Heat acclimation responses of an ultra-endurance running group preparing for hot desert-based competition

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Heat acclimation responses of an ultra-endurance running group preparing for hot desert-based competition

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Abstract
Heat acclimation induces adaptations that improve exercise tolerance in hot conditions. Here we report novel findings into the effects of ultra-marathon specific exercise load in increasing hot ambient conditions on indices of heat acclimation. Six male ultra-endurance runners completed a standard pre-acclimation protocol at 20°C ambient temperature (Tamb), followed by a heat acclimation protocol consisting of six 2 h running exercise-heat exposures (EH) at 60% VO2max on a motorised treadmill in an environmental chamber. Three EH were performed at 30°C Tamb followed by another three EH at 35°C Tamb. EH were separated by 48 h within Tamb and 72 h between Tamb. Nude body mass (NBM), blood and urine samples were collected pre-exercise; while NBM and urine were collected post-exercise. Rectal temperature (Tr), heart rate (HR), thermal comfort rating (TCR) and rating of perceived exertion were measured pre-exercise and monitored every 5 min during exercise. Water was provided ad libitum during exercise. Data were analysed using a repeated measures and one-way analysis of variance (ANOVA), with post hoc Tukey’s HSD. Significance was accepted as P < 0.05. Overall mean Tr was significantly lower during 30°C EH3 and 35°C EH3 compared with their respective EH1 (–0.20 and –0.23°C, respectively; P < 0.05). Similarly, overall mean HR was significantly lower during 30°C EH3 and 35°C EH3 compared with their respective EH1 (8 and 7 bpm respectively; P < 0.05). A significant decrease in overall mean TCR was observed during 35°C EH3, compared with 35°C EH1 (P < 0.05). Significant increases in resting pre-exercise plasma volume (estimated from Hb and Hct) were observed by 30°C EH3 (7.9%; P < 0.05). Thereafter, plasma volume remained above baseline throughout the experimental protocol. Two EH of 2 h at 60% VO2max at 30°C Tamb was sufficient to initiate heat acclimation in all ultra-endurance runners. Further, heat acclimation responses occurred with increasing EH to 35°C Tamb. Preventing exertional heat illnesses and optimising performance outcomes in ultra-endurance runners may occur with exposure to at least 2 h of exercise-heat stress on at least two occasions in the days leading up to multi-stage ultra-marathon competition in the heat.

Keywords: Heart rate, body temperature, thermal comfort, hydration, sweat rate

Introduction
In recent years, multi-stage ultra-marathon events have become increasingly popular amongst the recreationally active endurance enthusiast. Multi-stage ultra-marathon events consist of consecutive days (e.g. 5–8 days) of prolonged loaded running (e.g. 30–80 km day–1, totalling ≥200 km running with 5–15 kg load), and are habitually performed in extreme environmental temperatures [e.g. ≥30°C ambient temperature (Tamb)]. Competing in such hot environmental conditions results in accelerated increases in core temperature; subsequently promoting cardiovascular, thermoregulatory and metabolic strain that hinders exercise performance compared with competing in thermoneutral conditions (American College of Sports Medicine et al., 2007; Hargreaves, 2008). It is well established that time to exercise fatigue in the heat corresponds with the attainment of an increased core temperature to a critical set-point, exacerbating physiological strain possibly through neuroendocrine mechanisms (e.g. hypothalamic control, sympathetic and parasympathetic tone, stress
and renal hormone responses) (Hargreaves, 2008; Nielsen et al., 1993; Tatterson, Hahn, Martin, & Febbraio, 2000).

Participating in such a physiologically demanding event poses a formidable challenge for the body in regulating normal homeostatic temperature. High rates of metabolic heat production due to exercise stress and attenuated heat losses due to environment conditions are likely to accelerate core temperature increases to its critical set-point whereby performance is perturbed, and more importantly, health risk is potentially viable (e.g. heat illnesses) (American College of Sports Medicine et al., 2007; Wendt, van Loon, & Lichtenbelt, 2007). Preparing the body to cope with exercising in hot ambient conditions is a fundamental strategy, ultra-endurance runners must adopt to optimise performance and avoid unwanted heat-related illnesses; especially since a large majority of ultra-endurance runners reside in countries with mean $T_{amb}$ of $\leq 20^\circ C$.

Heat acclimation is the artificial exposure to heat, which promotes adaptations that enables the body to cope with exercising in hot conditions (Nielsen et al., 1993; Pandolf, 1998; Patterson, Stocks, & Taylor, 2004). Heat acclimation primarily induces extracellular hypervolaemia (plasma volume expansion), enhanced sweat rate and sensitivity and improved perceived tolerance to heat (Hargreaves, 2008; Magalhaes et al., 2010; Wendt et al., 2007). Subsequently, these adaptations reduce cardiovascular and thermoregulatory strain whilst exercising in the heat (Garrett, Goosens, Rehrer, Patterson, & Cotter, 2009; King, Costill, Fink, Hargreaves, & Fielding, 1985; Nielsen et al., 1993). For example, consecutive days of moderate exercise in $40^\circ C$ $T_{amb}$ results in plasma volume expansion (estimated from capillary method using haemoglobin and haematocrit) (Dill & Costill, 1974; Maughan, Leipers, & Greaves, 2001), lowered heart rate (HR) and rectal temperature ($T_{re}$) during exercise at the same workload, improved sweat function and redistribution, and promotes greater exercise time to exhaustion at the same exercise-heat exposure (EH) (Magalhaes et al., 2010; Nielsen et al., 1993).

Traditional heat acclimation protocols of $\geq 6$ exposures for at least 60 min of exercise at between 50% and 60% $\dot{V}O_{2max}$ in $\geq 30^\circ C$ $T_{amb}$ have been shown to expose the body to a substantial time and degree of heat stress that induces plasma volume expansion, reducing cardiovascular and thermoregulatory strain to exercise-heat stress (Wendt et al., 2007). However, due to lifestyle and occupation barriers, the majority of ultra-marathon competitors arrive at the race location 1 to 3 days prior to the commencement of competition in the heat. In addition, ultra-endurance runners’ typical training bouts consist of moderate intensity running lasting $\geq 2$ h. Therefore, traditional heat acclimation exercise protocols are not specific, nor practical, for ultra-endurance runners. Moreover, heat illnesses (e.g. heat exhaustion, exertional heat stroke) have been commonly reported when prolonged strenuous exercise is conducted within 24 to 48 h of initial heat exposure in the absence of heat acclimation or acclimatisation (American College of Sports Medicine et al., 2007). This suggests that recreational ultra-endurance runners are a high risk population for confirming incidence of heat illnesses. Interestingly, ultra-marathon runners have anecdotally reported improved thermal tolerance perception within two days into competition (e.g. 6–8 h EH; Al Andalus Ultra-trail; www.alandalus-ut.com). Heat acclimation responses of ultra-endurance runners preparing for multi-stage ultra-marathon competition in a hot ambient environment have not previously been investigated. Moreover, it is unknown whether increasing exercise-heat stress would induce accumulative heat acclimation responses that may be of benefit prior to and during such a physically demanding event.

With this in mind, the aim of the current study was to investigate the effects of an ultra-marathon specific heat acclimation protocol performed with increasing hot ambient conditions on indices of heat acclimation in a group of ultra-endurance runners preparing for hot desert-based competition.

**Methods**

**Participants**

Six non-heat acclimatised male ultra-endurance runners [mean age 27 years, $s = 8$; nude body mass (NBM) 81 kg, $s = 5$; height 1.83 m, $s = 0.05$; body fat 12%, $s = 3$; $\dot{V}O_{2max}$ 63 ml · kgBM$^{-1}$ · min$^{-1}$, $s = 5$] volunteered to participate in the study. All runners were healthy recreational club-level athletes with an average of 10 years competition experience, and weekly running training loads ranged from 60 to 140 km · week$^{-1}$. All participants gave written informed consent before the study, which received approval from the local ethics committee and conforms to the 2008 Helsinki declaration for research ethics.

**Preliminary measurements**

One week prior to starting the experimental procedure, participants were asked to report to the laboratory, where height (stadiometer, Bodycare Ltd, Warwickshire, UK) and NBM (STW-150KE, Hampe Electronics, Zhonghe, Taiwan) were measured. Body composition was determined by dual-emission x-ray absorptiometry (DEXA; Hologic QDR1500,
Bedford, USA). Maximal oxygen uptake ($VO_{2\text{max}}$) was established by means of a continuous incremental exercise test to volitional exhaustion on a motorised treadmill (h/p(cosmos Mercury 4.0, Nussdorf-Traunstein, Germany), as previously described (Costa et al., 2009). From the $VO_{2}$-work rate relationship, the treadmill speed at 60% $VO_{2\text{max}}$ was extrapolated and verified (10.2 km · h⁻¹, $s = 1.4$; and 1% gradient).

**Experimental trials**

During the 24 h period prior to and throughout the pre-acclimation (PA) and heat acclimation protocol, participants were required to refrain from their structured training programmes. To control dietary intake, each participant was asked to adhere to the same habitual dietary patterns along the experimental procedure which was monitored using a food and fluid log (2983 Kcal, $s = 135$; American Dietetics Association et al., 2009), and provided 58% carbohydrate, 25% fat, 17% protein and consume at least 35 ml · kgBM⁻¹ · day⁻¹ of water (2839 ml, $s = 170$) (Todorovic & Mickelwright, 2004). In addition, prior to each running exercise-bout, participants reported to the laboratory at the indicated time, and consumed a standard pre-exercise meal (526 Kcal; 118 g carbohydrate, 9 g protein, 2 g fat) 2 h prior to the onset of each running exercise-bout.

The PA protocol consisted of three running exercise-bouts (PA1, PA2 and PA3, respectively) at 60% $VO_{2\text{max}}$ for 2 h at 20°C $T_{\text{amb}}$ and 40% relative humidity (RH). This was followed by the heat acclimation protocol, consisting of six running EH at the same intensity and duration; three EH at 30°C $T_{\text{amb}}$ and 29% RH (30°C EH1, EH2 and EH3, respectively); followed by three EH at 35°C $T_{\text{amb}}$ and 27% RH (35°C EH1, EH2 and EH3, respectively). Three bouts of exercise-heat stress performed for 2 h provided similar total physiological strain as previously studies reporting significant improvements in indices of exercise-heat tolerance (Garrett, Rehre, & Patterson, 2011; Magalhaes et al., 2010; Nielsen et al., 1993; Nielsen, Strange, Christensen, Warberg, & Saltin, 1997). All running exercise-bouts were performed on a motorised treadmill in an environmental chamber (WIR Series, Design Environmental Ltd., Ebbw Vale, UK).

PA exercise-bouts and EH were separated by 48 h within each $T_{\text{amb}}$ and 72 h between $T_{\text{amb}}$. To avoid circadian variations, participants performed the 2 h exercise-bouts at the same time of day throughout the whole experimental procedure (Shido, Sugimoto, Tanabe, & Sakurada, 1999). In accordance with participants’ normal training habits, three participants performed the experimental procedure in the morning (start time 8:00 h), and three participants performed the experimental procedure in the afternoon (start time 16:00 h). To reduce any seasonal heat acclimatisation, the experimental procedure was conducted in January and February (winter-spring season; Garrett et al., 2009). The mean daily maximum temperatures during these months ranged from 2–7°C. In addition, to observe any changes in fitness level along the experimental design, a continuous incremental exercise test to volitional exhaustion on a motorised treadmill, as previously described, was performed two weeks following the completion of the experimental design ($n = 4$; $VO_{2\text{max}}$ 65 ml · kgBM⁻¹ · min⁻¹, $s = 5$).

**Measurements of heat acclimation**

Prior to each experimental exercise-bout, participants were requested to empty their bladder and bowels. Resting NBM measurement, blood and urine samples were collected prior to each exercise-bout. To monitor $T_{\text{re}}$ during exercise, a thermocouple was inserted 12 cm beyond the external anal sphincter (Grant REC soft insertion probe thermocouple; Grant 2020 Squirrel data logger, Shepreth, UK). To monitor HR during exercise, participants were fitted with a HR monitor (Polar Electro, Kempele, Finland). Urine measures of hydration (urine specific gravity, Atago Urical-Ne, NSG Precision Cells, New York, USA; urine osmolality, Osmocheck, Vitech Scientific, West Sussex, UK; urine colour) from a mid-flow urine sample collected into 30 ml universal tubes were used to determine hydration status (Armstrong et al., 1994). Whole blood samples were also collected by venepuncture without venestasis from an antecubital vein into a lithium heparin (4 ml, 1.5 IU · ml⁻¹ of heparin; Becton Dickinson, Oxford, UK) vacuutainer tube. Plasma volume changes were estimated from changes in haemoglobin and haematocrit content of whole blood samples by capillary method (Dill & Costill, 1974; Maughan et al., 2001) in triplicate using lithium heparin blood samples and a micro-haematocrit reader (Hawksley & Sons Limited, Lancing, UK), as previously described (Garrett et al., 2009; Garrett, Creasy, Rehre, Patterson, & Cotter, 2011; Nielsen et al., 1993; Watt, Febbraio, Garnham, & Hargreaves, 1999). Between 5 to 10 min after resting pre-exercise measurements and sample collection, participants initiated exercise on a motorised treadmill in the environmental chamber for 2 h at the previously determined treadmill speed that elicited 60% $VO_{2\text{max}}$, dressed in athletic shorts, socks and shoes.

$T_{\text{re}}$, HR, McGinnis 13-point thermal comfort rating (TCR; Hollies & Goldman, 1977) and rating of perceived exertion (RPE; Borg, 1982) were measured pre-exercise and monitored every 5 min.
during exercise. Water was provided ad libitum during exercise (overall $T_{\text{amb}}$ mean: 20°C 575 ml · h$^{-1}$, $s = 200$; 30°C 922 ml · h$^{-1}$, $s = 404$; 35°C 1233 ml · h$^{-1}$, $s = 699$), whilst urine output during exercise was also collected into a urine collection flask. In addition, exercise-induced NBM loss (pre to post-exercise NBM difference), total urine produced and total fluid intake during exercise were used in determining estimated fluid losses [estimated fluid losses = pre to post-exercise NBM difference (1 kg NBM equivalent to 1 L body water) + fluid intake during exercise – total urine output], as previously described (Maughan, Shirreffs, & Leiper, 2007; Shirreffs, 2000; Sunderland, Morris, & Nevill, 2008). After completion of the experimental running exercise bout, NBM measurements, blood and urine samples were collected, as previously described. Prior to leaving the laboratory after each exercise-bout, participants were also provided with a standard recovery drink (American Dietetic Association et al., 2009; Kerksick et al., 2008).

**Statistical analysis**

Data in text and tables are presented as mean value ± standard deviation ($s$). For clarity data in figures are presented as mean values ± standard error of the mean ($s_e$). In addition, open bars have been used in Figures 1–3 to identify the magnitude of change from exercise-bout one to three. A repeated measures analysis of variance (ANOVA; SPSS v.17.0.2) was used to investigate time and exercise-bout interactions. While, a one-way ANOVA (SPSS v.17.0.2) was used to investigate time and exercise-bout interactions, nor significant difference in overall mean, were observed for $T_{\text{amb}}$ during PA. In addition, when comparing $T_{\text{re}}$ between 20°C, 30°C and 35°C $T_{\text{amb}}$, a significantly greater overall mean $T_{\text{re}}$ was observed during 35°C EH1, compared with overall mean $T_{\text{re}}$ during 20°C PA1 ($P < 0.05$; Figure 1).

**Cardiovascular stability**

No significant differences in pre-exercise HR were observed between PA exercise-bouts and EH (20°C: 72 bpm, $s = 15$; 30°C: 73 bpm, $s = 17$; 35°C: 71 bpm, $s = 17$). A significant main effect of EH was observed for HR at 30°C and 35°C $T_{\text{amb}}$ ($F_{(2,10)} = 12.4, P = 0.002$; $F_{(2,10)} = 4.5, P = 0.04$, respectively; Figure 2). Overall mean HR during 30°C EH2 and EH3 were significantly lower (5 and 8 bpm, respectively) than 30°C EH1 ($P < 0.05$). Likewise, overall mean HR during 35°C EH2 and EH3 were significantly lower (5 and 7 bpm, respectively) than 35°C EH1 ($P < 0.05$). No significant time and exercise-bout interactions, nor significant difference in overall mean, were observed for HR during PA. Additionally, when comparing HR responses between 20°C, 30°C and 35°C $T_{\text{amb}}$, significantly greater overall mean HR was observed during 30°C EH1 and 35°C EH1, compared with overall mean HR during 20°C PA1 ($P < 0.05$; Figure 2).

**Thermal comfort rating**

No significant differences in pre-exercise TCR were observed between PA exercise-bouts and EH (20°C: 6.4, $s = 0.6$; 30°C: 7.2, $s = 0.5$; 35°C: 7.4, $s = 0.5$). A significant main effect of EH was observed for TCR at 35°C ($F_{(2,10)} = 4.6, P = 0.03$; Figure 3). Overall mean TCR score during 35°C EH3 was significantly lower ($-0.8$) than 35°C EH1 ($P < 0.05$). No significant time and exercise-bout interactions, nor significant difference in overall mean, were observed for TCR during PA and EH at 30°C $T_{\text{amb}}$. In addition, when comparing TCR responses between 20°C, 30°C and 35°C $T_{\text{amb}}$, significantly greater overall mean TCR was observed during 30°C EH1, compared with overall mean TCR during 20°C PA1. Similarly, significantly greater overall mean TCR was observed during 35°C EH1 and EH2, compared with overall mean TCR during 20°C PA1 and PA2, respectively ($P < 0.01$; Figure 3).

**Rating of perceived exertion**

No significant interactions or differences in overall mean RPE during exercise were observed between 35°C EH1 ($P < 0.05$; Figure 1).
PA1, PA2 and PA3 at 20°C \(T_{\text{amb}}\) (11, 10 and 10, respectively); and EH1, EH2 and EH3 at 30°C (11, 11, 10; respectively) \(T_{\text{amb}}\). In addition, no significant differences in overall mean RPE during exercise were observed between \(T_{\text{amb}}\).
No significant changes in resting pre-exercise plasma volume were observed subsequent to PA exercise-bouts. Whereas, compared with baseline (pre-exercise 20°C PA1) a significant increase in plasma volume ($F_{(2,15)} = 3.6, P = 0.05$) was evident pre-exercise at 30°C EH3 (7.9%, $s = 3.9$), 35°C
EH2 (9.6%, s = 5.2) and 35°C EH3 (7.0%, s = 5.8; P < 0.05 vs. pre-exercise 20°C PA1).

No significant differences in pre-exercise NBM were observed between PA exercise-bouts and EH at 30°C and 35°C T<sub>amb</sub>. Exercise-induced NBM loss was observed following all exercise-bouts (P < 0.01; Table I). A significantly greater exercise-induced NBM loss (F<sub>2,15</sub> = 2.9; P = 0.03) was observed between 20°C PA1 and PA3 (P < 0.05). Whilst, no significant differences in exercise-induced NBM loss were observed between EH at 30 and 35°C T<sub>amb</sub> (Table I). In addition, no significant differences in

Figure 3. Thermal comfort rating (TCR) during 2 h running exercise at 60% \( \dot{V}O_2\text{max} \) at (A) 20°C [pre-acclimation exercise-bouts (PA): PA1 ◆, PA2 ○, and PA3 ■, respectively], (B) 30°C and (C) 35°C T<sub>amb</sub> [exercise-heat exposures (EH): EH1 ◆, EH2 ○ and EH3 ■, respectively], on three consecutive occasions separated by 48 h between bouts and 72 h between ambient temperatures. Open box represents magnitude of change from exercise-bout one to three. Mean ± s (n = 6): "P < 0.05 vs. EH3; ""P < 0.01 vs. 20°C PA1; """"P < 0.01 vs. 20°C PA2.
respectively, on three consecutive occasions separated by 48 h between bouts and 72 h between ambient temperatures.

20 runners. While, exercise-heat stress at 35 °C and thermoregulatory strain in all ultra-endurance runners. The results from the current study suggest that two bouts of 2 h running exercise at 60% \( V_{\text{O}_2\text{max}} \) conducted in 30°C \( T_{\text{amb}} \) was sufficient to reduce cardiovascular and thermoregulatory strain in all ultra-endurance runners. While, exercise-heat stress at 35°C \( T_{\text{amb}} \) was required to reduce TCR in all ultra-endurance runners.

### Table I. Change in nude body mass (NBM), total fluid intake and estimated fluid losses induced by 2 h running exercise at 60% \( V_{\text{O}_2\text{max}} \) at 20°C [pre-acclimation (PA): PA1, PA2 and PA3, respectively], 30°C and 35°C \( T_{\text{amb}} \) [exercise-heat exposures (EH): EH1, EH2 and EH3, respectively], on three consecutive occasions separated by 48 h between bouts and 72 h between ambient temperatures

<table>
<thead>
<tr>
<th>( T_{\text{amb}} )</th>
<th>NBM change (%)</th>
<th>Total fluid intake (ml)</th>
<th>Estimated fluid losses (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( 20^\circ C )</td>
<td>-1.3 ± 0.8( ^{11} )</td>
<td>998 ± 412</td>
<td>2112 ± 457</td>
</tr>
<tr>
<td>( 30^\circ C )</td>
<td>-0.7 ± 0.3</td>
<td>1750 ± 912( ^{11} )</td>
<td>2323 ± 775( ^{11} )</td>
</tr>
<tr>
<td>( 35^\circ C )</td>
<td>-0.4 ± 1.2( ^{11} )</td>
<td>2504 ± 1391( ^{11} )</td>
<td>2835 ± 942( ^{11} )</td>
</tr>
</tbody>
</table>

Mean \( \pm s (n=6): \) \( ^{1} P < 0.05 \) and \( ^{11} P < 0.01 \) vs. EH3; \( ^{1} P < 0.05 \) and \( ^{11} P < 0.01 \) vs. 20°C \( T_{\text{amb}} \); \( ^{1} P < 0.05 \) and \( ^{11} P < 0.01 \) vs. 30°C \( T_{\text{amb}} \).

Overall mean exercise-induced NBM loss were observed between \( T_{\text{amb}} \) (Table I).

No significant differences in fluid intakes during running exercise were observed between PA exercise-bouts and EH at 30°C and 35°C \( T_{\text{amb}} \) (Table I). However, as \( T_{\text{amb}} \) increased (e.g. 20–35°C \( T_{\text{amb}} \)), significantly greater fluid intakes during exercise were observed \((P < 0.01; \) Table I). Significantly greater estimated fluid losses \((F_{2,15} = 4.6, P = 0.006)\) were observed during 30°C EH3 compared with 30°C EH1 and EH2 \((P < 0.01; \) Table I). Whereas, no significant differences in estimated fluid losses were observed between PA exercise-bouts and EH at 35°C \( T_{\text{amb}} \) (Table I). As \( T_{\text{amb}} \) increased, significantly greater overall mean estimated fluid losses during exercise were observed \((P < 0.01; \) Table I). In addition, no significant differences in urine measures of hydration were observed pre- and post-exercise between PA exercise-bouts and EH at 30°C and 35°C \( T_{\text{amb}} \); and also between \( T_{\text{amb}} \) (Overall mean: urine specific gravity 441 mOsmol · kg\(^{-1}\), \( s = 247; \) urine osmolality 1.013 g · ml\(^{-1}\), \( s = 0.008; \) urine colour 3, \( s = 1 \)).

### Discussion

The aim of the current study was to investigate the effects of ultra-marathon specific exercise load with increasing hot ambient temperatures on indices of heat acclimation in a group of ultra-endurance running preparing for competition. The results from the current study suggest that two bouts of 2 h running exercise at 60% \( V_{\text{O}_2\text{max}} \) conducted in 30°C \( T_{\text{amb}} \) was sufficient to reduce cardiovascular and thermoregulatory strain in all ultra-endurance runners. While, exercise-heat stress at 35°C \( T_{\text{amb}} \) was required to reduce TCR in all ultra-endurance runners.

In regard to practical relevance, the outcomes of the current study are especially important to recreational ultra-endurance runners, whom are increasing in numbers amongst sporting populations, along with greater organisations of ultra-endurance events and up-taking of active tourism in hot locations (e.g. active holidays). Evidence indicates that this population of athletes are at high risk of heat-related illnesses (American College of Sports Medicine et al., 2007), since barriers exists (lifestyle and occupational) in following traditional heat acclimatisation protocols (arriving at the competition destination at least a week in advance) (Wendt et al., 2007). Therefore, these findings have practical relevance recommending that two EH of at least 2 h of moderate exercise at \( \geq 30^\circ C \) \( T_{\text{amb}} \) are sufficient to initiate adaptations that improves tolerance to exercising in the heat, and potentially protecting ultra-endurance runners against heat illnesses.

The novel findings of the current study are in accordance with previous studies investigating the effects of longer heat acclimation protocols on exercise performance and exercise-heat tolerance (Armstrong & Maresh, 1991; Houmard et al., 1990; Nielsen et al., 1993, 1997). Nevertheless, an important feature of the current study is that participants performed three PA exercise-bouts in 20°C \( T_{\text{amb}} \), which produced no significant changes in measured variables. Whereas, subsequent EH at 30°C \( T_{\text{amb}} \) induced significant acclimation responses. It is unlikely that these responses are due to a training effect, since habitual training workloads of participants consist of daily running exercise bouts, ranging from 1 h high intensity interval session to 6 h mountain trail run sessions (McNicol, O’Brien, Paton, & Knez, 2009). In addition, no significant changes in \( V_{\text{O}_2\text{max}} \) were observed two weeks following the completion of the experimental...
design in four runners who re-performed the continuous incremental running exercise test to volitional exhaustion. Moreover, even in the presence of heat tolerance adaptations induced by 30°C $T_{amb}$ EH, a further novel finding in the current study was that heat acclimation responses occurred with subsequent increases in $T_{amb}$ (reduced TCR, cardiovascular and thermal strain at 35°C $T_{amb}$ EH). This observation has practical relevance indicating that ultra-endurance runners may have the in situ ability to thermally adapt to changes in exercise-heat stress (increases in $T_{amb}$) that may occur along consecutive days of multi-stage ultra-marathon competition.

It has previously been reported that the enhanced reductions in cardiovascular and thermoregulatory strain, and possibly sweating responses, seen with heat acclimation are primarily due to extracellular hypervolaemia (Fallmann, 1992; Hargreaves, 2008; Patterson et al., 2004; Wendt et al., 2007). It is speculated that exercise-heat stress induced extracellular hypervolaemia is possibly due to raised circulatory osmotic pressure associated with a vascular influx of plasma protein (e.g. albumin), drawing fluids into intravascular compartments and/or an up-regulation of fluid regulatory hormones (e.g. aldosterone, anti-diuretic hormone) increasing the renal resorption of water and sodium (Armstrong & Maresh, 1991; Patterson et al., 2004; Wendt et al., 2007). Subsequently, these mechanisms improve muscle perfusion, increase stroke volume (reducing HR) and improve thermoregulation through enhanced skin blood flow and increased sweating responses (Magalhaes et al., 2010; Patterson et al., 2004; Wendt et al., 2007). In combination, these adaptations may attenuate large perturbations in core temperature, reducing TCR during exercise in the heat (Armstrong & Maresh, 1991; Fallmann, 1992; Helyar, Green, Zappe, & Sutton, 1996; Nielsen et al., 1993).

With this in mind, a potential limitation in the current study was that we did not measure extracellular hypervolaemia directly (plasma volume expansion). However, changes in whole blood haemoglobin and haematocrit have previously been used to estimate plasma volume expansions (Dill & Costill, 1974; Maughan et al., 2001) and is reported to be a valid and reliable method of indirectly establishing plasma volume expansion during heat acclimation protocols (Garrett et al., 2009; Garrett, Creasy, et al., 2011; Nielsen et al., 1993). In the current study, resting pre-exercise plasma volume significantly increased in all participants after two exercise-bouts in 30°C $T_{amb}$ (+7.9%). Similar significant increases in plasma volume were observed in all participants after one exercise-bout at 35°C $T_{amb}$ (+9.6%). Whereas, exercising at 20°C $T_{amb}$ produced no significant change in resting plasma volume in all participants.

The changes in plasma volume observed are likely attributed to heat-stress, since dietary and fluid intakes were controlled during the experimental design; no substantial differences in urine measures of hydration and exercise-induced NBM loss were observed along the experimental procedure; and no differences in total fluid intake during exercise were observed within exercise-bouts at 30°C and 35°C $T_{amb}$ (Harrison, 1985; Senay, 1972). From a practical viewpoint, for the majority of ultra-endurance runners who commonly travel to the competition destination 1 to 3 days before the race start, it may be advantageous to induce the final EH 48 h prior to travel, since a 72 h resting lifestyle in a thermoneutral (indoor) to cold (outdoor) environment was sufficient to abolish the increases in plasma volume previously induced by EH. Interestingly, the 72 h decay in plasma volume (return to baseline) is reversed by further EH, albeit at higher ambient temperatures (35°C $T_{amb}$).

Estimated fluid losses were observed to be significantly greater after two exercise-bouts in 30°C $T_{amb}$ only, in all participants. It is speculated that the exercise-heat stress was substantial to enhance sweat responses, possibly through stimulation of neuroendocrine mechanisms (e.g. cholinergic and adrenergic control of cutaneous vasodilation and sweat gland activity) and increased plasma volume (Armstrong & Maresh, 1991; Magalhaes et al., 2010; Nielsen et al., 1993; Pandolf, 1998; Sato & Sato, 1983; Wendt et al., 2007; Yamazaki & Hamasaki, 2003). A reduced $T_{re}$ at the same exercise workload was observed after two EH at 30°C $T_{amb}$ (−0.20°C) in five participants. Similar responses were also seen after only one EH at 35°C $T_{amb}$ (−0.31°C) in all participants. While, a reduced HR at the same exercise workload was observed after one EH at 30°C (5 bpm) and 35°C (5 bpm) $T_{amb}$ in all participants. These results are in accordance with a previous study that reported −0.3°C $T_{re}$ and −13 bpm HR after a short-term heat acclimation protocol (Garrett et al., 2009). This suggests that reductions in cardiovascular and thermoregulatory strain were likely attributed to the heat-induced extracellular hypervolaemia indicated by increased plasma volume (which ranged from +2.5% to +11.5%); subsequently improving heat transfer through improved aqueous environment and stroke volume (Armstrong & Maresh, 1991; Patterson et al., 2004; Wendt et al., 2007). In addition, it has also been reported in previous literature that euhydration is a pre-requisite for optimal heat acclimation to occur (Armstrong & Maresh, 1991; Armstrong et al., 1994; Shirreffs, 2000). Possibly, the maintenance of euhydration during rest and exercising periods (as previously discussed) assisted with reducing cardiovascular and thermoregulatory strain observed in the current study.
Artificially induced extracellular hypervolaemia is consistently shown to lower HR at the same exercise-heat stress, but occasionally fails to improve thermoregulatory function and consistently fails to improve perceived heat tolerance (O’Sullivan, 2003; Sawka, Hubbard, Francesconi, & Horstman, 1983; Sawka, Convertino, Eichner, Schnieder, & Young, 2000). This suggests that other mechanisms, besides increases in plasma volume, are also responsible in improving exercise performance in the heat, possibly through neuroendocrine connections (Hargreaves, 2008; Wendt et al., 2007). The heat acclimation of other mechanisms that contribute to thermoregulation; for example: up-regulated cholinergic and adrenergic activity, and increased hypothalamic temperature set-point are both involved with thermal comfort tolerance and peripheral vasodilation (Hargreaves, 2008; Wendt et al., 2007). Reductions in TCR were only observed after two exercise-bouts at 35°C Tamb having previously performed three exercise-bouts at 30°C Tamb. These current findings are in accordance with the literature taking into account that improved thermal comfort are not linked with plasma volume changes, and is likely attributed to a combination of thermoregulatory and neuroendocrine mechanisms (Hargreaves, 2008; Magalhaes et al., 2010; Wendt et al., 2007). Due to the thermoregulatory-neuroendocrine link, thermal comfort has been shown to take longer for heat adaptation to occur (e.g. 7–14 exposure days), compared with cardiovascular and thermoregulatory measurements and sweating responses (Pandolf, 1998; Wendt et al., 2007).

In conclusion, two bouts of 2 h running exercise at 60% VO2max at 30°C Tamb were sufficient to induce significant heat acclimation responses including reduced cardiovascular and thermoregulatory strain at the same exercise-heat stress in all ultra-endurance runners. Heat acclimation responses also occurred with two bouts of 2 h running exercise at 60% VO2max at 35°C Tamb which also included reductions in TCR at the same exercise-heat stress.

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References


Heat acclimation in ultra-endurance runners


