

Predicting an Athlete's Physiological and Haematological Response to Live High-Train High Altitude Training using Hypoxic Sensitivity Methods

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Abstract

Purpose: Elite endurance runners frequently utilise live high-train high (LHTH) altitude training. Individual differences in response to the hypoxic exposure have resulted in contradictory findings. In the present study, we aimed to test if 4-weeks of LHTH enhanced tHbmass and physiological capacity in elite endurance runners. Secondly, to test if the Richalet hypoxic sensitivity test (HST) could predict the haematological and physiological responses of LHTH. **Method:** Twelve elite runners completed a 4-week altitude training camp (~2,300 m; ALT) and a further five highly trained runners completed similar training at sea level (CON). All participants visited the laboratory once for preliminary testing (PRE), to determine lactate threshold (LT), lactate turn point (LTP), $\dot{V}O_{2max}$ and tHbmass (using the oCOR-method). Repeat testing was completed post-altitude training camp or post-sea level training (POST). Additionally, the ALT group completed the HST prior to the altitude training camp. **Results:** LHTH found a difference ($P < 0.05$) within ALT, but not CON from PRE to POST in average LT ($6.1 \pm 4.6\%$ vs. $1.8 \pm 4.5\%$) and LTP ($5.4 \pm 3.8\%$ vs. $1.1 \pm 3.2\%$), respectively. No difference was found within ALT or CON in $\dot{V}O_{2max}$ ($2.7 \pm 3.5\%$ vs. $-3.3 \pm 6.3\%$) or tHbmass ($1.9 \pm 2.9\%$ vs. $-0.1 \pm 3.3\%$), respectively. The HST revealed hypoxic ventilatory response at rest (HVRr) and hypoxic cardiac response at rest (HCRr) predicted individual changes tHbmass and $\dot{V}O_{2max}$, respectively. **Conclusion:** In elite endurance runners, 4-weeks of LHTH enhanced some measures of physiological capacity, but not mean tHbmass compared to CON. Although there was no significant change in mean tHbmass or mean $\dot{V}O_{2max}$, the mean changes were predicted by HST variables.

Introduction

Endurance athletes report altitude training as an important part of their overall training regime (Álvarez-Herms et al. 2015; Turner et al. 2019) and if adequately performed and monitored, altitude training is recommended (Millet and Brocherie 2020). Indeed, the world's most successful endurance athletes include altitude training in their planning (Solli et al. 2017; Tjelta 2019; Schmitt et al. 2020) and it is periodized around Olympic cycles (Mujika et al. 2019).

The optimal "hypoxic dose" has been extensively summarised (Rusko et al. 2004; Millet et al. 2010; Chapman et al. 2014), with 4-weeks at 2,000-2,500 m sufficient to improve performance (Bonetti and Hopkins 2009) and enhance haematological adaptations (Gore et al. 2013). Although this has been disputed (Lundby and Robach 2016), concluding that altitude training works for some but certainly not in all athletes (Lundby et al. 2012). This argument is an ongoing discussion in altitude training research. It is likely that, 'non-responder' athletes are probably a product of 'one-off' camps and/or inadequate planning, periodization, programming, and monitoring of altitude training (Mujika et al. 2019).

The primary goal of altitude training is to improve the oxygen carrying capacity of the blood and the oxygen utilisation at the muscle, primarily through an increase in total haemoglobin mass (tHbmass) (Chapman and Levine 2007). An increase in tHbmass of 1% will result in a 0.6–0.7% change in $\dot{V}O_{2\max}$ (Saunders et al. 2013), and this may improve performance in endurance exercise (Bassett and Howley 2000; Jones and Carter 2000). However, there are multiple factors that impact upon altitude adaptation, such as, absolute and relative intensity of training and its distribution, iron availability, injury/ illness status, and inter-individual variability (Mujika et al. 2019), which all need to be understood.

The individual variability in response to hypoxia has been proposed as key component to a successful altitude training camp (Sinex and Chapman 2015). Pre-altitude camp hypoxic screening may be useful to predict which athletes would best respond to altitude training, but has received relatively little attention (Friedmann 2005; Chapman et al. 2010). Parameters, such as erythropoietin (EPO) response, ventilatory acclimatisation and training ability under hypoxic conditions have shown individual variation (Chapman 2013) and, the balance between those adaptations may determine whether the athlete will experience improvements in tHbmass or $\dot{V}O_{2\max}$ and following chronic hypoxic exposure (Constantini et al. 2017).

The aim of the present study was to investigate the effect of 4-weeks of LHTH at ~2,300 m on physiological determinants of performance and tHbmass in elite endurance runners. Furthermore, the study aimed to assess the predictive ability of the Richalet hypoxic sensitivity test (HST) against the hypoxia-induced changes in physiological and haematological markers.

Methods

Participants

British Athletics selected 16 endurance runners to attend a LHTH altitude training camp. Participants had represented Great Britain in either U23 or senior cross country and track competitions. Four participants withdrew due to injury or illness. The resultant 12 runners travelled to Iten, Kenya (~2,300 m above SL) for 4-weeks (ALT). Five nationally competitive endurance runners formed a SL control group (CON). Participant characteristics are shown in Table 1. All participants were SL residents and had not been to altitude above 1,500 m in the previous three months. Ethical approval, informed consent and medical questionnaires were completed following the principles outlined by the Declaration of Helsinki, as revised in 2013.

In the 3-4 months before the study period, participants were engaged in national and international track racing. Six weeks before the commencement of the study, participants had an end of year break (~14 days) and were training for one month before the study period. Participants were screened for iron status (Ferritin: $90.5 \pm 62.6 \text{ ng}\cdot\text{mL}^{-1}$) two weeks prior to the study period. According to organisational guidelines, one capsule of Ferrous Fumarate (305 mg) 3 times per day, was consumed for the experimental testing period if ferritin was below $30 \mu\text{g}\cdot\text{mL}^{-1}$ ($n = 1$). All participants were instructed to continue with their normal iron supplementation programme (Garvican-Lewis et al. 2016).

Table 1: Participant characteristics.

	ALT	CON
Male / Female	7 / 5	4 / 1
Age (yr)	23 ± 4	23 ± 3
Body Mass (kg)	63.0 ± 7.0	65.6 ± 5.7
Height (cm)	174.9 ± 6.7	180.7 ± 9.1
Sum of 8 skinfolds (mm)	60.1 ± 19.9	48.4 ± 11.5
tHbmass ($\text{g}\cdot\text{kg}^{-1}$)	12.9 ± 1.8	12.9 ± 1.0
Serum Ferritin ($\mu\text{g}\cdot\text{L}^{-1}$)	91.3 ± 66.2	88.0 ± 59.2
Performance time (%) *	94.3 ± 1.7	93.0 ± 1.5
Weekly Training Volume (miles)	52.8 ± 17.4	42.0 ± 18.9

*performance time (season's best) is expressed as percentage (%) of the male and female British record time in their primary event.

Experimental design

All participants visited the laboratory on two occasions for baseline physiological testing, and a second tHbmass and blood test (8 ± 5 d between baseline tHbmass tests). There were 10 ± 4 d between the physiological testing and departure to altitude. Upon arriving back at SL participants completed the post-tHbmass test within 5 days and repeat physiological testing was completed after 11 ± 4 d at SL. The SL (UK; 43 m) CON group completed the same physiological testing and tHbmass testing before a block of training (PRE) and post-training (POST). There were 61 ± 8 days and 51 ± 4 days between PRE and POST in the CON and ALT group, respectively. All participants

followed their own personal training programmes prescribed by their coach. Participants were given training zone guidance common to those characterised by Seiler (2010). Training data were recorded in training diaries and GPS watches depending on the athlete's preference.

Physiological and haematological testing

Anthropometric data were collected with body mass measured using digital scales (GFK 150, Adam Equipment Inc., Danbury, CT, USA) and body fat assessed from eight sites (biceps, triceps, subscapular, supra-spinal, iliac crest, abdominal, quadriceps and calf) using skinfold callipers (Harpenden, Burgess Hill, UK). Participants then performed a submaximal and maximal running assessment (Shaw et al. 2015) for the determination of training zones, the $\dot{V}O_2$ -speed relationship and $\dot{V}O_{2max}$. Following this tHbmass (TEM of 1.0%) was determined using the oCOR-method (Schmidt and Prommer 2005; Prommer and Schmidt 2007) as previously described in (Turner et al. 2014a, b). Venous blood was collected via venepuncture of an antecubital vein in the forearm. Venous blood samples were collected and analysed for haematocrit (Hct) and haemoglobin concentration ([Hb]) (Pentra ES 60, Horiba Medical; Kyoto, Japan), and serum Ferritin (Randox Daytona Rx, Randox Laboratories, County Antrim, UK).

Daily physiological measures and subjective questions

During the altitude training camp arterial oxygen saturation (Nonin Medical, Onyx Vantage 9590 Finger Pulse Oximeter, Plymouth, USA), a urine osmolality (Pocket PAL-OSMO; Vitech Scientific, Horsham, UK) and body mass (Seca 875, Seca, Hamburg, Germany) were recorded daily upon waking. Finally, participants were required to complete 7 subjective questions on sleep quality, energy levels, motivation to train, irritability, dizziness, muscle soreness and appetite. Each scale ranked from 1 (positive) to 5 (negative).

Hypoxic sensitivity test

The HST was completed ~2 h after the treadmill testing to allow the participants to recover. Eleven ALT participants completed the HST, as previously described (Richalet et al. 2012; Bourdillon et al. 2014). Due to injury one participant withdrew from the HST. Briefly, participants rested (RN) whilst standing on the treadmill for 4-min breathing normoxic air, the valve was then opened, and the participants rested for a further 4 min breathing hypoxic air (RH). The treadmill was started, and participants walked uphill at a speed a gradient targeting a heart rate (HR) of ~130 bpm ($5.8 \pm 0.4 \text{ km}\cdot\text{h}^{-1}$ and $7.5 \pm 0.7\%$ gradient) whilst breathing hypoxic air (EH) for 4 min. The valve was then closed, and the participants continued walking for a final 4 min whilst breathing normoxic air (EN). A wrist pulse oximeter MD300W (ChoiceMMed; Hong Kong) measured SpO_2 continuously and HR was also measured continuously (Polar H1 heart rate sensor with FT3 wristwatch; Kempele, Finland). Ventilation (VE), HR and SpO_2 responses during the last 30 seconds of each phase were used to characterise subjects' sensitivity to hypoxia (Richalet et al. 2012) by calculating: desaturation at rest (ΔSp_r)/exercise (ΔSp_e), ventilation changes at rest (ΔVE_r)/exercise (ΔVE_e), hypoxic ventilatory response at rest (HVR_r)/exercise (HVR_e) and hypoxic cardiac response at rest (HCR_r)/exercise (HCR_e). For a typical physiological response to the HST see Figure 9.2.

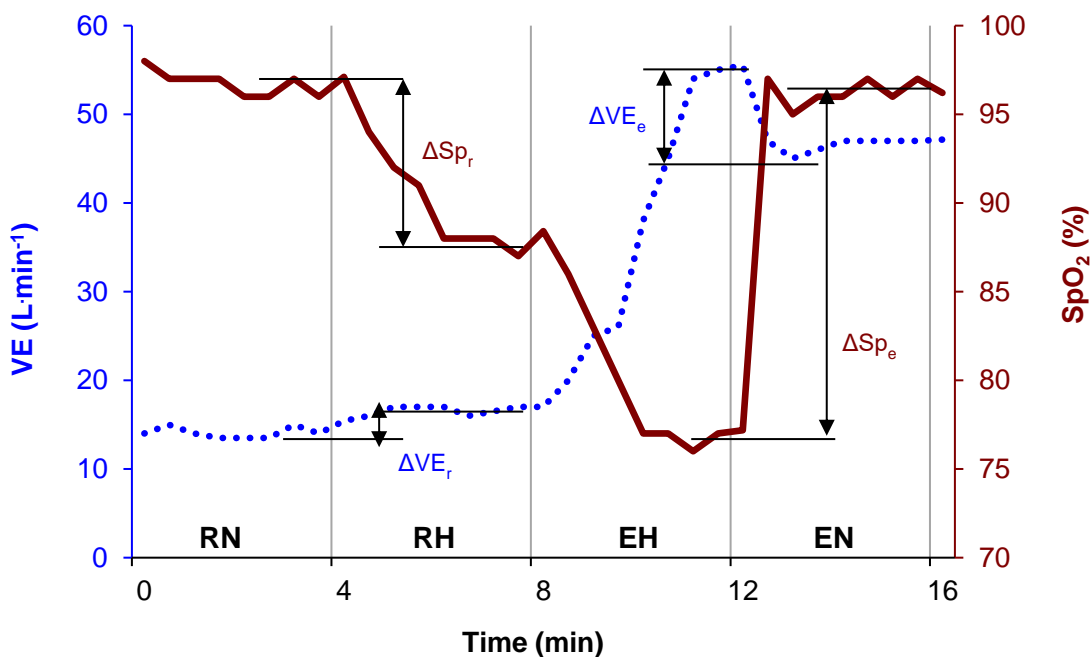


Figure Error! No text of specified style in document..1: A typical physiological response from the Hypoxic Sensitivity Test (Richalet et al. 2012). RN = rest in normoxia; RH = rest in hypoxia; EH = exercise in hypoxia; EN = exercise in normoxia; Hypoxia was generated by breathing a normobaric hypoxic gas mixture (Hi-FiO₂: 0.115). Dotted blue line = VE and solid red line = SpO₂. ΔSp_r = change in desaturation at rest; ΔSp_e = change in desaturation during exercise; ΔVE_r = change in pulmonary ventilation at rest; ΔVE_e = change in pulmonary ventilation during exercise.

Statistical analyses

Data were assessed for normality and sphericity and adjusted where necessary using the Huynh-Feldt method. A mixed measures ANOVA was used to compare ALT and CON measures from PRE and POST. A stepwise multiple regression analysis was used for the eight dependant variables of the HST (ΔSp_r, ΔSp_e, ΔVE_r, ΔVE_e, HVR_r, HVR_e, HCR_r and HCR_e) with the percentage change in tHbmass and $\dot{V}O_{2max}$ from PRE to POST. The following classification system refined by Hopkins et al. (2009) was used to interpret the magnitude of the relationship: trivial 0.0–0.1; small 0.1– 0.3; moderate 0.3–0.5; large 0.5–0.7; very large 0.7–0.9; almost perfect 0.9–1; and perfect 1. All data were recorded as mean ± SD, with significance accepted at $P < 0.05$.

Results

Training

Training data were collected for 8-10 weeks. The training consisted of weeks 1-3 at SL (48 ± 17 miles/week), weeks 4-7 at altitude (50 ± 17 miles/week), and weeks 8-10 at SL (44 ± 15 miles/week). The CON athletes were instructed to continue training as they would during the winter phase of the season.

Haematological and physiological responses to LHTH

There were no differences between baseline measurements of ALT and CON groups in tHbmass ($P = 0.646$), $\dot{V}O_{2\max}$ ($P = 0.527$), LT ($P = 0.981$), LTP ($P = 0.908$) and sum of 8 skinfolds ($P = 0.142$). **Age? Ferritin? Training Volume?** Total Hbmass increased by $1.9 \pm 2.9\%$ and $0.1 \pm 3.3\%$ from PRE to POST in the ALT and CON group, respectively (see figure 1B). There was no effect of time on tHbmass ($P = 0.21$) and there no interaction effect was found between time*condition ($P = 0.24$).

ALT had no effect on average $\dot{V}O_{2\max}$ ($P = 0.95$) from PRE to POST, however an interaction effect was found between time*condition ($P = 0.03$). An increase of $2.7 \pm 3.4\%$ in the ALT group was found compared to and a decrease of $3.3 \pm 6.3\%$ in the CON group (see figure 1A).

There was a main effect of time on LT ($P = 0.01$) and LTP ($P = 0.004$), however there was no interaction effect on LT between time*condition ($P = 0.11$). In the ALT group LT increased by $6.1 \pm 4.6\%$, and in the CON group LT increased by $1.8 \pm 4.5\%$ (see figure 1C). An interaction effect was found on LTP between time*condition ($P = 0.04$), with LTP increasing by $5.4 \pm 3.8\%$ and $1.1 \pm 3.2\%$ in the ALT and CON, respectively (see figure 1D).

Table 3 illustrates the HR and B[La] measured during the treadmill testing in the ALT and CON groups at PRE and POST. ALT had no effect on average HR or B[La] at LT, LTP or $\dot{V}O_{2\max}$ from PRE to POST or compared to CON. Differences were found ($P = 0.010$) between ALT and CON within PRE and POST, however there was no interaction effect.

Table 3: Heart rate (HR) and blood lactate (B[La]) responses in ALT and CON groups from PRE to POST. * denotes significant difference ($P < 0.05$) between conditions.

	ALT		CON	
	PRE	POST	PRE	POST
HR at LT ($\text{b}\cdot\text{min}^{-1}$)	159 ± 8	162 ± 10	161 ± 7	167 ± 7
HR at LTP ($\text{b}\cdot\text{min}^{-1}$)	170 ± 10	174 ± 10	172 ± 7	176 ± 7
HR_{max} ($\text{b}\cdot\text{min}^{-1}$)	183 ± 12	183 ± 10	190 ± 4	188 ± 4
B[La] at LT ($\text{mmol}\cdot\text{L}^{-1}$)	1.3 ± 0.5	1.1 ± 0.2	1.4 ± 0.5	1.3 ± 0.3
B[La] at LTP ($\text{mmol}\cdot\text{L}^{-1}$)	2.6 ± 0.5	2.4 ± 0.4	2.4 ± 0.8	2.6 ± 0.5
B[La]_{max} ($\text{mmol}\cdot\text{L}^{-1}$)	8.2 ± 2.5	8.0 ± 2.1	10.4 ± 2.0 *	10.5 ± 1.7 *

Table 2: Heart rate (HR) and blood lactate (B[La]) responses in ALT and CON groups from PRE to POST. * denotes significant difference ($P < 0.05$) between conditions.

	ALT				CON			
	PRE	POST	% Change	CI 95%	PRE	POST	% Change	CI 95%
tHbmass (g)	812 ± 147	830 ± 160	1.9 ± 3.2		847 ± 128	847 ± 122	0.1 ± 3.3	
Blood Volume (ml)	6162 ± 893	5992 ± 1107	-1.7 ± 7.0		6227 ± 717	6258 ± 643	0.4 ± 5.8	
Plasma Volume (ml)	3675 ± 497	3533 ± 668	-2.8 ± 9.6		3659 ± 400	3720 ± 295	1.6 ± 8.0	
$\dot{V}O_{2max}$ (ml·kg·min⁻¹)	68.5 ± 8.1	70.4 ± 7.7	2.7 ± 3.4		70.9 ± 3.2	68.9 ± 5.8	-3.3 ± 6.3	
LT (km·h⁻¹)	14.9 ± 1.3	15.9 ± 1.1	6.1 ± 4.6		14.9 ± 1.2	15.2 ± 1.3	1.8 ± 4.5	
LTP (km·h⁻¹)	16.9 ± 1.3	17.9 ± 1.1	5.4 ± 3.8		17.0 ± 1.3	17.2 ± 1.4	1.1 ± 3.2	
Sum8 Skinfolds (mm)	60.1 ± 19.9	56.5 ± 17.7	-6.6 ± 9.1		45.6 ± 8.3	44.3 ± 9.6	-3.8 ± 11.7	

Daily physiological measures and subjective questions

Measure	Mean \pm SD	vs. Δ tHbmass		vs. $\Delta\dot{V}O_{2\max}$	
		r	p-value	r	p-value
Oxygen Saturation (%)	94 \pm 1	-0.54	0.07	-0.52	0.09
Body Mass Loss (kg)	-0.5 \pm 0.7	0.01	0.98	0.19	0.56
Sleep Quality	2.1 \pm 0.5	0.32	0.31	0.22	0.48
Motivation	1.6 \pm 0.5	0.21	0.50	0.24	0.46
Energy Levels	1.9 \pm 0.4	0.36	0.25	0.46	0.14
Dizziness	1.1 \pm 0.2	0.02	0.95	0.31	0.32
Irritability	1.5 \pm 0.4	0.21	0.21	0.52	0.52
Muscle Soreness	2.1 \pm 0.4	0.34	0.28	0.25	0.44
Appetite	1.3 \pm 0.4	0.36	0.25	0.34	0.29
Subjective Average	1.6 \pm 0.3	0.39	0.21	0.45	0.14

Hypoxic sensitivity test (HST)

A stepwise multiple regression was run to predict post-ALT change in tHbmass (Δ tHbmass) from Δ Sp_r ($r = 0.514$), Δ Sp_e ($r = 0.446$), Δ VE_r ($r = 0.396$), Δ VE_e ($r = 0.216$), HVR_r ($r = 0.788$), HVR_e ($r = 0.065$), HCR_r ($r = -0.680$) and HCR_e ($r = -0.028$). The only predictor variable to enter the model was HVR_r; The variables predicted Δ tHbmass, $F(1,9) = 14.746$, $P = 0.004$, with an adjusted R² of 0.58 and standard error of the estimate of 1.852. The general form equation to predict Δ tHbmass (%) = $0.494 + (1.730 * HVR_r)$.

Further to this, another stepwise multiple regression was run to predict post-ALT change in $\dot{V}O_{2\max}$ ($\Delta\dot{V}O_{2\max}$) Δ Sp_r ($r = -0.091$), Δ Sp_e ($r = 0.385$), Δ VE_r ($r = 0.566$), Δ VE_e ($r = 0.513$), HVR_r ($r = 0.611$), HVR_e ($r = 0.394$), HCR_r ($r = -0.681$) and HCR_e ($r = 0.117$). The only predictor variable to enter the model was HCR_r. The variable predicted $\Delta\dot{V}O_{2\max}$, $F(1,9) = 7.786$, $P = 0.021$, with an adjusted R² of 0.404 and standard error of the estimate of 2.649. The general form equation to predict $\Delta\dot{V}O_{2\max}$ (%) = $5.131 - (1.795 * HCR_r)$.

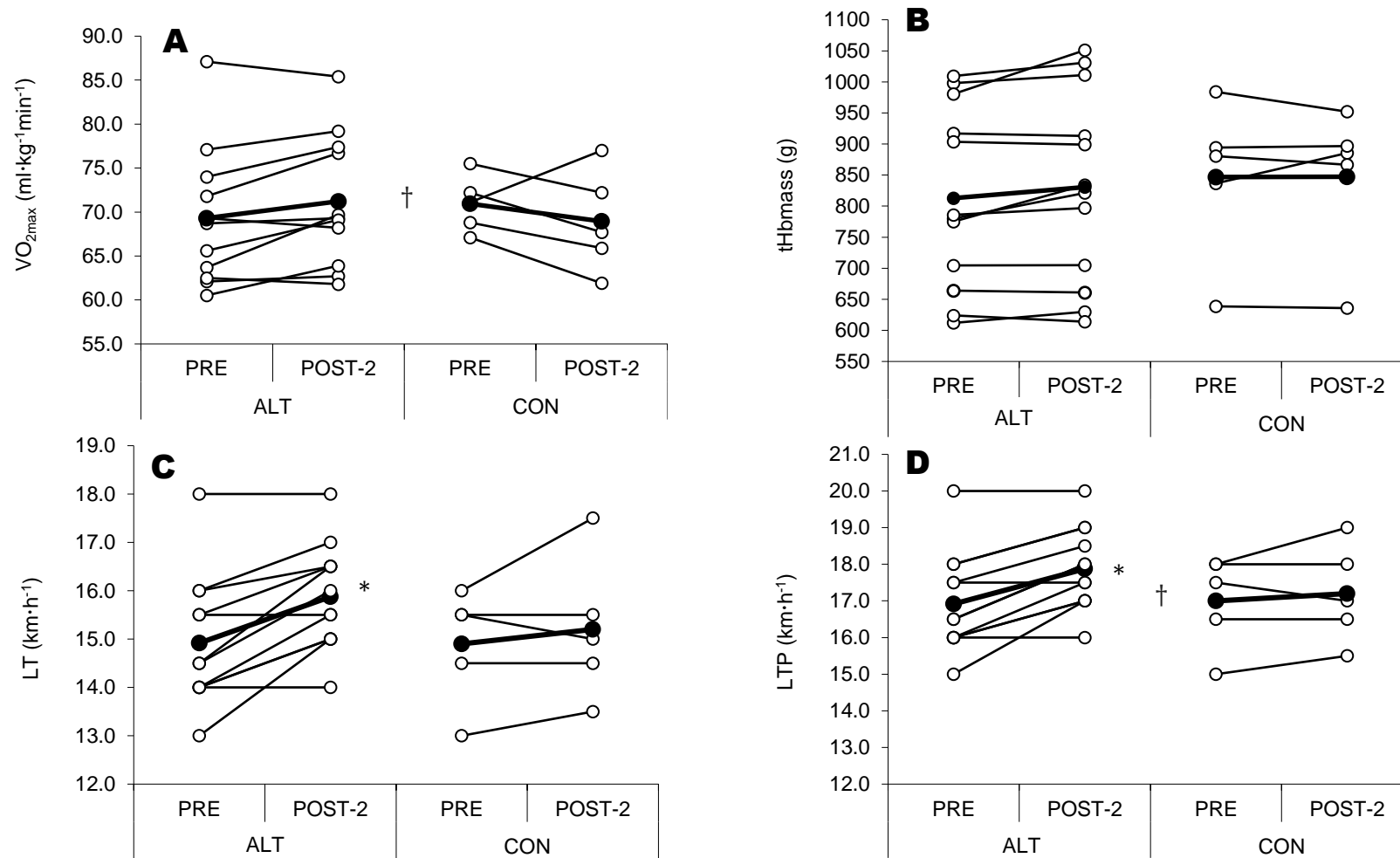


Figure 1: Individual (open circle) and mean (closed circle) differences from PRE to POST in $\dot{V}O_{2max}$ (Plot A), tHbmass (Plot B), LT (Plot C), LTP (Plot D) from ALT and CON groups. * denotes main effect of time and † denotes interaction effect of time*condition.

Discussion

The present study investigated the effect of 4-weeks of LHTH at ~2,300 m on physiological determinants of performance and tHbmass in elite endurance runners. Furthermore, the study aimed to assess if the Richalet HST could predict hypoxia-induced changes in tHbmass and $\dot{V}O_{2max}$. Despite nine out of twelve in the ALT group presenting an increase in tHbmass, a significant difference was not found. Four weeks of LHTH did however improve LTP and $\dot{V}O_{2max}$, versus CON. Pre-screening using the Richalet HST predicted subsequent changes in tHbmass and $\dot{V}O_{2max}$ using the variables of HCR_r and HVR_r, respectively.

Haematological and physiological responses to LHTH

Four weeks of LHTH altitude training at ~2,300 m did not result in increased tHbmass from PRE to POST. Although, LHTH did increase tHbmass by $1.9 \pm 3.2\%$ compared $0.1 \pm 3.3\%$ in CON. Whilst the findings of the present study appear to be contrary to previous studies measuring tHbmass in endurance runners after LHTH (Frese and Friedmann-Bette 2010; Garvican-Lewis et al. 2015; Sharma et al. 2019), there was a marked individual variation (range: -4.6 to 5.7%), with nine out of twelve in the ALT group eliciting an increase in tHbmass (including >2.0% in 8 athletes). With a moderate effect size (0.31), the observed increase is likely to be a true physiological change.

Both natural LHTH and simulated LHTL camps have reported average increases of tHbmass ranging from 2.0-4.1% (Frese and Friedmann-Bette 2010; Garvican-Lewis et al. 2015; Sharma et al. 2019) and 2.8-3.8% (Saunders et al. 2009; Robertson et al. 2010a, b), respectively. Similarly, the authors have reported high individual variation in tHbmass response to natural LHTH and simulated LHTL (Frese and Friedmann-Bette 2010; Robertson et al. 2010a). An increase in total red cell volume, or tHbmass, is thought to be associated with a greater acute and sustained increase in EPO, which has been linked to improved endurance performance after LHTH (Chapman et al. 1998). The reason for a more augmented EPO response at 2,500 m is not readily clear, variable and influenced by individual differences in hypoxic ventilatory drive, sensitivity to hypoxia at the point of EPO release and genetically inherited traits (Chapman et al. 1998). Unfortunately, the present study was unable to measure EPO during the altitude training camp.

Sperlich et al. (2016) suggested that adequately designed and controlled training loads, and continuously monitoring an individual athlete's adaptation and health status may result in a more successful altitude training experience. If markers of stress are monitored and training load (i.e., intensity or volume) is adjusted appropriately, there would be reduced signs of a maladaptive response (Sperlich et al. 2016). The present study monitored hydration status, resting oxygen saturation (with pulse-oximetry; SpO₂), body mass, sleep quality and subjective markers of wellness daily. The day-to-day changes in subjective markers and body mass were minimal. There were also no significant correlations in the level of desaturation, body mass losses, decline in sleep quality and reported wellbeing versus change in tHbmass or $\dot{V}O_{2max}$. Previously, illness and injury (McLean et al. 2013; Gough et al. 2013; Wachsmuth et al. 2013; Heikura et al. 2018) have been associated with detrimental increases in tHbmass. None of the ALT runners reported any incidences of illness or compromised training load during the camp period.

Retrospective analysis found a difference in Δ tHbmass from PRE to POST when the ALT group was split into males and females. The male (n = 7) participants increased from 909 ± 98 g to 936 ± 103 g ($+2.8 \pm 2.0\%$) with the females (n = 5) increasing from 675 ± 69 g to 680 ± 84 g ($+0.6 \pm 4.3\%$). Male to female comparison has been reported in elite athletes training for 3-4 weeks at ~2,000-2,300 m, with tHbmass increasing by ~5.5% vs ~3.4% (Heikura et al. 2018), ~8.3 vs ~8.7% (Heinicke et al. 2005) and ~6.3 vs ~8.0% (Wachsmuth et al. 2013), respectively. Heikura et al. (2018) also found superior increases in tHbmass in those with lower initial tHbmass values, as previously suggested by (Robach and

Lundby 2012). This contradicts the findings of the present study, which found no relationship between baseline tHbmass levels and subsequent changes in tHbmass.

The present study found a significant improvement in $\dot{V}O_{2max}$ compared to CON. This is likely due to the concomitant increase of $\dot{V}O_{2max}$ in ALT (+2.7%) and decrease in CON (-3.3%). Improvements in $\dot{V}O_{2max}$ of 2.5-4.2% have been found after 4-weeks of LHTH at 2,500 m (Levine and Stray-Gundersen 1997), after 17 days at 3,090 m (Dill and Adams 1971) and after 4-weeks at 1,740 m (Gore et al. 1997). There were, however, no improvements in $\dot{V}O_{2max}$ after 3-weeks at 2,300 m (Adams et al. 1975) and after 2-weeks at 2,000 m (Svedenhag et al. 1991). Individual variation, training status and achieving an adequate “hypoxic dose” appear to play a key role in enhancing $\dot{V}O_{2max}$ after LHTH.

Improvements in sea-level performance after a period of altitude training may have a multifactorial and are not solely dependent on increasing tHbmass via erythropoiesis (Gore et al. 2007). The ALT group improved both LT and LTP by $1 \text{ km}\cdot\text{h}^{-1}$ compared to no change in CON. There is limited comparison in submaximal physiological responses to LHTH in elite runners, however, Bailey et al. (1998) reported improvements in running velocity of 7.7% (16.7 to $18.1 \text{ km}\cdot\text{h}^{-1}$) at $2 \text{ mmol}\cdot\text{L}^{-1}$ and 10.8% (21.5 to $24.1 \text{ km}\cdot\text{h}^{-1}$) at $4 \text{ mmol}\cdot\text{L}^{-1}$ after 4-weeks at 1,500–2,000 m.

Hypoxic Sensitivity Test

After 4-weeks of altitude training at $\sim 2,300 \text{ m}$ results from regression analysis were that HVR_r ($< 0.14 \text{ L}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$) and HCR_r ($< 1.39 \text{ beats}\cdot\text{min}^{-1}\cdot\%$) best predicted the increase in tHbmass and $\dot{V}O_{2max}$, respectively. The findings suggest that athletes who experience a reduced HVR and HCR at rest during the HST are likely to produce a greater increase in tHbmass and $\dot{V}O_{2max}$, respectively. A high HVR is thought to help in performing work at high altitude (West et al. 2000) due to the extreme increase in ventilation required to remove carbon dioxide and increase arterial oxygen partial pressure (PaO_2) (Bernardi et al. 2006). However, to complete the required training intensity and load at altitude athletes would require a lower HVR to hypoxia. Previously, climbers who were characterised by a lower ventilatory sensitivity to hypoxia had more successful summits of mountain peaks without supplemental oxygen (Bernardi et al. 2006).

The present study found HRV and HCR to predict subsequent changes in tHbmass and $\dot{V}O_{2max}$ after LHTH altitude training. Bourdillon et al. (2014) found that Richalet HST did not predict the performance decrement of a 15 km time trial in severe hypoxia ($\text{FiO}_2 = 0.11$), however Pla et al. (2020) reported that ΔSp_e was moderately correlated with the change in performance at altitude ($r = 0.54$). Other variables, HVR_e ($r = -0.38$) and HCR_e ($r = -0.20$), were poorly correlated to the change in performance. Swimming performance, measured with a 100 or 200 m time trial, was completed after 8 days at 1,850 m and only arterial oxygen desaturation during exercise was correlated with the changes in performance. The present study found a moderate ($r = 0.44$) and small ($r = 0.27$), but insignificant, correlations between ΔSp_e and change in tHbmass and $\dot{V}O_{2max}$, respectively. Chapman (2013) stated individuals who are least able to maintain SaO_2 likely end up being the ones with the largest drop in $\dot{V}O_{2max}$, which may inhibit their ability to train at altitude.

Arterial oxygen saturation is the end product of both pulmonary ventilation and gas exchange (Beidleman et al. 2014) and the ventilatory response to exercise in hypoxia is correlated to SaO_2 (Benoit et al. 1995), therefore individuals with a strong ventilatory response to acute hypoxia are able to alleviate a fall in arterial oxygen content by increasing ventilation (Chapman et al. 2010). The present study has found a lesser HVR at rest during the HST was associated with an enhanced tHbmass after LHTH altitude training. Groups of athletes typically travel to the same altitude to train together, therefore it is important to understand how each athlete may respond to altitude prior to training camp. Those who are more sensitive may require additional interventions, such as pre-acclimatisation with heat (White et al. 2014) or hypoxia

(Chapman et al. 2013), nutritional strategies (Shannon et al. 2016) or remote ischemic preconditioning (Paradis-Deschênes et al. 2018).

Limitation and future directions

Changes in $\dot{V}O_{2max}$ (range: -2.0% to 8.6%), LT (range: 0% to 13.3%) and LTP (range: 0% to 11.8%) post-LHTH showed large variability, which may be associated with the timing of the testing after altitude exposure (Robertson et al. 2010b; Sharma et al. 2019). The post-LHTH treadmill test was completed at 10 ± 3 d at sea level. Unfortunately, existing competition commitments and the residential location of the participants, we were unable to complete post-testing on the same day. A meta-analysis by Bonetti and Hopkins (2009) found that by manipulating the study characteristics, such the test day (after altitude exposure), the chance of an enhancement of maximal endurance power output increased from 1.6% to 5.2% with LHTH. This highlights the importance of understanding the return to sea level period following altitude training when interpreting performance tests, although the mechanisms for this variation are unclear. Chapman et al. (2014a) believed that individual rates of decay in haematological, biomechanical, and ventilatory adaptations were central to the understanding.

The training completed during this camp was predominantly 'aerobic base' work where the intensity of training lower or equal to LT (Seiler and Kjerland 2006). Therefore, it is possible that the improvements in LT and LTP reflected this type of training. The extent to which an athlete may benefit from altitude training will differ according to their general and specific training focus (i.e. between types of endurance training; and between different periods of the training year) (Millet et al. 2010). Unfortunately, the ALT and CON groups completed different volumes of training and we were unable to quantify the relative contributions of different training zones. This is common in altitude training studies (Sharma et al. 2018) and should be considered in the future. The present study attempts to understand the outcome of one altitude training camp, whereas most endurance programmes now commit to multiple altitude training camps per season (Turner et al. 2019). Repeated testing around multiple altitude training camps would further enhance our understanding of the physiological and haematological responses. Performance-enhancing strategies for elite endurance athletes should determine the long-term effects of accumulated altitude training through repeated exposures (Mujika et al. 2019). Therefore, it would be beneficial to track changes in haematology and endurance performance during the entire season, paying particular attention to the athletes training load and how this influences the resultant adaptations (Sperlich et al. 2016).

Conclusions

The present study observed the physiological and haematological responses to a live high-training high altitude training at ~2,300 m. Altitude training resulted in improvements in LTP and $\dot{V}O_{2max}$, but not tHbmass, in elite endurance runners. Pre-screening for hypoxic sensitivity using Richalet's HST found that ventilatory and cardiac responses to hypoxia at rest predicted increases in tHbmass and $\dot{V}O_{2max}$, respectively. The findings suggest that pre-screening for hypoxic sensitivity may help elite athletes and coaches understand the response to hypoxia. It may then be possible to provide individualised training programmes and acclimatisation strategies, within groups of athletes training at the same altitude, however further research is required.

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