Journal of Science and Medicine in Sport 22 (2019) 117–122

Contents lists available at ScienceDirect

Journal of Science and Medicine in Sport

journal homepage: www.elsevier.com/locate/jsams

Original research

Intermittent exercise-heat exposures and intense physical activity sustain heat acclimation adaptations

J. Luke Pryor[a,b,*], Riana R. Pryor[a,b], Lesley W. Vandermark[b], Elizabeth L. Adams[b], Rachel M. VanScoy[b], Douglas J. Casa[b], Larry E. Armstrong[b], Elaine C. Lee[b], Lindsay J. DiStefano[b], Jeffrey M. Anderson[b,1], Carl M. Maresh[c]

[a] Central California Sports Sciences Institute, Department of Kinesiology, California State University, United States
[b] Korey Stringer Institute, Department of Kinesiology, University of Connecticut, United States
[c] Department of Human Sciences, The Ohio State University, United States

ARTICLE INFO

Article history:
Received 30 December 2017
Received in revised form 2 May 2018
Accepted 12 June 2018
Available online 19 June 2018

Keywords:
Thermoregulation
Exertional heat illness
Decay
Induction
Heat acclimatization

ABSTRACT

Objectives: To determine if intermittent exercise-heat exposures (IHE) every fifth day sustain heat acclimation (HA) adaptations 25 days after initial HA.
Design: Randomized control trial.
Methods: Sixteen non-heat acclimatized men heat acclimated during 10–11 days of exercise in the heat (40 °C, 40% RH). A heat stress test (120 min, 45% $\dot{V}O_{2peak}$) before (Pre HA) and after HA (Post HA) in similar hot conditions assessed HA status. Pair-matched participants were randomized into a control group (CON; n = 7) that exercised in a temperate environment (24 °C, 21%RH) or IHE group (n = 9) that exercised in a hot environment (40 °C, 40%RH) every fifth day for 25 days following HA (+25d) with out-of-laboratory exercise intensity and duration recorded. Both groups completed +25d in the hot condition.
Results: Both groups heat acclimated similarly ($p > 0.05$) evidenced by lower heart rate (HR), thermoregulatory, physiological, and perceptual responses (perceived exertion, fatigue, thermal sensation) Pre HA vs. Post HA ($p \leq 0.05$). At +25d, post-exercise HR ($p = 0.01$) and physiological strain index ($p < 0.05$) but neither T_{re} ($p = 0.18$) nor sweat rate ($p = 0.44$) were lower in IHE vs. CON. In IHE only, post-exercise T_{re} and perceptual responses at Post HA and +25d were lower than Pre HA ($p \leq 0.01$). +25d post-exercise epinephrine was higher in CON vs. IHE ($p = 0.04$). Exercise intensity during out-of-lab exercise and +25d post-exercise HR were correlated ($r = -0.89$, $p = 0.02$) in IHE.
Conclusions: Exercise-heat exposures every fifth day for 25 days and regular intense physical activity after HA sustained HR and T_{re} adaptations and reduced perceptual and physiological strain during exercise-heat stress ~1 month later.

1. Introduction

Heat acclimation (HA) is induced by repeated exercise-heat exposures that elicit temporary physiological adaptations that improve heat dissipation mechanisms, lessen thermal load, and reduce cardiovascular strain during exercise in hot and humid environments. These adaptations improve exercise-heat tolerance, enhance aerobic performance, and most critically, reduce the risk of exertional heat illness.[21] However, these HA-induced benefits are transient and decay within days to weeks.[1–9]

The thermoregulatory and cardiovascular adaptations derived from HA must be sustained for the continued health and safety of occupational and recreational athletes who periodically physically exert themselves in hot environments. For example, sustaining HA adaptations between deployments in hot climates when individuals live in cooler climates would be beneficial for military personnel.[2] Collegiate and professional athletes may live and train in cooler climates and artificially heat acclimate periodically to compete in hot environments,[10] such as the 2020 Tokyo Olympics and 2022 Qatar World Cup. Given the rapid decay of HA adaptations it has been suggested that high-level athletes should heat acclimate immediately preceding competition.[11] However, training and travel schedules can be limited by time and available resources not allowing all athletes to heat acclimate. Furthermore, HA during the taper period may interfere with the purpose of tapering, particu-

* Corresponding author.
E-mail address: lukepryor@csufresno.edu (J.L. Pryor).
[1] Deceased August 30, 2017.

https://doi.org/10.1016/j.jsams.2018.06.009
1440-2440/

larly in elite endurance athletes. Recently, it has been suggested that HA should occur several weeks prior to competition to avoid this conflict and allow for recovery after HA.[9,12] Exploring methods to sustain HA adaptations for ~2–4 weeks would then be extremely valuable given the rapid decay rate.[1–9]

A handful of studies show re-acclimation to heat requires fewer consecutive days of exercise-heat exposure than initial induction.[2,5,13] Daanen et al. reviewed all nine re-acclimation studies and observed in general, that the classical HA adaptations require 2–5 consecutive days of exercise-heat exposures within 2–4 weeks after initial HA to regain full adaptation.[9] Again, 2–5 days of consecutive exercise-heat exposure may be problematic for athletes immediately prior to competition.

In a review of literature, Taylor[14] postulated that one day of exercise-heat exposure for every five days without exposure preserves adaptations after initial HA, however this remains untested. The effectiveness of such a HA maintenance protocol via regular intermittent exercise-heat exposures is unknown. Therefore, this study aimed to investigate the efficacy of an intermittent exercise-heat exposure intervention 25 days after initial HA. We hypothesize that regular intermittent exercise-heat exposures would retain HA adaptations 25 days after initial HA. In addition to evaluating system level HA adaptations, epinephrine was assessed to provide insight into the overall physiological strain experience by the subjects and aid in determining intervention effectiveness. This knowledge could guide best practices to mitigate heat adaptation decay for sustained performance and protection against thermal injury.

2. Methods

We used a randomized control trial design to evaluate the effectiveness of intermittent exercise-heat exposures (every 5th day for 25 days after initial HA) on HA adaptation maintenance. Participants were pair-matched by self-reported physical activity, highest measured oxygen consumption ($\dot{V}O_{2peak}$), and body surface area to control factors known to affect thermoregulation and likely HA decay.[15] Matched participants were randomized into either an intermittent exercise-heat exposure (IHE) or no heat exposure (CON) group.

Sixteen recreationally active (physical activity 2–5 days $week^{-1}$; $\dot{V}O_{2peak} > 45$ mL kg^{-1} min^{-1}) non-heat acclimated college-aged males volunteered to participate (Table A.1). The study was completed from October to March in the northeastern United States where ambient conditions averaged 10.4 ± 2.2 °C and 70.5 ± 6.5% RH. No participant reported employment or frequent heavy exercise in hot environmental conditions for one month prior to the study. Participants were free of disease or injury limiting exercise, medication that may influence thermoregulation or fluid balance, and history of exertional heat illness within the past three years. Written informed consent was obtained prior to testing according to University institution review board policy for human participant testing (#H14-188).

During baseline testing height, body mass, body fat, $\dot{V}O_{2peak}$, and self-reported physical activity measurements were obtained. Height and body mass were used to calculate body surface area.[16] Percent body fat was estimated using a 3-site (chest, abdomen, thigh) skinfold technique using Lange calipers. Participants were familiarized with all perceptual scales: OMNI scale of perceived exertion,[17] thermal sensation,[18] and fatigue scale which ranged from 0 (no fatigue at all) to 10 (completely fatigued) in 1.0 increments. A treadmill ramping protocol (0.8–1.6 km h^{-1} every 2 min) while monitoring expired gases (TrueOne 2400, Parvo-Medics Inc., Provo, UT) was used to determine $\dot{V}O_{2peak}$. From these data, treadmill speed was calculated to elicit 45% $\dot{V}O_{2peak}$ during heat stress tests. $\dot{V}O_{2peak}$ was reassessed one day after Post HA and +25d.

Participants refrained from alcohol and unaccustomed, strenuous exercise for 24 h and caffeine for 8 h before all laboratory visits. Participants drank 500 mL of water the night before and 250 mL the morning of each laboratory visit to ensure euhydration, defined as urine specific gravity ≤1.020 (A300CL, Atago, Bellevue, WA). Pre- and post-exercise nude body mass was measured to calculate body mass loss and sweat rate while accounting for fluid intake (ad libitum) and urine output. A flexible rectal thermometer (model 401, Measurement Specialties, Beavercreek, OH) inserted 10–12 cm beyond the anal sphincter measured rectal temperature (T_{re}). A thermochron (DS1921G, Embedded Data Systems, Lawrenceburg, KY) was placed on the right chest, deltoid, thigh, and calf to determine whole body mean skin temperature (T_{sk}).[19] A chest mounted telemetry unit provided heart rate (HR) data (RaceTrainer™, Timex, Middlebury, CT).

All participants completed the same heat stress tests before (Pre HA) and after (Post HA) the HA protocol to assess acclimation status. Before exercise, participants sat in the hot chamber (40 °C, 40%RH) for 20 min allowing for stabilization of physiological variables and fluid compartment equilibration. Half way through the equilibration period, antecubital blood was collected into EDTA-containing tubes. Participants then performed treadmill exercise at 45% $\dot{V}O_{2peak}$ with a 2% grade for two 60 min bouts separated by 10 min of rest. During the first 5–10 min of exercise in Pre HA, expired gases were collected and analyzed to verify relative exercise intensity (45% $\dot{V}O_{2peak}$). If necessary, treadmill speed was adjusted to elicit 45% $\dot{V}O_{2peak}$ and remained consistent for all subsequent heat stress tests. Post-exercise measures were acquired immediately after exercise termination to enable comparisons across all trials regardless of duration. Post-exercise blood was drawn after ~10 min of seated rest in the heated chamber. The physiological strain index was calculated using baseline and immediate post-exercise T_{re} and HR.[20]

After Pre HA, all participants heat acclimated with 10 days of exercise (90–240 min; 40 °C, 40%RH) within a 11–12 day period (Table A.2). Briefly, the induction protocol included 4 days of interval exercise and 6 days of controlled hyperthermia.[21] Inclusion of various HA induction pathways allowed for additional research aims published elsewhere.[22]

After HA induction and Post HA, participants completed four additional heat stress tests in a hot (IHE; n = 9, 40 °C, 40% RH) or temperate (CON; n = 7, 24 °C, 21% RH) environment every fifth day (+5d, +10d, +15d, +20d). On day 25 (+25d), both groups performed the final heat stress test in the hot environment. Twenty-five days represents the time frame in which most HA associated adaptations are expected to decay.[9,21] To control diurnal variations in T_{re}, lab visits were scheduled within 6–7 h time blocks. To document the influence of physical activity on HA adaptation decay during the 25-day intervention,[13] participants were instructed to return to their normal training routine and record HR and duration during training using a chest mounted HR strap and watch provided to them by the researchers.

Percent plasma volume change was estimated by hematocrit and hemoglobin (HB 201+, Hemocue, Lake Forest, CA) shifts within each trial.[23] Epinephrine was determined by ELISA in duplicate (Rocky Mountain Diagnostics, Colorado Springs, CO) and plasma volume corrected.[24] Intra-assay CV was ≤19.3%. Epinephrine data were not normally distributed (Shaprio–Wilk, $p \leq 0.003$) and were natural log transformed prior to statistical analysis. All other data met parametric testing assumptions.

Separate group by time repeated measures ANOVAs with preplanned within (dependent) and between (independent) group

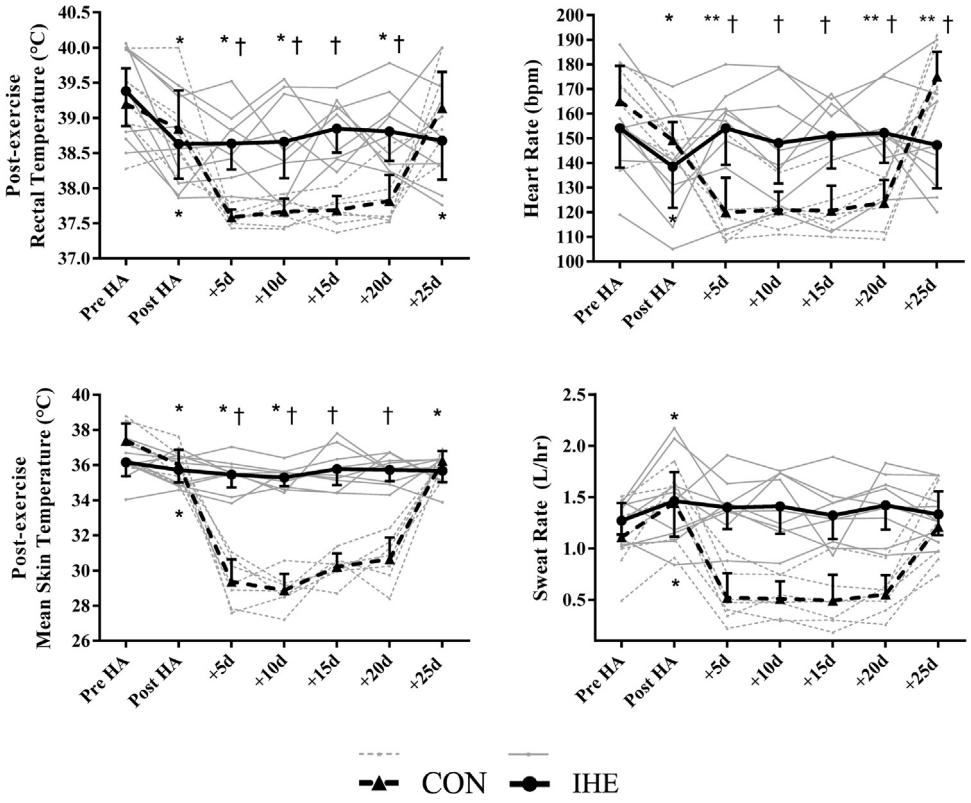

Fig. 1. Thermoregulatory and cardiovascular responses before and after HA and the 25-day intervention. During +5d, +10d, +15d, +20d CON exercised in a mild environment while IHE exercised in a hot environment. * $p < 0.05$ from Pre HA, ** $p = 0.001$ from Post HA, † $p \leq 0.01$ between groups. CON = no heat exposure group; IHE = intermittent exercise-heat exposure group. Group means with 95%CI are bolded black while individual data are light gray.

t-tests evaluated thermoregulatory, cardiovascular, and physiological responses to heat stress tests. Because exercise influences HA status,[13,15,25] Pearson's correlation was used to assess the relationship between out-of-lab physical activity and physiological responses at +25d. Data are reported as means or mean differences (CON–IHE) and 95% confidence intervals (95%CI). Effect size (ES) was calculated to determine the magnitude of difference between groups at +25d using Hedges' g equation. Small, medium, and large ES's were considered 0.2, 0.5, and 0.8, respectively. Percent gain or loss of HA adaptations at +25d were calculated.[13] Analyses were completed using SPSS version 21.0 (Armonk, NY, IBM Corp.) with $\alpha = 0.05$.

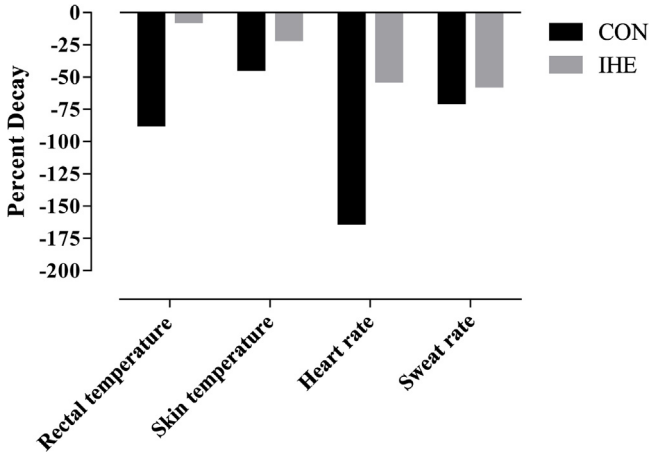

Fig. 2. Group comparison of adaptation decay 25 days after initial heat acclimation. Negative value denotes a loss of adaptation. CON = no heat exposure group; IHE = intermittent exercise-heat exposure group. Decay (%) calculated using an equation from Pandolf et al.[13]

3. Results

Groups were not different across baseline variables including pair-matching criteria (Table A.1). Across all subjects, the target T_{re} of 38.5 °C was achieved within 24–41 min and remained for 56.7 ± 16.8 min during each HA trial. HA was induced similarly between groups indicated by post-exercise T_{re}, T_{sk}, and HR, and increased sweat rate, resulting in a lower physiological strain index Pre HA vs. Post (Fig. 1 and Table A.3). Perceptual responses (perceived exertion, fatigue, thermal sensation) were lower after HA, with no between group differences (Table A.3). Epinephrine concentrations were lower Post vs. Pre HA in CON ($p = 0.05$) and trended so in IHE ($p = 0.06$; Fig. A.1).

After Post HA, subsequent heat stress tests were performed with 4.1 ± 0.8 days rest between lab visits. By design, post-exercise T_{re}, T_{sk}, and HR (Fig. 1) as well as physiological strain index, thermal sensation, sweat rate, body mass loss, and percent plasma volume change (Table A.3) were greater in IHE vs. CON at +5d, +10d, +15d, and +20d. Responses in both groups show high inter-individual variation.

IHE sustained select HA adaptations better than CON with post-exercise HR and T_{re} more preserved than T_{sk} and sweat rate adaptations (Fig. 2). At +25d in the same hot environment, post-exercise T_{re} was 0.47 °C ([−0.24,1.19], ES = 0.68) lower in IHE vs. CON, although this almost half degree difference did not reach statistical significance ($p = 0.18$). Because we were interested in changes throughout the 25-day intervention, we evaluated within group differences to determine if patterns diverged. Indeed, IHE experienced lower post-exercise T_{re} at Post HA, +5d, +10d, +20d,

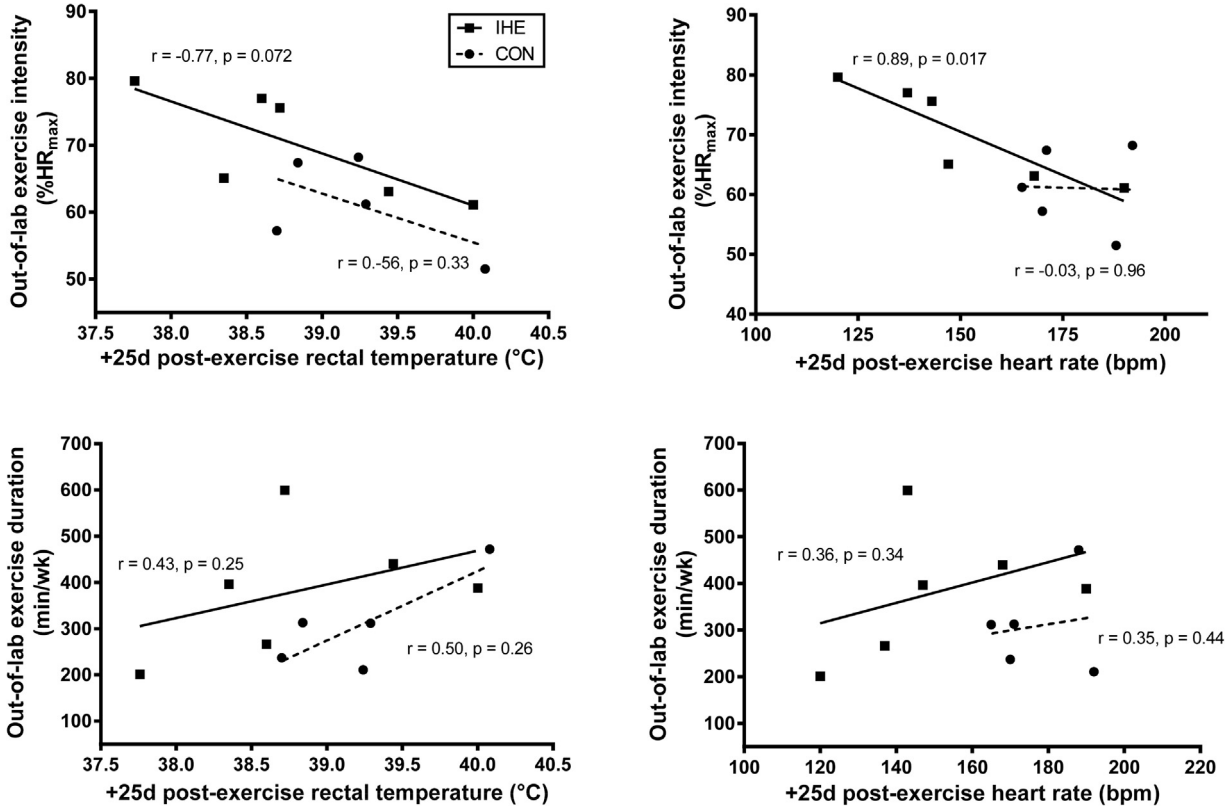

Fig. 3. Correlations among +25d post-exercise responses and out-of-lab physical activity recorded during the 25-day decay period. Squares with solid line indicate IHE (n = 6) and circles with dashed line indicate CON (n = 5).

and +25d compared to Pre HA. In contrast, CON post-exercise T_{re} was lower at Post HA, but not at +25d compared to Pre HA (Fig. 1). At +25d, post-exercise T_{sk} was not different between groups ($p = 0.11$) although a moderate-to-large effect size of 0.85 and mean difference of 0.65 °C [−0.17,1.47] were observed (Fig. 1).

Post-exercise HR was 28 bpm ([8,48], ES = 1.41, $p = 0.01$) lower in IHE compared to CON at +25d (Fig. 1). In CON, post-exercise HR was higher on +25d compared to Post HA ($p = 0.001$) but was similar to Pre HA ($p = 0.057$). At +25d, sweat rate was not different between groups ($p = 0.44$). +25d sweat rate was not different from Pre HA or Post HA in either group ($p \geq 0.15$). Post-exercise +25d physiological strain index was lower in IHE than CON (Table A.3), with IHE levels not different from Post HA, while CON returned to Pre HA levels. Corroborating this physiological strain in the CON group, +25d epinephrine levels were greater in CON vs. IHE ($p = 0.04$), with IHE concentrations not different from Post HA, while CON returned to Pre HA levels (Fig. A.1).

Post-exercise +25d perceived exertion, fatigue, and thermal sensation were not different between groups ($p \geq 0.14$). In IHE at +25d, these perceptual variables were not different to Post HA but remained lower than Pre HA, indicating decreased perceived strain (Table A.3).

Weekly physical activity duration (IHE: 245.6 min [18.4,471.6]; CON: 220.6 min [98.4,342.8], $p = 0.74$) and intensity (IHE: 61.9% HR_{max} [52.3,71.5]; CON: 68.3% HR_{max} [60.2,76.5], $p = 0.07$) recorded outside-of-laboratory testing during the 25-day intervention was highly varied and statistically not different between groups. Exercise intensity during out-of-lab exercise and +25d post-exercise HR were inversely correlated (Fig. 3) in IHE. Exercise intensity during out-of-lab exercise and +25d post exercise T_{re} had a strong but statistically non-significant inverse relationship (Fig. 3) in IHE. Despite these differences in out-of-lab exercise habits between groups, +25d $\dot{V}O_{2peak}$ (CON: 57.3 mL kg^{-1} min^{-1} [53.8,60.1]; IHE: 55.2 mL kg^{-1} min^{-1} [50.0,60.3]) remained unchanged from Pre (Table A.1) and Post HA (CON: 57.8 mL kg^{-1} min^{-1} [53.2,62.4]; IHE: 56.7 mL kg^{-1} min^{-1} [52.7,60.7]) values in both groups ($p > 0.23$). Because we recorded, but did not control, out-of-lab exercise, we assessed if out-of-lab exercise duration or intensity influenced +25d post-exercise HR responses between groups. Using these variables as separate or combined covariates, we found that +25d post-exercise HR was not different between groups ($p \geq 0.18$).

4. Discussion

After HA induction, IHE completed exercise-heat exposures every fifth day for 25 days, while CON (also heat acclimated) completed a similar protocol in a temperate environment. We observed a lower post-exercise HR, epinephrine, and physiological strain index for IHE vs. CON 25 days after HA. Further, only those exposed to intermittent exercise-heat exposures saw lower T_{re}, thermal sensation, thirst, and perception of effort 25 days after HA compared to the non-heat acclimated heat stress test (Pre HA). During the 25-day decay period, out-of-lab exercise intensity and +25d post-exercise HR were correlated in IHE only. The combination of exercise-heat exposure at least once every five days and intense out-of-lab physical training better sustained some HA adaptations that reduced thermal, physiological, cardiovascular, and perceptual strain during exercise-heat stress one month after induction.

We are the first to record (but not control) out-of-lab physical activity for 25 days after HA and explore the relationship of physical training to adaptation decay. Engaging in high intensity exercise out-of-lab appeared to aid in HR adaptation retention in IHE. This finding suggests regular exercise that sufficiently elevates body temperature initiating a strong sweat response and

skin blood flow may not only evoke partial HA,[13,15,25] but may also be important in sustaining adaptations. $\dot{V}O_{2peak}$ was not different between Post HA and +25d in either group implying it is exercise intensity (and frequency) that are related to adaptation retention and not fitness ($\dot{V}O_{2peak}$) per se.[15] A strength of this study was the inclusion of the CON group that did not receive exercise-heat exposures during the 25-day decay period. This allowed us to evaluate the importance of regular exercise-heat exposures in sustaining HA adaptations. The treadmill exercise (45% $\dot{V}O_{2peak}$) completed by CON in temperate conditions every 5th day after HA did not impart sufficient adaptive thermal stimuli as T_{re}, T_{sk}, and sweat rate did not exceed 38.2 °C, 31.0 °C, and 1.0 L h^{-1}, respectively. Albeit with a small sample size, frequent intense exercise along with intermittent exercise-heat exposures after HA may be a prudent practice to reduce adaptation decay. In two elite sailors, shorter higher intensity repeated exercise-heat exposures (40–60 min at 60% $\dot{V}O_2$max) along with regular sport training maintained HA adaptations for ~21 days,[26] corroborating our findings. Both low intensity/long duration and higher intensity/shorter duration exercise-heat exposures appear effective but with only two studies to draw conclusions addition research is required to clarify exercise-programing recommendations for adaptation retention after HA.

Although post-exercise T_{sk} and T_{re} were statistically similar between groups at +25d, the moderate-to-large effect sizes of 0.65–0.68 suggest key thermoregulatory differences existed affecting HR responses, a notion supported by the lower physiological strain index in IHE. Even when euhydrated, heat stress combined with exercise that raises T_{re} and T_{sk} creates competition for limited cardiac output between cutaneous and muscular vascular beds, elevating HR.[27] The higher epinephrine concentrations in CON also drove HR differences as epinephrine is released in response to high levels of exercise-heat stress.[28] Finally, HR variability studies attribute lower HR responses in HA individuals to increased parasympathetic activity,[29] but this was not measured in the current study. Hydration status appeared inconsequential concerning +25d HR responses as both groups were euhydrated before and dehydrated equally (according to body mass loss and percent plasma volume change) during the trial.

The +25d post-exercise T_{re} was not statistically different between groups but the mean difference of 0.47 °C may have practical importance given the moderate-to-large effect size. Previous studies modulating body temperature with either ice slurry ingestion or water perfused suits have shown that a T_{re} reduction of comparable magnitude (~0.50 °C) mitigated cardiovascular strain, perceived exertion, and improved time to exhaustion during exercise in heat.[30] Thus, a 0.47 °C between group and 0.71 °C within group (Pre HA vs. +25d in IHE) T_{re} reduction appears advantageous, especially in unacclimated hyperthermic individuals, and may limit hyperthermia-induced fatigue and exertional heat illness risk.

It is generally accepted that HA-induced adaptations are lost 3-4 weeks after HA without exercise-heat exposures.[6–9,13] With one exception,[2] the differences in decay rates among the earlier[6–8] and more recent HA decay studies[1,3,4,13] may be that HA adaptation decay was mitigated in studies[1,3,4,13] that periodically assessed physiological responses during the decay period via exercise-heat exposure. These intermittent exercise-heat exposures serve to elicit adaptive stimuli to effector organs (sweat glands, heart, cutaneous vascular beds) and in turn minimize decay. Periodically assessing physiological responses during HA decay was an unavoidable methodological consequence given the directives of previous studies but important in that they support our data and contention that intermittent exercise-heat exposure prolongs the beneficial adaptations of HA.

5. Conclusions

Intermittent exercise-heat exposures at least once every fifth day and regular intense physical activity after HA sustained some thermoregulatory, cardiovascular, and perceptual adaptations one month after induction. Periodic exercise-heat exposures afford logistical flexibility to sustain HA adaptations, contrasting re-acclimation models requiring several consecutive days of exercise-heat stress[2,5,13] or HA immediately prior to competition, which may place additional burden on already congested training and travel schedules. Intense physical activity appears to aid in HA adaptation retention. These novel data provide initial evidence to guide evidence based recommendations to mitigate HA adaptation decay.

Practical implications

- Sustaining heat acclimation adaptations is important for athletes who periodically compete in the heat or who heat acclimate several weeks prior to competition.
- Periodic exercise-heat exposure at least once every fifth day along with intense exercise in temperate conditions on other days sustain select thermoregulatory adaptations resulting in reduced physiological, cardiovascular, and perceptual strain during exercise-heat stress 25 days after initial heat acclimation.
- The mitigated decay of thermoregulatory adaptations and reduced cardiac strain implies performance could be enhanced for at least one month using this intervention.

Acknowledgments

This study was partially funded by research grants from a National Athletic Trainers' Association Research and Education Fund Doctoral Grant (#14DGP011), Eastern Athletic Trainers' Association Research Grant, and the Center for Health, Injury, and Prevention Graduate Researcher Grant (University of Connecticut).

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jsams.2018.06.009.

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