4 was $\sim 16\%$ greater in HOT ($P < 0.001$). Oxygen uptake ($\dot{V}O_2$) was lower throughout HOT than COOL ($P < 0.05$), except at 5 min and during the end-spurt in trial 1. In HOT, $\dot{V}O_{2\text{peak}}$ reached 97, 89, 85, and 85% of predetermined maximal $\dot{V}O_2$, whereas in COOL 97, 94, 93, and 92% were attained. Relative exercise intensity ($\% \dot{V}O_{2\text{peak}}$) during trials 1 and 2 was lower in HOT ($\sim 84\%$) than COOL ($\sim 86\%$; $P < 0.05$), decreasing slightly during trials 3 and 4 (~ 80 and $\sim 85\%$, respectively; $P < 0.05$). However, heart rate was higher throughout HOT ($P = 0.002$), and ratings of perceived exertion greater during trials 3 and 4 in HOT ($P < 0.05$). Consequently, the regulation of self-paced exercise appears to occur in conjunction with the maintenance of $\% \dot{V}O_{2\text{peak}}$ within a narrow range (80–85% $\dot{V}O_{2\text{peak}}$). This range widens under heat stress, however, when exercise becomes protracted and a disassociation develops between relative exercise intensity, heart rate, and ratings of perceived exertion.

cardiovascular strain; cycling; fatigue; hyperthermia; pacing; thermoregulation; time trial; $\dot{V}O_{2\text{max}}$

SELF-PACED (I.E., TIME TRIAL) exercise requires the completion of a known distance in the quickest possible time or the production of the greatest amount of work in a set duration (1, 11). When undertaken in the heat, prolonged self-paced exercise performance is significantly impaired relative to cool conditions (10, 27, 28, 42, 46). The impairment in performance is mediated by the complex interplay of multiple physiological systems (e.g., nervous, metabolic) (8, 34, 45), with the development of thermal strain potentially modulating a performance-limiting increase in cardiovascular strain (5, 27, 28, 30). Indeed, a narrowing core-to-skin temperature gradient during exercise-heat stress increases the skin blood flow requirement for heat dissipation (5, 35, 36). The combination of an increase in cutaneous blood volume and a temperature-mediated rise in intrinsic heart rate decreases cardiac filling, which leads to reductions in stroke volume and maximal cardiac output when heart rate approaches maximum (6, 13, 15, 36). During pro-

longed constant-load exercise to exhaustion, for example, this forces the cardiovascular system toward a functional limit at submaximal workloads (e.g., power output), ultimately reducing maximal oxygen uptake ($\dot{V}O_{2\text{max}}$) (2, 24, 29). This decrement in cardiovascular reserve is purported to be the primary determinant limiting aerobic exercise in the heat (5, 36, 37).

When performing prolonged self-paced exercise under heat stress, a thermoregulatory-mediated rise in cardiovascular strain is associated with a lower sustainable power output, compared with exercising in cool conditions (27). Furthermore, peak oxygen uptake ($\dot{V}O_{2\text{peak}}$) measured during the final maximal end-spurt of a 45- to 60-min time trial is 11–18% lower in hot (35°C) than in cool (20°C) conditions (27, 28). It has been suggested that this decrease in $\dot{V}O_{2\text{peak}}$ leads to a progressive increase in relative exercise intensity (i.e., $\% \dot{V}O_{2\text{peak}}$ utilized) for any given power output (i.e., absolute workload) sustained throughout self-paced efforts (27). However, given that $\dot{V}O_{2\text{max}}/\dot{V}O_{2\text{peak}}$ is not reduced during short (≤ 15 min) bouts of exercise in the heat (35), the decrease in power output appears to occur progressively in conjunction with the maintenance of a given $\% \dot{V}O_{2\text{peak}}$. This relative intensity corresponds to exercising at the physiological limit, or fatigue threshold (1, 19, 23), which requires balancing the relative contributions of aerobic and anaerobic metabolism to maintain a “performance oxygen uptake ($\dot{V}O_2$)” within a given range for a specific task (7). Although a decrease in $\dot{V}O_{2\text{peak}}$ has been suggested to regulate self-paced exercise in the heat by mediating adjustments in absolute workload (i.e., power output) to maintain $\% \dot{V}O_{2\text{peak}}$ within a narrow range (5, 27), the time course and extent of decrease in $\dot{V}O_{2\text{peak}}$ throughout the duration of a prolonged time trial have yet to be determined. Understanding the pathway(s) via which self-paced exercise in the heat is regulated and the factors that affect this regulation have the potential to influence the pacing strategies utilized by athletes, as well as enhance our understanding of how humans modulate their behavior in a performance setting.

Therefore, the purpose of this study was to examine $\% \dot{V}O_{2\text{peak}}$ utilized during four consecutive 15-min time trials with 1.5-min end-spurts (for the measurement of $\dot{V}O_{2\text{peak}}$) in highly trained cyclists in hot/humid and cool/dry conditions, interspersed by 5 min of active recovery. The protocol and environmental conditions were selected as they replicate the effort associated with a prolonged time trial (i.e., ~ 60 min or ~ 40 km) in which a decrease and clear difference in $\dot{V}O_{2\text{peak}}$ occurred between conditions on completing exercise (27, 28). The protocol also allows for the measurement of a reference $\dot{V}O_{2\text{peak}}$ at regular intervals to determine the relative exercise intensity maintained during prolonged self-paced exercise. It was hypothesized that $\dot{V}O_2$ and power output in well-conditioned athletes would remain similar between conditions during the first time trial, whereas they would decrease to greater

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extents in the heat during the remaining trials. As such, it was further hypothesized that $\dot{V}O_{2\text{peak}}$ would remain similar between conditions, due to a greater decrease in $\dot{V}O_{2\text{peak}}$ under heat stress.

METHODS

Subjects. Ten (nine men, one woman: amenorrheic) well-trained (>250 km/wk) cyclists volunteered for this study. The subjects were accustomed to cycling in the heat, as the study was conducted in the spring in Qatar. They were also experienced in time trialing, having participated in various cycling and triathlon races in the previous months. Their characteristics were as follows (mean \pm SD): age, 36 ± 7 yr; body mass, 77.2 ± 10.2 kg; height, 178.0 ± 7.97 cm. Subjects were fully informed of the experimental procedures and potential risks before giving written, informed consent. They were also required to complete a Medical History Questionnaire and Physical Activity Readiness Questionnaire before being admitted to the study. The protocol was approved by the Anti-Doping Lab Qatar Institutional Review Board. All procedures conformed to the standards of the Declaration of Helsinki.

Preexperimental $\dot{V}O_{2\text{max}}$ assessment. Subjects visited the laboratory on two occasions, separated by 3–7 days, before the two experimental trials. During these visits, height and nude body mass were measured using a precision stadiometer and balance (Seca 769, Hamburg, Germany). An incremental $\dot{V}O_{2\text{max}}$ assessment and familiarization time trial were then performed in COOL (18°C and 40% relative humidity) and HOT (35°C and 60% relative humidity) conditions with an airflow of 12.5 km/h. The $\dot{V}O_{2\text{max}}$ test was undertaken following a 5-min rest period in the environmental chamber, and the protocol consisted of cycling on an electronically braked cycle ergometer (Schoberer Rad Messtechnik, Jülich, Germany) at a starting power output of 100 W and increasing every minute by 25 W until volitional fatigue. $\dot{V}O_{2\text{max}}$ and maximum heart rate were obtained over a 60-s period during the classic plateau phase (17, 43), with a respiratory exchange ratio > 1.10 . After the $\dot{V}O_{2\text{max}}$ assessment (~ 5 min), subjects performed a 15-min time trial with an additional 1.5-min maximal end-spurt to familiarize with the procedures of the experimental protocol and equipment. This was undertaken in each condition. Rectal (T_{re}) and mean skin temperatures (\bar{T}_{sk}) were continuously monitored. During all assessments and trials, subjects wore cycling shorts, socks, and cycling shoes. Saddle and handlebar position were adjusted by the subjects to their preferred cycling position before undertaking the first $\dot{V}O_{2\text{max}}$ assessment and remained unchanged for all trials.

Experimental trials. Subjects visited the laboratory at the same time of day on two occasions separated by 4–7 days. On arrival (~ 60 min before testing), they emptied their bladder and provided a urine sample for the measurement of urine specific gravity (Pal-10-S, Vitech Scientific, West Sussex, UK). They were then weighed (i.e., nude body mass) before changing into cycling attire, and then inserted a rectal thermistor probe (MRB rectal probe, Ellab, Hilleroed, Denmark). Subjects then rested in the seated position in temperate conditions (20 – 22°C , 50% relative humidity) while being instrumented. Following this period, they mounted a cycle ergometer and rested in a cycling position for 5 min before resting baseline data were collected. Subjects then performed a standard warm-up of 5 min at 150 W, which included 3×10 s spin-ups (i.e., high cadence accelerations) at 2, 3, and 4 min. Once the warm-up was completed, they entered a climate chamber (Tescor, Warminster, PA) set to either COOL or HOT conditions with an airflow of 12.5 km/h. The transition time from the end of the warm-up to entering the chamber was ~ 2 min. As with the preexperimental assessment, the experimental trials were counterbalanced. On entering the climate chamber, subjects sat resting on the ergometer for 5 min before baseline measures were collected. Subjects then undertook four 16.5-min time trials interspersed by 5 min of passive/active recovery. Specifically, subjects were asked to maintain the highest sustainable effort during 15 min and then reach maximal effort during a 1.5-min end-spurt, in order for $\dot{V}O_{2\text{peak}}$ to be

measured. This was conducted to evaluate the time course and extent of decrease in $\dot{V}O_{2\text{peak}}$, which was assumed to be attained if heart rate rose to within 5 beats/min of that observed in the preexperimental $\dot{V}O_{2\text{max}}$ assessment in the same condition (2, 48). $\dot{V}O_{2\text{peak}}$ and peak heart rate were taken as the highest averaged 30-s segment during the final 45 s of the progressive maximal end-spurt. Given that a plateau was not sustained, the term $\dot{V}O_{2\text{peak}}$ refers to the highest $\dot{V}O_2$ measured during the maximal end-spurt, whereas $\dot{V}O_{2\text{max}}$ refers to the $\dot{V}O_2$ measured during the preexperimental $\dot{V}O_{2\text{max}}$ assessment. Subjects were informed of every 5 min elapsed during the 15-min time trials and were able to change between gears (i.e., resistance). This was the only form of feedback provided until the final 1.5 min (i.e., end-spurt from 15 to 16.5 min) was reached. At this point, strong verbal encouragement was given, and the ergometer visual display (power, heart rate, cadence, speed, distance) was revealed to the subjects, so that they could reach and maintain a high power output and heart rate. The recovery period consisted of subjects cycling at ~ 75 W after having recovered from the maximal end-spurt, typically from 2.5 to 5 min.

Cardiovascular and temperature measurements. $\dot{V}O_2$ and heart rate were measured at 5, 10, and 15 min and during the final maximal end-spurt of each time trial. $\dot{V}O_2$ was measured using an online breath-by-breath cardiopulmonary system (Oxycon Pro, CareFusion, Rolle, Switzerland). Heart rate was monitored telemetrically with a Polar transmitter-receiver (T-31 Polar Electro, Lake Success, NY). Measurements at 5, 10, and 15 min were conducted over 1 min. Systolic (SBP) and diastolic blood pressure (DBP) were recorded manually by the same investigator at 5 and 10 min of each time trial using a sphygmomanometer (Gamma G5, Heine Optotechnik, Herrsching, Germany) and used to calculate mean arterial pressure: $\text{DBP} + 1/3 \times (\text{SBP} - \text{DBP})$.

Body core temperature was monitored with a T_{re} probe inserted 12 cm beyond the anal sphincter. Skin temperatures of the chest, upper arm, thigh, and lower leg were monitored with iButton temperature sensors/data loggers (Maxim Integrated Products, Sunnyvale, CA) and used to calculate \bar{T}_{sk} (31). Skin blood flow was monitored at the level of the right medioventral forearm and upper scapula (Moor Instruments, Axminster, UK) and expressed as the mean of these sites following the application of a software-integrated smoothing factor and averaging over 1 min for each site. Measurements were conducted at rest, at 5, 10, and 15 min, and during the final maximal end-spurt of each time trial in conjunction to those of $\dot{V}O_2$. To minimize movement artifacts and the accumulation of saliva in the mouthpiece, subjects were instructed to maintain an upright cycling position and keep their head up.

Hydration and perceptual measurements. Subjects were instructed to drink 6 ml of water/kg body mass every 2.5 h on the day before each experimental session to ensure euhydration at the start of exercise. During the time trial, they were permitted to drink ad libitum. Body mass changes were evaluated at the conclusion of each trial to determine sweat production with corrections for fluid ingested and sweat trapped in clothing. The rating of perceived exertion (RPE) using the Borg 6–20 scale (4) and thermal comfort using the Bedford Thermal Comfort Scale (3) were recorded at 5, 10, and 15 min and during the final maximal end-spurt of each time trial. Subjects were familiarized with the scales before testing.

Statistical analysis. All statistical calculations were performed using PASW software version 21.0 (SPSS, Chicago, IL). Two-way repeated-measures ANOVAs were performed to test significance between and within treatments. ANOVA assumptions were verified preceding all statistical procedures; however, none of the data violated the assumption of sphericity; therefore Greenhouse-Geisser corrections were not applied. Effect size was measured using partial η^2 values, with $\eta^2 > 0.06$ representing a moderate effect and $\eta^2 \geq 0.14$ a large effect. Where significant effects were established, pairwise differences were identified using the Bonferroni post hoc analysis procedure adjusted for multiple comparisons. Hydration and time were evaluated using Student's paired *t*-tests, as were preexperimental

Table 1. *Cardiorespiratory, performance, and temperature responses during the preexperimental $\dot{V}O_{2\max}$ test in COOL and HOT conditions*

	COOL	HOT
Time, min	12.82 ± 0.92	12.27 ± 1.08*
PO _{max} , W	395.4 ± 23.1	382.0 ± 27.5*
$\dot{V}O_{2\max}$, l/min	4.7 ± 0.3	4.7 ± 0.4
HR _{max} , beats/min	182 ± 9	183 ± 9
RER	1.17 ± 0.03	1.17 ± 0.03
Initial T _{re} , °C	37.1 ± 0.3	37.0 ± 0.2
Final T _{re} , °C	37.5 ± 1.2	37.5 ± 0.3
Initial \bar{T}_{sk} , °C	28.5 ± 1.2	34.5 ± 0.9*
Final \bar{T}_{sk} , °C	28.4 ± 1.2	35.9 ± 0.5*
Initial T _{re} -to- \bar{T}_{sk} , °C	8.6 ± 1.1	2.5 ± 0.9*
Final T _{re} -to- \bar{T}_{sk} , °C	9.2 ± 1.2	1.6 ± 0.5*

Values are means ± SD. COOL, 18°C and 40% relative humidity condition; HOT, 35°C and 60% relative humidity condition; PO_{max}, maximal power output; $\dot{V}O_{2\max}$, maximal oxygen uptake; HR_{max}, maximal heart rate; RER, respiratory exchange ratio; T_{re}, rectal temperature; \bar{T}_{sk} , mean skin temperature; T_{re}-to- \bar{T}_{sk} , core to skin temperature gradient. *Significant difference between COOL and HOT ($P < 0.01$).

assessment values. P values < 0.05 were considered statistically significant. All values are expressed as means ± SD.

RESULTS

Preexperimental $\dot{V}O_{2\max}$ assessment responses. Subjects attained a similar $\dot{V}O_{2\max}$ in the HOT and COOL preexperimental assessments ($P = 0.723$), despite peak power output being slightly lower and time to fatigue shorter in the heat ($P = 0.01$; Table 1). Moreover, while initial and final T_{re} were similar between conditions, \bar{T}_{sk} was higher throughout the $\dot{V}O_{2\max}$ assessment in HOT ($P < 0.001$). Consequently, the T_{re}-to- \bar{T}_{sk} gradient was narrower at the onset and termination of the $\dot{V}O_{2\max}$ assessment in HOT ($P < 0.001$). Of note, the female participant in the present study demonstrated a similar pattern of response in thermoregulatory and cardiovascular function to that of the male subjects.

Time trial responses. Significant condition ($P < 0.001$, $\eta^2 = 0.88$), time ($P < 0.001$, $\eta^2 = 0.90$), and interaction ($P < 0.001$, $\eta^2 = 0.33$) effects were observed for power output (Fig. 1A). From the onset of exercise, power output was lower in HOT compared with COOL ($P < 0.001$). Within each condition, mean power output during the first time trial was higher than that during the second, third, and fourth trials ($P < 0.05$). Furthermore, mean power output was similar in the third and fourth trials. The decrease in mean power output from the first to the fourth time trial was greater in HOT ($-25.9 \pm 10.2\%$) compared with COOL ($-10.0 \pm 6.5\%$; $P < 0.001$). Pacing during COOL remained relatively even, varying only by $\sim 11\%$ (range: 6.9 to -4.5%) relative to average power output ($P < 0.05$; Fig. 1B). In HOT, the variation was significantly greater ($P < 0.01$), ranging from 23.2 to -11.7% ($P < 0.01$). In both conditions, power output during the progressive maximal end-sprints increased significantly relative to the power output maintained in the preceding 15-min time trial ($P < 0.05$). In COOL, power output was similar during the first and second maximal end-sprints ($P = 0.055$), decreasing significantly in the third ($P = 0.004$) and fourth ($P = 0.006$). In HOT, power output during the maximal end-sprints decreased significantly following the first time trial ($P < 0.001$).

Time trial temperature responses. There were significant condition ($P = 0.001$, $\eta^2 = 0.71$), time ($P < 0.001$, $\eta^2 = 0.95$), and interaction ($P < 0.001$, $\eta^2 = 0.71$) effects for T_{re} (Fig. 2A). Baseline T_{re} was similar between conditions before the subjects entered the chamber ($P = 0.677$). However, after 5 min of baseline rest ($P = 0.005$) in the climate chamber and also after 5 min of exercise ($P = 0.007$), T_{re} was lower in HOT relative to COOL. Thereafter, T_{re} increased in both conditions ($P < 0.001$) and significantly more so in HOT, reaching final values of $38.6 \pm 0.3^\circ\text{C}$ (COOL) and $39.4 \pm 0.7^\circ\text{C}$ (HOT) ($P < 0.001$). Although slightly greater, the rate of T_{re} increase was similar during the first 15-min time trial in HOT ($3.7 \pm 1.4^\circ\text{C/h}$) and COOL ($3.2 \pm 1.0^\circ\text{C/h}$; $P = 0.239$). During the second time trial, the rate of increase in T_{re} was significantly greater in HOT ($2.7 \pm 1.1^\circ\text{C/h}$) compared with COOL ($1.2 \pm$

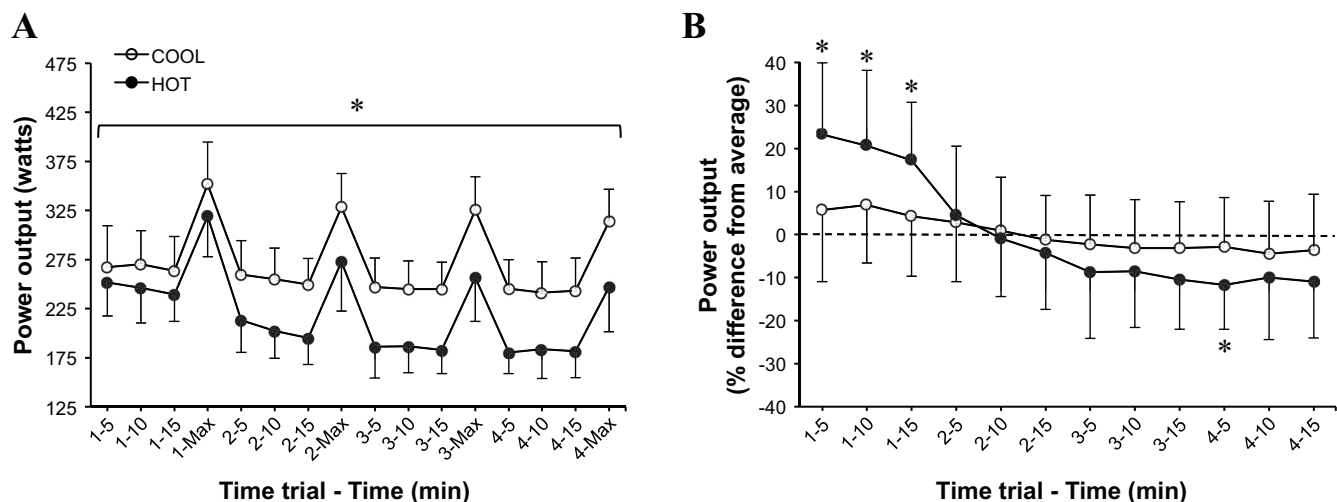


Fig. 1. Power output (A) and normalized power output (B) during four consecutive 15-min self-paced efforts with an additional 1.5-min maximal end-sprint, interspersed by 5 min of recovery in HOT and COOL conditions. Power output was normalized to the average power sustained during the four 15-min efforts. Dashed line represents average power output. COOL, 18°C and 40% relative humidity condition; HOT, 35°C and 60% relative humidity condition. Values are means ± SD. *Significant difference between COOL and HOT ($P < 0.05$).

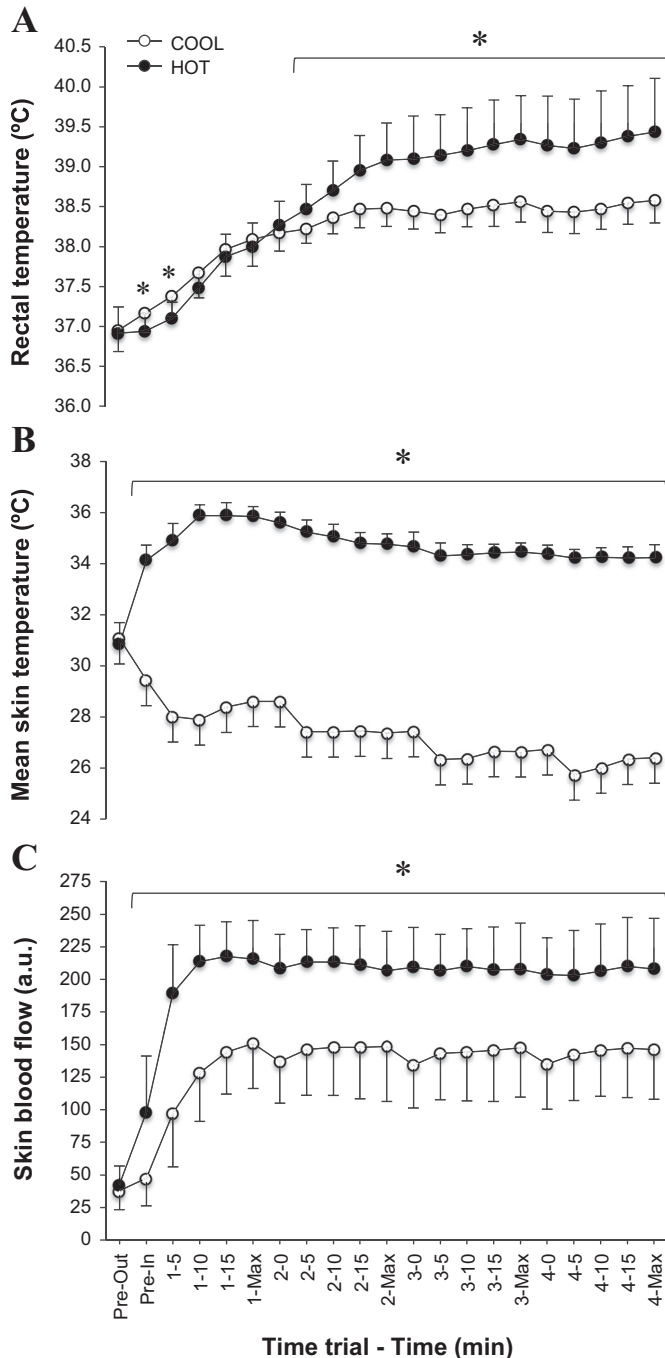


Fig. 2. Rectal temperature (A), mean skin temperature (B), and skin blood flow (C) before (Pre) and during four consecutive 15-min self-paced efforts with an additional 1.5-min maximal end-spurt, interspersed by 5 min of recovery in HOT and COOL conditions. a.u., Arbitrary units. Values are means \pm SD. *Significant difference between COOL and HOT ($P < 0.05$).

0.5°C/h; $P = 0.003$), remaining similar in the third (HOT: 0.7 ± 0.6 and COOL: 0.3 ± 0.5 °C/h; $P = 0.074$) and fourth (HOT: 0.5 ± 0.4 and COOL: 0.4 ± 0.3 °C/h; $P = 0.834$) trials.

Significant condition ($P < 0.002$, $\eta^2 = 0.98$), time ($P < 0.001$, $\eta^2 = 0.65$), and interaction ($P < 0.001$, $\eta^2 = 0.84$) effects were also observed for \bar{T}_{sk} (Fig. 2B). Baseline \bar{T}_{sk} values were similar before the subjects entered the climate chamber ($P = 0.384$), but then increased in HOT and decreased in COOL ($P < 0.005$), leveling off after 5–10 min.

Skin blood flow responses indicate significant condition ($P < 0.001$, $\eta^2 = 0.81$), time ($P < 0.001$, $\eta^2 = 0.88$), and interaction ($P < 0.001$, $\eta^2 = 0.39$) effects (Fig. 2C). Baseline values for skin blood flow were similar before the subjects entered the climate chamber ($P = 0.456$), but then increased in both conditions and significantly more in HOT ($P < 0.005$).

Time trial cardiovascular responses. Significant condition ($P = 0.002$, $\eta^2 = 0.68$), time ($P < 0.001$, $\eta^2 = 0.97$), and interaction ($P < 0.001$, $\eta^2 = 0.25$) effects were observed for heart rate (Fig. 3A). Throughout the HOT time trials, heart rate was higher than during COOL ($P < 0.05$), except at 5 min during the first ($P = 0.234$), third ($P = 0.053$), and fourth ($P = 0.054$) time trials. During the progressive maximal end-spurts in COOL, heart rate reached $98 \pm 2\%$ of the maximum recorded during the preexperimental $\dot{V}O_{2max}$ assessment, whereas $101 \pm 3\%$ was attained in HOT. Relative to the peak heart rate recorded during each progressive maximal end-spurt, the percent heart rate maintained in the time trials was similar between conditions (Fig. 3B).

Significant condition ($P = 0.001$, $\eta^2 = 0.71$), time ($P < 0.001$, $\eta^2 = 0.86$), and interaction ($P < 0.001$, $\eta^2 = 0.37$) effects were observed for $\dot{V}O_2$ (Fig. 3C). Throughout the HOT time trials, $\dot{V}O_2$ was lower than during COOL ($P < 0.05$), except at 5 min ($P = 0.56$) and during the maximal end-spurt of the first time trial ($P = 0.62$). During the progressive maximal end-spurts in COOL, $\dot{V}O_{2peak}$ reached 97 ± 4 , 94 ± 4 , 93 ± 4 , and $92 \pm 5\%$ of the maximum recorded during the preexperimental $\dot{V}O_{2max}$ assessment, whereas 97 ± 5 , 89 ± 7 , 85 ± 10 , and $85 \pm 10\%$ were attained in HOT. A significant condition ($P = 0.047$, $\eta^2 = 0.37$) effect was observed for relative exercise intensity, whereas nonsignificant time ($P = 0.351$, $\eta^2 = 0.11$) and interaction effects ($P = 0.296$, $\eta^2 = 0.13$) were noted (Fig. 3D). Hence, relative to the $\dot{V}O_{2peak}$ recorded during each progressive maximal end-spurt, the $\% \dot{V}O_{2peak}$ maintained in each time trial was slightly greater in COOL (86 ± 7 , 86 ± 6 , 85 ± 5 , and $86 \pm 5\%$) compared with HOT (84 ± 5 , 84 ± 5 , 80 ± 4 , and $80 \pm 8\%$, respectively, for time trials 1–4).

Mean arterial blood pressure data showed significant condition ($P < 0.001$, $\eta^2 = 0.87$), time ($P < 0.001$, $\eta^2 = 0.58$), and interaction ($P < 0.001$, $\eta^2 = 0.67$) effects (Fig. 4). From 10 min onwards, mean arterial blood pressure was lower in HOT compared with COOL ($P < 0.005$). Mean arterial blood pressure decreased significantly during HOT ($P < 0.001$), whereas it remained stable in COOL.

Time trial hydration and perceptual responses. Preexercise body mass (HOT: 77.4 ± 10.3 and COOL: 77.3 ± 10.1 kg) and urine specific gravity (HOT: 1.014 ± 0.008 and COOL: 1.015 ± 0.007) were similar between conditions and indicative of euhydration. While percent body mass losses were greater in HOT ($2.0 \pm 1.0\%$) compared with COOL ($1.3 \pm 0.6\%$; $P = 0.02$), the difference was minimal ($-0.7 \pm 0.8\%$). This was due to the greater sweat production in HOT (2.3 ± 0.4 l/h) compared with COOL (1.3 ± 0.2 l/h; $P < 0.001$), compensated for by a larger fluid intake (HOT: 2.2 ± 0.9 vs. COOL: 1.1 ± 0.4 l/h; $P = 0.001$).

Significant condition ($P = 0.015$, $\eta^2 = 0.50$), time ($P < 0.001$, $\eta^2 = 0.90$), and interaction ($P < 0.001$, $\eta^2 = 0.32$) effects were observed for RPE (Fig. 5A). During each time trial, RPE increased from 5 min to maximal end-spurt ($P < 0.05$), reaching similar peak values across trials. However,

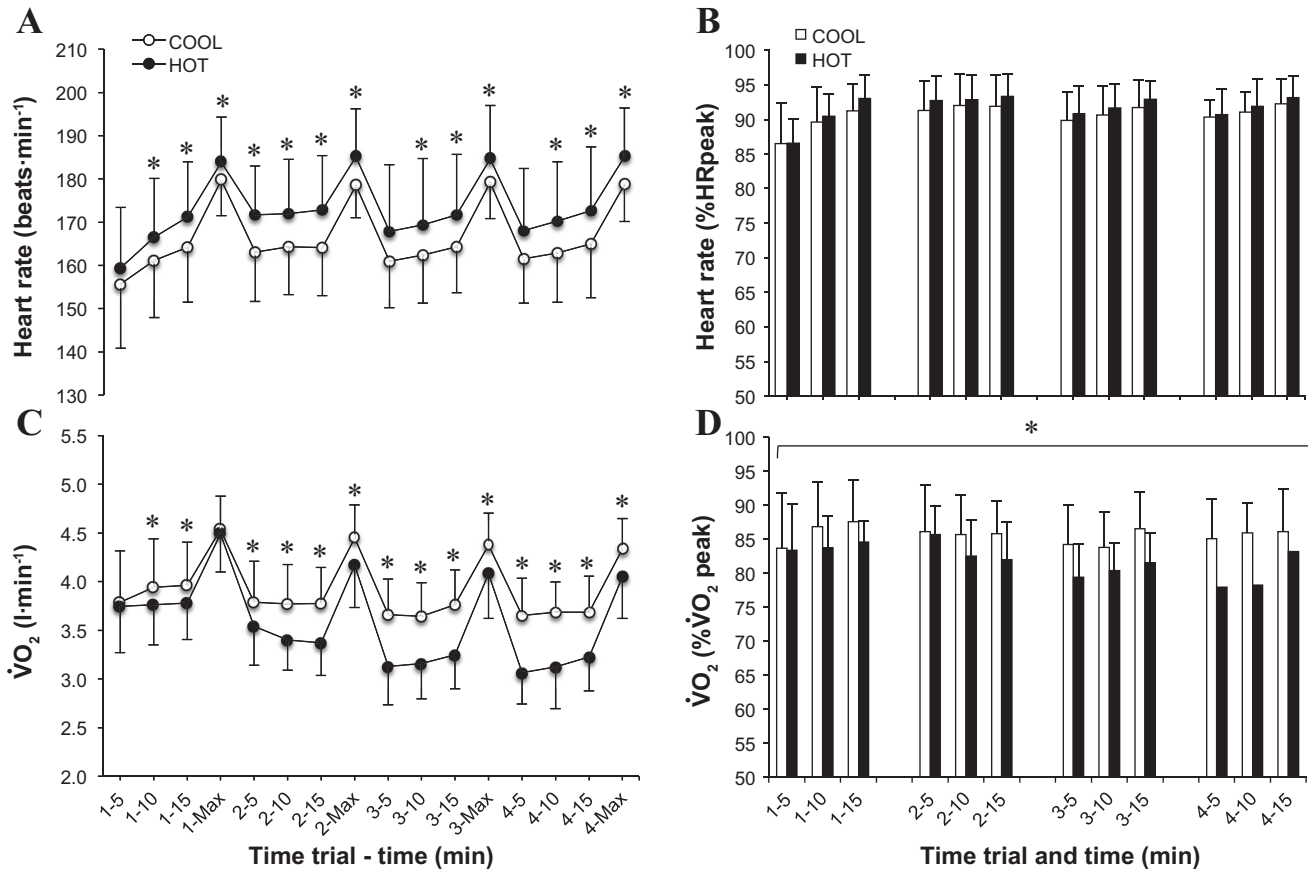


Fig. 3. Heart rate (A), normalized heart rate (B), oxygen uptake ($\dot{V}O_2$; C), and normalized $\dot{V}O_2$ (D) during four consecutive 15-min self-paced efforts with an additional 1.5-min maximal end-spurt, interspersed by 5 min of recovery in HOT and COOL conditions. Heart rate and $\dot{V}O_2$ were normalized as a function of the peak values attained during the 1.5-min maximal efforts. $\dot{V}O_{2peak}$, peak oxygen uptake; HR_{peak}, peak heart rate. Values are means \pm SD. *Significant difference between COOL and HOT ($P < 0.05$).

RPE was higher in HOT during the third time trial at 5, 10, and 15 min and during the fourth time trial at 10 and 15 min ($P < 0.05$). Significant condition ($P < 0.001$, $\eta^2 = 0.87$), time ($P < 0.001$, $\eta^2 = 0.70$), and interaction ($P < 0.001$, $\eta^2 = 0.41$) effects were observed for thermal comfort (Fig. 5B). Although

preexercise thermal comfort was similar before entering the climate chamber (COOL: 3.2 ± 0.9 and HOT: 3.1 ± 1.1), a significant difference was noted before exercise was initiated while inside the chamber (COOL: 2.1 ± 0.9 and HOT: 4.8 ± 0.4 ; $P < 0.001$). Thereafter, thermal comfort was higher throughout exercise in the heat ($P < 0.001$).

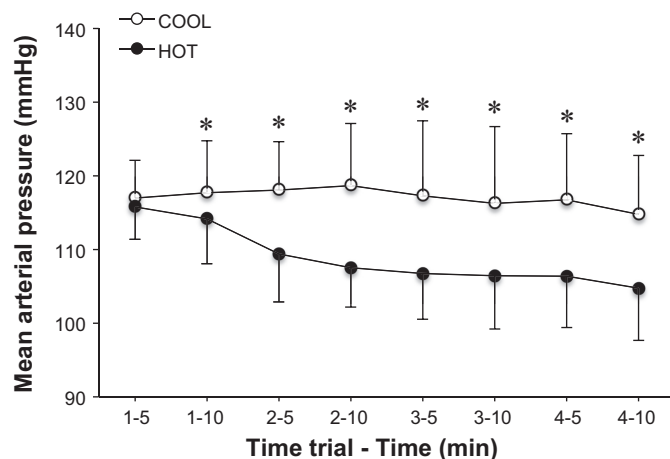


Fig. 4. Mean arterial pressure during four consecutive 15-min self-paced efforts with an additional 1.5-min maximal end-spurt, interspersed by 5 min of recovery in HOT and COOL conditions. Values are means \pm SD. *Significant difference between COOL and HOT ($P < 0.005$).

DISCUSSION

This study is the first to examine the time course and extent of decrease in $\dot{V}O_{2peak}$ during prolonged self-paced cycling in HOT and COOL conditions, and determine the relative exercise intensity (i.e., % $\dot{V}O_{2peak}$) maintained during such efforts in relation to changes in performance. The novel findings of this study are that: 1) $\dot{V}O_{2peak}$ decreased progressively throughout self-paced exercise in both HOT and COOL conditions, with the majority of the decrement occurring at the end of time trial 2 after ~30 min of exercise; 2) the relative intensity of exercise maintained during the initial two time trials (0–30 min) was ~2% lower in the HOT condition, despite a greater decrease in $\dot{V}O_{2peak}$ occurring in the heat from the second time trial onward; and 3) relative exercise intensity during the final two time trials (30–60 min) was ~5% lower in the HOT compared with COOL condition, yet accompanied by a higher heart rate and RPE. These results suggest that the regulation of self-paced exercise in experienced and motivated individuals is

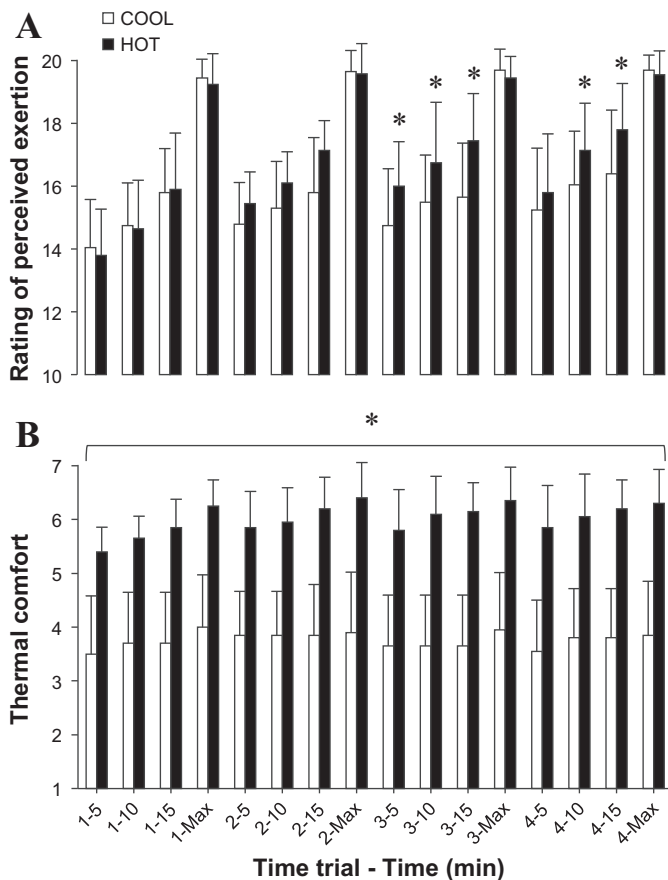


Fig. 5. Rating of perceived exertion (A) and thermal comfort (B) during four consecutive 15-min self-paced efforts with an additional 1.5-min maximal end-spurt, interspersed by 5 min of recovery in HOT and COOL conditions. Values are means \pm SD. *Significant difference between COOL and HOT ($P < 0.05$).

associated with the maintenance of $\dot{V}O_{2\text{peak}}$ within a fairly narrow range in response to sensory information stemming from a thermal strain-mediated increase in cardiovascular strain. This range is extended under heat stress, as exercise duration increases and a disassociation develops between $\dot{V}O_{2\text{peak}}$, heart rate, and RPE. This dissociation indicates that other factors, such as those related to temperature regulation, autonomic control, and local effects of temperature on intrinsic heart rate, may also contribute to influence prolonged self-paced exercise performance in the heat.

In agreement with previous observations (27, 28), $\dot{V}O_2$ and power output (Fig. 1) decreased throughout the time trials in the HOT condition, concomitant with the development of thermal (Fig. 2) and cardiovascular strain (Figs. 3 and 4). Interestingly, however, while $\dot{V}O_{2\text{peak}}$ was within $\sim 2\%$ between conditions during the first 30 min of exercise, an $\sim 5\%$ lower $\dot{V}O_{2\text{peak}}$ was sustained in the HOT condition during the final 30 min (i.e., time trials 3 and 4). This was observed despite $\dot{V}O_{2\text{peak}}$ decreasing significantly more in the HOT than in the COOL condition. As indicated in Fig. 3D, $\dot{V}O_{2\text{peak}}$ between conditions remained within a narrow range for the first two time trials (COOL: $\sim 86\%$ and HOT: $\sim 84\%$), widening slightly in the final two time trials (COOL: $\sim 85\%$ and HOT: $\sim 80\%$). Although these findings demonstrate the clear influence of heat stress on performance and $\dot{V}O_2$, it remains to be

determined whether more severe (i.e., hot and/or humid) environmental conditions exacerbate the magnitude of decrease in these parameters and the relative exercise intensity sustained during prolonged self-paced efforts. Notwithstanding, data from the present study are within range of the relative exercise intensity ($\sim 86\%$) maintained in other studies during a 40-km time trial in temperate conditions (27, 44). Moreover, the percentage of peak heart rate maintained in all time trials, as well as the RPE responses (Fig. 5), were similar to those of experienced cyclists undertaking self-paced efforts of similar length/duration in field and laboratory settings (26, 30, 42).

In recent years, considerable focus has been placed on developing models to describe the regulation of pacing and performance during time trial efforts (1, 8, 33, 34, 41, 45, 46). These models have proposed that performance is modulated to various extents by 1) the conscious awareness of physiological disturbances (e.g., afferent feedback from cardiorespiratory responses), which evoke behavioral adjustments in work rate, and by 2) the subconscious anticipation of potentially harmful challenges to homeostasis (e.g., heat storage), which down-regulate muscle recruitment in a feedforward manner to avoid catastrophic system failure. However, the premise on which the latter concept was founded has been shown to be fundamentally flawed, due to erroneous calculations of the rate of body heat storage (20, 32). In the present study, a similar rate of rise in T_{re} was calculated during the first 15 min of the time trials, despite a power output difference of ~ 21 W between conditions. The lower power output noted at the start of the time trial in the HOT condition was somewhat atypical and may stem from subjects consciously adopting a conservative approach, knowing that four separate time trials had to be performed. Indeed, power output in the heat usually matches that of cooler conditions for the first 10–15 min of time trials (10, 27, 28, 30, 42, 46), unless participants are inexperienced at performing such tasks (40), or the self-paced effort is preloaded with exercise in the heat (9). Hettinga et al. (18) also showed that cycling in the heat decreases gross efficiency by 0.9% relative to cooler conditions. While changes in core temperature did not account for this decrement, the authors indicated that thermoregulatory adjustments (i.e., increased skin blood flow) might have contributed to increase cardiorespiratory strain and concomitantly decrease gross efficiency. Such a decrease may have occurred at the start of exercise in the heat in the present study, thereby lowering power output but maintaining a similar $\dot{V}O_2$ and $\dot{V}O_{2\text{peak}}$ to that of the COOL condition. Notwithstanding, while a lower starting power output was observed in HOT conditions, the pacing profile demonstrated in Fig. 1B under COOL (i.e., even pace) and HOT (i.e., positive pace) environmental conditions is indicative of the typical pattern adopted by trained cyclists in laboratory settings, which includes an end-spurt.

In the heat, it appears that a positive pacing profile was observed because $\dot{V}O_{2\text{peak}}$ did not decrease until thermal strain developed significantly enough to impact on cardiovascular function. This is corroborated not only by the similar $\dot{V}O_{2\text{peak}}$ values noted during the end-spurt of the first time trial in each condition, but also during the preexperimental assessment, where $\dot{V}O_{2\text{max}}$ was matched between the HOT and COOL conditions (Table 1). The similarity in response was observed, despite the initial and final $T_{\text{re-to-}T_{\text{sk}}}$ gradients being significantly narrower in the heat. This indicates that $\dot{V}O_{2\text{max}}$ is

unaffected when skin temperature is elevated, but core temperature remains relatively low (e.g., $<38^{\circ}\text{C}$) (35, 40). Accordingly, $\dot{V}\text{O}_{2\text{peak}}$ during the first maximal end-spurt was comparable between conditions, albeit slightly lower than $\dot{V}\text{O}_{2\text{max}}$ by $\sim 3\%$. During the second time trial, $\dot{V}\text{O}_{2\text{peak}}$ decreased significantly more in the heat, which coincided with a much greater T_{re} in the HOT ($\sim 39.1^{\circ}\text{C}$) than COOL ($\sim 38.5^{\circ}\text{C}$) condition. Thereafter, the difference in $\dot{V}\text{O}_{2\text{peak}}$ between conditions was attenuated, as the rate of rise in T_{re} tapered off and \bar{T}_{sk} decreased slightly (Fig. 2). This observation reinforces the relationship between thermal and cardiovascular strain, environmental compensability, and pacing.

Interestingly, while $\% \dot{V}\text{O}_{2\text{peak}}$ was lower in the heat during the latter stages of exercise, heart rate was ~ 7 beats/min higher throughout all of the HOT time trials, which is a finding often reported in trained individuals performing self-paced exercise (27, 28, 42). The maintenance of a lower $\% \dot{V}\text{O}_{2\text{peak}}$ during the final two time trials in combination with the higher heart rate suggests a dissociation between heart rate and relative exercise intensity during prolonged self-paced efforts in the heat (Fig. 3). Accordingly, it has been suggested that the increase in heart rate during sustained constant-load exercise in the heat is only partly related to the reduction in $\dot{V}\text{O}_{2\text{peak}}$, with the remainder of the increase associated with other factors (2). The greater increase in heart rate is proposed to occur as a result of an elevation in sympathetic nervous activity and vagal withdrawal, as well as via the direct effect of blood temperature on the sinoatrial node (i.e., pacemaker tissue located in the right atrium) (16, 21, 22). The latter has been shown to produce an increase in heart rate of $7\text{--}8\text{ beats}\cdot\text{min}^{-1}\cdot^{\circ}\text{C}^{-1}$ (14, 16, 21). Hence, with progressive increases in ambient and concomitantly core temperature, the increase in heart rate during exercise-heat stress may be more pronounced for a given increase in $\% \dot{V}\text{O}_{2\text{peak}}$ than in cooler conditions (2). Thus the proportional relationship between heart rate and relative exercise intensity that exists in COOL conditions (12) does not appear to extend to prolonged exercise performed in the heat, especially self-paced exercise.

A dissociation was also observed between $\% \dot{V}\text{O}_{2\text{peak}}$ and RPE. Indeed, similar RPE values were noted during the first 30 min of exercise (i.e., time trials 1 and 2), but then increased throughout the final two time trials in the heat, despite the maintenance of a lower $\% \dot{V}\text{O}_{2\text{peak}}$ (Fig. 5). The higher RPE recorded during the final two time trials, the point at which thermal strain was greatest, may partly relate to sensations of thermal discomfort exacerbating those of perceived exertion (38, 39). It has also been suggested that the central nervous system integrates afferent sensory information to align work rate with an acceptable RPE during self-paced exercise (41, 45). This does not appear to be the case in the heat though, as time trials are often completed at a higher RPE than in cooler conditions (27, 28, 42). Conversely, it is well established the central command during exercise is associated with the parallel activation of cardiovascular and motor control centers. However, the magnitude of the central command-mediated cardiovascular response can be dictated by the perception of effort, independent of adjustments in motor activation (e.g., power output) (47). As such, the degree of central command (i.e., motor outflow) representing a given RPE and heart rate in HOT and COOL conditions may not necessarily result in identical muscle recruitment patterns.

Ultimately, pacing relates to making decisions based on prior experience, accurate knowledge of a task, and an understanding of one's physical abilities, while interpreting and reacting to physiological cues (i.e., sensory information) associated with effort. As such, it is a process informed by awareness and influenced by a variety of factors, such as the development of hyperthermia, the perception of effort, neuromuscular function, and metabolism (8, 25, 34). Accordingly, the regulation of self-paced exercise performance in motivated and experienced individuals under heat stress appears to be associated with sensory information stemming from a thermal strain-mediated increase in cardiovascular strain. As highlighted in the present study, $\dot{V}\text{O}_{2\text{peak}}$ decreased progressively during self-paced exercise in both HOT and COOL conditions, but the decrement was greater in the heat after 15 min of exercise. Although $\% \dot{V}\text{O}_{2\text{peak}}$ maintained during the time trials was lower throughout the HOT condition, it was within $\sim 2\%$ of the COOL condition during the first 30 min of exercise, decreasing thereafter to $\sim 5\%$. This decrease was nonetheless accompanied by a higher heart rate and RPE. These results suggest that prolonged (~ 60 min) self-paced exercise performance is associated with the maintenance of $\% \dot{V}\text{O}_{2\text{peak}}$ within a relatively narrow range, which widens under heat stress when exercise becomes protracted and a disassociation develops between relative exercise intensity and heart rate and RPE.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: J.D.P. and S.R. conception and design of research; J.D.P. and S.R. performed experiments; J.D.P. analyzed data; J.D.P. interpreted results of experiments; J.D.P. prepared figures; J.D.P. drafted manuscript; J.D.P. and S.R. edited and revised manuscript; J.D.P. and S.R. approved final version of manuscript.

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