Heat acclimation with controlled heart rate: influence of hydration status

Gavin Travers[1, 2], David Nichols[3], Nathan Riding[1], José González-Alonso[2, 4], Julien D. Périard[1,5*]

[1]Athlete Health and Performance Research Centre, Aspetar Orthopedic and Sports Medicine Hospital, Doha, Qatar

[2] Centre for Sports Medicine and Human Performance, Brunel University London, United Kingdom

[3] Sport Development Centre, Loughborough University, Loughborough, United Kingdom

[4] Division of Sport, Health and Exercise Sciences, Department of Life Sciences, Brunel University London, Uxbridge, UK

[5] University of Canberra Research Institute for Sport and Exercise, University of Canberra, Bruce, Australia

Short title: Acclimation with controlled heart rate

*** Correspondence:**
Julien Périard

Research Institute for Sport and Exercise

University of Canberra

Bruce, ACT, 2617

Australia

Email: julien.periard@canberra.edu.au

Phone: +61 (0)2 6206 8540

ABSTRACT

Purpose: To characterize the adaptive responses to heat acclimation (HA) with controlled heart rate (HR) and determine whether hydration strategy alters adaptations. The influence of HA on $\dot{V}O_{2max}$ in cool conditions and self-paced exercise in the heat was also determined.

Methods: Eight males ($\dot{V}O_{2max}$: 55±7 $ml \cdot kg^{-1} \cdot min^{-1}$) completed two 10-day interventions in a counterbalanced cross-over design. Fluid intakes differed between interventions to either maintain euhydration (HA-EUH) or elicit similar daily body mass deficits (2.85±0.26%; HA-DEH). HA consisted of 90 min of cycling in 40°C and 40% RH. Initial workload (172±22 W) was adjusted over the last 75 min to maintain exercising HR equivalent to 65% $\dot{V}O_{2max}$. A $\dot{V}O_{2max}$ test in cool conditions and 30 min time-trial in hot-humid conditions were completed before and after HA.

Results: HR at the end of the initial 15 min workload was 10±5 $beats \cdot min^{-1}$ lower on day 10 in both interventions (P<0.001). The workload necessary to maintain exercising HR (145±7 $beats \cdot min^{-1}$) increased throughout HA-EUH (25±10 W, P=0.001) and HA-DEH (16±18 W, P=0.02). There was a main effect of HA on sweat rate (P=0.014), which tended to increase with HA-EUH (0.19±0.18 $L \cdot h^{-1}$, P=0.06) but not HA-DEH (P=0.12). Skin temperature decreased during HA-EUH (0.6 ± 0.5°C, P=0.03), but not HA-DEH (P=0.30). There was a main effect of HA on $\dot{V}O_{2max}$ (~3 $ml \cdot kg^{-1} \cdot min^{-1}$, P=0.02); however, neither intervention independently increased $\dot{V}O_{2max}$ (both P=0.08). Time-trial performance increased following HA-EUH (19±16 W, P=0.02), but not HA-DEH (P=0.21).

Conclusions: Controlled HR exercise in the heat induces several HA adaptations, which may be optimized by maintaining euhydration. HA-EUH also improves self-paced exercise performance in the heat. However, HA does not appear to significantly increase $\dot{V}O_{2max}$ in cool conditions.

Keywords: acclimatization, hypohydration, dehydration, thermoregulation, fluid

INTRODUCTION

Endurance exercise in a hot environment is associated with increased thermoregulatory and cardiovascular strain that contributes to impaired performance (1,2). Heat acclimation (HA) leads to significant reductions in physiological strain associated with exercising heat stress and can, at least in part, restore the observed decrements in performance (3). Adaptations are acquired via the frequent, repeated elevation of whole-body temperature to a level that sufficiently stimulates vasomotor and sudomotor thermoeffector responses (4). Typically, these adaptations are evident via increased sweat sensitivity and output, increased skin blood flow, lowered body temperatures, reduced cardiovascular strain and improved fluid balance (5,6).

Several strategies to induce HA in a safe and practicable manner have been explored, including sauna bathing (7), post-exercise hot water immersion (8) and isothermic/controlled hyperthermia HA (4). The latter approach utilizes alterations in exogenous (i.e. ambient) or endogenous (i.e. metabolic) heat stress to maintain the thermal stimulus for adaptation; a core temperature ≥38.5°C (4,9–11). As exposures are repeated and adaptations develop, workload is increased to achieve a stable core temperature while HR remains unchanged (10,12) or slightly decreases (13). This response is observed whether euhydration is maintained or dehydration via fluid restriction occurs (13), highlighting the interplay between HR, core temperature and hydration status during exercising heat stress. Therefore, exercise in the heat at a target HR may offer a practical approach to HA (5). However, the responses to such a protocol are yet to be fully described.

Purposely inducing dehydration via minimizing fluid intake throughout HA may enhance the adaptive process (5,14). Acutely, exercise-induced dehydration leads to hyperosmotic hypovolemia (15) and greater release of aldosterone compared to euhydration (16); an important mediator of post-exercise plasma volume (PV) expansion (17). Therefore, permissive dehydration during HA may promote a sustained expansion of PV via increased fluid-electrolyte retention, as well as the oncotic effects of intravascular protein (9,18). To date, restricting fluid intake during short-term HA has been shown to result in greater PV expansion and reductions in HR during exercise in the heat compared to maintaining euhydration (10). However, others have shown no additional benefit of dehydration during a similar time frame (11,19). In addition, medium- and long-term interventions have either not compared responses to euhydrated HA (9), investigated these responses between independent groups (20), or have not standardized the degree of daily dehydration throughout HA (13). Consequently, the adaptive responses to HA with differing hydration strategies remains unclear.

HA has also been reported to increase $\dot{V}O_{2max}$, the power output at lactate threshold and improve mechanical efficiency by 5-7% in cool ambient conditions (11,21). Many of the adaptations to HA have been proposed to cause these increases in aerobic exercise performance (22). For instance, a more pronounced hypervolemia following dehydrated HA (10) may increase maximal cardiac output. Furthermore, HA may increase red cell volume (RCV; 7) and enhance the oxygen carrying capacity of the blood. Whether HA increases $\dot{V}O_{2max}$ independently of exercise training in temperate conditions is unclear. Investigations comparing matched groups performing exercise HA and temperate aerobic training have shown no improvement in $\dot{V}O_{2max}$ or exercise performance in cool conditions following HA (23). Furthermore, euhydrated HA with controlled HR has been shown to improve $\dot{V}O_{2max}$ and exercise performance in hot, but not cool conditions in a randomized cross-over trial (24). However, the effects of hydration status during HA with controlled HR on $\dot{V}O_{2max}$ in a temperate environment is unknown.

Therefore, this study sought to characterize the responses to exercising HA with controlled HR and determine the influence of hydration status on the adaptive process. The effects of each regimen on time-trial performance in the heat and $\dot{V}O_{2max}$ in cool conditions were also investigated to identify the potential for each intervention to enhance performance in hot and cool conditions. It was hypothesized that i) matched daily levels of dehydration throughout HA with controlled HR would lead to similar adaptations compared to HA with maintained euhydration. In addition, ii) both HA strategies would enhance self-paced exercise performance in the heat, however iii) neither intervention would increase $\dot{V}O_{2max}$ in cool conditions.

METHODOLOGY

Participants

Eight males (Performance level 2, 25). with an average age, height, body mass, $\dot{V}O_{2max}$, W_{max} and maximal HR of 33 ± 5 years, 176 ± 5 cm, 75.7 ± 4.5 kg, 4.01 ± 0.61 $L \cdot min^{-1}$, 394 ± 28 W and 192 ± 7 $beats \cdot min^{-1}$, respectively volunteered to take part in the study following completion of a pre-screening health questionnaire. All were non-native residents of Qatar predominately originating from western Europe and North America, while two participants were Caucasians of South African nationality. All had resided in the Middle East for 2.1 years (range 0.3-5.6 years) before taking part in the study. Participants were all trained cyclists and triathletes regularly training ≥5 h per week with experience in cycling time-trial events. Participants provided written informed consent prior to taking part in this study, which was approved by Anti-Doping Lab Qatar Research Ethics Committee (Approval no. F201500105) and conducted in accordance with the Declaration of Helsinki.

Due to high average ambient temperatures in Qatar (Latitude: 25° North) where average daily peak temperatures range from 22-42°C for the coldest and warmest months of the year, respectively (26), participants were encouraged to minimize outdoor exercising exposures to once per week for a minimum of three weeks prior to the first visit to the laboratory. Over this period participants attended the laboratory to perform their training on cycle ergometers, and this timeframe was used to familiarize participants to the equipment and procedures of the study. All participants also reported deliberate avoidance of outdoor training in the summer months, instead opting for indoor training, cross training (e.g. swimming), travel from the region or a combination of the above.

Experimental Design

A within participant, counterbalanced crossover design study was completed, with participants undergoing two exercise HA interventions with controlled HR. Each intervention differed in the fluid intake strategy employed during HA, with participants maintaining euhydration (HA-EUH) or progressively reaching similar levels of dehydration (HA-DEH) each day via individualized prescribed fluid intakes. Before and after each HA period a graded maximal exercise test and self-paced laboratory cycling time-trial were conducted to determine the effects each intervention on exercise performance. Changes in RCV, blood volume (BV) and PV were determined via carbon monoxide (CO) rebreathing. Each intervention was separated by a washout period of 10 ± 3 weeks. All experiments were conducted at a similar time of day to minimize effects of circadian variation. Food and fluid intake were recorded in the 24 h period prior to experimental performance trials and days 1, 5 and 10 of the first HA intervention and was replicated throughout the second intervention.

Experimental procedures

A minimum of 72 h prior to the first HA exposure of each intervention an experimental trial was conducted to determine $\dot{V}O_{2max}$ in cool conditions and self-paced exercise performance in the heat. On arrival, participants provided a urine sample to determine their hydration status via urine specific gravity (USG; PAL-10s, ATAGO, Tokyo, Japan). Measurements of stature to the nearest 1 cm and nude body mass to the nearest 100 g were then taken using a stadiometer (SECA 798, Germany). Afterwards, participants self-inserted a rectal thermistor (DM 852, Ellab, A/S, Hillerød, Denmark) and dressed in cycling shorts and socks. Participants then sat quietly in the laboratory for a period of 10 min before measurements of resting rectal temperature (T_{rec}) and HR were taken. Participants then completed an incremental cycling test to exhaustion (Lode Excalibur Sport, Groningen, The Netherlands) in an environmental chamber (LowOxygen Systems, LOXY International Ltd., Berlin, Germany) with ambient temperature and relative humidity (RH) of 19.2 ± 1.9°C and 63 ± 10%. The incremental test consisted of five sub-maximal stages that began at 90 W and increased by 30 W every 4 min. Immediately after the final stage, a ramp increment of 30 $W \cdot min^{-1}$ occurred until volitional fatigue despite strong verbal encouragement. Breath-by-breath pulmonary gas exchange (Oxycon Pro, Jaeger, Höchberg, Germany), HR, T_{rec} and mean skin temperature (T_{sk}) were continuously recorded. Ratings of perceived exertion (RPE; Borg 6-20 scale, (27)) and thermal comfort (TC; 1-7 scale, (28)) were recorded at the end of each stage and immediately after the test. Sub-maximal values were averaged over the final minute of each stage while $\dot{V}O_{2max}$ was defined as the highest minute average recorded and maximal power output (W_{max}) was defined as the average minute power output prior to volitional fatigue.

Following the maximal exercise test, participants rested for 30 min in the main laboratory. Over this period 5 $ml \cdot kg^{-1}$ of water was consumed before participants entered a second environmental chamber (TEMI 1000, Sanwood environmental chambers co., Taiwan) and mounted a cycle ergometer (SRM, Jülich, Germany). Conditions within the chamber averaged 35.0°C ± 0.2°C and 60 ± 2% RH. Participants performed 5 min of unloaded pedaling before a zero-offset was performed according to manufacturer guidelines. Participants were then instructed to complete 30 min of self-paced cycling at the highest sustainable power output. Feedback was limited to time remaining in the form of a digital stopwatch. Plain water was consumed *ad libitum* throughout the trial. A fan provided convective airflow at 3 $m \cdot s^{-1}$. Power output and HR were measured continuously while T_{rec} and T_{sk} were measured at 6 min intervals. Overall RPE and TC was recorded immediately after the test.

HA with controlled HR

HA consisted of 90 min cycling exercise per day over 10 consecutive days in 40°C and 40% RH. Target exercise intensity was determined via linear regression of the $\dot{V}O_2$-power and $\dot{V}O_2$-HR relationships of the experimental visit. Each HA session involved an initial 15 min period of cycling at a constant workload equivalent to 65% $\dot{V}O_{2max}$ on the same electronically braked cycle ergometer used during the maximal incremental tests. Over the final 75 min of each session computer software (Lode ergometry manager 9.0) adjusted resistance every 30 s so that an exercising HR associated with 65% $\dot{V}O_{2max}$ was maintained. Participants were instructed to maintain a steady cadence ≥80 $rev \cdot min^{-1}$. The initial constant workload period was designed to raise HR and promote the onset of sweating while preventing the initial increase in T_{rec} being dampened by excessive increases in HR and corresponding adjustments in power. A fan provided 3 $m \cdot s^{-1}$ of convective airflow throughout. HR and power output were measured continuously while T_{rec}, T_{sk} and ambient conditions were recorded every 5 min, and RPE and TC every 10 min.

A minimum of 24 h prior to the first HA exposure participants attended the laboratory and completed 60 min of constant workload cycling in the heat (33°C and 50% RH). Nude body mass was recorded before and after exercise and corrected for fluid intake to determine sweating rate. On day one of each intervention either 90% (HA-EUH) or 10% (HA-DEH) of expected hourly sweat losses was provided to the nearest 1 ml in the form of a 0.1% electrolyte drink (HIGH5 ZERO, H5, Bardon, UK). Fluid was divided into six equal aliquots that were provided at the onset of exercise and every 15 min thereafter. Following each exposure, participants towel dried non-evaporated sweat and body mass was measured to determine sweat lost. Fluid volumes were then adjusted for each subsequent exposure to match changes in sweating rate and standardize end-exercise hydration status. Following HA-EUH participants were permitted to drink *ad libitum*. In HA-DEH, participants were also provided with water equaling ~150% of their body mass deficit and encouraged to consume this within ~2-3 h. No other instructions were given for control of posture or physical activity.

Measurement procedures

Hydration was assessed via USG, with a value ≤1.020 considered to be indicative of euhydration (29). All other resting measurements during HA were conducted at the end of a 10 min period in the supine position. Average HR was recorded over the final minute of rest via a chest strap and coded monitor (RS800CX, Polar Electro, Kempele, Finland). T_{rec} was measured via a sterilized re-usable medical grade thermistor located 15 cm beyond the anal sphincter. Area weighted T_{sk} was calculated from four sites (30) and was measured using iButton™ data loggers (Maxim Integrated Products, Sunnyvale, CA, USA) held in place on the skin surface using a thin strip of non-porous tape (Opsite Flexifix, Smith&Nephew Medical Ltd. Hull, UK) to allow convective airflow around the sensor.

Hemoglobin mass was determined via a modified optimized CO rebreathing technique (31). Between-day duplicate measurements were taken prior to each HA intervention, with a single measurement conducted ~24 h after day 10 of each HA protocol. Briefly, participants rebreathed a 1.2 $ml \cdot kg^{-1}$ bolus of CO for 2 min after a period of seated rest. Arterialized capillary fingertip blood samples were analyzed in quintuplicate (ABL 90 FLEX, Radiometer, BrØnshØj, Denmark) prior to and 7 min following the start of the rebreathing period to determine the percentage of hemoglobin saturated with CO. Absolute hemoglobin mass (in g) was then calculated following corrections for remaining CO in the rebreathing apparatus, washout of CO following the procedure and estimates of residual lung volume (31). In any case where hemoglobin mass differed by >2% between pre-HA measurements, the test was repeated. The typical measurement error for this technique with the investigators in our laboratory was 0.63%.

On days 1, 5 and 10 of each HA intervention a venous blood sample was collected from an antecubital vein without stasis via venipuncture (BD Eclipse 21 g, BD, Utah, USA). An 8 ml sample was collected into two 4 ml K^+ EDTA tubes (BD Vacutainer, BD, Utah, USA). Samples were immediately analyzed in duplicate in a Coulter counter (UniCell DxH 800 Coulter Analysis System, Beckman Coulter, CA, USA). Hemoglobin concentration was directly measured calorimetrically, and hematocrit was derived using the following equation

$$RBC \times (\frac{MCV}{10})$$

where, RBC is red blood cell count and MCV is mean corpuscular volume. These parameters had a typical error of 0.1 $g \cdot dL^{-1}$ and 0.34%, respectively. Values were paired with most recent hemoglobin mass measurements for calculations of RCV, BV and PV. Post exercise samples were collected immediately after exercise from a bed adjacent to the ergometer.

Statistical analyses

Two-way ANOVA with repeated measures analyses were used to determine differences in resting and exercising responses to each HA intervention on days 1, 5 and 10. Separate two-way ANOVA tests were conducted to analyze effects of HA interventions on exercise capacity in cool conditions and self-paced exercise performance in the heat. Mauchley's test was used to test the assumption of Sphericity. In cases where this assumption was violated a Greenhouse-Geisser correction factor was applied. Bonferroni *post-hoc* testing was employed to determine where differences occurred. Wilcoxon signed rank tests were used to analyze ordinal (RPE and TC) data. Separate *t*-tests were conducted between HA interventions for HR and T_{rec} at rest and after 15 min of exercise on days 2 and 3 to determine if there was an order effect or insufficient washout of HA adaptations between interventions. All statistical analyses were conducted using SPSS (Version 21, IBM, Armonk, US). Results are reported as mean ± SD. The level of significance was set at $P < 0.05$. Effect sizes are presented using partial eta squared values for analyses of variance ($\eta_p^2 \leq 0.02$: small; 0.02-0.13: medium; 0.13-0.26: large; (32)).

RESULTS

HA intervention summary

Ambient conditions throughout HA averaged 40.0 ± 0.3°C and 40.1 ± 1.6% RH. Initial 15 min power output was 171 ± 20 and 173 ± 22 W ($P = 0.75$, $\eta_p^2 = 0.09$), while target exercising HR for the remaining 75 min was 146 ± 7 and 145 ± 7 beats·min^{-1} ($P = 0.45$, $\eta_p^2 = 0.12$) in HA-EUH and HA-DEH, respectively. By design, fluid intakes differed between interventions ($P <0.001$, $\eta_p^2 = 0.98$) and increased throughout the 10-days ($P = 0.009$, $\eta_p^2 = 0.6$) by 334 ± 316 ml from 1803 ± 362 ml on day 1 of HA-EUH and by 39 ± 23 ml from 214 ± 35 ml on day 1 of HA-DEH, respectively. Body mass was maintained within -0.60 ± 0.26% of resting values during HA-EUH exposures, whereas HA-DEH resulted in daily body mass deficits of 2.85 ± 0.48% ($P < 0.001$, $\eta_p^2 = 0.97$). Body mass changes within each HA intervention did not differ between days ($P = 0.22$, $\eta_p^2 = 0.40$). PV and BV decreased by 7.7 ± 3.8 and 4.4 ± 2.3% respectively during exercise in HA-EUH and to a significantly greater extent in HA-DEH (14.0 ± 3.2%; $P = 0.006$, $\eta_p^2 = 0.74$ and 8.1 ± 1.9%; $P = 0.006$, $\eta_p^2 = 0.75$, respectively). The relative decreases in PV and BV from supine rest to the end of exercise did not differ between days in either intervention ($P = 0.30$, $\eta_p^2 = 0.39$ and $P = 0.49$, $\eta_p^2 = 0.11$, respectively).

Resting HA responses

A summary of resting measurements throughout each HA intervention is presented in Table 1. USG measurements indicated participants attended the laboratory in a euhydrated state each day ($P = 0.49$, $\eta_p^2 = 0.03$). Neither intervention altered resting T_{rec} ($P = 0.17$, $\eta_p^2 = 0.22$) and there was a tendency for a main effect of HA to decrease resting HR ($P = 0.07$, $\eta_p^2 = 0.31$). There was no effect of hydration strategy ($P = 0.85$, $\eta_p^2 = 0.006$) or HA-hydration interaction effects ($P = 0.38$, $\eta_p^2 = 0.13$) on resting HR. A significant main effect of HA was observed on both PV ($P = 0.048$, $\eta_p^2 = 0.38$) and BV ($P = 0.041$, $\eta_p^2 = 0.38$); however, pairwise analyses did not identify any significant differences between days (all $P > 0.05$). No effects of hydration strategy or interaction effects were observed on PV ($P = 0.64$, $\eta_p^2 = 0.03$ and $P = 0.40$, $\eta_p^2 = 0.12$, respectively) and BV ($P = 0.64$, $\eta_p^2 = 0.03$ and $P = 0.44$, $\eta_p^2 = 0.24$, respectively). RCV did not change with HA ($P = 0.27$, $\eta_p^2 = 0.17$). Similar T_{rec} and HR responses were observed between the first and second HA intervention on days 2 and 3 (all $P > 0.05$), indicating a lack of order and re-induction effects of the second HA intervention.

Exercising HA responses

The responses to days 1, 5 and 10 of each HA intervention are displayed in Figures 1 and 2. HR immediately after the initial 15 min of exercise at a constant workload decreased throughout HA (P < 0.001, η_p^2 = 0.75). This occurred after 10 days in HA-EUH, decreasing from 156 ± 9 on day 1 to 144 ± 9 beats·min^{-1} on day 10 (P = 0.02, Figure 1A). In contrast, a significant decrease in HR was observed by day 5 of HA-DEH from 154 ± 13 to 147 ± 9 beats·min^{-1} (P = 0.04) and did not decrease further with HA (P = 1.00, Figure 1B). Neither intervention altered the T_{rec} response to 15 min constant workload cycling in the heat (P = 0.31, η_p^2 = 0.15, Figure 1E and F). Similar 15 min exercising HR and T_{rec} responses on days 2 and 3 were also observed between the first and second HA interventions (all P > 0.05), suggesting the absence of an order and re-induction effect.

Average HR for the final 75 min of exercise each day of HA was 147 ± 6 and 146 ± 7 beats·min^{-1} in HA-EUH and HA-DEH, respectively (P = 0.72, η_p^2 = 0.11) and did not differ between interventions (P = 0.76, η_p^2 = 0.04, Figure 1A and B). Average power over this period was greater in HA-EUH compared to HA-DEH (P = 0.001, η_p^2 = 0.82) and increased to a greater extent throughout HA when euhydration was maintained (P < 0.001, η_p^2 = 0.81, Figure 1C and D). The average T_{rec} during HR controlled exercise was 38.4 ± 0.2°C in both HA-EUH and HA-DEH and did not differ between days (P = 0.49, η_p^2 = 0.23) or interventions (P = 0.90, 0.003, Figure 1E and F). There was a main effect of HA on T_{sk} (P = 0.03, η_p^2 = 0.69) with an average decrease of 0.63 ± 0.50°C between days 1 and 10 in HA-EUH (P = 0.03, Figure 1G). This was accompanied by a tendency for sweating rate to increase from 1.55 ± 0.17 L·h^{-1} on day 1 to 1.74 ± 0.21 L·h^{-1} on day 10. (P = 0.06, η_p^2 = 0.46). However, neither T_{sk} (-0.35 ± 0.16°C, P = 0.30, Figure 1H) nor sweat rate (+0.15 ± 0.16, from 1.51 ± 0.23 L·h^{-1}, P = 0.12) changed significantly between days 1 and 10 of HA-DEH. On day 10 sweat rate was significantly greater in HA-EUH compared to HA-DEH (P = 0.02). Perceptual responses to exercise remained unchanged between days of HA and did not differ between interventions (all P > 0.05), with RPE and TC during controlled HR exercise averaging 12.3 ± 1.4 ("somewhat hard") and 5.3 ± 0.7 ("comfortable warm"), respectively (Figure 2).

Maximal aerobic capacity and self-paced exercise performance with HA

The HR and $\dot{V}O_2$ responses to graded incremental exercise in cool conditions were similar prior to each HA intervention and were unaltered by either protocol (all P > 0.05). W_{max} did not change with HA (P = 0.25, η_p^2 = 0.18), averaging 385 ± 35 and 393 ± 27 W prior to and 397 ± 24 W and 399 ± 23 W following HA-EUH and HA-DEH, respectively. $\dot{V}O_{2max}$ was 3.92 ± 0.49 and 4.05 ± 0.47 $L \cdot min^{-1}$ prior to HA-EUH and HA-DEH, respectively (*P* = 0.12). There was a main effect of HA on $\dot{V}O_{2max}$ (*P* = 0.017, η_p^2 = 0.58), however pairwise analyses only identified a tendency for a slight ~0.16 $L \cdot min^{-1}$ increase following HA-EUH and HA-DEH, reaching 4.09 ± 0.44 and 4.19 ± 0.39 $L \cdot min^{-1}$, respectively (both *P* = 0.08, Figure 3A).

There was no main effect of hydration on time-trial power output in the heat (*P* = 0.94, η_p^2 = 0.001). However, there was a significant main effect of HA (*P* = 0.03, η_p^2 = 0.69) and no interaction effects (*P* = 0.63, η_p^2 = 0.04). Power during the time-trials was similar prior to each HA intervention, averaging 231 ± 33 and 235 ± 32 W before HA-EUH and HA-DEH, respectively (*P* = 0.62). Average power output increased significantly by 19 ± 16 W following HA-EUH (*P* = 0.012), but not following HA-DEH (13 ± 26 W, *P* = 0.21, Figures 3B and 4A and B). HR (*P* = 0.97, η_p^2 = 0.011), T_{rec} (*P* = 0.85, η_p^2 = 0.13) and T_{sk} (*P* = 0.33, η_p^2 = 0.47) responses did not differ between time-trials (Figure 4).

DISCUSSION

This study sought to characterize the adaptive responses to HA with controlled HR and determine whether hydration strategy altered the adaptation process. The study also sought to determine the potential ergogenic effects of each intervention on $\dot{V}O_{2max}$ in a cool environment and self-paced exercise performance in humid heat. Our findings indicate that i) HA led to decreases in HR during the initial 15 min of constant workload exercise and, ii) decreased average T_{sk}, and increased sweat rate and power output for the same exercising HR over the final 75 min. However, iii) these adaptations occurred when euhydration was maintained, as HA-DEH did not result in a significant increase in sweat rate or reduce T_{sk}. Furthermore, iv) neither HA intervention independently led to changes in resting T_{rec}, HR, PV nor BV. Finally, v) while there was a trend for an increase in $\dot{V}O_{2max}$, neither HA-EUH nor HA-DEH independently resulted in notable effects on sub-maximal or maximal $\dot{V}O_2$ responses to exercise in cool conditions, whereas vi) time-trial performance in the heat was improved following HA-EUH, but not HA-DEH .

Adaptations to HA with controlled HR

The purpose of utilizing the controlled HR approach was to ensure the maintenance of a constant adaptive stimulus across each day of HA (5). By adopting a target HR associated with sub-maximal exercise, trained individuals achieved a stable elevation in T_{rec} coupled with high T_{sk}, regardless of hydration status (Figure 1). A stable and sustained elevation in RPE and TC was also attained (Figure

2). The level of heat strain achieved in the current study was similar to that observed with the isothermal/controlled hyperthermia approach (4,9,11,18) and facilitated several favorable adaptations. These included a ~10 beats·min^{-1} reduction in HR during constant workload exercise, ~0.5°C reduction in T_{sk}, ~0.17 L·h^{-1} increase in sweat rate and an overall ~21 W increase in power output when cycling at a given HR (Figure 1). Our findings are in line with those reported elsewhere using this HA approach. For example, Keiser et al., (24) observed a 10% increase in exercise performance in the heat, 6% increase in PV, a reduction in sweat sodium concentration and a much larger 26% increase in sweat output compared to the present study (13%) after 10 days of HA in similar conditions. One possibility for the relatively smaller increases in sweat rate with HA in the present study may relate to differences in fitness status (~8 ml·kg^{-1}) and performance levels (Level 2 vs. 3 (25)) between our participants and those of Keiser et al., (24). For example, although trained, untrained and unfit individuals exhibit similar increases in sweat rate with HA (33), fitter individuals exhibit larger whole body and local sweat rates than those who are less fit during exercise at the same relative intensity (34). Despite these differences, our results indicate significant favorable adaptations to heat stress and improved exercise performance in a hot environment, particularly when euhydration was maintained. Therefore, exercise heat stress at a target HR via alterations in workload is a practicable and effective approach to HA.

Influence of hydration status on adaptation

Dehydration has been proposed to facilitate HA adaptations (6) as it increases fluid regulatory responses during exercise (16). Controlled hyperthermia with permissive dehydration has been shown to enhance electrolyte retention and expand PV (9), and this has been suggested to be beneficial for short-term interventions (10) as a greater BV may facilitate a decrease in HR via imporvements in diastolic filling (21). However, few investigations have directly assessed the influence of hydration status on the development of HA (10,11,13) and the proposed benefit is unclear (14). In the present investigation HR during initial constant workload exercise was decreased in both interventions, but the average significant increase in sweat rate and decrease in T_{sk} that occurred in HA-EUH was not observed with HA-DEH (Figure 1). The 17 ± 19 W difference in absolute workload during HR controlled exercise between interventions on day 10, and therefore the level of metabolic heat production (34), may explain the small ~0.08 L·h^{-1} differences in sweat rate observed. It is therefore unclear if this difference is due to adaptations specific to each intervention, or our experimental design.

Although a main effect of HA day on resting BV and PV was noted, no significant absolute or relative differences between days within either intervention was identified (Table 1). The reason for this lack of an effect is unclear, as HA typically results in a 4% expansion in PV (35), similar to the average changes observed on day 5 with HA-DEH. Although not significant, the ~200 ml greater BV may have facilitated the ~7 beats·min^{-1} decrease in HR in HA-DEH compared to HA-EUH on this day (Figure 1B). However, BV and PV were similar between days 1 and 10, suggesting a return towards baseline values. HA protocols with permissive dehydration have reported large, sustained expansions in the region of 5-10% (11,14,34). By design, HA-DEH resulted in a daily ~3% reduction in body mass; surpassing the 2% change expected to stimulate a fluid regulatory response (36). Other investigations standardized the volumes of fluids consumed, resulting in progressively greater daily body mass deficits as interventions progressed (9,10,13). It is possible this approach may be necessary to repeatedly stimulate increases in the extracellular fluid compartment following exercise (18). However, Neal and colleagues (13) induced body mass deficits between 2.3 to 3.1% over 10 days of HA. Although they observed slight increases in plasma osmolality, these were not greater than the 2% required to stimulate renal water conservation (36). We did not measure plasma osmolality in the present investigation, but this may explain the observed similarities in PV between days.

Effects of HA on maximal aerobic capacity in cool conditions

In the present study there was a small main effect of HA on $\dot{V}O_{2max}$ (~0.16 L·min^{-1}) in a cool environment. However, neither intervention independently increased $\dot{V}O_{2max}$ (Figure 3A), nor altered the sub-maximal responses during incremental exercise. Furthermore, the significant main effect of HA on $\dot{V}O_{2max}$ (~4%) was also similar in magnitude to the differences observed between interventions pre-acclimation (~3.6% or 0.13 L·min^{-1}). Together, this suggests that the observed effect in the current investigation is not meaningfully larger than the intra-individual between-day responses to maximal incremental exercise in these individuals. This lack of change is supported by the similarities in resting and exercising cardiovascular, thermoregulatory, ventilatory and metabolic responses observed following HA. For instance, an increase in oxygen delivery and/or maximal cardiac output may result from increases in RCV and/or PV with HA (7,21). However, neither parameter was significantly altered by HA in the present study (Table 1). Given these similarities, it does not seem likely either intervention altered oxygen carrying capacity or delivery to locomotor muscles. Instead, these findings agree with those of others (23,24), that HA does not alter maximal incremental exercise performance in cool conditions.

Self-paced exercise performance following HA

HA-EUH led to significant improvements in time-trial performance in the heat. Average power was 19 W (9%) greater following HA-EUH, with all participants demonstrating an increase in performance (Figures 3B and 4A). This finding is typical of the effects of HA on closed-loop self-regulated performance tasks, with others reporting improvements in cycling time-trials between 8 and 15% (21,23,24,37). Conversely, HA-DEH did not significantly improve time-trial performance, with 3 participants achieving slightly lower average power outputs following the intervention (Figures 3B and 4B). This finding further supports the notion that more complete thermoregulatory adaptations occurred in HA-EUH compared to HA-DEH that facilitated improved performance in hot-humid conditions. There are several reasons for this possibility, such as $\dot{V}O_{2max}$ and time-trial performance being similar prior to HA-DEH and HA-EUH, and post-intervention $\dot{V}O_{2max}$ being similarly influenced by both HA interventions. Also, the pacing profiles, thermoregulatory and cardiovascular responses to exercise did not differ between trials. Self-paced exercise in both hot and cool conditions tends to be conducted at a relative exercise intensity commensurate with distance/duration (38), and decrements in power output occur over time with the development of hyperthermia and cardiovascular strain (1). Although $\dot{V}O_2$ was not measured during the time-trials in this study, given there were similar thermal and cardiovascular responses at a greater average power output following HA-EUH, it is likely that HA facilitated improved heat exchange between the body and the environment. However, the possibility of altered training stimuli between HA-EUH and HA-DEH affecting the exercise performance may not be dismissed entirely. For example, during HR controlled exercise ~560 kJ more work was completed throughout HA-EUH compared to HA-DEH. Therefore, the presence of a training effect cannot be ruled out and is an accepted limitation.

Limitations

Future studies may seek to determine the effectiveness of interventions against appropriately work-matched controls (22). Acutely, to maintain a stable HR during exercise in the heat, large reductions in workload must occur (39), which result in a significantly lower stroke volume, cardiac output and whole-body oxygen consumption during dehydrated compared to euhydrated exercise (40), in addition to less work being performed during HA-DEH. One further consideration is that participants began each exposure fully hydrated, and therefore only experienced moderate levels of dehydration during the final ~15 min of exercise in HA-DEH compared to HA-EUH. While HA-EUH increased sweat rate and improved performance, this was not to a significantly greater extent compared to HA-DEH. Therefore, we cannot conclusively state that the differences in observed adaptations are merely due to altered fluid intake strategies. Instead, considering our findings it is recommended that euhydration is maintained during exercise at a target HR in the heat, as it mitigates cardiovascular strain and facilitates the exercising component of HA.

Conclusions

Both euhydrated and dehydrated exercise at a controlled HR in the heat result in an elevated and sustained whole-body temperature and sweat rate that facilitate several adaptive responses typical of HA. These responses were associated with sustained elevations in the perception of exertion and thermal comfort throughout acclimation. Therefore, such an intervention may be easily implemented by athletes as a means of safely regulating exercise intensity during heat exposures. Maintaining euhydration during HA appears to be more beneficial than allowing dehydration to occur, as demonstrated by an enhanced sweat rate, decreased T_{sk} and improved self-paced exercise performance in the heat. Indeed, dehydration consistently impaired the exercising component of HA at a controlled HR and limited adaptations beyond day 5 of the intervention. Finally, neither HA-EUH nor HA-DEH independently altered the thermal, cardiovascular, ventilatory or metabolic responses to sub-maximal and maximal exercise in cool conditions.

Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author Contributions

GT and JP designed the study. GT, DN, NR and JP collected the data. GT, JP, JG-A and DN interpreted and analysed the data. GT wrote the initial manuscript and all authors provided input and approved the final version.

Funding

This study was funded by Aspetar Orthopaedic and Sports Medicine Hospital, Doha, Qatar.

Acknowledgments

The authors wish to thank Prof. Mathew Wilson, Prof. Sebastien Racinais, Dr. Nathan Townsend, Dr. Lee Taylor and Miss Bernadette Burke for their assistance and technical expertise. The authors also extend a special thank you to the participants who made this study possible. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation, and statement that results of the present study do not constitute endorsement by ACSM.

References

1. Périard JD, Cramer MN, Chapman PG, Caillaud C, Thompson MW. Cardiovascular strain impairs prolonged self-paced exercise in the heat. Exp Physiol. 2011;96(2):134–44.

2. Sawka MN, Knowlton RG, Critz JB. Thermal and circulatory responses to repeated bouts of prolonged running. Med Sci Sports Exerc. 1979;11(2):177–80.

3. Racinais S, Périard JD, Karlsen A, Nybo L. Effect of heat and heat acclimatization on cycling time trial performance and pacing. Med Sci Sports Exerc. 2014;47(3):601–6.

4. Fox RH, Goldsmith R, Kidd DJ, Lewis HE. Acclimatization to heat in man by controlled elevation of body temperature. J Physiol. 1963;166(3):530–47.

5. Périard JD, Racinais S, Sawka MN. Adaptations and mechanisms of human heat acclimation: Applications for competitive athletes and sports. Scand J Med Sci Sport. 2015;25(S1):20–38.

6. Taylor NAS. Human heat adaptation. Compr Physiol. 2014;4(1):325–65.

7. Scoon GSM, Hopkins WG, Mayhew S, Cotter JD. Effect of post-exercise sauna bathing on the endurance performance of competitive male runners. J Sci Med Sport. 2007;10(4):259–62.

8. Zurawlew MJ, Walsh NP, Fortes MB, Potter C. Post-exercise hot water immersion induces heat acclimation and improves endurance exercise performance in the heat. Scand J Med Sci Sports. 2016;26(7):745–54.

9. Patterson MJ, Stocks JM, Taylor NAS. Sustained and generalized extracellular fluid expansion following heat acclimation. J Physiol. 2004;559(1):327–34.

10. Garrett AT, Goosens NG, Rehrer NJ, Patterson MJ, Harrison J, Sammut I, et al. Short-term heat acclimation is effective and may be enhanced rather than impaired by dehydration. Am J Hum Biol. 2014;26(3):311–20.

11. Neal RA, Corbett J, Massey HC, Tipton MJ. Effect of short-term heat acclimation with permissive dehydration on thermoregulation and temperate exercise performance. Scand J Med Sci Sport. 2016;26(8):875–84.

12. Gibson OR, Mee JA, Tuttle JA, Taylor L, Watt PW, Maxwell NS. Isothermic and fixed intensity heat acclimation methods induce similar heat adaptation following short and long-term timescales. J Therm Biol. 2015;49–50:55–65.

13. Neal RA, Massey HC, Tipton MJ, Young JS, Corbett J. Effect of permissive dehydration on induction and decay of heat acclimation, and temperate exercise performance. Front Physiol. 2016;7:1–11.

14. Akerman AP, Tipton M, Minson CT, Cotter JD. Heat stress and dehydration in adapting for performance: Good, bad, both, or neither? Temperature. 2016;3(3):412–36.

15. Sawka MN, Toner MM, Francesconi RP, Pandolf KB. Hypohydration and exercise: Effects of heat acclimation, gender, and environment. J Appl Physiol Respir Environ Exerc Physiol. 1983;55(4):1147–53.

16. Kenefick RW, Maresh CM, Armstrong LE, Riebe D, Echegaray ME, Castellani JW. Rehydration with fluid of varying tonicities: Effects on fluid regulatory hormones and exercise performance in the heat. J Appl Physiol. 2007;102(5):1899–905.

17. Nagashima K, Wu J, Kavouras SA, Mack GW. Increased renal tubular sodium reabsorption during exercise-induced hypervolemia in humans. J Appl Physiol. 2001;91(3):1229–36.

18. Patterson MJ, Stocks JM, Taylor NAS. Whole-body fluid distribution in humans during dehydration and recovery, before and after humid-heat acclimation induced using controlled hyperthermia. Acta Physiol. 2014;210(4):899–912.

19. Schleh MW, Ruby BC, Dumke CL. Short term heat acclimation reduces heat stress, but is not augmented by dehydration. J Therm Biol. 2018;78:227–34.

20. Pethick WA, Murray HJ, McFadyen P, Brodie R, Gaul CA, Stellingwerff T. Effects of hydration status during heat acclimation on plasma volume and performance. Scand J Med Sci Sport. 2019;29(2):189–99.

21. Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improves exercise performance. J Appl Physiol. 2010;109(4):1140–7.

22. Corbett J, Neal RA, Lunt HC, Tipton MJ. Adaptation to heat and exercise performance under cooler conditions: a new hot topic. Sport Med. 2014;44(10):1323–31.

23. Karlsen A, Racinais S, Jensen M V., Nørgaard SJ, Bonne T, Nybo L. Heat acclimatization does not improve VO2max or cycling performance in a cool climate in trained cyclists. Scand J Med Sci Sport. 2015;25(S1):269–76.

24. Keiser S, Flück D, Hüppin F, Stravs A, Hilty MP, Lundby C. Heat training increases exercise capacity in hot but not in temperate conditions: A mechanistic counter-balanced cross-over study. Am J Physiol - Hear Circ Physiol. 2015;309(5):H750–61.

25. Decroix L, De Pauw K, Foster C, Meeusen R. Guidelines to classify female subject groups in sport-science research. Int J Sports Physiol Perform. 2016;11(2):204–13.

26. Climate and Weather Averages in Doha, Qatar [Internet]. 2018. Available from: https://www.timeanddate.com/weather/qatar/doha/climate

27. Borg GA. Pyshophysical bases of perceived exertion. Med Sci Sports Exerc. 1982;14(5):377–81.

28. Bedford T. The warmth factor in comfort at work. MRC Industrial Health Board Report. 1936.

29. Armstrong LE, Maresh CM, Castellani JW, Bergeron MF, Kenefick RW. Urinary indices of hydration status. Int J Sport Nutr. 1994;4:265–79.

30. Ramanathan NL. a New Weighting System for Mean Surface Temperature of the Human Body. J Appl Physiol. 1964;19:531–3.

31. Schmidt W, Prommer N. The optimised CO-rebreathing method: A new tool to determine total haemoglobin mass routinely. Eur J Appl Physiol. 2005;95(5–6):486–95.

32. Cohen JS. Statistical power analysis for the behavioural sciences. 2nd ed. New York Academic Press; 1988. 579 p.

33. Shvartz E, Shapiro Y, Magazanik A, Meroz A, Birnfiesd H, Mechtinger A, et al. Heat acclimation, physical fitness, and responses to exercise in temperate and hot environments. J Appl Physiol. 1977;43(4):678–83.

34. Jay O, Bain AR, Deren TM, Sacheli M, Cramer MN. Large differences in peak oxygen uptake do not independently alter changes in core temperature and sweating during exercise. Am J Physiol - Regul Integr Comp Physiol. 2011;301(3):832–41.

35. Tyler CJ, Reeve T, Hodges GJ, Cheung SS. The Effects of heat adaptation on physiology, perception and exercise performance in the heat: a meta-analysis. Sport Med. 2016;46(11):1699–724.

36. Cheuvront SN, Kenefick RW. Dehydration: Physiology, assessment, and performance effects. Compr Physiol. 2014;4(1):257–85.

37. Garrett AT, Creasy R, Rehrer NJ, Patterson MJ, Cotter JD. Effectiveness of short-term heat acclimation for highly trained athletes. Eur J Appl Physiol. 2012;112(5):1827–37.

38. Périard JD, Racinais S. Self-paced exercise in hot and cool conditions is associated with the maintenance of %VO2peak within a narrow range. J Appl Physiol. 2015;118(10):1258–65.

39. Wingo JE, Cureton KJ. Maximal oxygen uptake after attenuation of cardiovascular drift during heat stress. Aviat Sp Environ Med. 2006;77(7):687–94.

40. Nybo L, Jensen T, Nielsen B, González-Alonso J. Effects of marked hyperthermia with and without dehydration on VO2kinetics during intense exercise. J Appl Physiol. 2001;90(3):1057–64.

Figure Legends

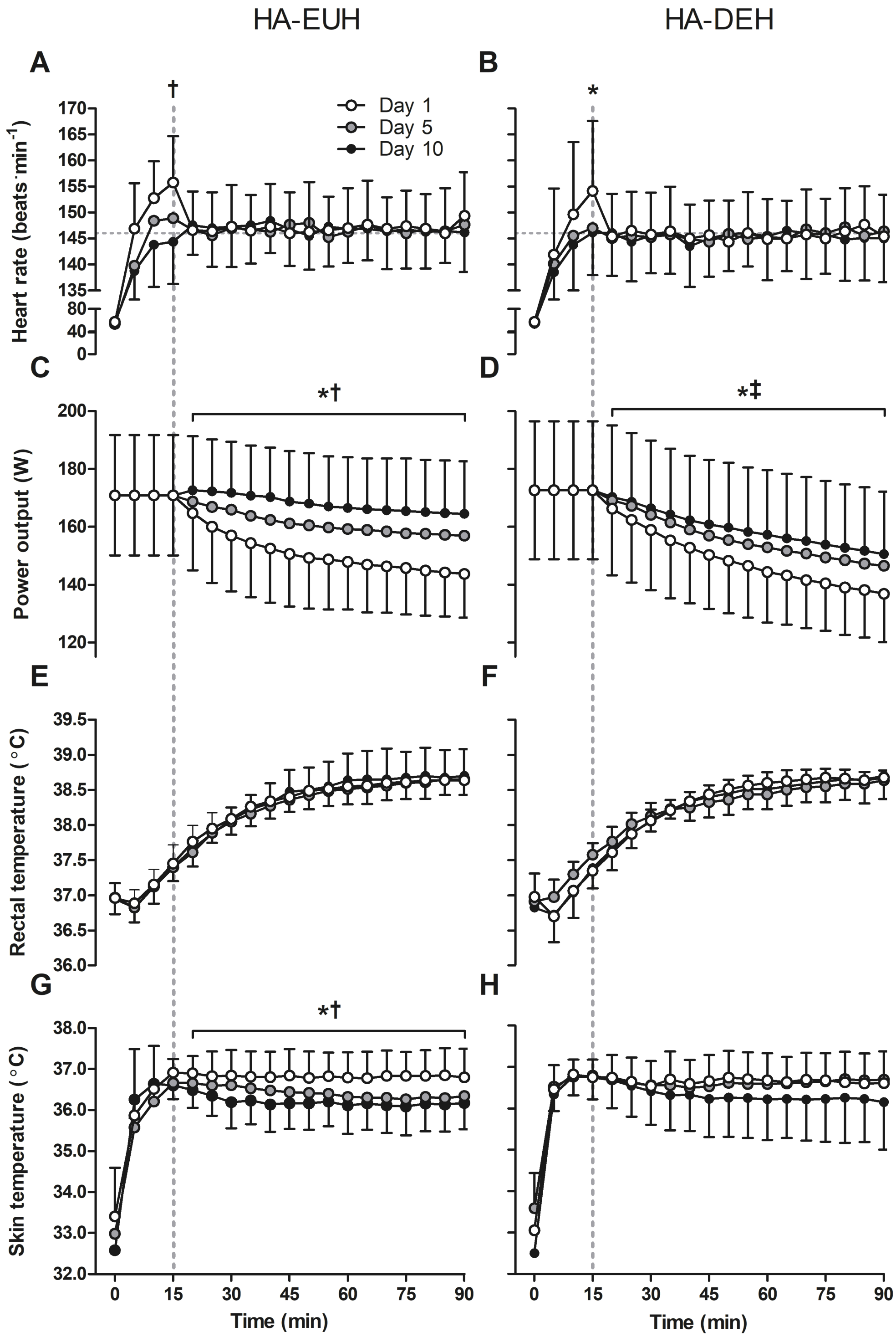
Figure 1. Day 1, 5 and 10 responses to euhydrated (EUH) and dehydrated (DEH) heat acclimation (HA) with controlled heart rate. Horizontal dotted lines show target exercising heart rates (A, B) that were achieved via manipulations in workload (C, D). Vertical dotted lines show point at which heart rate control began. Rectal (E, F) and skin temperatures (G, H) were recorded throughout each exposure. *Significant difference between day 5 and day 1 ($P \leq 0.04$). †Significant difference between day 10 and day 1 ($P \leq 0.04$). ‡Significantly different from EUH during heart rate-controlled exercise each day ($P \leq 0.04$). Solid lines indicate differences are between 75 min average values.

Figure 2. Average rating of perceived exertion (A, B) and thermal comfort (C, D) throughout days 1, 5 and 10 of euhydrated (EUH) and dehydrated (DEH) heat acclimation (HA) with controlled heart rate. Vertical dotted lines show point at which heart rate control began.

Figure 3. Individual changes in $\dot{V}O_{2max}$ in cool conditions (A) and average power output during a 30 min time-trial in hot-humid conditions (B) pre- to post-HA with controlled heart rate. *Significantly different from pre-HA ($P = 0.012$).

Figure 4. Power output (A, B) heart rate (C, D), core temperature (E, F) and mean skin temperature (G, H) during 30 min cycling time-trials conducted in hot humid conditions before and after euhydrated (EUH) and dehydrated (DEH) heat acclimation (HA) with controlled heart rate. *Significant overall difference from pre-HA ($P = 0.012$).

Figure 1.



HA-EUH
HA-DEH
A
B
C
D
E
F
G
H
Day 1
Day 5
Day 10
Heart rate (beats·min-1)
Power output (W)
Rectal temperature (°C)
Skin temperature (°C)
Time (min)
*†
*‡

Figure 2.

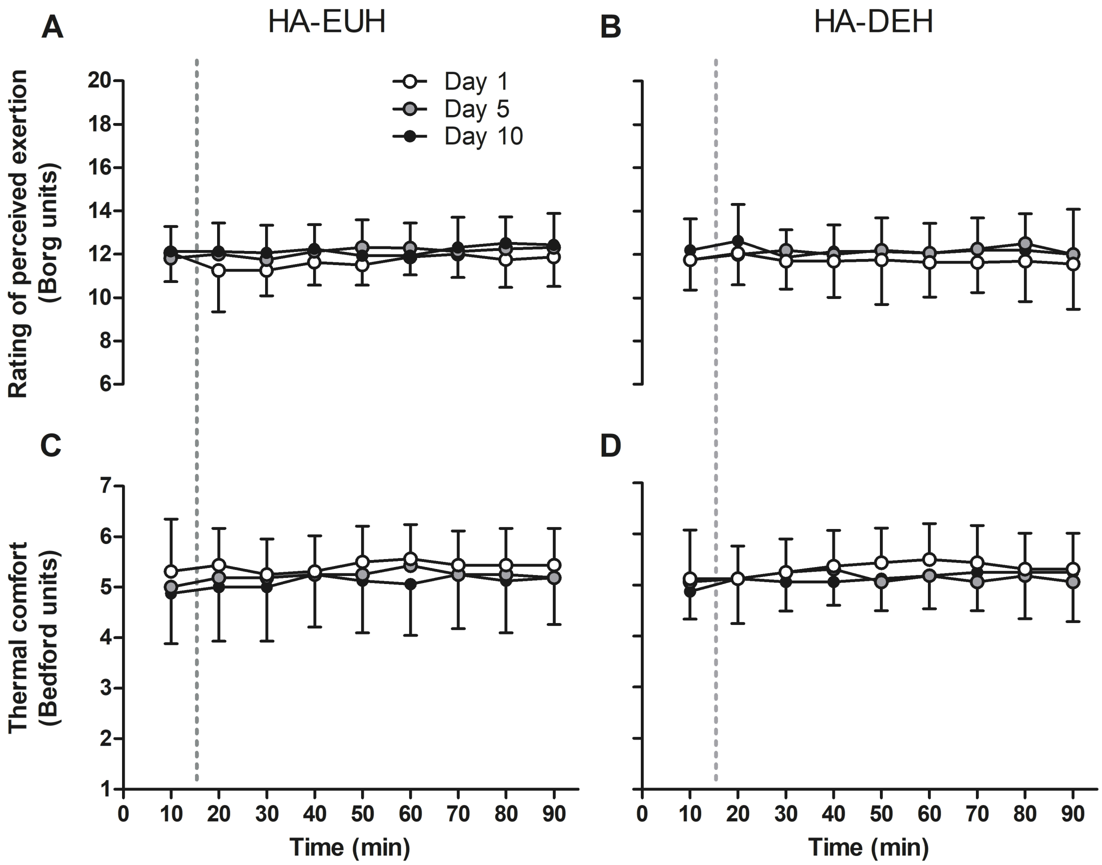
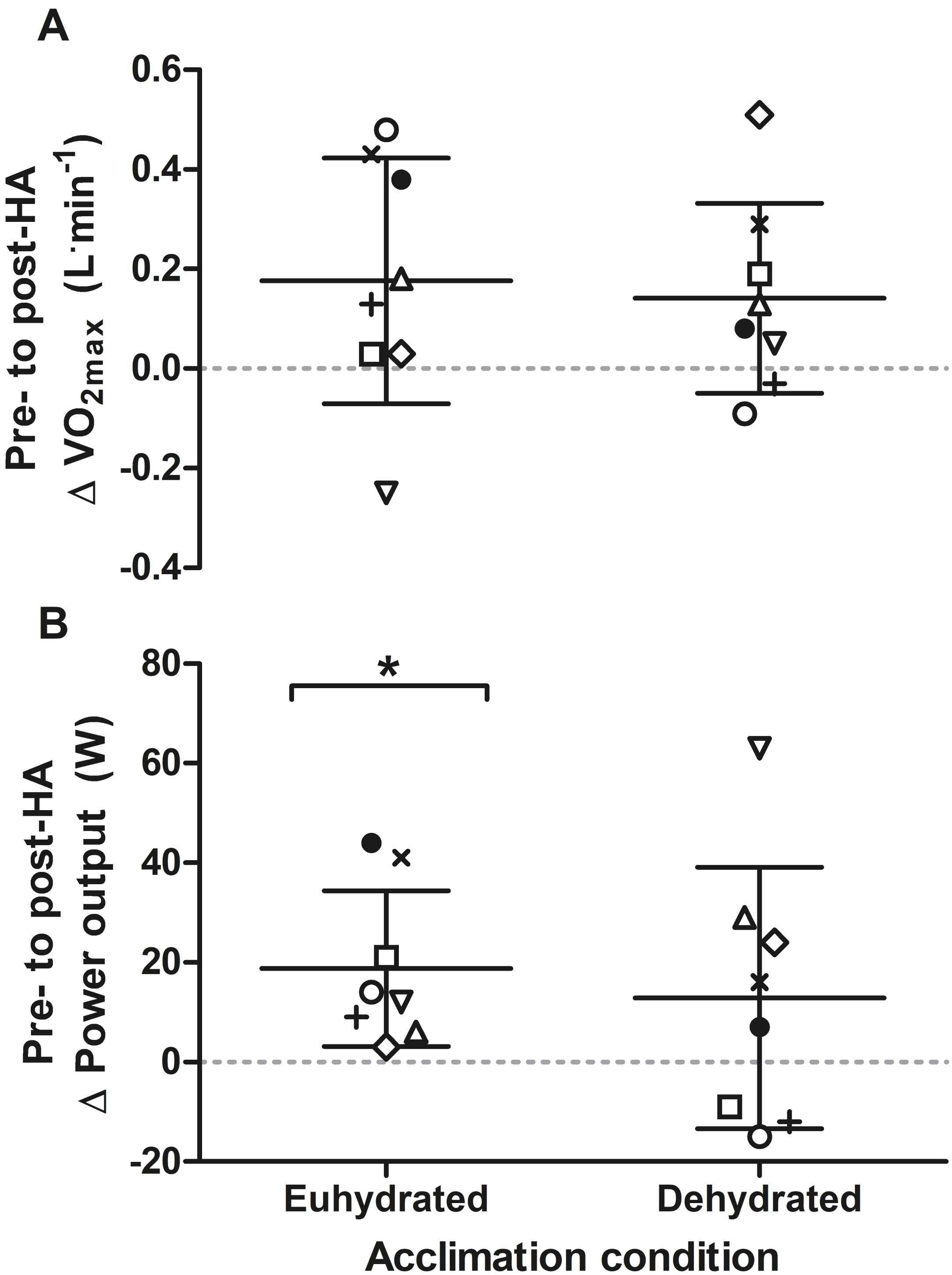
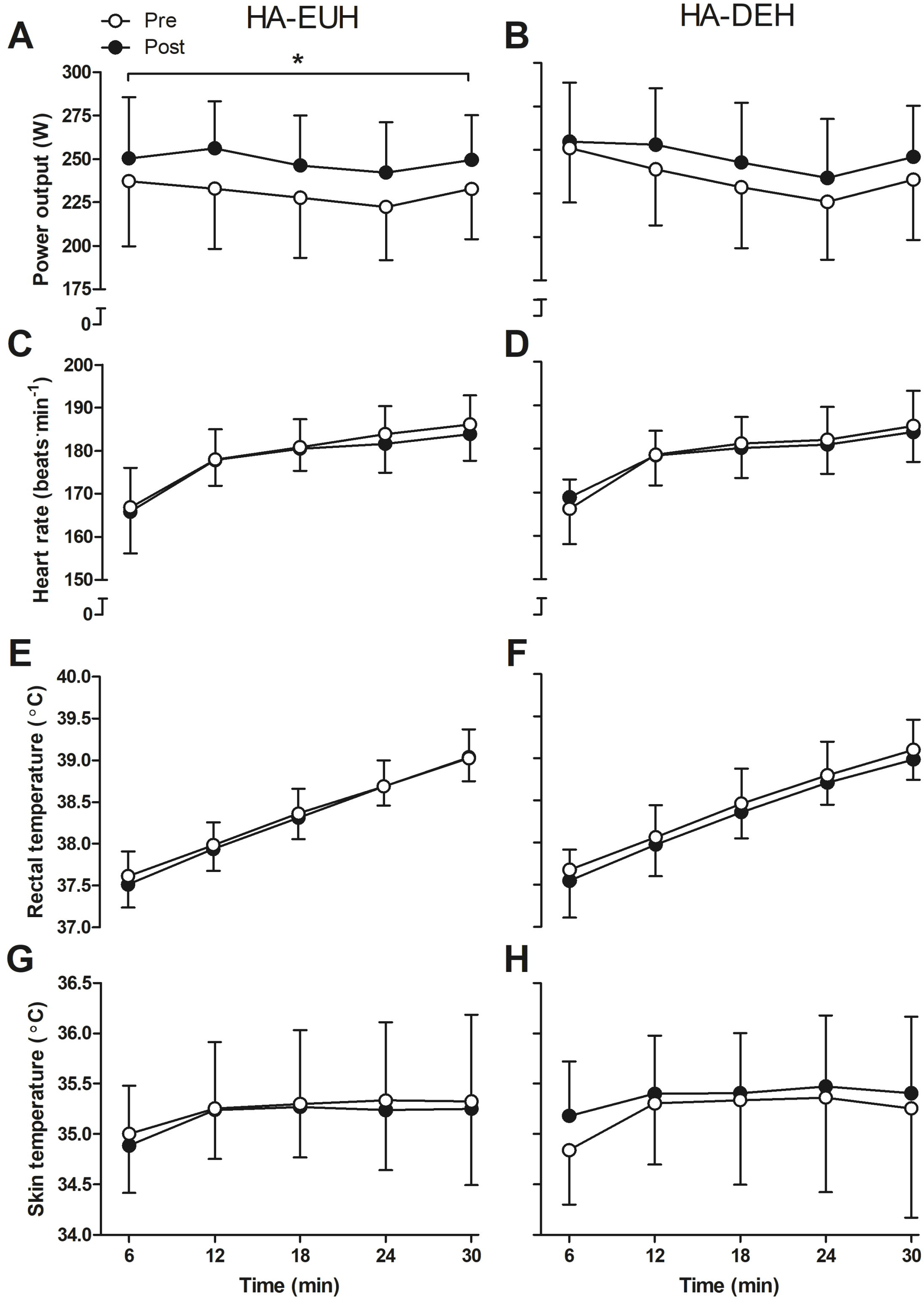

A
HA-EUH
B
HA-DEH
Day 1
Day 5
Day 10
Rating of perceived exertion (Borg units)
20
18
16
14
12
10
8
6
C
D
Thermal comfort (Bedford units)
7
6
5
4
3
2
1
0 10 20 30 40 50 60 70 80 90
Time (min)
0 10 20 30 40 50 60 70 80 90
Time (min)

Figure 3.



A
0.6
0.4
0.2
0.0
-0.2
-0.4
Pre- to post-HA
Δ VO2max (L·min-1)
B
80
60
40
20
0
-20
*
Pre- to post-HA
Δ Power output (W)
Euhydrated
Dehydrated
Acclimation condition

Figure 4.



A
B
C
D
E
F
G
H
HA-EUH
HA-DEH
Pre
Post
*
Power output (W)
300
275
250
225
200
175
0
Heart rate (beats·min-1)
200
190
180
170
160
150
0
Rectal temperature (°C)
40.0
39.5
39.0
38.5
38.0
37.5
37.0
Skin temperature (°C)
36.5
36.0
35.5
35.0
34.5
34.0
6
12
18
24
30
Time (min)