# Heat Reacclimation Using Exercise or Hot Water Immersion

NICOLA GERRETT, PUCK ALKEMADE, and HEIN DAANEN

*Faculty of Behavioural and Movement Sciences, Vrije Universiteit Amsterdam, Amsterdam, THE NETHERLANDS*

## ABSTRACT

GERRETT, N., P. ALKEMADE, and H. DAANEN. Heat Reacclimation Using Exercise or Hot Water Immersion. *Med. Sci. Sports Exerc.*, Vol. 53, No. 7, pp. 1517–1528, 2021. **Introduction:** The aim of this study was to compare the effectiveness of exercise versus hot water immersion heat reacclimation (HRA) protocols. **Methods:** Twenty-four participants completed a heat stress test (HST; 33°C, 65% RH), which involved cycling at a power output equivalent to 1.5 W·kg$^{-1}$ for 35 min whereby thermophysiological variables were measured. This was followed by a graded exercise test until exhaustion. HST1 was before a 10-d controlled hyperthermia (CH) heat acclimation (HA) protocol and HST2 immediately after. Participants completed HST3 after a 28-d decay period without heat exposure and were then separated into three groups to complete a 5-d HRA protocol: a control group (CH-CON, $n = 8$); a hot water immersion group (CH-HWI, $n = 8$), and a controlled hyperthermia group (CH-CH, $n = 8$). This was followed by HST4. **Results:** Compared with HST1, time to exhaustion and thermal comfort improved; resting rectal temperature ($T_{re}$), end of exercise $T_{re}$, and mean skin temperature ($T_{sk}$) were lower; and whole body sweat rate (WBSR) was greater in HST2 for all groups ($P < 0.05$). After a 28-d decay, only WBSR, time to exhaustion, and mean $T_{sk}$ returned to pre-HA values. Of these decayed variables, only WBSR was reinstated after HRA; the improvement was observed in both the CH-CH and the CH-HWI groups ($P < 0.05$). **Conclusion:** The data suggest that HRA protocol may not be necessary for cardiovascular and thermal adaptations within a 28-d decay period, as long as a 10-d CH-HA protocol has successfully induced these physiological adaptations. For sweat adaptations, a 5-d CH or HWI-HRA protocol can reinstate the lost adaptations. **Key Words:** CONTROLLED HYPERTHERMIA, DECAY, PERFORMANCE, ATHLETES, ADAPTATION

D espite the well-reported benefits of heat acclimation (HA) protocols for athletic performance in hot conditions (1,2), they are logistically and practically difficult to use alongside other preparation strategies before competition. The thermoregulatory benefits of HA can be obtained after approximately 10–14 consecutive days of training in the heat (2), but for many athletes, it is not always feasible to incorporate this prolonged and challenging exposure into their training schedule. In recent years, several studies have investigated alternative strategies to address these issues: fewer days (3), twice daily sessions to reduce the total number of days (4), and lowering the training load and implementing a post-workout hot water

immersion (HWI) protocol (5). The physiological adaptations that occur from HA, such as a reduced resting core temperature, a lower heart rate, and an expanded plasma volume will begin to decay once the stimulus has been removed and the rate of decay varies for each of the phenotypic markers of HA (1). It is thus beneficial for athletes to use their HA protocol as close to competition as possible to take full advantage of the adaptations acquired. The dilemma for athletes and coaches is how to implement a HA protocol alongside a tapering plan, which usually requires athletes to reduce their training volume 6–21 d before competition (6).

A strategy that has not been exploited is that of HA memory obtained during both the HA and the decay period (7). During HA, physiological adaptations occur as a result of altered gene expression and translational processes (8). During the decay period, the physiological phenotypes dwindle away to a preacclimation state, but at the molecular level, they remain in an altered state, referred to as dormant memory (7). This dormant memory is a key process for heat reacclimation (HRA), allowing a faster accrual of the adaptations during HRA in rats (7), and a similar phenomenon has been reported in humans (9,10). There is even some evidence to suggest a supercompensation, with lower resting/exercising heart rate after HRA compared with the initial HA (11,12). For 26–30 d away from the heat, just 4 d of HRA is reportedly required to accrue the lost physiological adaptations (9). Ashley et al. (13) suggested that a reacclimation period of 4 d is

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recommended after 2 wk absence from the heat and 5 d for 4 wk absence in the heat. Variations exist in the recommended time required for HRA, but they consistently point to a shorter HRA duration compared with the initial HA.

HRA strategies that are effective and practical and that minimize the interference with any precompetition preparation and/or tapering are required. Passive heating has been shown to be a practical strategy stimulating health benefits, to improve heat tolerance, to enhance exercise performance, and to induce hyperthermia (14–16). Although evidence suggests that HA adaptations are more complete when the program includes both exercise and heat exposure (2), there is a possibility that if the HA phenotypes have been previously acquired during an effective HA strategy, then an HRA protocol with heat alone (passive) may still be effective. This seems plausible given that HWI and exercise both stimulate heat shock protein synthesis, which provides cellular protection against exposure to high temperatures and improves thermal tolerance (16). It is possible then that the thermal stimulus alone from passive heating could be as effective, yet more practical, than an exercising controlled hyperthermia (CH) strategy for HRA.

The aim of this study was to assess the effectiveness of HRA on thermophysiological responses and performance during exercise in the heat. We hypothesized that a 5-d HRA, after a 28-d decay period, would result in a similar or even higher (i.e., supercompensation) adaptation response as that of HA. An additional aim was to determine whether HRA, using a practical HWI protocol, can be used instead of a CH protocol. We hypothesized that HWI would bring about the same physiological adaptations as a CH-HRA protocol.

## METHODS

### Participants

A power calculation was performed using G*Power software (Heinrich-Heine-Universität Düsseldorf, Germany). A partial eta-squared (large effect) of 0.14 for resting $T_{re}$ was determined using previously reported changes after CH in trained groups (9,17). This value, along with an $\alpha$ of 0.05, a $\beta$ of 0.80, a correlation among repeated measures set to 0.6, and a nonsphericity correction $\varepsilon$ set at 0.34 (1/[repetitions − 1]) indicated that a minimum total sample size of 21 (7 per group) was required to demonstrate a significant difference. We therefore recruited 24 unacclimated participants who were separated into three groups: control group (CH-CON), active (CH) HRA group (CH-CH), and passive (HWI) HRA group (CH-HWI). Participants between the three groups were matched for age, body surface area, and $\dot{V}O_{2peak}$ and had equal number of male ($n = 5$) and females ($n = 3$) per group (Table 1). According to previous classification guidelines (18,19), most participants were classified in performance levels 2 or 3 but few in levels 1 or 4. Because of this variation, we refer to them collectively as habitually trained, representative of their regular level of engagement with exercise training.

Participants were informed about the study purpose and procedures before providing verbal and written consent. The

TABLE 1. Participant characteristics of the three experimental groups.

| | CH-CON (n = 8, 5M, 3F) | CH-CH (n = 8, 5M, 3F) | CH-HWI (n = 8, 5M, 3F) |
|---|---|---|---|
| Age (yr) | 30 ± 8 | 34 ± 8 | 30 ± 8 |
| Height (cm) | 182.5 ± 10 | 181.2 ± 6.2 | 183.6 ± 9.6 |
| Weight (kg) | 74.8 ± 11 | 77.0 ± 7.2 | 75.2 ± 13.9 |
| BSA (m²) | 1.96 ± 0.2 | 1.97 ± 0.1 | 1.97 ± 0.2 |
| $\dot{V}O_{2peak}$ (mL·kg⁻¹·min⁻¹) | 52.8 ± 8.9 | 49.8 ± 7.8 | 53.0 ± 10.4 |

Data are presented as mean ± SD. No differences were observed between any of the variables listed ($P > 0.05$).
M, male; F, female; BSA, body surface area; CH-CH, HRA with controlled hyperthermia; CH-HWI, HRA with hot water immersion; CH-CON, control group.

Faculty of Behavioural Movement Science Ethical Committee at Vrije Universiteit Amsterdam approved the study (report no. VCWE-2018-160R1), which conforms to the standards set out by the Declaration of Helsinki. Participants were screened for preexisting medical conditions and specifically had no history of heat-related illnesses and cardiovascular complications and were nonsmokers. Three participants were taking medication: one participant was taking medication (70 mg alendronic acid weekly and 500 mg calcichew 7.5 mg and mirtazapine daily), one participant was taking Ritalin (ADHD), and one participant was taking methotrexate and folic acid. These participants were taking the medication consistently for the duration of the study. Female menstrual cycle was recorded but not controlled for as this would have been unfeasible given the required timings of each part of the protocol (10-d HA, 28-d decay, and 5-d HRA consecutively). Six females used the combined pill: one used a hormonal intrauterine device and two reported regular natural menstrual cycles (25–35 d).

### Experimental Design

After explaining and familiarizing participants with the experimental procedures and laboratory area, participants completed a graded exercise test to determine $\dot{V}O_{2peak}$. They were then familiarized with the heat stress test (HST). Approximately 4 to 7 d after the preliminary visit, participants completed the main experimental trials, which are illustrated in Figure 1. Participants completed the first HST (HST1), which was followed by a 10-d (consecutive) CH. Forty-eight hours after the final HA session, participants repeated the HST (HST2). All participants then completed a 28-d decay period where they were allowed to engage in their typical training regime but were not allowed to be exposed to any hot thermal stimulus (saunas, hot tubs, etc.). Although physical activity data were not recorded during the decay period, participants did provide us with a typical training week. Training mode varied but were predominately aerobic activities (running, cycling, swimming, and rowing) and some (but not all) engaging in strength training once a week. Participants averaged $5.5 \pm 2$ d of training per week, averaging $181 \pm 65$ min of training per week.

Twenty-eight days after completing HST2, they then completed another HST (HST3) to assess the level of decay. The following day, participants began their assigned 5-d (consecutive) HRA protocol: CH-CON, an active HRA group (CH-CH), or a passive HRA group (CH-HWI). Forty-eight hours after
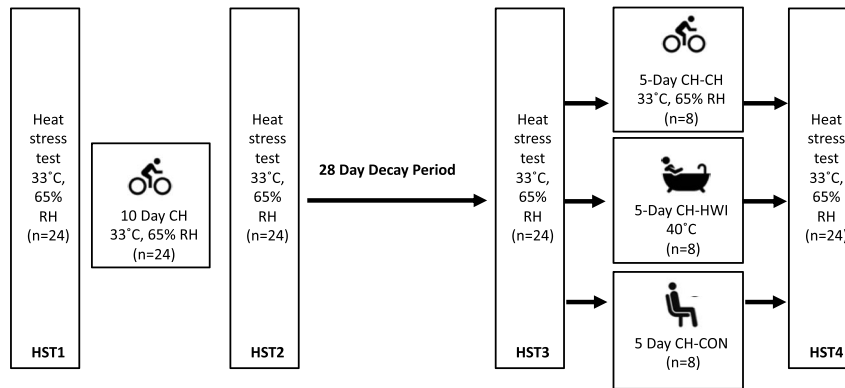
FIGURE 1—A schematic diagram illustrating the experimental protocol to examine the effects of three different HRA protocols on the adaptive responses to exercise in the heat. CH-CH, HRA with controlled hyperthermia; CH-HWI, HRA with hot water immersion; CH-CON, control group.

completing the final HRA protocol, participants completed the final HST (HST4).

The CH-CON were permitted to train during the allocated 5-d HRA period, but they were not allowed any exposure to a sustained hot thermal stimulus (saunas, hot tubs, etc.). Testing took place between the months of January and May in the Netherlands; the average outdoor temperature and relative humidity during this period was approximately 8°C and 78%, respectively.

## Experimental Trials

$\dot{V}O_{2peak}$, **anthropometric, and familiarization.** Participants' height (Seca217, Hamburg, Germany) and weight (SATEX 34 SA-1 250, Weegtechniek; Holland B.V., Zeewolde, The Netherlands) were measured initially. All participants then completed a graded exercise test to determine $\dot{V}O_{2peak}$ on an electrically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands) in temperate conditions (22°C, 32% RH). Participants started cycling at 25 W, with the intensity increasing 25 $W \cdot min^{-1}$ until volitional exhaustion. Heart rate (Polar Vantage-M, Kempele, Finland) and respiratory gases (Quark CPET; Cosmed, Rome, Italy) were continuously monitored throughout. $\dot{V}O_{2peak}$ was identified as the highest 15-s moving average over the entire exercise period. After a short break, participants moved to the environmental chamber to be familiarized with the experimental trials (i.e., the HST) in the heat. During this familiarization session, they were also familiarized with the perceptual scores: thermal sensation, thermal comfort, and RPE.

## HST

Testing took place throughout the day, but each participant completed their own respective four HST at the same time of day. Participants were asked to refrain from consuming caffeine or alcohol and to avoid any strenuous exercise 24 h preceding all HST. In addition, they were instructed to record their food and beverage intake during the preceding 24 h and asked to replicate this for all tests. They were required to ensure they were euhydrated by consuming 500 mL of water

the evening before testing and water equal to 10 $mL \cdot kg^{-1}$ of body weight (BW) 0–3 h before all HST. Hydration status, as indicated by a USG value ≤1.025 (20), was measured using a handheld refractometer (Atago Co. Ltd., Tokyo, Japan). Despite following the pretesting hydration guidelines, six participants had USG values slightly above 1.025. After confirming that they had followed the preexperiment hydration requirements, they were allowed to resume with the experiments after consuming a set volume of water (5 $mL \cdot kg^{-1}$ BW).

The HST was conducted in an environmental chamber (b-Cat b.v., Tiel, The Netherlands) set at 33°C, 65% RH, minimal air movement. Upon entering, they sat for 10 min to obtain a baseline sample of all physiological and perceptual data. They then mounted an electrically braked cycle ergometer (Lode Excalibur) and completed 35 min cycling at an external power output equivalent to 1.5 $W \cdot kg^{-1}$ BW. This was followed by a 5-min rest period, where participants could move off the bike, stretch, or sit but were required to consume a set volume of water (3 $mL \cdot kg^{-1}$ BW). They returned to the bike to complete a graded exercise test to exhaustion (GXT) as a performance measure, whereby the load was increased by 25 $W \cdot min^{-1}$, starting from 1.5 $W \cdot kg^{-1}$ BW until volitional exhaustion. No feedback or encouragement was provided. To assess the physiological responses to exercise in the heat, heart rate, rectal temperature ($T_{re}$), and mean skin temperature ($T_{sk}$) were continuously monitored, and perceptual scores (thermal sensation, thermal comfort, and RPE) were recorded at 5-min intervals and at the end of the GXT.

## Controlled Hyperthermia

Participants completed 10 HA sessions at approximately the same time of day (±3 h) in the same conditions as the HST (33°C, 65% RH). A CH-HA protocol was followed where the aim was to increase $T_{re}$ to 38.5°C (referred to as "thermal drive") within approximately 35 min and then hold it slightly above 38.5°C for 1 h (referred to as "thermal maintenance"). Thermal maintenance was regulated by adjusting the external power output or where necessary resting inside the chamber. Participants were free to drink water *ad libitum* during each

HA session, and the volume consumed was recorded. For the CH-CH group, this protocol was followed for another 5 d during the HRA. Before each session, a urine sample was collected to monitor hydration status during the 10-d HA. Nude BW was measured before and after each HA session, alongside fluid volume to calculate whole body sweat rate (WBSR). Heart rate, $T_{re}$, and mean $T_{sk}$ were continuously monitored, and perceptual scores (thermal sensation, thermal comfort, and RPE) were recorded at 5-min intervals.

## HWI

Passive heating, using hot water (40°C) immersion for 40 min, was selected based on its high practical value; baths are accessible to most, and the protocol will have minimal effect on training schedules. The water temperature of 40°C is below the thermal pain threshold, and pilot testing confirmed that the proposed protocol was challenging but tolerable. This seemed a good balance between providing a strong enough stimulus but still achievable for all to complete.

Participants sat in a neutral room for 5–10 min while a 5-min baseline value of physiological and perceptual responses was collected. They then entered the bath (Lay-Z-Spa BW54113 Monaco 2018 Model, Shanghai, China), which was set at 40°C, for 40 min. Water temperature was recorded using a PT100 sensor (GMH3750-SET1; Greisinger electronic GmbH, Germany) every 5 min. Participants were required to keep their shoulders under the water; however, this protocol is challenging and the risk of syncope is high. Participants were allowed "relief" breaks for 2 min, every 10 min, whereby they could sit on a stool, with the lower body still submerged to provide some relief from the oppressive bath temperature. If $T_{re}$ rose >39°C, participants were required to sit on a stool with the water to their waist. While immersed, participants were free to drink water *ad libitum*, and the volume consumed was recorded. During immersion, $T_{re}$ and heart rate were monitored continuously, and thermal sensation and thermal comfort were reported every 5 min. Immersion ended after 40 min unless the participants withdrew themselves or $T_{re}$ exceeded 39.5°C. After immersion, participants lay supine for a minimum of 10 min and sat upright for a minimum of 5 min to ensure $T_{re}$ began to decline and blood pressure appeared well regulated.

## Measurement and Calculations

For all sessions (HA, HST, and HRA), $T_{re}$ was measured as an indicator of core body temperature. Before all experimental trials, participants self-inserted a rectal thermometer (MSR, Seuzach, Switzerland, or Yellow Springs Instruments, Yellow Springs, OH) at least 10 cm past the anal sphincter. During all experimental trials, except the immersion trials, $T_{sk}$ was measured using iButtons (DS1922; Maxim Integrated Products, San Jose, CA) attached to the skin using tape (Fixomull Stretch ADH; BSN Medical GmbH, Hamburg, Germany) from four sites (chest, forearm, thigh, and calf), and a weighted mean $T_{sk}$ was calculated (21). Heart rate was continuously monitored in all trials using a heart rate monitor (Polar Vantage-M). Thermal

sensation was rated using an adapted scale with intermediary values ranging from +10 (extremely hot) to −10 (extremely cold), with 0 indicating thermal neutrality (22). Thermal comfort was rated on an adapted 6-point Likert scale with intermediary values as follows: 0 = comfortable, 2 = slightly uncomfortable, 4 = uncomfortable, and 6 = very uncomfortable (23). RPE was recorded using the 6- to 20-point Borg scale (24). Thermal sensation, thermal comfort, and RPE were assessed in all trials at 5-min intervals except the GXT, where they were recalled at the end of the test shortly after exercise termination. Before and after all sessions, nude BW was measured using platform scales (SATEX 34 SA-1 250, Weegtechniek, Holland B.V.), and WBSR calculated after correcting for duration and water volume consumption. All physiological data were averaged over 5 min. Exercise performance from the GXT was expressed in absolute terms, indicated as the total exercising time, i.e., time to exhaustion (TTE, in seconds). For further assessment of exercise performance, the data were also expressed as a percentage change, relative to performance from HST1.

To compare the two HRA protocols, the thermal impulse per session was quantified from $T_{re}$ data, using the following equation (25):

$$\text{thermal impulse} = \int (T_{\text{re-}i} - T_{\text{re-0}}) \, dt_1 \ [°C \cdot \text{min}^{-1}]$$

where $T_{\text{re-}i}$ is the rectal temperature at time $i$ (°C), $T_{\text{re-0}}$ is the initial (time 0) rectal temperature, and $dt_n$ is the duration of each stimulus (min).

## Data Analysis

All data were synced and formatted using MATLAB (R2019a; The MathWorks Inc., Natick, MA), figures were produced in GraphPad Prism (version 7; GraphPad Software, La Jolla, CA), and data were analyzed using the Statistical Package for the Social Sciences version 25 (IBM SPSS Statistics 20, Armonk, NY). Descriptive data are reported as mean ± SD. Significance was set at $P < 0.05$. The Shapiro–Wilk test was used to check if the data were normally distributed. To examine performance and physiological responses to the HST, we used a mixed ANOVA, with time (HST1–4) as within-subject factors and HRA protocols (CH-CH, CH-HWI, and CH-CON) as between-subject factors. To estimate the magnitude of the effects, partial eta-square ($\eta^2$) for ANOVA effects was calculated, with $\eta^2 > 0.06$ representing a moderate effect and $\eta^2 > 0.14$ a large effect. Specific *post hoc* analyses were conducted to answer specific questions related to our hypotheses, with the alpha adjusted accordingly. To assess whether the CH-HA was successful, comparisons were made between HST1 and HST2. To confirm whether decay had occurred, HST2 and HST3 were compared. To confirm whether decay was complete, HST3 and HST1 were compared. To assess whether HRA was successful, HST3 and HST4 were compared, and finally to determine whether a supercompensation had occurred, HST2 and HST4 were compared. The effect size of each pairwise comparison was calculated and reported as Hedges' *g* with 90% confidence intervals (CI); <0.19 is

TABLE 2. Daily physiological response to the initial 10-d controlled hyperthermia (CH) HA protocol completed by all three groups: control group (CH-CON, $n = 8$), hot water bathing group (CH-HWI, $n = 8$), and controlled hyperthermia group (CH-CH, $n = 8$).

| | | HA1 | HA2 | HA3 | HA4 | HA5 | HA6 | HA7 | HA8 | HA9 | HA10 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Time to 38.5°C $T_{re}$ (min) | CH-CON | 37 ± 8 | 37 ± 7 | 36 ± 7 | 36 ± 6 | 38 ± 8 | 36 ± 5 | 38 ± 8 | 37 ± 5 | 35 ± 5 | 38 ± 5 |
| | CH-HWI | 37 ± 4 | 38 ± 9 | 34 ± 6 | 37 ± 5 | 36 ± 5 | 36 ± 6 | 37 ± 6 | 35 ± 5 | 35 ± 6 | 40 ± 10 |
| | CH-CH | 36 ± 8 | 38 ± 8 | 38 ± 8 | 41 ± 10 | 40 ± 6 | 42 ± 7 | 42 ± 9 | 38 ± 5 | 38 ± 5 | 41 ± 6 |
| WBSR (L·h$^{-1}$)[a] | CH-CON | 1.00 ± 0.32 | 1.16 ± 0.32 | 1.27 ± 0.44 | 1.28 ± 0.40 | 1.24 ± 0.33 | 1.38 ± 0.42 | 1.42 ± 0.41 | 1.43 ± 0.46 | 1.53 ± 0.36 | 1.43 ± 0.40 |
| | CH-HWI | 1.06 ± 0.28 | 1.13 ± 0.26 | 1.23 ± 0.33 | 1.23 ± 0.25 | 1.14 ± 0.28 | 1.31 ± 0.31 | 1.34 ± 0.30 | 1.41 ± 0.28 | 1.43 ± 0.37 | 1.43 ± 0.34 |
| | CH-CH | 1.01 ± 0.53 | 1.02 ± 0.47 | 0.99 ± 0.51 | 1.07 ± 0.53 | 1.18 ± 0.57 | 1.13 ± 0.63 | 1.17 ± 0.61 | 1.25 ± 0.66 | 1.33 ± 0.66 | 1.30 ± 0.72 |
| Average heart rate (bpm)— thermal drive | CH-CON | 137 ± 20 | 141 ± 22 | 140 ± 20 | 139 ± 22 | 137 ± 21 | 137 ± 19 | 135 ± 20 | 137 ± 20 | 138 ± 21 | 137 ± 23 |
| | CH-HWI | 139 ± 23 | 136 ± 21 | 139 ± 21 | 136 ± 17 | 137 ± 17 | 140 ± 17 | 140 ± 18 | 138 ± 16 | 140 ± 18 | 138 ± 15 |
| | CH-CH | 137 ± 18 | 141 ± 20 | 138 ± 20 | 139 ± 21 | 136 ± 19 | 135 ± 20 | 138 ± 17 | 139 ± 19 | 137 ± 17 | 136 ± 17 |
| Average heart rate (bpm)— thermal maintenance | CH-CON | 128 ± 21 | 128 ± 19 | 124 ± 19 | 125 ± 20 | 123 ± 16 | 122 ± 18 | 135 ± 17 | 123 ± 18 | 127 ± 14 | 127 ± 17 |
| | CH-HWI | 133 ± 25 | 129 ± 20 | 128 ± 20 | 126 ± 15 | 130 ± 19 | 130 ± 16 | 140 ± 18 | 132 ± 16 | 130 ± 16 | 130 ± 16 |
| | CH-CH | 130 ± 22 | 135 ± 21 | 129 ± 21 | 132 ± 20 | 128 ± 16 | 130 ± 18 | 138 ± 17 | 129 ± 18 | 131 ± 14 | 135 ± 17 |

Data are presented as mean ± SD.
[a]A main effect of time indicated that WBSR was significantly highly than HA1, from days 4 to 10.

classified as "trivial," 0.2–0.49 as "small," 0.5–079 as "moderate," and >0.8 as a "large" effect.

The same statistical approach was used to examine physiological responses to the HA and HRA sessions, with days (HA days 1–10 and HRA days 1–5) as within-subject factors and HRA groups (CH-CH, CH-HWI, and CH-CON) as between-subject factors. For HA and HRA, *post hoc* analyses were made relative to HA1 or HRA1, with the alpha adjusted accordingly. Violations of sphericity were corrected for using the Greenhouse–Geisser adjustment. Nonparametric analyses of the perceptual responses (thermal sensation, thermal comfort, and RPE) were conducted using permutation tests.

## RESULTS

**Heat acclimation and HRA programs.** All participants completed 10 d of HA using a CH technique, and the daily physiological responses of each group are summarized in Table 2. $T_{re}$ increased to 38.5°C in 37 ± 7 min, which did not differ over HA days, nor were they different between groups ($P > 0.05$, $\eta^2 < 0.08$). Heart rate during the thermal drive phase and the thermal maintenance phase of CH did not differ over the number of HA days and were also similar between groups ($P > 0.05$, $\eta^2 < 0.07$). Although daily WBSR was not different between groups ($P = 0.57$, $\eta^2 = 0.09$; Table 2), there was a main effect of time for WBSR, with the improvements occurring from HA4-HA10 compared with HA1 ($P > 0.05$, $g > 0.60$). There was no interaction (group × time) effect for WBSR ($P = 0.62$, $\eta^2 = 0.05$).

All participants completed their respective HRA protocols. Table 3 summarizes the daily physiological responses of the CH-HWI and CH-CH groups. For the CH-CH group, $T_{re}$

increased to 38.5°C in 40 ± 7 min. Heart rates during the thermal drive phase were 141 ± 20 and 133 ± 20 bpm during the thermal maintenance phase of CH. The lower heart rate during thermal maintenance compared with the thermal drive is indicative of maintaining the elevated yet stable $T_{re}$ with rest periods and low-intensity exercise. For the CH-HWI group, the water temperature was 39.7°C ± 0.86°C, and all participants completed the required 40 min. Resting $T_{re}$ was 37.42°C ± 0.24°C and increased to 38.86°C ± 0.34°C at the end of HWI. Resting heart rate was 72 ± 12 bpm and increased to 102 ± 14 bpm at the end of HWI.

Daily WBSR values during HRA for CH-CH and CH-HWI were not different between groups ($P = 0.61$, $\eta^2 = 0.05$; Table 3), but there was a main effect of time, with the improvements occurring from HRA2–5 compared with HRA1 ($P > 0.05$, $g = 0.17$–0.38). There was no interaction effect for WBSR ($P = 0.43$, $\eta^2 = 0.29$). Heart rate and thermal impulse for $T_{re}$ were both significantly higher for the CH-CH group ($P < 0.001$, $\eta^2 > 0.75$), but the data were not different over time and no interaction effects were observed ($P > 0.05$, $\eta^2 < 0.15$).

**HST—exercise performance.** During HST3, one participant from the CH-HWI group felt unwell and did not complete the GXT. GXT data of this participant were removed before analysis. Figure 2 illustrates the GXT data, and the associated ES with 90% CI are illustrated in Figure 3. Analysis revealed main effects of time ($P < 0.001$, $\eta^2 = 0.28$), with the GXT during HST2 being longer than HST1 ($P = 0.04$, $g = 0.37$) and both HST3 and HST4 being shorter than HST2 ($P < 0.002$, $g = 0.25$ and 0.35, respectively), although no other differences were observed. There was no effect of HRA group ($P = 0.94$, $\eta^2 = 0.006$) nor an interaction effect ($P = 0.22$, $\eta^2 = 0.12$). A main effect of time ($P = 0.05$,

TABLE 3. Daily physiological response to the 5-d HRA protocol completed by either the hot water immersion (CH-HWI, $n = 8$) or the controlled hyperthermia (CH-CH, $n = 8$).

| | | HRA1 | HRA2 | HRA3 | HRA4 | HRA5 |
|---|---|---|---|---|---|---|
| Thermal impulse (°C·min$^{-1}$) | CH-HWI | 1325 ± 14 | 1324 ± 24 | 1311 ± 26 | 1320 ± 28 | 1322 ± 27 |
| | CH-CH | 3517 ± 244[b] | 3493 ± 249[b] | 3470 ± 234[b] | 3480 ± 317[b] | 3517 ± 298[b] |
| Heart rate (bpm) | CH-HWI | 95 ± 5 | 93 ± 7 | 88 ± 3 | 119 ± 3 | 114 ± 11 |
| | CH-CH | 135 ± 17 | 138 ± 16 | 139 ± 14 | 135 ± 14 | 134 ± 12 |
| WBSR (L·h$^{-1}$)[a] | CH-HWI | 1.1 ± 0.6 | 1.3 ± 0.6 | 1.3 ± 0.7 | 1.4 ± 0.7 | 1.4 ± 0.7 |
| | CH-CH | 1.3 ± 0.4 | 1.4 ± 0.4 | 1.4 ± 0.4 | 1.4 ± 0.5 | 1.5 ± 0.5 |

Data are presented as mean ± SD. Thermal impulse, WBSR, and the average heart rate of the entire HRA protocol data are presented.
[a]A main effect of time indicated that WBSR was significantly higher than HRA1, from days 2 to 5.
[b]A main effect of HRA protocol with CH-CH being systematically higher than CH-HWI.
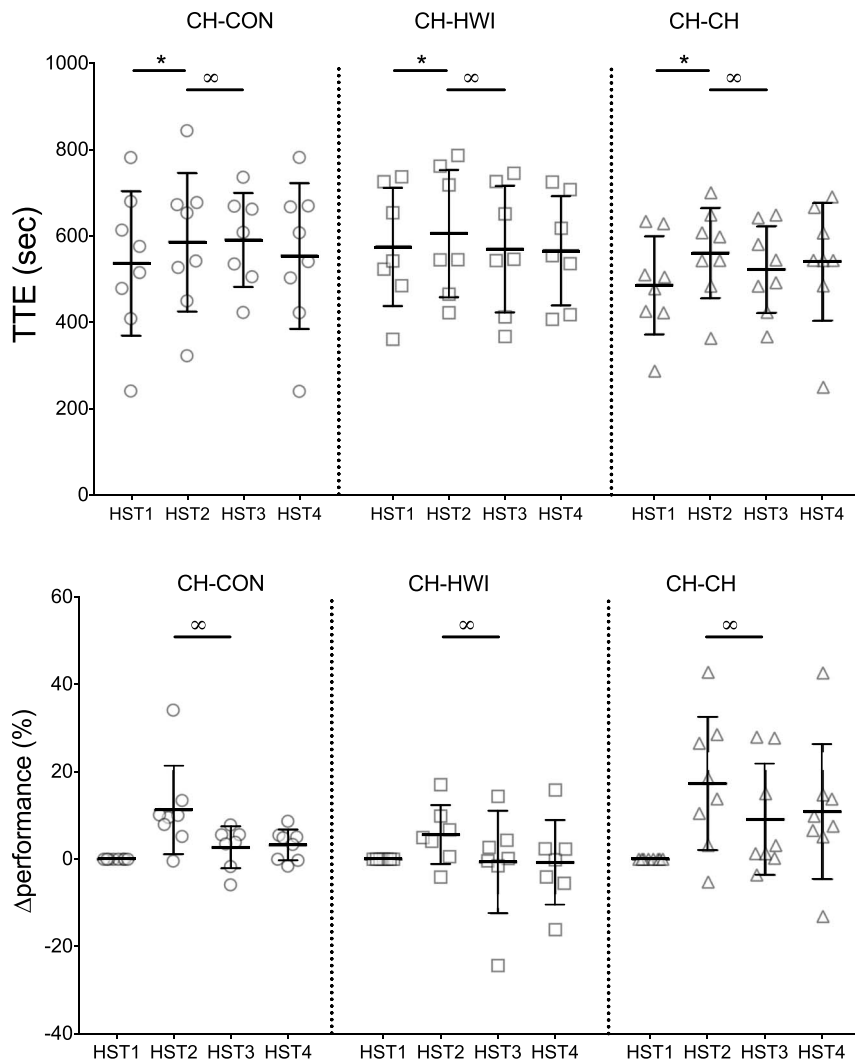
**FIGURE 2**—Top panel shows the TTE during the GXT test. The bottom panel shows exercise performance as a percentage change from HST1 for CH-CON ($n = 8$), CH-HWI group ($n = 7$), and CH-CH group ($n = 8$) during HST1, HST2, HST3, and HST4. *Gray plots* illustrate individual responses, whereas *solid black line* illustrates the mean ± SD. *Main effects of time between HST1 and HST2 ($P < 0.05$). ∞Main effects of time between HST2 and HST3 ($P < 0.05$).

$\eta^2 = 0.22$) and HRA group ($P = 0.036$, $\eta^2 = 0.27$) was found, but no interaction effect was found for performance as a percentage change from HST1 ($P = 0.43$ $\eta^2 = 0.03$). HST3 was significantly lower than HST2 ($P = 0.004$), but HST2 and HST3 were similar to HST4 ($P > 0.08$, $g = 0.24$ and $-0.64$, respectively).

**Physiological responses to exercise.** For the following data, the effect size and 90% CI are presented in Figure 3. Figure 4 illustrates $T_{re}$ at rest and at the end of the 35-min cycling protocol (1.5 W·kg$^{-1}$ BW). For resting $T_{re}$, there was a main effect of time ($P < 0.001$, $\eta^2 = 0.47$) with HST2 and HST3 being significantly lower than HST1 ($P < 0.009$, $g = -0.51$ and $-1.06$), but no other time effects were found ($P > 0.05$, $g > -0.33$). There were no differences between HRA groups ($P = 0.768$, $\eta^2 = 0.03$), nor an interaction effect ($P = 0.135$, $\eta^2 = 0.15$). A main effect of time was also observed for end of exercise $T_{re}$, and *post hoc* analysis indicated that $T_{re}$ was significantly lower after 10 d HA (HST2 vs HST1, $P = 0.046$, $g = -0.46$). $T_{re}$ did not return to pre-HA values, as HST2 and

HST3 were similar ($P = 0.0001$, $g = -0.28$), but $T_{re}$ was lower than HST1 after the decay (HST3, $P = 0.001$, $g = -0.76$). There were no differences in end of exercise $T_{re}$ between HST2, HST3, and HST4 ($P < 0.05$, $g = -0.01$ to $-0.31$). There were no differences between HRA groups ($P = 0.72$, $\eta^2 = 0.03$), nor an interaction effect ($P = 0.28$, $\eta^2 = 0.11$).

Figure 5 (left panels) illustrates WBSR, end of exercise mean $T_{sk}$, and heart rate. For WBSR, there was a main effect of time ($P = 0.0001$, $\eta^2 = 0.41$) with an elevated WBSR during HST2 compared with HST1 ($P = 0.0001$, $g = 0.60$). After the decay period, WBSR decreased, evidenced by a significant reduction during HST3 compared with HST2 ($P = 0.001$, $g = -0.51$), and decay was complete (HST1 and HST3, $P = 0.99$, $g = 0.09$). After HRA, WBSR increased with HST4 being greater than HST3 ($P = 0.008$, $g = 0.35$), but this was not greater than HST2 ($P = 0.285$, $g = -0.17$). There was no main effect of group ($P = 0.31$, $\eta^2 = 0.11$), but an interaction effect did exist for WBSR ($P = 0.050$, $\eta^2 = 0.184$). During HST4, the WBSR values for the CH-CH
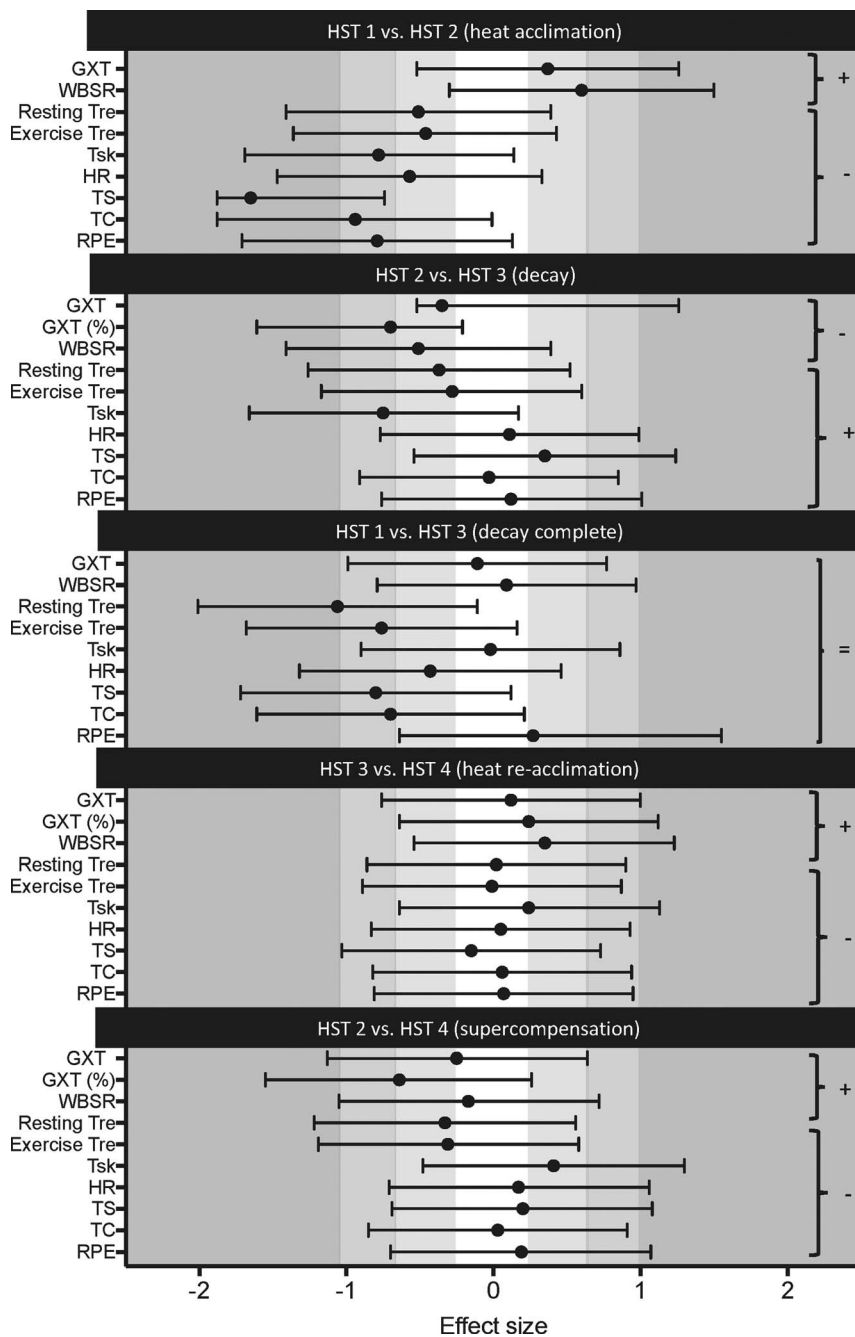
**FIGURE 3**—Effect size and 90% CI of the physiological and perceptual responses to HST and graded exercise performance test (GXT) ($n = 24$). $T_{sk}$, heart rate (HR), thermal sensation (TS), thermal comfort (TC), and RPE are the end of exercise response. The symbols ("+," "-," and "=") to the right of the graph indicate the expected (effect) direction of each dependent variable for each pairwise comparisons: HST1 and HST2 indicate HA adaptation; HST2 and HST3 indicate decay; HST1 and HST3 indicate complete decay; HST3 and HST4 indicate HRA response; HST2 and HST4 indicate supercompensation. The shaded vertical bars denote effect size ranges of trivial (<0.19, *white*), small (0.2–0.49, *lighter gray*), moderate (0.5–0.79, *gray*), and large (>0.8, *darker gray*) effects.

and CH-HWI groups were significantly higher than the CH-CON group ($P < 0.05$, $g = 0.5$ to 1.1).

For mean $T_{sk}$, there was a main effect of time ($P = 0.001$, $\eta^2 = 0.32$) but no effect of HRA group and no interaction effect ($P > 0.05$, $\eta^2 < 0.29$). Mean $T_{sk}$ during HST2 was lower than HST1 ($P = 0.004$, $g = -0.78$), and HST3 was higher than HST2 ($P = 0.003$, $g = -0.75$). Decay was complete as there were no differences observed between HST1 and HST3. No

differences were observed between HST2 and HST3 with HST4 ($P > 0.05$, $g < 0.41$).

For resting heart rate, there were no main effects for time, group, or interactions ($P > 0.05$, $\eta^2 < 0.13$). For heart rate at the end of exercise, there was a main effect of time ($P = 0.001$, $\eta^2 < 0.38$), no effect of group, and no interaction effect ($P < 0.05$, $\eta^2 < 0.12$). Heart rate at the end of exercise was lower during HST2 and HST3 compared with HST1
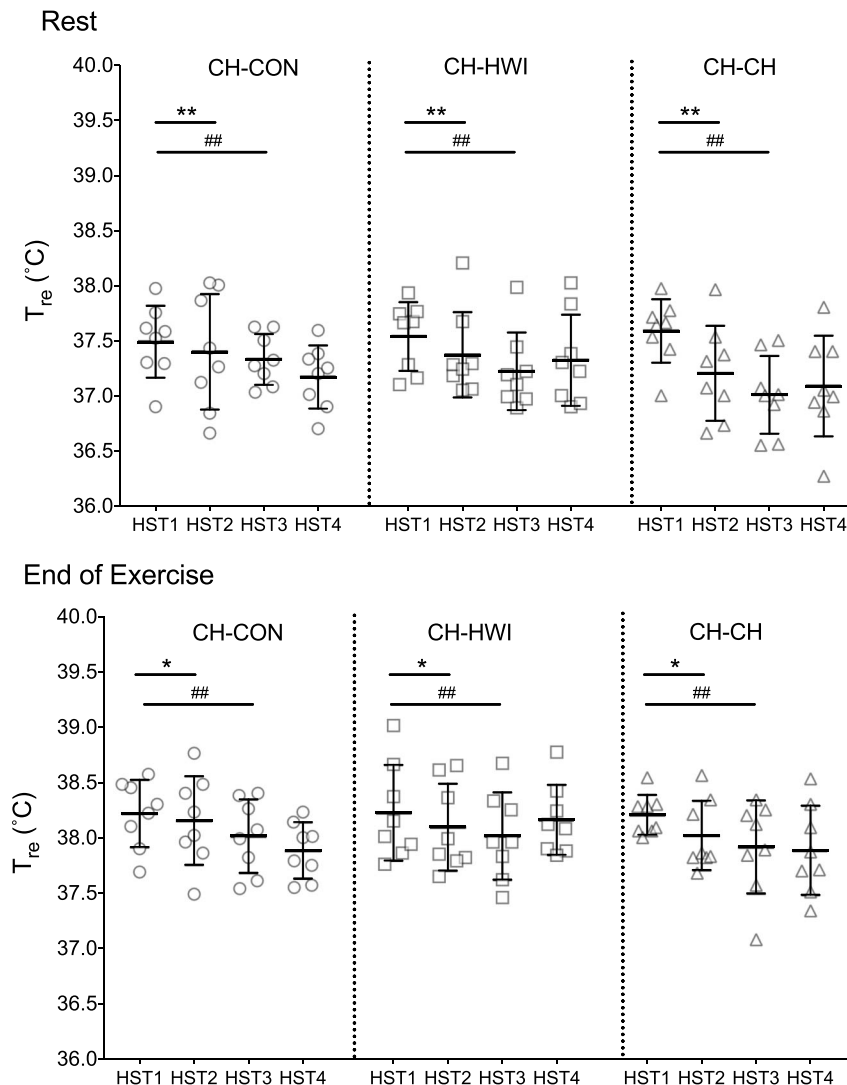
**FIGURE 4**—$T_{re}$ at rest (*top panel*) and at the end of exercise (*bottom panel*) for CH-CON ($n = 8$), CH-HWI group ($n = 8$), and CH-CH group ($n = 8$) during HST1, HST2, HST3, and HST4. *Gray plots* illustrate individual responses, whereas *solid black line* illustrates the mean ± SD. Main effects of time between HST1 and HST2 (*$P < 0.05$ and **$P < 0.001$). Main effects of time between HST1 and HST3 (##$P < 0.001$).

($P < 0.001$, $g = -0.43$ to $-0.57$). There were no other differences observed.

**Thermal sensation, thermal comfort, and RPE.** Figure 5 (right panels) illustrates the perceptual measures at the end of the 35-min cycling (1.5 W·kg$^{-1}$ BW). For thermal sensation, there was no effect of group, time, or interaction ($P > 0.3$, $\eta^2 < 0.12$). For thermal comfort, there was a main effect of time with all HST2 and HST3 being lower than HST1 ($P < 0.001$, $g < 0.70$), but there was no group or interaction effect ($P > 0.5$). For RPE, there was no group, time, or interaction effect ($P > 0.2$).

## DISCUSSION

The present study aimed to assess the effectiveness of HRA on thermophysiological responses and performance during exercise in the heat. In summary, the main findings indicated that after a successful CH-HA protocol, most of the adaptations

were retained during the 28-d decay period. Of the phenotypes to decay, the only variable to improve after HRA was WBSR. Additional exposure to the heat via active (CH-CH) or passive (CH-HWI) exposure did increase WBSR to a similar extent, both greater than no additional heat exposure (CH-CON). The data suggest that in habitually trained individuals, HRA may not be necessary within a 28-d decay period, as long as a 10-d CH-HA protocol has been successful in bringing about these physiological adaptations.

**Heat acclimation.** All participants completed the initial 10-d CH-HA protocol, which resulted in a similar stimulus between groups, evidenced by the lack of any group effects (Table 2). To address our research questions, it was important that the HA phenotypes were observed in all groups. Compared with HST1, TTE improved, and resting and end of exercise $T_{re}$, end of exercise mean $T_{sk}$, and thermal comfort were lower and WBSR greater in HST2 for all groups. The effect sizes (Fig. 3) indicated that these adaptations were all more
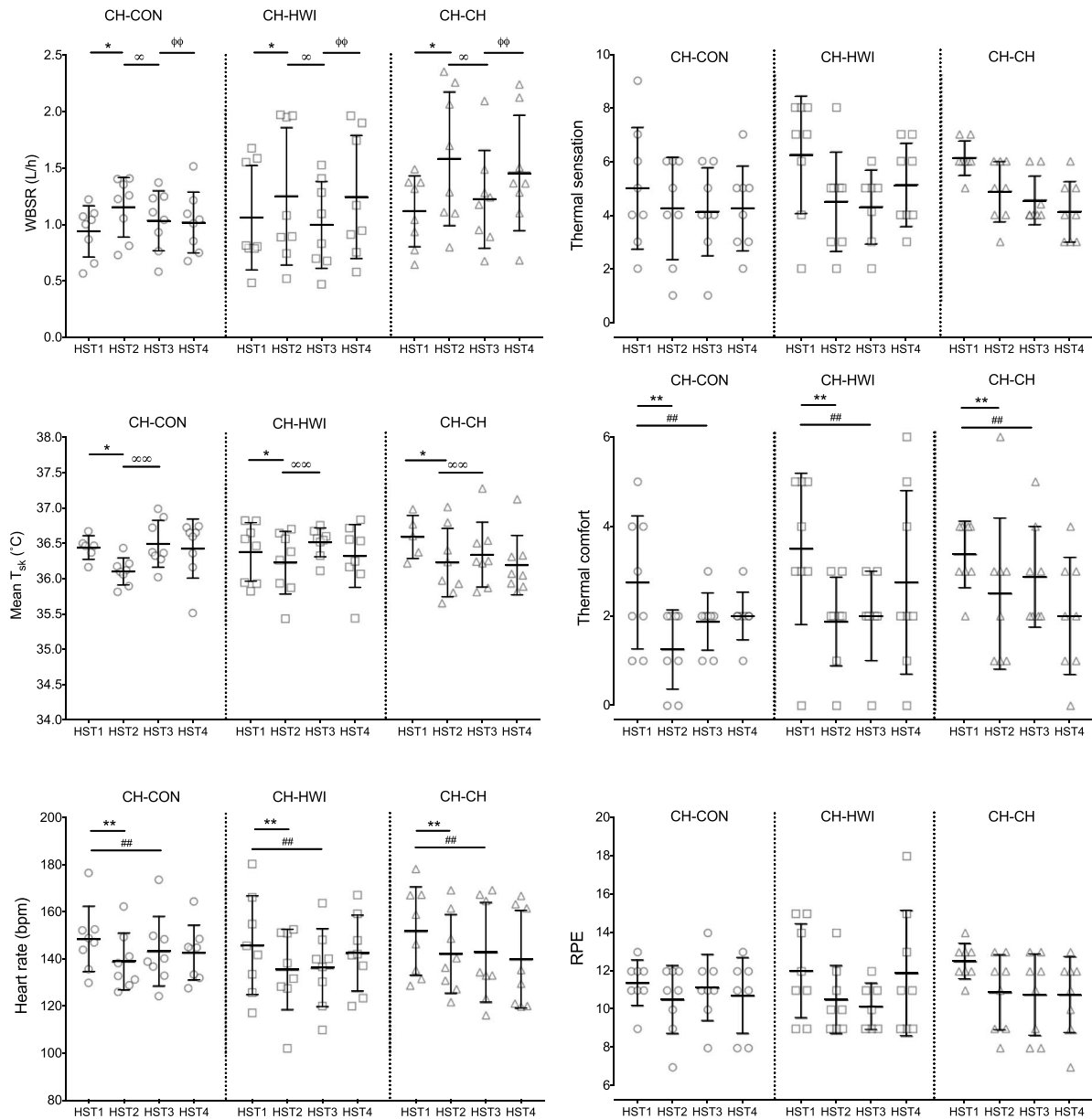
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**FIGURE 5**—WBSR, end of exercise mean $T_{sk}$, heart rate, thermal comfort, thermal sensation, and RPE for CH-CON ($n = 8$), CH-HWI group ($n = 8$), and CH-CH group ($n = 8$) during HST1, HST2, HST3, and HST4. *Gray plots* illustrate individual responses, whereas *solid black line* illustrates the mean ± SD. Main effects of time between HST1 and HST2 (*$P < 0.05$ and **$P < 0.001$). Main effects of time between HST1 and HST3 (##$P < 0.001$). Main effects of time between HST2 and HST3 ($\infty P < 0.05$ and $\infty\infty P < 0.001$). Main effects of time between HST3 and HST4 ($\phi P < 0.05$ and $\phi\phi P < 0.001$).

than trivial effects, but the large CI values reflect the uncertainty within the data. Neither thermal sensation nor RPE decreased after completing the HA protocol, although the effect sizes were moderate. Overall, most of the adaptations were acquired, and there were no differences between groups. The adaptations were comparable with other studies. Weller et al. (9), who used a mixed fixed load and a CH protocol, reported a reduction in resting $T_{re}$ of $-0.26°C$, and the average of our group was $-0.20°C$. However, they reported greater improvements after 60 min exercise in $T_{re}$ ($-0.50°C$) compared with our moderate ($g = -0.46$) improvements after HA ($-0.17°C$). The attenuation in $T_{re}$ at the end of exercise was most likely associated with the parallel decrease in resting

$T_{re}$ rather than a reduced heat storage during exercise (26). This was unexpected, given the increased sweat production and lower heart rate, but our shorter cycling protocol (35 min) may have masked the thermophysiological adaptations during exercise. End of exercise heart rate was 10 bpm lower for all groups, and WBSR increased by 0.3 $L \cdot h^{-1}$, which is comparable with other studies (9,27).

An interesting observation during the initial 10-d CH-HA protocol was the attainment of an increased WBSR after just 4 d of CH, although a meta-analysis showed that sweat rate adaptations are greatest after at least 8 d of HA (2,28,29). The aforementioned time course for HA phenotypes is generally based on studies using fixed load protocols. Controlled hyperthermia

is becoming increasingly common, but the time course and the magnitude of the adaptations are difficult to evaluate because of the limited number of studies (29). Neal et al. (17) used a CH-HA protocol and also noted elevated sweat output after just 4 d. It is unclear why we both observed a faster adaptation response; perhaps the training status of our participants allowed for a faster sweat rate adaptation. This warrants more research.

**Decay.** We used a 28-d decay period, with the expectation that most of the adaptations would be lost or at least lower than postacclimation values. Exercise performance (TTE), WBSR and mean $T_{sk}$ were the only HA phenotypes to decay; the nonsignificant differences between HST1 and HST3 and the trivial effect size suggest that this decay was complete. The decay observed for mean $T_{sk}$ may be associated with the concomitant reduction in WBSR, which would result in reduced evaporative cooling at the skin. However, it is unclear why performance (TTE) decayed, despite a lowered $T_{re}$ and improved thermal comfort even after the decay period.

It has previously been suggested that the time course for HA and decay vary for different phenotypes (2). It has been suggested that sudomotor adaptations have the longest accrual time (>7 d), but along with cardiovascular adaptations, they are also quickly lost (30). Although we have already questioned the delayed attainment of the sudomotor adaptations after CH-HA, we did observe a sudomotor decay that is consistent with the literature (9,12,31,32). Because local sweat production is fundamental for sweat gland adaptations (33), it stands to reason that the removal of the heat stimulus resulted in a sweat gland detraining response. This may account for the fast sudomotor decay observed here and by others.

It has been suggested that 1 d of HA is lost for every 2 d away from the heat (34), but accumulating evidence from this study and others (9,10) seems to suggest that decay occurs less quickly than originally thought. There are some studies showing very little decay after short periods (7 d) away from the heat after both CH and fixed load HA protocols. Only a few studies have used longer decay periods, and the findings are inconclusive. A consistent finding among many studies is that heart rate declines at a faster rate than core body temperature. Our findings are at odds with this, as neither $T_{re}$ nor heart rate decayed. The cardiorespiratory fitness status of our participants may have accounted for the minimal decay observed in our study (10). Furthermore, their habitual training schedule, which predominately included endurance-based training sessions, may have occasionally elevated core body temperature and thus attenuated decay.

An interesting observation from our study was the continued adaptation (i.e., gains) in $T_{re}$ during the so-called decay period. Daanen et al. (30) also found that the adaptations for $T_{re}$ were more pronounced after a 3-d decay period than they were immediately after HA. Weller et al. (9), showed small gains in resting and end of exercise $T_{re}$ after the 26-d decay period. Why $T_{re}$ is able to maintain this adaptation throughout a decay period, whereas sudomotor and cardiovascular responses are lost more quickly, is unclear. Daanen et al. (30) suggested that the short recovery time (<24 h) between heat exposures was insufficient for the adaptations to manifest. This may have resulted in a latency period before this particular HA phenotype became evident.

**HRA.** Although the HA phenotypes disappear after prolonged nonheat exposure, it has been suggested that at the molecular level they remain in an altered state (7,8). It was our aim to see whether this dormant memory could be harnessed to offset the practical and logistical challenges for athletes when trying to combine a successful HA protocol into the tapering phase of training. This memory would allow athletes to adopt an HA protocol several weeks before competition to use a decay period alongside their tapering phase and in a few days before competition acclimate faster. For these reasons, passive heating, using HWI, was investigated as a potential HRA protocol as it would expose athletes to a heat stimulus, which may be sufficient to trigger the dormant memory and bring about the reinduction. The protocol used was adapted from previous research (5) to ensure compliance. HWI is accompanied by a high risk of syncope, and completing the full duration was challenging for some of our participants. To alleviate the risk and to decrease discomfort, participants were allowed "relief" breaks whereby they sat partially submerged for 2 min, every 10 min. All participants of the CH-HWI group successfully completed the protocol.

Several studies suggest that HRA is faster than HA, but no study has been able to conclusively confirm this as decay because all adaptations were incomplete (9–13,32,35,36). In the present study, exercise performance (TTE), WBSR, and mean $T_{sk}$ were the only variables that significantly decayed, and this decay appears complete. However, after HRA, the only variable to improve was WBSR. The shorter period (5 vs 10 d) suggests a faster HRA for sudomotor adaptations. This is supported by the faster WBSR increase during HRA (1 d) compared with HA (4 d). Furthermore, this improved sudomotor response was observed in both the CH and the HWI-HRA groups. Although it is generally accepted that exercise in the heat is more effective than passive heat exposure for developing HA phenotypes (2), our findings suggest that this does not hold true for sweat rate, which improved during HST4 in both the CH-HWI and the CH-CH groups. WBSR values during the two HRA protocols were similar, which suggests that as long as the exposure results in a WBSR of 1.1–1.5 L·h$^{-1}$, then this is enough to stimulate the dormant memory and bring about the readaptations faster. This value may even exceed the required sweat rate, as Taylor et al. (37) suggested that the minimum WBSR for sudomotor adaptation is approximately 0.4–0.8 L·h$^{-1}$.

Compared with CH-CH, the duration of exposure was considerably shorter for the CH-HWI group (~90 vs 40 min, respectively), and the heart rate and the thermal impulse for $T_{re}$ were considerably lower. Despite this, CH-CH was not superior in reinstating the lost HA adaptations, nor in promoting a super compensation. Most HA protocols focus on elevating core body temperature as this is a key stimulus for HA, but Regan et al. (38) highlighted the importance of an elevated $T_{sk}$ on HA. $T_{sk}$ was not measured during the HWI, but we can assume it was similar to the water temperature (~40°C) and

APPLIED SCIENCES

fluctuated slightly during the "relief" periods. The mean $T_{sk}$ during CH-HRA (not reported) was 35.9°C ± 0.97°C. Although the daily duration was shorter and the thermal impulse for the CH-HWI group lower, the elevated $T_{sk}$ during the HWI was evidently sufficient to promote high sweat rates to initiate sudomotor adaptations. Sweating relies on central and cutaneous thermal afferents; the higher thermal impulse for the CH-CH group predominantly stimulated central afferents, but the dual stimulus (core and $T_{sk}$) from HWI stimulated both central and cutaneous thermal afferents to bring about the adaptations.

Exercise performance (TTE) and mean $T_{sk}$ also decayed but showed no further adaptations to HRA, regardless of HRA strategy. Although we hypothesized that the CH-HWI group would initiate similar adaptations to CH-CH, it is also plausible that the combination of exercise and heat would be superior in improving performance compared with passive heat alone. However, HRA, whether by HWI or CH, did not improve the TTE, and it is not clear why this did not occur in the presence of a lower $T_{re}$ and improved WBSR. Previous HRA studies have focused predominately on strategies for the benefit of occupational workers as opposed to athletic performance. As such, this is the first study to investigate the influence of HRA on a performance variable, and although TTE lacks ecologically validity for some sports, it is an important first step in discerning the effect of HRA on exercise performance.

The concept of supercompensation occurring with HRA was alluded to by Weller et al. (9), who found that after HRA, heart rate and $T_{re}$ tended to decrease below the values at the end of HA. They were not able to confirm whether this truly exists as decay had been minimal or nonexistent. In our study, we could test this hypothesis for WBSR, mean $T_{sk}$, and performance (TTE), all of which decayed completely. Our data indicate three important findings. First, in the presence of decay, not all of the HA phenotypes will regain the adaptations with a 5-d HRA protocol. Second, sudomotor adaptations that are lost during a decay period can be reinstated to a similar post-HA/predecay values, but supercompensation did not occur. Third, when decay has not occurred, super compensation does not exist with HRA, regardless of passive or active HRA protocols. The possibility that longer HRA protocols could result in supercompensation is unlikely, given that we used a 5-d HRA protocol and others have reported gains after as little as 1 d of HRA. Pandolf et al. (10) suggested that fitter individuals acclimate fast, decay slow, and rapidly reacclimate.

The success of HRA is most likely influenced by the success of the HA protocol and the duration of the decay period. It may be the case that when full HA has been achieved, the rate of decay is slower. Moreover, when decay is large, more HRA days are needed to return the loss, but this might vary for the different HA phenotypes. In the present study, $T_{re}$ did not change at all during decay, and no additional improvements were observed with HRA. By contrast, WBSR decayed, and the adaptation was reinstated in both HRA protocols. We have been unable to elucidate any mechanisms accounting for decay and reinduction because all studies to date, including this one, have not been able to examine the effect of HRA

when decay has been 100% complete. This is difficult to control, and it may be that future studies will use a >2-month decay period to ensure all adaptations are lost. Given that the two HRA protocols used in the present study did not show any differential superiority, it may be suggested that HWI could still be a successful HRA strategy in the presence of molecular dormant memory (i.e., when decay is complete) from a successful HA protocol; more research is required.

**Limitations.** We were unable to control for the menstrual cycle phase because of the predetermined timings of the protocol. As a result, females were only tested at a similar menstrual phase during HST2 and HST3. Core body temperature is usually higher during the luteal phase (39,40), and we did observe an elevated resting $T_{re}$ during at least one HST in three (out of nine) participants, which corresponded with their (self-reported) luteal phase. Although an elevated core temperature has been reported to impair incremental exercise performance in the heat (40), we did not observe any trends for this when tests were completed in the luteal phase. There is limited evidence for local or WBSR differences between menstrual cycle phase in hot and humid conditions (39,40). As such, we doubt that the observed WBSR adaptations were influenced by menstrual cycle phases during the four HST. This certainly warrants further investigation, although the challenge to align HA, decay, and HRA protocols with the menstrual cycle remains.

## CONCLUSION

In habitually trained individuals, most of the physiological adaptations acquired during an initial 10-d CH-HA protocol were not lost during a 28-d decay period. However, we did observe that sudomotor adaptations were lost during a 28-d decay period, but they can be reinstated to similar post-HA/predecay values. Most importantly, the sudomotor adaptations can be reinstated with either an active or a passive 5-d HA strategy, such as CH or HWI. It was clear that a super compensation does not exist in all the HA phenotypes measured, whether decay occurred or not, and regardless of a passive or active HRA protocol. Collectively, the data suggest that HRA protocol may not be necessary within a 28-d decay period, as long as a 10-d CH-HA protocol has been successful in bringing about the appropriate physiological adaptations. However, if there is reason to believe that the sweat adaptation has started to decay, then a 5-d passive or active HRA protocol can reinstate the lost adaptations.

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