EFFECT OF SHORT-TERM HEAT ACCLIMATION TRAINING ON KINETICS OF LACTATE REMOVAL FOLLOWING MAXIMAL EXERCISE

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Abstract

AIM—Heat acclimation (HA) evokes numerous physiological adaptations, improves heat tolerance and has also been shown to enhance lactate (LA) responses during exercise, similar to that seen with endurance training. The purpose of this study was to examine whether HA improves the body's ability to remove LA during recovery following maximal exercise.

METHODS—Ten healthy men completed two trials of maximal treadmill exercise (PRE- and POST-HA) separated by 5 days of HA. Each day of HA consisted of two 45 minute periods of cycling at ~50% VO_{2max} separated by a 15min rest period in an environmental chamber (T_{db} 45°C, RH 20%). In PRE-/POST-HA trials, venous blood was collected during 60 minutes of recovery to determine LA concentrations and removal kinetics (A2: amplitude and y2: velocity constant) using bi-exponential curve fitting.

RESULTS—Physiological adaptation to heat was significantly developed during HA, as evidenced by end-exercise T_{re} (DAY 1 vs. 5) (38.89±0.56 vs. 38.66±0.44 °C), T_{sk} (38.07±.51 vs. 37.66±.48 °C), HR (175.0±9.9 vs. 165.0±18.5 beats·min^{-1}), and sweat rate (1.24 ±.26 vs. 1.47 ±. 27 L·min^{-1}) (p<.05). However, there was no significant difference in either LA concentrations (LA_{0min}: 8.78±1.08 vs. 8.69±1.23; LA_{peak}: 10.97±1.77 vs. 10.95±1.46; and La_{60min}; 2.88±.82 vs. 2.96±.93 mmol·L^{-1}) or removal kinetics (A2: −13.05±7.05 vs −15.59±7.90 mmol.L^{-1} and y2: . 02±.01 vs .03±.01 min^{-1}).

CONCLUSION—The present study concluded that, while effective in inducing thermo-physiological adaptations to heat stress, short-term HA does not improve the body's ability to remove LA following maximal exercise. Therefore, athletes and workers seeking faster LA recovery from intense physical activity may not benefit from short-term HA.

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Keywords

heat stress; heat adaptation; lactate recovery; lactic acidosis; performance

Introduction

The exchange and removal of lactate (LA); consequently, the return of H^+ close to baseline levels, during recovery is critical for repetition of intense exercise [1–3]. LA can be removed into areas of high cellular respiration by metabolic transporters [4], such as monocarboxylate transporters (MCT-1) [5]. LA can also be exchanged intracellularly for substrate use in the mitochondria, or exchanged from cell-to-cell for substrate use in other tissues (e.g. from the skeletal muscle to the myocardium) [6]. The physiological process of LA exchange and removal over time, termed LA kinetics, has been studied at length since 1933 [7]. Investigations of LA kinetics during exercise have shown that LA production exceeds LA removal over the course of progressive exercise until peak values are reached [8, 9]. Thereafter, LA is removed in an exponential manner during recovery, yet remains elevated above baseline for about 60 minutes [2, 10–12].

LA exchange and removal ability is considered to be a valid criterion measure of aerobic performance, and can be improved with various training techniques, such as endurance training [11]. The enhancement of LA kinetic parameters through endurance training has been studied extensively in sedentary and athletic populations, both during [13–15] and after exercise [16–19]. Initially, the relative merits of endurance training were reported to elicit no significant effect on LA recovery [16]. However, more recent studies report that exercise intensity may alter the effects of endurance training on the mechanisms for LA kinetics [17–19]. For example, decreased LA production at lower intensities (<60% VO_{2Max}) and increased LA removal at higher intensities (>60% VO_{2Max}) are suggested to be the primary mechanisms for the LA levels observed at these differing exercise intensities [14]. Regardless of the mechanism, endurance training improves LA parameters, which can improve a person's ability to recover from intense exercise in order to perform repeated bouts.

Heat stress has been documented to attenuate exercise performance, and increase cardiovascular strain and a risk of heat injury and illness [20, 21], with a significantly elevated utilization of skeletal muscle glycogen and thus high plasma LA levels [20]. Therefore, heat stress and exercise-induced lactic acidosis have been labeled limiting performance by-products of prolonged, intense exercise [22]. A popular training strategy to prepare physical performance in heat stress conditions is heat acclimation (HA) training. Previous studies implementing HA observed improved thermoregulatory adaptions such as improved sweat production, reduced cardiovascular strain, and better heat tolerance [23, 24] that combined enhance exercise performance in the heat. HA has also been shown to decrease LA accumulation in both cool and hot conditions [25], decrease plasma LA concentrations at a given sub-maximal exercise intensity [20], and improve the LA threshold which is widely used as a marker to indicate exercise intensity and predict aerobic performance [11]. Although the

responsible mechanism(s) are debatable and uncertain, HA has been shown to elicit various physiological and metabolic adaptations that improve LA responses during exercise.

LA removal is an important marker for those who often perform multiple bouts of intense physical activity in the heat. By understanding the performance-limiting factors (hyperthermia and lactic acidosis) and available training methods, one can implement a program to augment physical performance. While endurance training has been shown to enhance LA removal abilities during recovery, the question remains whether HA training can elicit similar benefits. To our knowledge, no study has investigated the effects of HA on LA removal kinetics during exercise recovery. We hypothesized that HA training would enhance LA removal abilities (y2) during the recovery period after intense exercise. This study was undertaken utilizing the iterative method of bi-exponential modeling to examine the influence of HA on LA removal kinetics in repeated measures (within-subject) design.

Materials and Methods

Subjects

Ten healthy, non-smoking, males (age: 22.6 ± 2.6 years, mass: 78.3 ± 10.1 kg, height: 183 ± 0.7 cm, BMI 23.3 ± 2.6 kg·m^2), volunteered to participate in the study. Subjects were excluded from the study if they had an aerobic capacity (determined by VO$_{2max}$) less than 50 ml·kg·min^{-1}, smoked, or had a positive history of cardiovascular or metabolic diseases. Subjects were informed of the study procedures and associated risks, and written informed consent was obtained before the initiation of their study participation. The study protocol was reviewed and approved by the National Institute for Occupational Safety and Health (NIOSH) Human Subject Review Board (HSRB).

Procedures

Subjects undertook two maximal exercise test trials (PRE-/POST-HA) separated by five consecutive days of HA training. POST-HA was performed 3 days after the completion of HA training to assure that the subjects were fully recovered from the intervention.

Subjects reported to the laboratory between 7:00 and 8:00 AM after having been instructed to avoid strenuous exercise for at least 24 hours and caffeine drinks on the morning of testing, but to eat a light breakfast. To minimize circadian variation of the study variables, all experimental trials were performed at the same time of the day for each subject. As a general procedure upon arrival at the laboratory, subjects provided a mid-stream urine sample for dehydration assessment, drank an assigned amount of bottled water ($5mL \cdot kg^{-1}$ body weight) for hydration purposes, and underwent a brief medical checkup performed by a licensed physician. Subjects wore an athletic T-shirt, shorts, and athletic shoes for all seven trials.

Maximal Exercise Protocol

Following the initial medical exam, subjects rested quietly for baseline measurements including resting blood pressure, heart rate (HR), and a 12-lead electrocardiogram. Baseline blood samples were collected from the antecubital vein through a 22 gauge catheter which remained in situ during exercise and the subsequent recovery. Following baseline

measurements, subjects were connected by a mouthpiece to a standard metabolic cart (Vmax Spectra System, VIASYS, Yorba Linda, CA) and then started a three-minute warm-up (4.8 km·h^{-1}, 0% grade) on the treadmill. Thereafter, they performed a ramped Bruce protocol until volitional fatigue. During the exercise, cardiopulmonary responses were continuously monitored and Rating of Perceived Exertion (RPE) was recorded during the last 30 seconds of each stage of the protocol. After reaching volitional fatigue, treadmill speed was reduced and subjects completed three minutes of treadmill walking for active recovery, after which they sat quietly on a chair for the remaining period of recovery. Blood draws commenced immediately at the end of exercise (0 minute) and during one hour of recovery (minutes 1,2,3,4,5,7,9,11,13,15,20,25,30,45,60). Sterile saline solution (0.9% sodium chloride) was infused between blood draws (>2min) to prevent clotting. An additional amount of blood (>1mL) was discarded prior to the subsequent blood sampling to avoid misinterpretation of LA concentrations related to residual blood and saline solution in the catheter tube.

HA Protocol

Following the initial medical exam, subjects were instrumented with measurement sensors in an environmental chamber maintained at thermoneutral conditions (T_{db} 20°C, RH 30%). Rectal temperature (T_{re}) was measured by placing a rectal probe (REF-4491, YSI temperature, Dayton, OH) 13cm beyond the anal sphincter. Skin temperature sensors (T-type copper/constantan, Concept Engineering, Old Saybrook, CT) were affixed onto four body sites (chest, shoulder, thigh, and calf) to monitor and calculate weighted mean skin temperature (T_{sk}) according to Ramanathan [26]. Subjects also wore a previously validated portable breath-by-breath gas exchange analyzer (Model K4 b^2, Cosmed Rome, Italy) [27] with a Polar HR transmitter (Polar Electro Inc, Lake Success, NY) strapped on the chest to monitor exercise intensity and HR response during exercise. RPE (6–20 scale) and heat perception (HP) (1–7 scale; 1: Slightly cool – 7: Extremely hot) were also recorded every 15 minutes during exercise. After temperature sensors were applied, subjects quietly rested for stabilization and baseline measurements prior to performing the HA protocol, which consisted of two 45 minute periods of cycling separated by a 15 minute rest period in hot/dry environmental conditions (33°C WBGT; T_{db} 45°C, RH 20%). Subjects cycled using an electronically braked cycle ergometer (VIAsprint™ 150P, CareFusion, Hochberg, Germany) at approximately 50% of their VO_{2max}. Exercise intensity, in terms of the external workload, ranged between 100 and 135 Watts, at which subjects were asked to cycle between 60 and 80 RPM. Subjects were given a controlled amount of bottled water (5mL·kg^{-1} body weight) during the 15 minute rest period; however, no additional hydration was permitted during the test. Exercise was terminated if subjects expressed volitional fatigue or their core temperature exceeded 39°C. When this occurred, subjects were instructed to remain in the heat chamber and sit quietly for the remaining duration of the test for passive heat exposure. Whole body sweat rate (SR) was determined by a difference in pre- and post-test nude body weight measured nearest 1g on a calibrated scale (model: Electronic scale-4450, GSE, Farmington Hills, MI).

Blood Sampling and Analysis

Venous blood samples for LA measurements were collected in 2mL lithium heparin tubes at predetermined time points, as described above. It should be noted that varying methods

(arterial, capillary, venous) of blood sampling alter test results. Venous blood samples have lower LA concentrations than arterial and capillary blood samples [28]. Blood LA concentrations were analyzed in duplicate, using a standard laboratory LA analyzer (Model 2300; Yellow Springs Instrument Co., Yellow Springs, OH).

For each individual PRE-HA and POST-HA, a LA recovery curve was fitted to the below bi-exponential equation [1, 10] by an iterative nonlinear regression technique using R programming language. All inferences were performed using variance weightings and each of the curve fits were iterated with a deviation tolerance of $1e^{-6}$. The start values of the velocity constants (y1, y2) were fitted using log regression on the first 7 values and the last 6 values, respectively.

$$LA(t) = LA(0) + A1\,(1 - e^{-y1}) + A2\,(1 - e^{-y2})$$

Where, LA(0) and LA(t) (mmol·L^{-1}) represent the venous LA concentrations immediately after exercise and at time t (min) of the recovery period, respectively. The parameters A1 and A2 (mmol·L^{-1}) are the amplitudes of the two exponential functions; and y1 and y2 (min^{-1}) are their respective velocity constants. These velocity constants describe the exchange (y1) ability of LA between the previously worked muscle and the blood, and the body's overall removal (y2) capacity of LA during recovery. Venous blood sampling makes y2 the only physiological focus for analysis in this study [10].

Blood for catecholamine measurements was sampled in 4ml EDTA tubes at baseline and 0 minutes. After ten minutes of centrifugation at 3,500 RPM, the aliquoted plasma sample was stored at −40°C for later analysis. Catecholamine levels were determined via enzyme-linked immunosorbent assay (Human Bi-CAT ELISA, ALPCO, Salem, NH). Standards, controls, and sample absorbance were analyzed and read in duplicate using a microplate reader (VersaMax, Molecular Devices LLC, Sunnyvale, CA). The assay sensitivity was 50 pg·mL^{-1} and 10 pg·mL^{-1} for norepinephrine and epinephrine, respectively. Intra-assay coefficient of variation (Precision) was 9.8% for norepinephrine and 6.9% for epinephrine, respectively.

Statistical Analyses

All experimental data were calculated for descriptive statistics (mean ± SD). Physiological responses (T$_{re}$, T$_{sk}$, SR, and HR) measured during HA (Day 1 and 5), and catecholamine levels measured during PRE- and POST-HA were compared using two-way repeated measures ANOVA (condition × time) to determine if HA elicited a significant physiological adaptation. In addition, the end-exercise HP and RPE during HA were compared using Paired samples t-test. A LA recovery curve following PRE- and POST-HA was also compared using two-way repeated measures ANOVA (condition × time) at four LA measurement points (baseline, 0 minute: end-exercise, peak, and 60 minutes) while the parameters of the bi-exponential function were compared using Paired samples t-test. In all ANOVA analyses, Greenhouse-Geisser correction for assumption of sphericity was adopted and a post-hoc pair-wise comparison with LSD adjustments was carried out for a significant

F-ratio. A statistical significance was accepted when p<.05 and all analyses were performed using the Statistical Package for Social Sciences (v19.0, IBM, Somers, NY).

Results

HA

There was a significant main effect of condition (F=5.36, p=.04) and time (F=112.64, p<.001) on T_{re}. T_{re} significantly increased throughout the 105 minutes of HA on both DAY 1 and 5, but final T_{re} was significantly lower on DAY 5 (38.66±0.44 °C) than on DAY 1 (38.89±0.56 °C) (Figure 1a). There was no significant interaction between condition and time on T_{re} (F=.44, p=.87).

There was no significant main effect of condition (F=4.80, p=.05) on T_{sk}, though the final T_{sk} was significantly lower on DAY 5 (37.66±.48 °C) than on DAY 1 (38.07±.51 °C) of HA (Figure 1b). There was also a significant effect of time (F=118.33, p<.001) on T_{sk} which increased throughout the 105 minutes of exercise. There was no significant interaction between condition and time on T_{sk} (F=2.63, p=.11).

There was a significant main effect of condition (F= 5.49, p=.04) and time (F= 87.62, p<.001) on HR. Final HR was significantly lower on DAY 5 (165.0±18.5 beats·min^{-1}) than on DAY 1 (175.0±9.9 beats·min^{-1}) (Figure 1c). There was no significant interaction between condition and time on HR (F=2.63, p=.11).

There was a significant increase in SR from DAY 1 (1.24 ±.26 L·min^{-1}) to DAY 5 (1.47 ±.27 L·min^{-1}) of HA (t=−5.02, p<.001) (Figure 1d).

Subjects also exhibited significant improvements in subjective perceptions, as shown by significantly lower HP on DAY 5 (5.0±1.05) than on DAY 1 (5.70±.67) of HA (t=2.689, p=.025) (Figure 1e), and significantly lower RPE on DAY 5 (13.8±2.93) than on DAY 1 (15.8±2.29) of HA (t=3.586, p=.006) (Figure 1f).

Maximal Exercise

There was no significant difference in VO_{2max} between PRE-HA (61.3±5.4 ml·kg·min^{-1}) and POST-HA (60.6±4.17 ml·kg·min^{-1}) (t=.76, p=.46).

There was no significant main effect of condition (F=.11, p=.73), but a significant effect of time (F=198.45 p<.001) on LA (Table 1). There was also no significant interaction between condition and time on LA (F=368, p=.72).

There was no significant difference in measured parameters of LA kinetics during the 60 minutes recovery between PRE- and POST-HA (p>.05) (Table 2).

There was no significant main effect of condition on both epinephrine (F=.72, p=.41) and norepinephrine (F=.04, p=.84), but there was a significant effect of time on both epinephrine (F=25.74, p<.01) and norepinephrine (F=51.64, p<.001) from the baseline to the end of exercise (Figure 2).

Discussion

The present results showed that 5 days of HA training under hot-dry conditions (33°C WBGT; Tdb 45°C, RH 20%) effectively elicited both physiological and subjective adaptations to heat. This was evidenced by significantly lower end-exercise Tre, Tsk, and HR with improved SR, HP, and RPE responses on DAY 5 than DAY 1. However, as opposed to our hypothesis, these physiological adaptations did not result in significant changes in LA concentrations or kinetics of LA removal as analyzed by means of bi-exponential curve fitting (Freund and Gendry, 1978; Thomas et al., 2005) during recovery from maximal exercise.

The effects of HA on LA response are somewhat inconsistent with previous findings that reported significant changes in LA concentrations during and at the end of exercise, though some of the inconsistency may be related to methodological differences in the present study. The studies with a longer period of HA (7–10 days) [20, 25] showed a significant reduction in plasma and skeletal muscle LA concentrations during sub-maximal exercise (~70% VO_{2max}). These studies suggested that HA training may be responsible for a reduction in glycogenolysis and an elevation in blood flow (due to increased cardiac output) to the working muscle and splanchnic area, resulting in LA production and removal, respectively, thereby reducing LA concentration at a given exercise intensity. Lorenzo [24] also reported that trained cyclists, after 10 days of HA, showed a delayed LA threshold with increased VO_{2max} (approximately 3.3 ml·kg·min^{-1}) during exercise in a cool environment (13 °C, 30% RH), and speculated previously discussed HA-induced adaptations together with increased plasma volume (approximately 6.5%) as possible mechanisms for the improvement of the LA response to exercise.

It must be noted these previous studies investigated the effects of HA on LA concentration during or immediately after sub-maximal exercise; while the present study focused on kinetics of lactate removal during 60 minutes of recovery from maximal exercise. Indeed, HA may alter mechanisms influencing LA exchange and removal to a greater degree during sub-maximal exercise up to the lactate threshold than LA removal after exercise. This speculation is not verified by the present findings due to the absence of LA measurements during exercise, but may be partially supported by the previous study [29] that showed HA diminished LA concentration during a sub-maximal exercise, but no significant improvement in end-exercise LA concentration. Moreover, it was also shown that LA concentration is significantly lower in trained cyclists than in untrained subjects during submaximal exercise, but LA concentrations in the two groups became similar when exercise intensity increased to maximal levels beyond the LA threshold [30]. Therefore, a training effect on LA response to exercise may be more prominent during exercise than during recovery.

Catecholamine response to exercise is another measure to explain training-induced alteration in LA concentration as it is believed that LA kinetics are governed by the sympathetic nervous system [13], due in part to the direct linear relationship that exists between catecholamine and LA concentration during exercise [30, 31]. In the present study, epinephrine and norepinephrine levels were significantly elevated from baseline to the end-exercise, but

showed no significant changes between PRE- and POST-HA (Figure 2). This observation is in contrast to previous studies [20, 29] reporting HA reduced catecholamine levels during and at the end of exercise and was thought to be attributable to reduced muscle glycogenolysis and the concomitant decrease in LA concentration at a given sub-maximal intensity (50–70% VO_{2max}). At maximal exercise intensity however, the results are conflicting, with some studies showing improved catecholamine response [32, 33] and others reporting no differences between trained and untrained subjects [30]. However, it is not certain from these studies if diminished catecholamine secretion in response to sub-maximal exercise, would remain similar at maximal exercise and subsequently suppress LA release from working muscles and/or accelerate LA removal during recovery. Regardless of the relationship established between catecholamines and LA during exercise, their correlation during the recovery period is suggested to be less prominent [30].

Among the many fates of LA, oxidation is the primary means of LA removal during exercise and into the early phase of recovery from intensive exercise [4–6, 11, 18], accounting for 70–75% of LA removal and gluconeogenesis [6]. Endurance training has been known to improve LA oxidation by enhancing MCT-1 concentration, a mediator of lactate metabolism transporting LA to the mitochondria [14, 15, 34] that is found to be strongly linked with y2 of lactate kinetics [5]. In our study, we purposely differentiated the mode of exercise between PRE- and POST-HA on a treadmill and HA training on a cycle ergometer to isolate HA effects on LA kinetics from an endurance training effect; therefore, it is not surprising that VO_{2max} was attained similarly between PRE- and POST-HA. However, previous studies [18, 19] that showed improved kinetics of LA removal, specifically y2, also reported a concomitant improvement in aerobic capacity (e.g. VO_{2max}) resulting from endurance training (4 weeks cycling at 60–80% VO_{2max} [18] and 6 weeks continuous or intermittent running at 60–100% VO_{2max} [19]) similar to those who observed a lower LA concentration with improved VO_{2max} after HA training [24]. These findings implicate that training-induced alterations in physiological mechanisms affecting the body's ability to remove LA may be induced only when the training stimulus is more chronic and sufficient enough to improve aerobic capacity. Nonetheless, it is also possible that a relatively short period (5 days) of HA training in the present study may have not been sufficiently long enough to evoke some of the postulated physiological adaptations beyond thermoregulatory adaptations, such as decreased glycogenolysis or increased MCT-1 concentration that could improve LA kinetics during and/or after maximal exercise.

The present study includes some methodological limitations. First, the present HA training was implemented for only 5 consecutive days, shorter than traditional HA regimes lasting 7 – 12 days [20, 24, 25, 35]. This study was designed to reflect a practical application in the field for those who (e.g. athletes, firefighters) are in need of preparation for a comparative event and/or performing a job duty in hot environments, and are willing to be acclimated in a relatively short time (within a week) of their training/work schedule. Although this short-term HA training significantly elicited thermoregulatory adaptation to heat, it may have not been adequate to elicit other metabolic adaptations which may positively affect LA removal. Second, we have not included LA or catecholamine measurements during exercise, but focused on LA removal kinetics following the cessation of maximal exercise; which makes it difficult to determine whether HA, within our study design, could have affected LA kinetics

during exercise with concomitant effects on other aspects of LA, such as LA maximal steady state and threshold. Third, our subjects, despite not being athletes, were young, fit individuals who exhibited high VO_{2max} (group mean above 60 ml·kg·min^{-1}) and reported performing endurance exercise in a regular basis. Therefore, noticeable alterations in some physiological mechanisms might have been limited during a short period of HA training. Third, this study was conducted in a within-subject design implementing a single group of subjects, and no control group for the comparison of different training methods. Therefore, the study results need to be interpreted with caution within our study design.

Conclusions

The present study is the first to investigate the effect of a shortened period of HA training on the kinetics of LA removal during exercise recovery. It is concluded that 5 days of HA training in young, healthy men, successfully brought about physiological and subjective adaptations to heat, verified by significant improvement in end-exercise T_{re}, T_{sk}, HR, and SR and decreases in HP and RPE responses from Day 1 to Day 5 of HA. However, these adaptations did not result in the improvement of LA removal ability (y2) during 60 minutes of recovery from maximal exercise. While a longer period of HA training may enhance LA removal ability, as speculated from previously reported improvements in LA concentration and threshold during sub-maximal exercise, it is not clear whether these alterations would also accelerate LA removal during recovery. Future studies investigating how a longer period of HA training (> 5 days) would affect LA kinetics during exercise and recovery phases, with a clear identification of responsible physiological mechanisms, are warranted.

Acknowledgments

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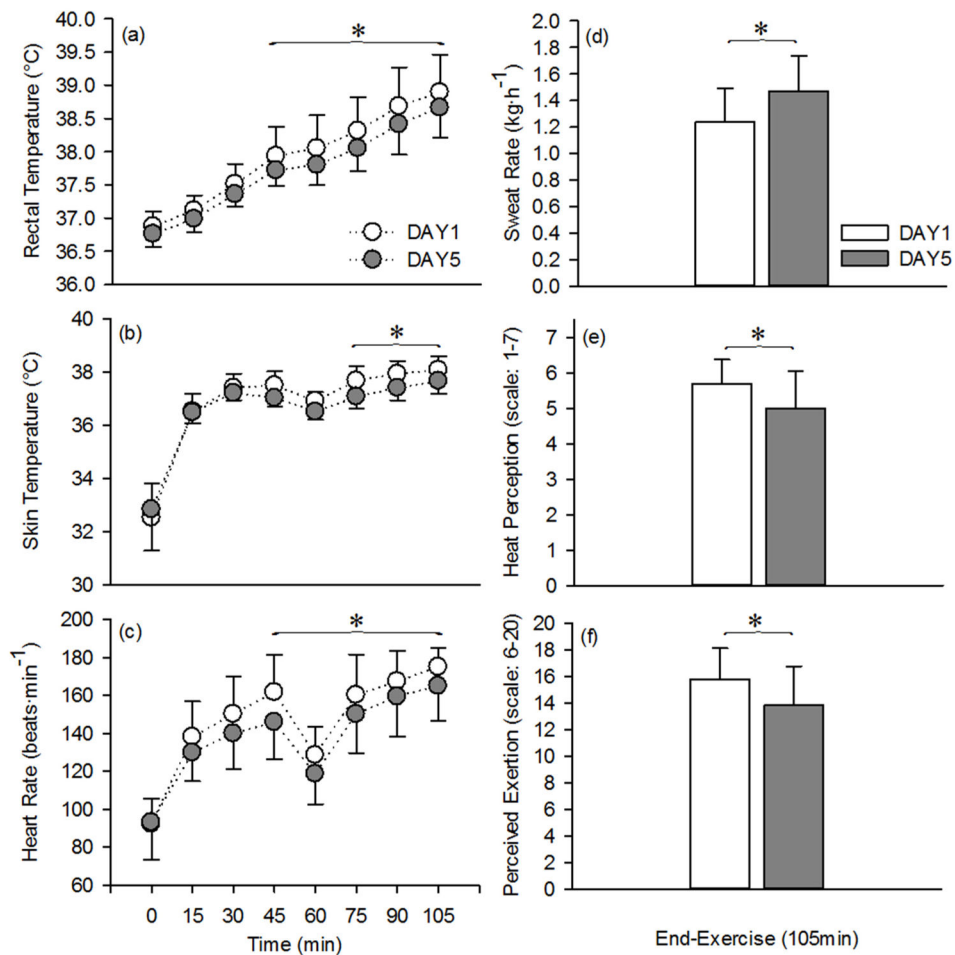
Figure 1.
Comparison of physiological and subjective measurement variables between Day 1 and Day 5 of HA training. Data are mean and standard deviation (n=10). *: Statistical difference in the variable between Day 1 and 5 (p<.05).
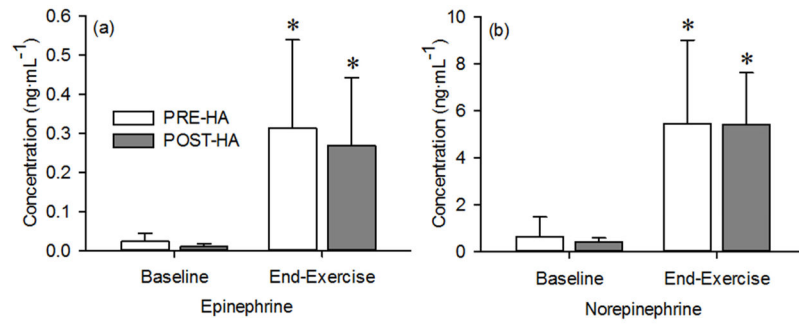
Figure 2.
Catecholamine responses during maximal exercise tests. Data are mean and standard deviation (n=10). *: Statistical difference between baseline and end-exercise (p<.05)

TABLE 1

Summary of LA concentrations at rest and following a maximal exercise (PRE-HA and POST-HA).

Trial	Parameters			
	LA_{base} (mmol.L^{-1})	LA_{0min} (min^{-1})	LA_{peak} (mmol.L^{-1})	LA_{60min} (min^{-1})
PRE-HA	1.75 ± 0.49	8.78 ± 1.08	10.97 ± 1.77	2.88 ± 0.82
POST-HA	1.54 ± 0.27	8.69 ± 1.23	10.95 ± 1.46	2.96 ± 0.93

Data are mean ± standard deviation (n=10).

TABLE 2

Summary of LA kinetics parameters following a maximal exercise (PRE-HA and POST-HA)

Trial	Parameters				
	$L(0)$ (mmol.L^{-1})	A1 (mmol.L^{-1})	$y1$ (min^{-1})	A2 (mmol.L^{-1})	$y2$ (min^{-1})
PRE-HA	8.04 ± 0.18	6.21 ± 0.80	0.24 ± 0.03	-13.05 ± 7.05	0.02 ± 0.01
POST-HA	8.25 ± 0.35	9.82 ± 1.67	0.22 ± 0.03	-15.59 ± 7.90	0.03 ± 0.01

Data are mean ± standard error. La(0) (venous blood LA concentration at the end of maximal exercise), A1 and A2 (amplitude of the two exponential time functions), y1 and y2 (velocity constants denoting LA exchange and remove abilities, respectively).