Abstract

Purpose: To investigate the effects of short-term, high-intensity interval training (HIIT) heat acclimation (HA).

Methods: Male cyclists/triathletes were assigned into either a HA (*n*=13) or a comparative (COMP, *n* = 10) group. HA completed three cycling heat stress tests to exhaustion (60% W_{max}) (HST1: pre HA; HST2: post HA; HST3: 7d post HA). HA consisted of 30 min bouts of HIIT cycling (6 min at 50% W_{max} then 12x1 min 100% W_{max} bouts with 1 min rest between each) on 5 consecutive days. COMP completed HST1 and HST2 only. HST and HA trials were conducted in 35°C/50% rh. Cycling capacity, physiological, and perceptual data were recorded.

Results: Cycling capacity was impaired following HIIT HA (77.2±34.2 min vs. 56.2±24.4 min, p=0.03) and did not return to baseline following 7d of no HA (59.2±37.4 min). Capacity in HST1 and HST2 was similar in COMP (43.5±8.3 vs. 46.8±15.7 min, p=0.54). HIIT HA lowered resting rectal (37.0±0.3°C vs. 36.8±0.2°C, p=0.05) and body temperature (36.0±0.3°C vs. 35.8±0.3°C, p=0.03) in HST2 compared to HST1 and lowered mean skin temperature (35.4±0.5°C vs. 35.1±0.3°C, p=0.02) and perceived strain on day 5 compared to day 1 of HA. All other data were unaffected.

Conclusions: Cycling capacity was impaired in the heat following 5 days of consecutive HIIT HA despite some heat adaptation. Based upon our data, this approach is not recommended for athletes preparing to compete in the heat; however, it is possible that it may be beneficial if a state of overreaching is avoided.

Key words

High-intensity interval training; heat adaptation, acclimatization, over-reaching, hyperthermia

INTRODUCTION

Exercise performance in the heat is often impaired due to the greater physiological strain experienced [1-3] but heat acclimation/acclimatisation (HA) can reduce this impairment by inducing a number of beneficial physiological (e.g., reduction in cardiovascular strain, lower core body temperature, greater electrolyte reabsorption facilitated by increases in aldosterone) and perceptual adaptations (e.g. lower perceived effort and thermal comfort) [3,4]. The extent to which these adaptations to heat occur depends on the magnitude of the thermal impulse, which in turn depends on the intensity, duration, and frequency of heat exposure [3]. HA can reduce actual and perceived thermal strain and improve exercise performance [4] but only ~15% of athletes surveyed undertook HA prior to the 2015 IAAF World Athletic Championships in Beijing [5]. The athletes that undertook HA prior to the championships did so for 17 – 30 days [5] and while longer HA protocols are more effective (mean performance improvement: ~+22% for 7+ days of HA) [4], prolonged HA may be difficult for many athletes to fit into their schedule. Smaller but important mean improvements of ~7% have also been observed following short-term HA (STHA: <7 days) in both time trial [4,6] and time to exhaustion/exercise capacity [4,8] performance measures. Such protocols would be attractive to both athletes and coaches.

Low-intensity/high-volume and high-intensity/low-volume training both form part of an endurance athlete's training structure [9,10]. Traditional HA protocols have consisted of low-intensity/high volume exercise [4] and have attempted to induce a sufficient thermal impulse by increasing the frequency and/or duration of heat exposure [3,4]. Manipulation of the exercise intensity of HA protocols has received less attention, and the few studies that have investigated different HA intensities have reported equivocal adaptation and exercise performance data [11-14]. Seven days of low-intensity/moderate-volume (60 min, 50% $\dot{V}O_{2max}$) HA reduced oxygen consumption, heart rate and core temperature to a similar extent as a moderate-intensity/low-volume protocol (~35 min, 75% $\dot{V}O_{2max}$) suggesting that elevating the exercise intensity can reduce the duration required for heat adaptation [11]. The effect of these physiological adaptations on subsequent exercise performance was not investigated in that study but recent data suggest that high intensity STHA can improve explosive exercise performance in the heat [14] and either has no effect [14] or impairs [13] prolonged exercise performance. Unfortunately, physiological data were limited in each study and discrepancies exist in the protocols used making it difficult to ascertain whether the performance adaptations are specific to HA adaptations or to the exercise type used in the HA. Wingfield et al. [14] adopted a submaximal "high-intensity" STHA approach (30 min at 40 – 70% maximal power) and concluded that adaptations were exercise-specific, however, 20 km cycling performance was unaffected and only explosive activity (maximal cycling sprint power and jump height) was improved. Minimal physiological adaptations were observed and it is worth noting that the thermal strain experienced (peak tympanic temperature = ~38°C) may not have been sufficient for adaptation [15]. Schmit et al [13] prescribed 60 min of high-intensity HA based upon the participant's highest intensity training sessions and observed positive physiological adaptations to the heat but reported that they were offset by functional over-reaching-related maladaptation.

Short-duration, high-intensity HA is an attractive proposition for a time-short athlete; however, data regarding its efficacy are less attractive. Wingfield et al. [14] suggested that performance improvements may be training-specific but it is also possible that the lack of performance improvements are explained by the lack of physiological adaptation. It is also possible that although an increase in physiological strain is good for adaptation, too much additional strain may cause maladaptation as a result of over-reaching. The purpose of the present study was to investigate the efficacy of a high intensity interval STHA protocol in highly trained male endurance athletes in inducing beneficial physiological and perceptual adaptations and on improving exercise capacity in the heat.

METHODS

Participants

Twenty-three well-trained non heat-acclimated, adult male cyclists/triathletes participated in the study. Participants were randomly assigned to either a heat acclimation group (HA; n = 13) or a comparative group (COMP; n = 10). Participants had to meet the following eligibility criteria so most of the differences between group demographics were trivial in size (g<0.2) (Table 1). Participants were required to undertake regular cycling-specific training (>60 $km \cdot wk^{-1}$; >3+ $h \cdot wk^{-1}$) and have a peak oxygen uptake ($\dot{V}O_{2peak}$) greater than 55 $ml \cdot kg^{-1} \cdot min^{-1}$. Participants completed a health history questionnaire [16] and provided written, informed consent prior to testing. Participants were blinded to the purpose of the experiment. HA participants were told that we were assessing the HA protocol whereas the COMP group were told that we were assessing cycling capacity test variability. The study was conducted in accordance with Helsinki Declaration II. Ethical approval was granted by the University of Roehampton's ethical committee (LSC 14/102).

Experimental overview *(Figure 1)*

Participants in the HA group visited the laboratory on nine occasions whereas those in the COMP group attended on three (Figure 1). During the preliminary visit, anthropometric data were collected and participants completed an incremental cycle test to determine work max (W_{max}). 3 – 7d after the W_{max} test all participants completed the first heat stress test (HST1). HA participants then completed 5 consecutive days of HA starting 3 – 4 days after HST1 while COMP participants maintained their normal training. Both groups undertook the second HST (HST2) 9–11d after HST1- for the HA group this was 3–4d after the final HA session. HA participants completed a third HST (HST3) 7–9d after HST2. Between HST2 and HST3 participants undertook their normal training (see Table 1) and avoided exposure to high temperatures. Trials were completed during the autumn and winter months. Participants recorded their dietary and activity patterns for the 24h before HST1 and repeated this for the 24h prior to subsequent HSTs. Participants maintained their usual dietary and physical activity patterns at all other times during the experiment.

Preliminary laboratory visit

Body mass (Seca, model 813, Germany) and stature (Harpenden Stadiometer, Holtain Ltd, UK) were recorded before percentage fat was measured using the whole body air displacement plethysmography method (BodPod, Cosmed, Italy). Anthropometric data were collected by an ISAK qualified technician. Maximal power output and oxygen uptake were then measured simultaneously using a modified version of the protocol used by Kuipers et al. [17] performed in ambient conditions (22 ± 1°C, 55 ± 3% rh). Participants cycled for 5 min at 100W on a cycle ergometer (Monark 874E, Vansbro, Sweden) before undertaking a continuous incremental maximal cycle test during which workload was increased by 50W every 2.5 min until a heart rate (HR) of 160 $b \cdot min^{-1}$ was reached and then by 25W every 2.5 min until volitional exhaustion. Maximum workload was calculated using the equation of Kuipers et al. [17]. Breath by breath gas exchange was continuously measured using a calibrated on-line metabolic cart (Oxycon Pro, Jaeger, Germany). Maximal oxygen uptake was the highest value over any 10 s period using a rolling 5 breath average.

Heat Stress Tests (HST)

Each HST involved cycling at 60% W_{max} on a cycle ergometer (Monark 874E, Vansbro, Sweden) at a self-selected cadence until volitional fatigue in hot conditions (35°C and 50% rh). The HSTs allowed for comparative physiological, perceptual, and exercise capacity data to be collected before and after the intervention. Cycling capacity was defined as the time at which participants voluntarily terminated exercise or experimenters ended the trial because the participant was unable to maintain the required cadence (± 5 rpm). Participants were not provided with any indication of the duration cycled until the completion of all visits. A fan was placed ~ 1 m in front of the participants providing airflow of ~4.0 $m \cdot s^{-1}$. Participants completed all HSTs at the same time of day (± 60 min), and without verbal encouragement.

Prior to the HST, participants sat in the chamber for 10 min during which time a capillary blood sample was taken. Rectal temperature (T_r), skin temperature (T_{sk}), HR, thermal sensation (TS) and rating of perceived exertion (RPE) were recorded at 5 min intervals and upon termination of the test. Participants were not informed of the time-points at which perceptual data were recorded. A post-exercise capillary blood sample was collected upon termination while the participant remained seated on the ergometer. Participants drank chilled (~6 – 8°C), water *ad libitum* and the volume consumed was recorded. Post-exercise, dry nude body mass was recorded once the participant had left the environmental chamber to estimate sweat loss and sweat rate.

Heat acclimation

HA participants sat in the chamber for 10 min to establish baseline values before cycling (Monark 874E, Vansbro, Sweden) for 30 min in the heat (35°C; 50% rh) without a fan. Each HA bout started with a 6 min sub-maximal (50% W_{max}) warm-up followed by 12 x 1 min intervals at 100% W_{max} interspersed by 1 min bouts of unloaded spinning. Participants cycled at a self-selected cadence and were encouraged to complete as much work as possible during each 30 min bout. Power output was recorded during each sprint and T_r, T_{sk}, HR, TS and RPE were recorded at 5 min intervals. A capillary blood sample was taken immediately before and after each HA bout. Participants drank chilled water *ad libitum* and the volume consumed was recorded. Post-exercise nude body mass was recorded upon the completion of each HA session.

Measurements

Pre-trial set-up involved participants recording their nude body mass (Seca, model 813, Germany), self-inserting a rectal thermistor (REC-U-VL3-0, Grant Instruments (Cambridge) Ltd., UK) ~10 cm past the anal sphincter, attaching a HR belt (Polar Electro Oy, Kempele, Finland) and having four skin thermistors (EUS-U-VL-3, Grant Instruments (Cambridge) Ltd., UK.) attached. The four surface skin thermistors were attached to the participant's sternal notch, forearm, thigh and calf using a transparent dressing (Tagaderm, 3M Health Care, USA) and water-proof tape (Transpore, 3M Health Care, USA) for the calculation of weighted mean T_{sk} using the equation of Ramanathan [18]. Rectal temperature and mean T_{sk} were then used to estimate mean body temperature using the equation of Burton [19]. Thermistors were connected to a portable data logger (Squirrel 2020 Series, Grant Instruments (Cambridge) Ltd., UK). Ratings of perceived exertion (RPE) were recorded using a 6 –

20 scale (2) and thermal sensation (TS) was rated with an nine-point scale, with 4 as comfortable (neutral) and 8 as unbearably hot (29). Sweat loss and sweat rate were estimated using changes in nude body mass accounting for the volume of fluid consumed and urine excreted.

Capillary blood samples collected before and after each HST and HA session were immediately analyzed for hemoglobin (Hb) and hematocrit (Hct) using reflectance photometry (Insight Hb testing system, ACON laboratories, San Diego, USA). Plasma volume (PV) was estimated using the methods of Dill and Costill [20]. Additional capillary samples were collected before each HST and on days 1 and 5 of the heat acclimation bouts in microvette tubes containing clotting activator (Microvette CB300Z, Sartstedt, Leicester, UK) for serum separation. These samples were left at room temperature for 1 h and then centrifuged at 3860 rpm for 15 min at room temperature as per the manufacturer's guidelines. Serum was removed and stored in Eppendorf tubes at -80°C for analysis of serum aldosterone concentrations via enzyme-linked immunosorbent essay (Aldosterone Parameter Assay Kit, KGE016, R&D Systems Europe Ltd, Abingdon, UK). The laboratory-specific coefficient of variation of the assay was 10.6%.

Statistical Analyses

Data are presented as mean ± standard deviation or median [25 – 75 % quartiles]. Physiological and performance data from the HA group were compared using one-way factorial analysis of variance. HST and HA data were compared separately- HST data had three levels (HST1, HST2, HST3) whereas HA data had two levels (day 1 and day 5). Following a significant F value, post hoc analyses with Bonferroni adjustments for multiple comparisons were conducted. Perceptual data were compared using Friedman's ANOVA with Wilcoxon signed-rank tests run following a significant main effect. Secondary correlation analysis was run between changes in capacity, maximal oxygen uptake and W_{max} to see whether capacity changes could be explained by either marker of fitness/training status

Physiological and performance data from the COMP group were compared using paired samples t-tests. Perceptual data were compared using Wilcoxon signed-rank tests. The variability of the capacity test was quantified by calculating the co-efficient of variation (CV) between COMP group performance times (HST1 vs. HST2).

SPSS (Version 22; SPSS, Inc., Chicago, IL, USA) was used and the alpha level was set a priori at 0.05. For parametric data, Hedges' g effect sizes were calculated and interpreted using the following classifications; medium effect: 0.5 < 0.8, and large effect: > 0.8 [21] . For non-parametric, perceptual data, r effect sizes were calculated and interpreted using the following classifications: medium effect: 0.3 < 0.5, large effect: 0.5 < 0.7, and very large effect: 0.7 – 1.0 [21].

RESULTS

Heat acclimation data (Table 2)

11 participants completed all 60 sprints, 1 participant completed 59 sprints and 1 completed 57. Mean and total work did not differ between day 1 and 5 ($p = 0.74$, $g = -0.05$ and $p = 0.48$, $g = -0.11$, respectively). Mean T_{sk} ($p = 0.02$, $g = 0.64$), TS ($p = 0.01$, $r = 0.71$), and RPE ($p = 0.04$, $r = 0.56$) were lower on day 5 compared to day 1 as were peak TS ($p = 0.01$, $r = 0.71$) and RPE ($p = 0.049$, $r = 0.44$). All other variables were unaffected.

Heat Stress Test data: Cycling Capacity *(Figures 2 and 3)*

For the HA group (Figure 2), cycling capacity was greatest in HST1 (77.2 ± 34.2 min) compared to HST2 (56.2 ± 24.4 min; $p = 0.03$, $g = 0.68$). HST1 capacity was greater than HST3 but this difference was not statistically significant (59.2 ± 37.4 min; $p = 0.11$, $g = 0.50$). HST2 and HST3 capacity times were similar ($p > 0.99$; $g = 0.09$). Two participants improved in HST2 following HA (+14.8% and +20.8%) but the other 11 participants did worse following HA (range: -2.6% to -64.1%). The mean percentage change following HA (HST1 vs. HST2) was -22.0 ± 25.7% There was no correlation between the percentage change in endurance capacity (HST1 v HST2) following HA and $\dot{V}O_{2peak}$ ($r = 0.42$, $p = 0.15$) or W_{max} ($r = 0.32$, $p = 0.28$).

For the COMP group (Figure 3), exercise capacity in HST1 and HST2 were statistically similar (43.5 ± 8.3 vs. 46.8 ± 15.7 min; $p = 0.54$; $g = 0.25$). The coefficient of variation between HST1 and HST2 in the COMP group was 15.1 ± 16.0%.

Heat Stress Test data: Physiological and perceptual responses (Tables 3 and 4)

HA participants started HST2 with a lower resting T_r ($p = 0.05$, $g = 0.67$) and T_{body} ($p = 0.03$, $g = 0.68$), than HST1. SR was not statistically lower in HST3 than HST1 but a medium effect size reduction was observed ($p = 0.16$, $g = 0.52$). TS at volitional termination was higher in HST1 than HST2 ($p < 0.01$; $r = 0.70$) and HST3 ($p = 0.02$; $r <0.65$). In COMP, resting PV was not statistically higher in HST2 than HST1 ($p = 0.15$; $g = 0.86$) and final TS was not statistically lower in HST2 compared to HST1 ($p = 0.33$; $r = 0.31$). All other data were similar between trials for HA and COMP groups ($p >0.05$; $g <0.5$/$r <0.3$).

DISCUSSION

The main findings from the present study indicate that five consecutive days of high intensity HA results in small reductions in actual and perceived strain but impairs subsequent exercise capacity. The capacity decrements reported in the current study are in line with other high intensity STHA data [13]; however, this is the first high-intensity STHA investigation to observe lower core body temperatures following such HA.

We observed a small reduction in resting core temperature between HST1 and HST2 indicating partial adaptation (Table 3); however, we did not observe changes in HR, PV, aldosterone, or SR. These data are in line with some [13,14], but not all [12] high intensity STHA studies. Prolonged bouts of STHA can increase PV [6,22] but medium-term HA is more effective at doing so [23] suggesting that longer heat exposure might be required for PV adaptations. Longer HA regimens may also be needed for other fluid-related heat adaptations such as increases in SR and aldosterone [8,12,24]. HR adaptations often occur first [25] and so it is a little surprising that no such adaptations were observed; however, HR adaptations are often observed with PV adaptations and so the lack of hypervolemia may explain the lack of bradycardia [26,27]. To initiate adaptation, it has been proposed that the thermal impulse must exceed a critical threshold [3] and this may explain the small physiological adaptation seen in the current study. Although individual variation exists, time spent with core temperatures $\geq$38.5°C may be required for heat adaptation [15] and the highest mean body core temperature recorded in the present study was only 38.3 ± 0.4°C. Other high intensity STHA studies reported similar thermal impulses and also failed to achieve a sustained elevation in body core temperature $\geq$38.5°C resulting in minimal physiological adaptations [12-14]. In combination, these data suggest that short duration HIIT in the heat provides an insufficient thermal impulse for extensive physiological heat adaptation. Perceptual data showed positive adaptations with reduced mean RPE and peak TS at day 5 compared to day 1 of HA. Reduced perceived levels of effort and thermal comfort/sensation are consistently reported after successful HA and such improvements would be expected to improve volitional exercise [4,12]; however, this is not always the case [12-14]. The lower peak TS in HST2 than HST1 is likely to be due to the lower core temperature and exercise duration in HST2 rather than being an indication of any perceptual adaptation.

The capacity impairments observed in our study are in line with, but much greater than, Schmit and colleagues [13] who reported that high-intensity HA impaired 20 km time trial performance (-1.7%). The authors proposed that high-intensity HA may induce over-reaching and maladaptation in non-acclimated athletes and our greater impairments (-22%) may be due to a greater over-reaching from the higher exercise intensity used. In temperate conditions, performance decrements have been reported in cyclists who were in a state of functional-overreaching (F-OR) following a high training load, but these decrements were reversed following a period of tapering [28].

The combined stress of HIIT in the heat will result in a greater risk of cumulative fatigue and may explain the performance decrements seen in our findings. We did not control our participants' training loads during the 5 days of HA and so it is possible that overall training load was greater, although participants reported reducing their training due to the demands of the HA. Training load data would have provided a useful insight into the possible role of cumulative fatigue during HIIT heat acclimation. Schmit et al. [13] reported that high-intensity HA impaired 20 km time trial performance but that the impairment was reversed following a one-week taper during which time participants had their normal training load reduced by ~50%. In the present study, there was a small recovery in exercise capacity in HST3 compared to HST2 but this did not reach, let alone surpass, baseline levels. We did not control the training loads of our participants during the 5-7 days between HST2 and HST3 and therefore it is possible that the sustained reduction was observed as a result of a greater overall training load and cumulative fatigue.

PRACTICAL APPLICATIONS

Although short-duration, high-intensity HA protocols would be attractive to time-short athletes and coaches, we found this approach to have minimal effects on key physiological and perceptual markers of heat adaptation and to markedly reduce subsequent exercise capacity. We did not regulate non-HA training and so it is possible that in such instances, this approach results in cumulative fatigue and overreaching. Achieving an optimal and appropriate thermal strain via a more traditional HA approach has been consistently demonstrated and so further investigation into developing a HA protocol that includes a blend of exercise intensities with an appropriate period of tapering may be beneficial to the time sensitive athlete in preparation for competition.

CONCLUSION

Despite some evidence of thermoregulatory and perceptual adaptations following high-intensity STHA, exercise capacity in the heat is impaired in well-trained endurance cyclists. Cumulative fatigue and insufficient recovery from 5 consecutive days of HIIT HA in conjunction with normal training may explain this impairment. Based upon our data, high intensity STHA is not recommended for individuals preparing to compete/exercise in endurance events in the heat; however, it is possible that it may be beneficial if careful consideration is paid to managing the overall training load and a state of overreaching is avoided.

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REFERENCES

1. Guy JH, Deakin GB, Edwards AM, Miller CM, Pyne DB. Adaptation to hot environmental conditions: an exploration of the performance basis, procedures and future directions to optimise opportunities for elite athletes. *Sports Med.* Mar 2015;45(3):303-311.
2. Tatterson AJ, Hahn AG, Martin DT, Febbraio MA. Effects of heat stress on physiological responses and exercise performance in elite cyclists. *J Sci Med Sport.* Jun 2000;3(2):186-193.
3. Taylor NA. Human heat adaptation. *Compr Physiol.* Jan 2014;4(1):325-365.
4. 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. *Sports Med.* Nov 2016;46(11):1699-1724.
5. Periard JD, Racinais S, Timpka T, Dahlstrom O, Spreco A, Jacobsson J, Bargoria V, Halje K, Alonso JM. Strategies and factors associated with preparing for competing in the heat: a cohort study at the 2015 IAAF World Athletics Championships. *Br J Sports Med.* Feb 2017;51(4):264-270.
6. Garrett AT, Creasy R, Rehrer NJ, Patterson MJ, Cotter JD. Effectiveness of short-term heat acclimation for highly trained athletes. *Eur J Appl Physiol.* May 2012;112(5):1827-1837.
7. Garrett AT, Rehrer NJ, Patterson MJ. Induction and decay of short-term heat acclimation in moderately and highly trained athletes. *Sports Med.* Sep 1 2011;41(9):757-771.
8. Sunderland C, Morris JG, Nevill ME. A heat acclimation protocol for team sports. *Br J Sports Med.* May 2008;42(5):327-333.
9. Laursen PB. Training for intense exercise performance: high-intensity or high-volume training? *Scand J Med Sci Sports.* Oct 2010;20 Suppl 2:1-10.
10. Seiler S. What is best practice for training intensity and duration distribution in endurance athletes? *Int J Sports Physiol Perform.* Sep 2010;5(3):276-291.
11. Houmard JA, Costill DL, Davis JA, Mitchell JB, Pascoe DD, Robergs RA. The influence of exercise intensity on heat acclimation in trained subjects. *Med Sci Sports Exerc.* Oct 1990;22(5):615-620.
12. Kelly M, Gastin PB, Dwyer DB, Sostaric S, Snow RJ. Short Duration Heat Acclimation in Australian Football Players. *J Sports Sci Med.* Mar 2016;15(1):118-125.
13. Schmit C, Duffield R, Hausswirth C, Brisswalter J, Le Meur Y. Optimizing Heat Acclimation for Endurance Athletes: High- vs Low-Intensity Training. *Int J Sports Physiol Perform.* Sep 5 2017:1-24.

14. Wingfield GL, Gale R, Minett GM, Marino FE, Skein M. The effect of high versus low intensity heat acclimation on performance and neuromuscular responses. *J Therm Biol.* May 2016;58:50-59.
15. Gibson OR, Dennis A, Parfitt T, Taylor L, Watt PW, Maxwell NS. Extracellular Hsp72 concentration relates to a minimum endogenous criteria during acute exercise-heat exposure. *Cell Stress Chaperones.* May 2014;19(3):389-400.
16. American College of Sports Medicine Position Stand and American Heart Association. Recommendations for cardiovascular screening, staffing, and emergency policies at health/fitness facilities. *Med Sci Sports Exerc.* Jun 1998;30(6):1009-1018.
17. Kuipers H, Verstappen FT, Keizer HA, Geurten P, van Kranenburg G. Variability of aerobic performance in the laboratory and its physiologic correlates. *Int J Sports Med.* Aug 1985;6(4):197-201.
18. Ramanathan NL. A New Weighting System for Mean Surface Temperature of the Human Body. *J Appl Physiol.* May 1964;19:531-533.
19. Burton AC. Human Calorimetry: The average temperature of the tissues of the body. *J Nutr.* 1935;9.
20. Dill DB, Costill DL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol.* Aug 1974;37(2):247-248.
21. Cohen J. *Statistical power analysis for the behavioral sciences*. New Jersey: Lawrence Erlbaum; 1988.
22. Armstrong LE, Francesconi RP, Kraemer WJ, Leva N, De Luca JP, Hubbard RW. Plasma cortisol, renin, and aldosterone during an intense heat acclimation program. *Int J Sports Med.* Feb 1989;10(1):38-42.
23. Gibson OR, Turner G, Tuttle JA, Taylor L, Watt PW, Maxwell NS. Heat acclimation attenuates physiological strain and the HSP72, but not HSP90alpha, mRNA response to acute normobaric hypoxia. *J Appl Physiol (1985).* Oct 15 2015;119(8):889-899.
24. Garrett AT, Goosens NG, Rehrer NJ, Patterson MJ, Cotter JD. Induction and decay of short-term heat acclimation. *Eur J Appl Physiol.* Dec 2009;107(6):659-670.
25. Pandolf KB, Burse RL, Goldman RF. Role of physical fitness in heat acclimatisation, decay and reinduction. *Ergonomics.* Jul 1977;20(4):399-408.
26. Senay LC, Mitchell D, Wyndham CH. Acclimatization in a hot, humid environment: body fluid adjustments. *J Appl Physiol.* May 1976;40(5):786-796.
27. Sawka MN, Wenger CB, Pandolf KB. Thermoregulatory Responses to Acute Exercise-Heat Stress and Heat

Acclimation. *Comprehensive Physiology*: John Wiley & Sons, Inc.; 2010.

28. Aubry A, Hausswirth C, Louis J, Coutts AJ, Y LEM. Functional overreaching: the key to peak performance during the taper? *Med Sci Sports Exerc.* Sep 2014;46(9):1769-1777.