Temperate Performance Benefits after Heat, but Not Combined Heat and Hypoxic Training

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ABSTRACT

MCCLEAVE, E. L., K. M. SLATTERY, R. DUFFIELD, P. U. SAUNDERS, A. P. SHARMA, S. J. CROWCROFT, and A. J. COUTTS. Temperate Performance Benefits after Heat, but Not Combined Heat and Hypoxic Training. *Med. Sci. Sports Exerc.*, Vol. 49, No. 3, pp. 509–517, 2017. **Purpose**: Independent heat and hypoxic exposure can enhance temperate endurance performance in trained athletes, although their combined effects remain unknown. This study examined whether the addition of heat interval training during "live high, train low" (LHTL) hypoxic exposure would result in enhanced performance and physiological adaptations as compared with heat or temperate training. **Methods**: Twenty-six well-trained runners completed 3 wk of interval training assigned to one of three conditions: 1) LHTL hypoxic exposure plus heat training (H + H; 3000 m for 13 $h \cdot d^{-1}$, train at 33°C, 60% relative humidity [RH]), 2) heat training with no hypoxic exposure (HOT, live at <600 m and train at 33°C, 60% RH), or 3) temperate training with no hypoxic exposure (CONT; live at <600 m and train at 14°C, 55% RH). Performance 3-km time-trials (3-km TT), running economy, hemoglobin mass, and plasma volume were assessed using magnitude-based inferences statistical approach before (Baseline), after (Post), and 3 wk (3wkP) after exposure. **Results**: Compared with Baseline, 3-km TT performance was likely increased in HOT at 3wkP (−3.3% ± 1.3%; mean ± 90% confidence interval), with no performance improvement in either H + H or CONT. Hemoglobin mass increased by 3.8% ± 1.8% at Post in H + H only. Plasma volume in HOT was possibly elevated above H + H and CONT at Post but not at 3wkP. Correlations between changes in 3-km TT performance and physiological adaptations were unclear. **Conclusion**: Incorporating heat-based training into a 3-wk training block can improve temperate performance at 3 wk after exposure, with athlete psychology, physiology, and environmental dose all important considerations. Despite hematological adaptations, the addition of LHTL to heat interval training has no greater 3-km TT performance benefit than temperate training alone. **Key Words:** HEAT ACCLIMATION, HYPOXIA, PLASMA VOLUME, ENDURANCE, HEMOGLOBIN MASS

Substantial training loads (TL) are undertaken by endurance athletes to maximize physiological adaptations and physical performance. However, both high and unaccustomed loads can increase risks of overreaching and injury, which are counterproductive to maximizing performance (11). Therefore, interventions that enhance the physiological and performance outcomes in the absence of increased training volume are attractive to coaches and athletes. Accordingly, considerable interest exists on the effects of living and training in altered environments (i.e., heat and hypoxia). This approach can be used to increase the physiological stress without the need for large increases in external TL (20). Although studies have examined the performance benefits of independent heat (18,32) and hypoxic exposure (4,17), the combined effects of heat and hypoxia are not yet well understood (5).

Repeated exposure to hypoxia can have both ergogenic effects on endurance performance and amplify systemic physiological adaptations (20). The "live high, train low" (LHTL) model traditionally incorporates 12–14 $h \cdot d^{-1}$ of altitude exposure (i.e., >2000 m), with training conducted at low–moderate altitude (i.e., <1250 m) to allow the maintenance of training intensity (20). This model has been shown to improve sea-level endurance performance (4,28), hemoglobin mass (Hb_{mass}), and maximal aerobic capacity ($\dot{V}O_{2max}$) in well-trained endurance athletes (17). Several studies have demonstrated small but significant improvements in run time-trial performance for 3 km (28,37) and 5 km (17) after 2–4 wk of LHTL. However, not all studies have shown improvements for similar distances (27). This lack of consistent improvement is suggested to be related to a number factors, not limited to the extent of physiological adaptation incurred, the hypoxic dose, and the training status of the athletes (4).

In addition to hypoxia, repeated heat exposure has been shown to have a positive ergogenic benefit in hot (16,18)

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and temperate environments (18,26,36). However, a recent debate in the literature highlights the uncertainty surrounding the capacity of heat to improve temperate performance (21,22). The proposed mechanisms for heat exposure improving temperate performance are not clearly understood but are suggested to be related to elevated plasma volume (PV), reduced cardiovascular and thermoregulatory strain, enhanced lactate threshold, and $\dot{V}O_{2max}$ (18). In addition, lower perceptions of heat stress are also evident after heat exposure, which may also be related to performance improvements (34).

In a previous study investigating concurrent heat and intermittent hypoxic exposure in untrained individuals, it was apparent that the combined stimuli elevated PV but had no impact on $\dot{V}O_{2peak}$ (38). However, the combination of LHTL hypoxia and heat training has suggested possible positive physiological and temperate performance adaptations. Buchheit et al. (5) conducted a 2-wk preseason training camp incorporating LHTL plus heat training in team sport athletes. Compared with training in a hot environment alone, the LHTL plus heat group had a greater Hb_{mass} increase, with no difference between groups in PV or Yo-Yo Intermittent Recovery Test 2 performance. Interestingly, 4 wk later, there was a better maintenance of performance, PV, and Hb_{mass} in the combined LHTL plus heat training group (5). The possibility of greater and longer lasting adaptations after concurrent heat and hypoxic exposure makes it an attractive training method. However, this study was limited by the lack of a control group and the early preseason training status of the athletes. Given these limitations, the impact of combined heat and hypoxic training remains equivocal and is yet to be examined on well-trained endurance athletes.

Accordingly, the aim of this study was to examine performance and physiological adaptations to 3 wk of LHTL combined with heat interval training in well-trained runners. In addition, we aimed to assess the time course of these adaptations in the 3 wk after exposure. It was hypothesized that LHTL combined with heat interval training would elicit greater and longer lasting physiological adaptations and 3-km time-trial (3-km TT) performance improvements than training in the heat alone or temperate conditions.

METHODS

Participants. Twenty-eight well-trained male and female middle distance runners were recruited for the study, with 26 included for final analyses. Of the excluded participants, one did not complete all testing requirements, and one participant reported illness during the study. Participants were matched based on previous TL, peak oxygen uptake ($\dot{V}O_{2peak}$), and associated velocity ($v\dot{V}O_{2peak}$) obtained during preliminary testing. After taking into account the participants' geographic proximity to the testing centers, they were randomly assigned (coin toss/number) by an independent associate to one of three groups: 1) LHTL hypoxic exposure plus training in a hot environment (H + H; F_iO_2 = 14.4% [3000 m] for 13 $h \cdot d^{-1}$; train at <600 m, 33°C, 60% relative humidity [RH]); 2) heat training with no hypoxic exposure (HOT; live and train at <600 m, 33°C, 60% RH); or 3) temperate training with no hypoxic exposure (CONT; live and train at <600 m, 14°C, 55% RH). Participants had ≥2 yr running experience and regularly completed 10–20 h of training each week. All groups contained a mix of male and female athletes, and no participants had heat or hypoxic exposure in the 4 wk prior. All differences in baseline characteristics between training groups were unclear (Table 1). Before the study, all participants were informed of all procedures and potential risks involved in the study, and a written informed consent was obtained. The study was approved by the Human Research Ethics Committee of the University of Technology Sydney (trial no. UTS HREC 2014000203).

Experimental overview. This study was a multicenter, parallel, matched group design, with all training and testing conducted during winter and early spring months in Sydney or Canberra, Australia (June–November 2014). The study included a 3-wk period (exposure) whereby participants lived and trained in their assigned environmental conditions. This was followed by a 3-wk period (nonexposure) in which all individuals lived and trained in temperate, normoxic conditions. During the exposure period, individuals in the H + H group spent 21 d (13 $h \cdot d^{-1}$, F_iO_2 = 14.4%,) in a normobaric hypoxic facility at the Australian Institute of Sport (AIS, Canberra). All participants completed 3 × 90 min treadmill sessions per week, including two interval sessions and one moderate continuous run (a total of nine sessions). H + H and HOT participants completed heat sessions in a climate-controlled chamber (Altitude Training Systems, Lidcombe, Australia). Canberra-based participants trained at the University of Canberra (32.5°C ± 0.7°C, 59% ± 7% RH), whereas Sydney-based participants trained at the New South Wales Institute of

TABLE 1. Baseline physical characteristics of participants in heat and hypoxia (H + H), heat (HOT), and temperate (CONT) training groups.

	H + H *n* = 10; M = 6, F = 4	HOT *n* = 9; M = 6, F = 3	CONT *n* = 7; M = 5, F = 2
Age (yr)	28.7 ± 9.5	29.8 ± 5.2	30.7 ± 5.1
Body mass (kg)	65.4 ± 8.6	71.5 ± 11.9	69.9 ± 10.6
Height (cm)	174.9 ± 8.2	176.7 ± 9.4	179.1 ± 9.3
Sum of 7 (mm)	63.1 ± 30.1	58.6 ± 15.5	50.6 ± 17.2
$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	4.1 ± 0.8	4.7 ± 1.2	4.5 ± 1.1
$\dot{V}O_{2peak}$ ($mL \cdot min^{-1} \cdot kg^{-1}$)	62.6 ± 8.0	65.1 ± 7.2	64.9 ± 9.3
v-4 $mmol \cdot L^{-1}$ ($km \cdot h^{-1}$)	15.6 ± 1.5	16.5 ± 1.8	16.4 ± 2.7
$v\dot{V}O_{2peak}$ ($km \cdot h^{-1}$)	18.0 ± 2.3	18.6 ± 1.8	19.0 ± 3.4
Baseline 3-km TT (s)	643 ± 72	652 ± 76	651 ± 127

Data are presented as group mean ± SD of the raw values. Sum of 7, total 7 sites of skinfolds; $\dot{V}O_{2peak}$, peak oxygen consumption; v-4 $mmol \cdot L^{-1}$, velocity corresponding to lactate at 4 $mmol \cdot L^{-1}$; $v\dot{V}O_{2peak}$, velocity corresponding to $\dot{V}O_{2peak}$; Baseline 3-km TT, initial performance time in 3-km TT; M = males; F = females.

Sport (NSWIS, 32.9°C ± 0.5°C, 56% ± 3% RH). Sydney-based participants assigned to the CONT group completed treadmill sessions in an air-conditioned room (14.4°C ± 1.9°C, 51% ± 13% RH), whereas Canberra-based participants trained in an outdoor covered area (12.6°C ± 4°C, 56% ± 13% RH). In addition to the treadmill sessions, all participants maintained aerobic training in a temperate, normoxic environment during the study to maintain aerobic conditioning. As part of additional testing not described in the current study, each participant undertook a heat tolerance test with 75 min exposure to 33°C at the start and end of each 3-wk period (two in exposure and two in nonexposure, data not reported here). Core temperature was assessed via a temperature probe (Mon-a-therm, Mansfield, MA) inserted 10 cm beyond the anal sphincter, with temperature elevated to an average of 38.3°C ± 0.4°C across all groups (average peak 39.1°C ± 0.5°C), suggesting that the heat dose was sufficient to elicit an adaptive response (25). Performance tests were completed a minimum of 4 d after any heat exposure, and the control group received no more than one 75 min heat exposure within a 7-d period. Thus, this testing was not expected to induce any heat acclimation adaptations (2).

Within 2 wk before the exposure period, participants undertook an incremental treadmill test for assessment of running economy (RE) and $\dot{V}O_{2peak}$. A double baseline measure of Hb_{mass} was assessed during the same period, along with a resting venous blood sample for the measurement of ferritin concentration. Approximately 5 d before the exposure period, performance was assessed via 3-km TT (Baseline). RE, Hb_{mass}, and 3-km TT were repeated immediately (Post) and 3 wk after (3wkP) the exposure period. An additional Hb_{mass} test was conducted 1 wk (1wkP) after the exposure period to further quantify the decay timeline of adaptations (as shown in Fig. 1). All equipment was matched between locations, with participants completing testing and treadmill sessions at the same location and at a similar time of day.

Incremental treadmill test. Participants completed a progressive 4 × 4 min incremental run (0% gradient, 1-min recovery between stages) on a motorized treadmill (Canberra: custom-built motorized treadmill, AIS; Sydney: Payne Treadmill, Stanton Engineering, Girraween, Australia). Starting speed was determined based on participant's ability (between 11 and 17 $km \cdot h^{-1}$) with each stage increased by 1 $km \cdot h^{-1}$. HR (Suunto T6, Vantaa, Finland) and oxygen consumption ($\dot{V}O_2$) were measured continuously throughout the test (Canberra: in-house automated metabolic system as described previously [29]; Sydney: Moxus Modular Metabolic System, AEI Technologies, Pittsburgh, PA). RE was determined as the mean $\dot{V}O_2$ during the last minute of the first two submaximal stages (14). At Baseline testing only, participants completed an incremental run to maximal volitional fatigue for the determination of $\dot{V}O_{2peak}$, corresponding velocity at $\dot{V}O_{2peak}$ ($v\dot{V}O_{2peak}$), and HR_{max} (39).

Performance time trial. In both training locations, 3-km TT values were conducted on a 400-m outdoor athletics track (MONDO synthetic track, Mondo S.p.A., Italy). Participants completed a self-selected warm-up that was replicated at each 3-km TT. Participants were blinded to all pacing and timing information, with verbal feedback given only to notify when one lap remained. Time splits were recorded via hand held stopwatch (Seiko, Tokyo, Japan), with RPE CR-10 (8) collected immediately after. Environmental temperature, RH, and wind speed (Kestrel 3500 Delta T Meter; Nielsen-Kellerman, Boothwyn, PA) were recorded during each 3-km TT (Canberra: 13.5°C ± 4.3°C, 55.2% ± 18% RH, 1.0 ± 1.0 $m \cdot s^{-1}$ wind speed; Sydney: 19.5°C ± 3.4°C, 53.3% ± 16% RH, 1.5 ± 0.9 $m \cdot s^{-1}$). To minimize the effects of diet on physical performance, participants recorded their diet for the 24 h before the Baseline 3-km TT and replicated this diet for each subsequent test. Further, before each 3-km TT, participants completed a series of questions pertaining to muscle soreness, general fatigue, and motivation (5-point Likert scale) (35). In addition, participants were asked the specific question "How important is this upcoming 3-km TT to you?" with answers scaled on a 10-point Likert Scale (1), ranging from 0 (not important at all) to 10 (highly important). Participants also rated "What

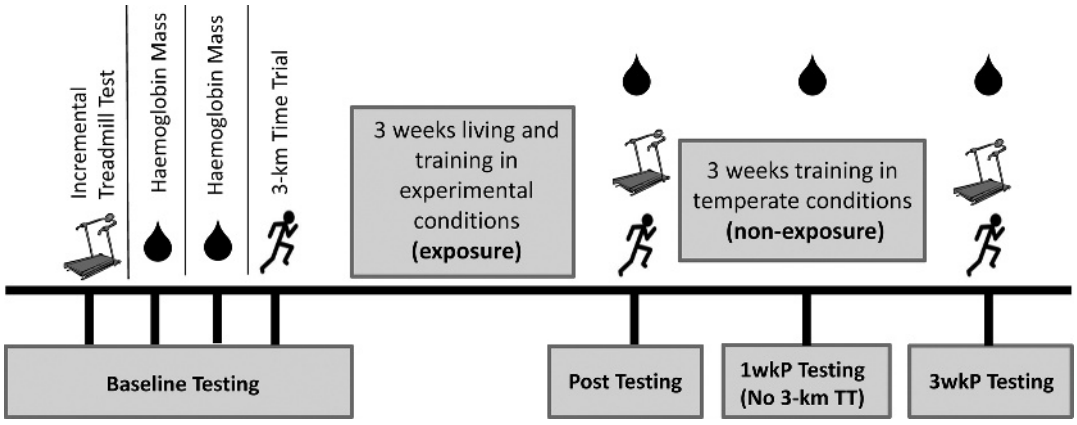

FIGURE 1—Outline of study design, illustrating the exposure and nonexposure training periods. Along with the incremental treadmill testing, Hb_{mass} (CO rebreathing) and a 3-km TT performance were conducted. Testing protocols were conducted after exposure (Post), 1 wk (1wkP), and 3 wk after exposure (3wkP).

percentage [0–100] of your full potential do you think you can run today?"

Training monitoring. Daily TL arbitrary unit was monitored using the session RPE (sRPE) method, calculated as the product of training duration (min) and the mean training intensity (RPE CR-10). Treadmill interval sessions were conducted on motorized treadmills (Canberra: Trackmaster TMX58, Newtown, KS; Sydney: Life Fitness 9500HR, Brunswick Corporation, Lake Forrest, IL), with participants completed a standardized and individualized 20 min warm-up before each session. An outline of the treadmill sessions is presented in Table 2. Interval intensities were matched across all groups based on the percentage of $v\dot{V}O_{2peak}$ as determined from Baseline testing. Intensities ranged from 80% to 100% $v\dot{V}O_{2peak}$, with the only exceptions being sessions 1, 5, and 9, which were conducted as 45 min of continuous running at 65% $v\dot{V}O_{2peak}$. Participants completed their own standardized cooldown and remained in the heat chamber or air-conditioned room until 90 min of exposure was completed. HR was recorded continuously, with sRPE recorded after each session. Participants were allowed to drink water *ad libitum* during training sessions.

Participants recorded all training throughout the study, commencing 2 wk before the exposure period to capture participants' habitual training programs. Participants were instructed to continue with their normal aerobic training during the study in temperate normoxic conditions, in addition to the prescribed three weekly treadmill sessions, and were instructed to replace regular high intensity sessions with the treadmill sessions. As part of this additional aerobic temperate training during the exposure period, all participants reported completing one long duration and one aerobic interval session per week. During the nonexposure period, participants were prescribed an individualized training program based on their previous TL.

Hb_{mass}. Hb_{mass} was measured via the optimized carbon monoxide (CO) rebreathing method (33). Briefly, a CO dose of 1.2 $mL \cdot kg^{-1}$ body mass was rebreathed for 2 min through a glass spirometer. Capillary fingertip blood samples (200 μL) were obtained before CO administration and 7 min after CO inhalation. An average of five blood samples were used for the measurement of percent carboxyhemoglobin (%HbCO) via a CO-oximeter (OSM3, Radiometer, Copenhagen, Denmark), with Hb_{mass} determined as the mean change in %HbCO (9). Duplicate measures were obtained at Baseline on 23 of 26 participants, with the typical error of measurement (TE) for Hb_{mass} calculated at 1.8% (1.4%–2.4%, 90% confidence interval [CI]). The duplicate measures were obtained with a minimum of 48 h between tests (maximum 2 wk), with these values averaged into a single time point for analysis. PV and BV were indirectly calculated by the optimized CO-rebreathing procedure as described previously. All measures were performed by three experienced researchers, with the same tester completing tests on the same participants where possible.

TABLE 2. Treadmill training sessions completed during exposure period.

Session	Description	Pace
1	45-min heat run	65% $v\dot{V}O_2$
2	4 × 5 min with 90 s recovery	80% $v\dot{V}O_2$
3	8 × 90 s at 6% gradient with 90 s recovery	80% $v\dot{V}O_2$
4	6 × 3 min with 60 s recovery	85% $v\dot{V}O_2$
5	45-min run	65% $v\dot{V}O_2$
6	12 × 1 min with 60 s recovery	100% $v\dot{V}O_2$
7	2 × 10 min with 3-min recovery	80% $v\dot{V}O_2$
8	1, 2, 3, 4, 3, 2, and 1 min with 60-s recovery between each interval	90% $v\dot{V}O_2$
9	45-min heat run	65% $v\dot{V}O_2$

All participants completed an individualized 20-min warm-up and 20-min cooldown during each session, except session 1, 5, and 9, completed as 45 min of continuous run in heat (33°C) for all participants.

Blood biochemistry. Venous blood was collected from the antecubital vein 2–3 wk before commencement of the study for the determination of blood ferritin levels. Blood was collected into serum separation tubes (SST; Vacuette®, Greiner Bio-One, Frickenhausen, Germany), centrifuged at 3000 rpm and 4°C for 10 min (2-16 K, Sigma Laborzentrifugen GmbH, Osterode am Harz, Germany), and sent to the laboratory for same day analysis (Sydney: Douglass Hanly Moir Pathology, Macquarie Park, Australia; Canberra: AIS Biochemisty Lab). Sydney samples were assessed on an Abbott i2000 (Abbott Diagnostics, Lake Forest, IL) and Canberra on a Cobas Integra 400 plus analyzer (Roche Diagnostics Ltd., Forrenstrasse, Switzerland). Any participants with ferritin levels <100 $\mu g \cdot L^{-1}$ were provided a daily oral iron supplement to take throughout the duration of the study to maintain adequate iron levels required for accelerated erythropoiesis (Ferrograd C, 325 mg dried ferrous sulfate + 562.4 mg sodium ascorbate; Abbott, Botany, Australia).

Statistical analyses. Data are presented as mean ± SD unless otherwise stated. Data were log-transformed to reduce bias from any nonuniformity of error and assessed for practicality according to magnitude-based inferences (3). Effects were deemed unclear if the CI overlapped the thresholds for both the smallest positive and negative effects (>5%), with clear effects assessed as follows: <1%, almost certainly note; 1%–5%, very unlikely; >5%–25%, unlikely; >25%–75%, possibly; >75%–95%, likely; >95%–99%, very likely; and >99%, almost certainly (12). The smallest worthwhile change in performance was half the typical within-athlete coefficient of variation (CV), or 1.0% in elite runners (13). For measures not directly related to performance, the smallest worthwhile change was calculated as a standardized small effect size (ES = 0.20) multiplied by the pretest between-subject standard deviation (6). ES = 0.20, 0.50, and 0.80 were considered as small, medium, and large, respectively. The TE for outcome measures was calculated from the SD of the change scores divided by the mean and presented as a coefficient of variation (%). Pearson product–moment correlation analyses were calculated to assess the relationship between 3-km TT and physiological parameters. The following thresholds were used to assess the magnitude of correlation (r [90% CI]) between measures: <0.30, trivial

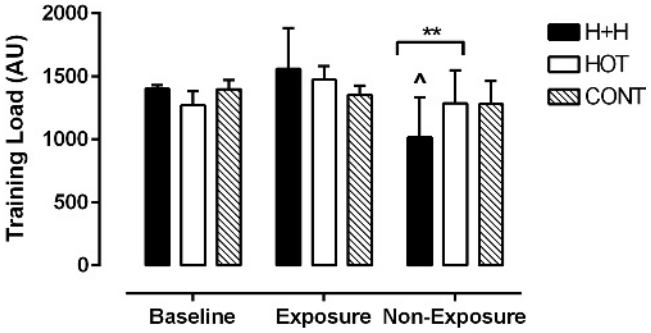

FIGURE 2—Mean ± SD weekly internal TL, expressed as sRPE (RPE × duration in minutes). Data are divided into the 2 wk prior (baseline), 3 wk of environmental stimuli (exposure), and the 3 wk after exposure where all training was conducted in temperate, sea-level conditions (nonexposure). No difference between groups in TL across the study period was found. **Likely within-group reduction in TL in HOT and H + H from exposure to nonexposure. ^Likely between-group reduction from exposure to nonexposure in H + H compared with both HOT and CONT. AU, arbitrary units.

to small; 0.30–0.49, moderate; 0.50–0.69, large; 0.70–0.89, very large; and 0.90–1.00, almost perfect. If the 90% CI overlapped the positive and negative values, the magnitudes were deemed unclear. An *a priori* power analysis was completed using G*Power (G*Power version 3.1.9.2, Universität Kiel, Germany) based on time-trial data obtained from previous similar studies, which demonstrated that 10 subjects per group is the minimum required to achieve a power of 0.8, and as such we recognize the potential limitation of reduced power of this study.

RESULTS

TL. During the exposure period, HOT and H + H received 13.5 h total heat exposure, with control receiving 2.5 h. Both groups had an additional 2.5 h heat during the nonexposure period (heat response testing, data are not presented here). Participants in H + H spent 291.0 ± 13.4 h in normobaric hypoxia, averaging of 13.9 ± 0.6 h·d^{-1}.

During the Baseline period, there were no clear differences between groups in weekly TL as determined from sRPE (H + H vs HOT: ES = −0.44 [−1.22 to 0.34]; H + H vs CONT: ES = −0.17 [−1.04 to 0.70]; HOT vs CONT: ES = −0.21 [−1.05 to 0.630]) (Fig. 2). Across the entire 6 wk of the study, no clear TL differences existed between groups (HOT vs H + H: ES = 0.02 [−0.76 to 0.80]; CONT vs H + H: ES = 0.20 [−0.63 to 1.02]; HOT vs CONT: ES = −0.11 [−0.93 to 0.71]). However, when comparing the exposure to nonexposure period, HOT and H + H had a within-group reduction in TL during the nonexposure period (HOT: ES = −0.31 [−0.53 to −0.08] likely; H + H: ES = −1.75 [−2.12 to 1.37] most likely; CONT: ES = −0.08 [−0.5 to 0.33] unclear). During the same period, H + H had a very likely TL reduction in H + H compared with both CONT and HOT (H + H vs CONT: ES = −1.26 [−2.00 to −0.53]; H + H vs HOT: ES = −0.8 [−1.19 to −0.40]), with unclear differences between HOT and CONT (ES = −0.26 [−0.80 to 0.28]).

Time-trial performance. Improvement in 3-km TT performance occurred only in HOT, with a likely faster completion time by −3.3% ± 1.3% (mean ± 90% CI) from Baseline to 3wkP (652 ± 76 vs 629 ± 67 s; ES = −0.26 [−0.36 to −0.16]; Fig. 3). This improvement was possibly greater when compared with both H + H (643 ± 72 vs 639 ± 74 s; ES = −0.24 [−0.40 to −0.08]) and CONT (651 ± 118 vs 649 ± 127 s; ES = −0.19 [−0.32 to −0.07]) (Fig. 3). There were no substantial changes from Baseline in performance in any group at POST, and also in H + H and CONT at 3wkP. There were no clear between- or within-group differences in RPE after each respective 3-km TT.

Pre–time-trial questionnaires. The perceived capacity of H + H to fulfill their 3-km TT performance potential was likely reduced from Baseline to Post (ES = −0.48

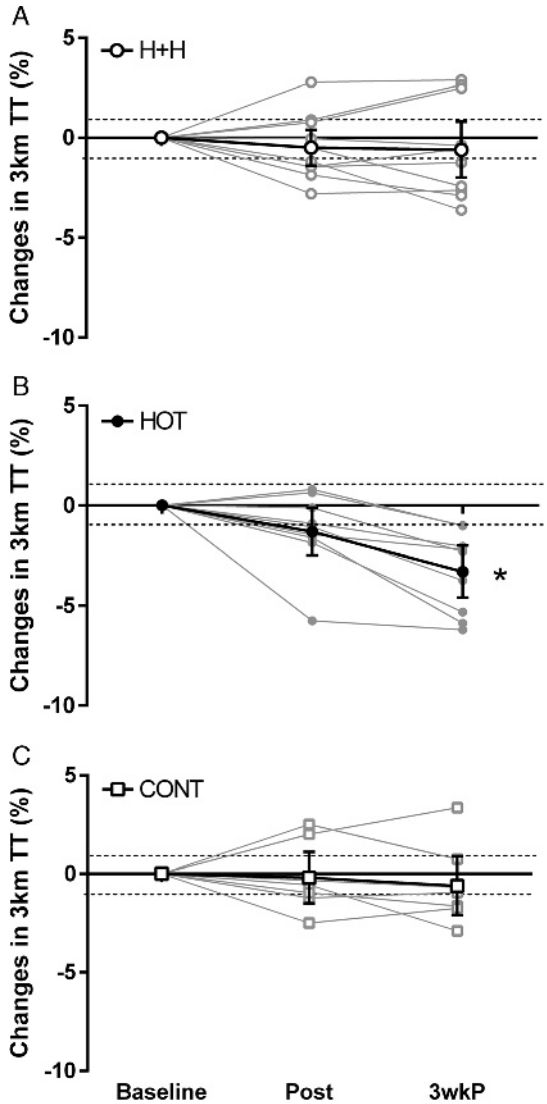

FIGURE 3—Change in 3-km TT performance expressed as a percent change (%) from Baseline ± 90% CI for H + H (A), HOT (B), and CONT (C). *Likely within-group difference from Baseline.

[−1.02 to 0.06]), resulting in a likely greater reduction compared with HOT at Post (ES = −0.85 [−1.70 to 0.00]) and CONT at 3wkP (ES = −1.53 [−3.04 to 0.01]). Motivation likely increased in HOT from Baseline to Post (ES = 0.43 [−0.06 to 0.92]) and in CONT from Post to 3wkP (ES = 0.20 [−0.19 to 0.60]); however, it was likely reduced in H + H during the same period (ES = −1.12 [−2.12 to −0.12]). This resulted in very likely reduction in motivation from Post to 3wkP in CONT compared with H + H (ES = 1.12 [0.24–1.99]).

Perceived importance of the 3-km TT likely increased both in HOT (ES = 0.45 [−0.17 to 1.08]) and H + H (ES = 0.46 [−0.09 to 1.01]) from Baseline to Post but was unclear in CONT. Although perceived importance remained likely elevated in HOT until 3wkP (ES = 0.49 [0.16–0.82] vs Baseline), it decreased from Post to 3wkP in H + H (ES = −0.38 [−0.72 to 0.05]). General fatigue was likely reduced from Post to 3wkP in HOT (ES = −0.43 [−0.98 to 0.11] and possibly reduced in CONT (ES = −0.16 [−0.46 to 0.15]). However, H + H had likely greater increase in general fatigue both from Post to 3wkP (ES = 0.54 [0.09–0.99]), as well as Baseline to 3wkP (ES = 0.60 [0.02–1.18]). As a result, 3wkP fatigue was likely lower in both HOT and CONT when compared with H + H at both Baseline and Post (CONT vs H + H: ES = −0.76 [−1.32 to −0.20] vs Post; ES = −0.83 [−1.50 to −0.16] vs Baseline; HOT v H + H: ES = −1.06 [−1.76 to −0.35] vs Post; ES = −0.69 [−1.43 to 0.05] vs Baseline). All other between- and within-group differences were unclear.

RE. All RE between- and within-group differences were trivial, unlikely, or unclear. HR was likely reduced in all groups when comparing Baseline to Post (expressed as a percentage of maximum HR), with no clear between-group differences (HOT: 79.4% ± 4.7% vs 76.8% ± 4.6% ES = −0.49 [−0.90 to −0.07]; H + H: 86.0% ± 3.6% vs 82.6% ± 5.2% ES = −0.57 [−1.07 to −0.07]; CONT: 84.8% ± 3.1% vs 82.8% ± 3.8% ES = −0.49 [−1.01 to 0.03]). HR was possibly further reduced at 3wkP in H + H and CONT and maintained in HOT. As a result, all groups had a reduced submaximal HR from Baseline to 3wkP (HOT: 79.4% ± 4.7% vs 76.6% ± 5.2%, ES = −0.52 [−1.04 to 0.00], likely; H + H: 86.0% ± 3.6% vs 81.0% ± 6.2%, ES = −0.85 [−1.46 to −0.24], very likely; CONT: 84.8% ± 3.1% vs 81.8% ± 3.8%, ES = −0.72 [−1.21 to −0.24], very likely).

Hematology. PV increased by 3.8% ± 6.0% in HOT during the exposure period (ES = 0.13 [−0.07 to 0.34]), with this change possibly greater when compared with both H + H (ES = 0.20 [−0.12 to 0.52]) and CONT (ES = 0.17 [−0.13 to 0.47]) (Fig. 4). At 1wkP, PV remained likely elevated in HOT compared with H + H (ES = 0.68 [−0.09 to 1.46]). All differences in HOT and H + H were deemed unclear by 3wkP, and all CONT time course differences throughout the study duration were unlikely or trivial. BV increased in HOT by 3.3% ± 3.9% (ES = 0.11 [−0.02 to 0.24]) during the exposure period, which was possibly greater when compared with H + H during the same period (ES = 0.15 [−0.05 to 0.35]). However, all other within- and between-group differences were unclear or trivial.

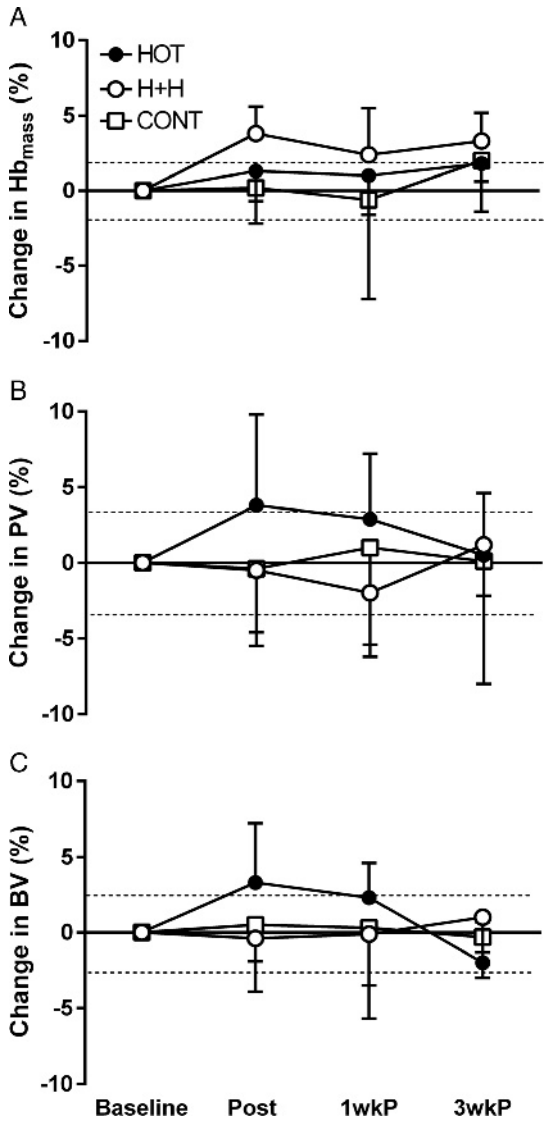

FIGURE 4—Percent change (%) from Baseline in Hb_{mass} (A), PV (B), and BV (C). Groups are indicated by the symbols HOT (•), H + H (○), and CONT (□).

Hb_{mass} was increased by 3.8% ± 1.8% in H + H during the exposure period (784 ± 197 vs 813 ± 203 g; ES = 0.14 [0.08–0.21]) and remained elevated from Baseline by 3.3% ± 1.9% at 3wkP (ES = 0.12 [0.05–0.19]). This change was greater than the TE from Baseline. However, all within- and between-group differences were trivial, unlikely, or unclear. There were no clear correlations in any group between 3-km TT performance and PV, BV, Hb_{mass}, HR, or RE.

DISCUSSION

This study investigated the effects of 3 wk of independent heat interval training or LHTL hypoxic exposure combined with heat interval training in well-trained middle distance runners. The main finding was that 3-km TT performance was only improved 3 wk after HOT training, despite small but positive physiological adaptations (i.e., PV) lasting up to

1 wk postexposure. Despite H + H demonstrating positive hematological adaptations (i.e., Hb_{mass}) higher than that of temperate training alone, there were no performance improvements. Accordingly, the initial hypothesis that LHTL combined with heat training would be of greatest performance benefit was not supported.

The 3-km TT performance was improved in temperate conditions after heat interval training in all HOT participants at 3wkP. This adds further support to previous research indicating enhanced temperate performance after heat exposure (18,26,36). A novel finding was that the performance peak in all participants occurred 3 wk after heat exposure, but combining LHTL and heat training did not further enhance 3-km TT performance. Direct comparison to previous studies investigating combined LHTL and heat (5) or studies that did not find enhanced temperate performance after heat training (15,16) should be done so tentatively. This is due to a lack of control group (5,26), the absence of TL data before or during the study (18), the assessment of performance within 2 wk of exposure (15,16), and/or the high number of fatiguing maximal tests in a short time frame, which could have reduced the athletes motivation to perform (15). The current protocol of intermittent heat exposure for a 3-wk period, with several weeks of temperate training before competition, is a practical protocol that can be used to enhance performance in well-trained endurance athletes.

It is apparent that heat interval training provides greater 3-km TT performance improvements than combining with LHTL, although physiological explanations for these observations remain elusive. Indeed, there was no clear relationship between any of the physiological measures and 3-km TT performance. As further exploration, heat acclimation can induce several cardiovascular (24) and thermoregulatory (32) adaptations to tolerate heat stress, including increased PV (18), $\dot{V}O_{2max}$, RE, and power at lactate threshold (18). These adaptations have been suggested to be ergogenic in both hot (18,26) and temperate conditions (18). We suggest that the 270-min·wk^{-1} heat exposure (i.e., 3 × 90 min sessions per week) was sufficient to increase in PV in HOT (by 3.8% ± 6.0%), although only until 1wkP, and not at 3wkP when 3-km TT performance improved. By contrast, PV values in both H + H and CONT were not increased by more than 1.2% above baseline values at any time during the study, despite H + H receiving the same heat dose as HOT. Such absence of PV expansion in H + H contrasts with previous combined heat and hypoxic findings (5) and warrants further exploration.

As athletes with lower training status have a greater adaptive potential than highly trained athletes (40), it is possible the early season training status of athletes in previous combined heat and LHTL research (5) contributed to the greater PV increases compared more established training status of the current participants. The suggestion of an optimal PV volume to enhance performance (7) may provide background as to why performance in HOT did not occur until PV values returned to normal at 3wkP. In addition to training status, the PV response in the present study may also relate to the nature and dose of the environmental stimuli. Hypoxia has been shown to induce hemoconcentration and reduce PV (31). The heat dose in the present study was sufficient to prevent PV reduction in H + H; however, it was unable to match the PV increase in HOT. Thus, heat stimuli seem to prevent hypoxic induced hemoconcentration; however, it may be that a greater dose of heat stimuli is required to compensate PV beyond the losses from hypoxia. Further research is required to assess if any other heat training benefits could be negated due to hypoxic exposure. However, on the basis of the current data, we recommend that when combining heat and hypoxia, a greater heat dose may elicit PV responses equivalent to heat exposure alone.

RE has been shown to be improved with endurance performance and has been reported to improve after simulated LHTL exposure in elite middle distance runners (30). In the present study, there were only trivial improvements in RE in all training conditions. Moreover, similar to previous research (5), submaximal HR remained unchanged between groups. Although RE has been reported to be increased immediately after LHTL alone (14), there does not seem to be any benefit of concurrent heat and altitude or heat alone on RE. Accordingly, the improvements observed in 3-km TT performance observed in the heat group cannot be explained by changes in RE.

A recent meta-analysis has shown that Hb_{mass} increases by ~1.1% per 100 h of altitude exposure and remains elevated by 3.3% for up to 20 d after exposure (10). Similarly, the present study revealed that H + H had a 3.8% ± 1.8% increase in Hb_{mass} with ~290 h of hypoxic exposure, whereas no increases occurred in HOT and CONT. Despite H + H having an increase in Hb_{mass}, the lack of performance changes in H + H supports previous research showing that the changes in Hb_{mass} from the hypoxic exposure have minimal impact on 3-km TT performance (27).

Considering no associations were observed between the measured physiological adaptations and 3-km TT performance, other unmeasured physiological adaptations, not limited to enhanced thermoregulatory regulation, increased cardiac and skeletal muscle metabolic efficiency (21), or nonphysiological factors may provide explanations for the observed performance responses. The uncoupling of performance and physiology changes is not uncommon in trained individuals (27), and factors such as perception of effort, motivation, and fatigue can contribute to overall endurance performance outcomes (23). At the 3-km TT at 3wkP, fatigue was increased in H + H, despite TL being reduced during the nonexposure period. At the same time point, motivation and perceived time-trial importance was reduced in H + H but increased in HOT and CONT. It is likely that the combined psychophysiological changes in the HOT underlie the observed performance changes. Although speculative, the combined perceptions of increased motivation and importance of the 3-km TT garnered HOT contributed positively to improved 3-km TT performance. Physiological adaptations to training were mostly trivial in CONT, whereas

any beneficial effect of the physiological adaptations associated with the H + H may have been minimized by a negative psychological response. Potentially, the combined stress of heat and hypoxia prevented appropriate recovery from the hard training sessions in the heat, thus lingering to suppress performance outcomes. Although it could be argued that the combined stress of heat and hypoxia may have been reduced if the treadmill sessions were matched for cardiovascular strain rather than absolute workload (%v$\dot{V}O_2$), the absolute TL provides a more practical application of training prescription in trained individuals, particularly because of the intermittent nature of the sessions. Future investigations incorporating a staggered or reduced combination of heat and hypoxia are required (i.e., reduction in number of heat sessions or an incremental hypoxic dose). These findings illustrate the importance of considering both physiology and psychological aspects when aiming to elicit performance enhancements in well-trained athletes.

LIMITATIONS

Despite the previously mentioned findings, some limitations should be acknowledged. Although participants were blinded to the specific temperature and oxygen concentrations during the study, they were unable to be blinded to their assigned environmental conditions. Furthermore, the heat and hypoxic environmental stimuli in the study were simulated and, therefore, may not be replicated in natural heat or hypoxic environments. Specifically, physiological adaptations resulting from hypobaric hypoxia or simulated normobaric hypoxia are suggested to differ (19); however, recent evidence suggests no difference in $\dot{V}O_{2max}$ or 3-km run time-trial (28). However, we recommend future research to investigate if similar results would occur in athletes living and training a natural environment. Another limitation is that we only investigated 3-km TT running performance benefits in a temperate environment. The physiological adaptations resulting from heat and LHTL exposure often enhance athlete's aerobic capacity. To assess this, future research could assess endurance performance for a longer duration in which there is a greater reliance on energy provision from aerobic sources.

CONCLUSIONS

In summary, 3 wk of interval training in a hot environment may enhance 3-km TT performance in a temperate environment in the weeks after exposure. The present results showed that although adding LHTL to heat interval training can elicit a hematological response, these physiological changes do not result in improved 3-km TT performance. Collectively, these findings indicate that combining LHTL with heat exposure does not provide additional benefit over heat training alone, and the incorporation of heat into a training camp may be a simple approach to improving athletic performance. However, factors such as psychology of the athlete, dose of stimuli, environment, and training status should be considered when including heat or hypoxia as part of an athlete's training program.

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