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ORIGINAL ARTICLE

Biochemical responses and physical performance during high-intensity resistance circuit training in hypoxia and normoxia

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Abstract
Purpose The aim of this study was to analyze the effect of hypoxia on metabolic and acid–base balance, blood oxygenation, electrolyte, and half-squat performance variables during high-resistance circuit (HRC) training.
Methods Twelve resistance-trained subjects participated in this study. After a 6RM testing session, participants performed three randomized trials of HRC: normoxia (NORM: $FiO_2 = 0.21$), moderate hypoxia (MH: $FiO_2 = 0.16$), or high hypoxia (HH: $FiO_2 = 0.13$), separated by 72 h of recovery in normoxic conditions. HRC consisted of two blocks of three exercises (Block 1: bench press, deadlift and elbow flexion; Block 2: half-squat, triceps extension, and ankle extension). Each exercise was performed at 6RM. Rest periods lasted for 35 s between exercises, 3 min between sets, and 5 min between blocks. Peak and mean force and power were determined during half-squat. Metabolic, acid–base balance, blood oxygenation and electrolyte variables, arterial oxygen saturation (SaO_2), and rating of perceived exertion (RPE) were measured following each block.
Results During the first set, peak force and power were significantly lower in HH than MH and NORM; whereas in the second set, mean and peak force and power were significantly lower in HH than NORM. At the end of the HRC training session, blood lactate and RPE in HH were significantly higher than in MH and NORM. SaO_2, pH, HCO_3^-, and pO_2 values were significantly lower in all hypoxic conditions than in NORM.
Conclusion These results indicate that simulated hypoxia during HRC exercise reduce blood oxygenation, pH, and HCO_3^-, and increased blood lactate ultimately decreasing muscular performance.

Keywords Hypoxic · HRC · Lactate · Power · Resistance training

Abbreviations

ACSM	American college of sports medicine
ATP	Adenosine triphosphate
Ca^{2+}	Calcium
Cl^-	Chloride
cm	Centimeter
FiO_2	Fraction of inspired oxygen
Glu	Glucose
H^+	Hydrogen
HCO_3	Bicarbonate
HH	High hypoxia
HRC	High-resistance circuit
K^+	Potassium
l	Litre
m	Meter
MH	Moderate hypoxia
min	Minute
kg	Kilogram
Na^+	Sodium

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NORM	Normoxia
pCO_2	Carbon dioxide partial pressure
PCr	Phosphocreatine
pO_2	Oxygen partial pressure
RM	Maximum repetition
RPE	Rating of perceived exertion
RT	Resistance training
RTH	Resistance training under hypoxia
s	Second
SaO_2	Arterial oxygen saturation
SPSS	Statistical package for the social sciences
W	Watt

Introduction

Resistance training (RT) is an effective method to modify muscle morphology (i.e., increasing muscle mass) and to stimulate neuromuscular adaptations to increase strength, power, and local muscular endurance, ultimately leading to enhanced athletic performance (Garber et al. 2011; Scott et al. 2015a). Structural and functional adaptations of skeletal muscle can be finely tuned by modifying the exercise stimuli, such as training volume, training intensity, and/or environmental condition (Kon et al. 2014). Recently, some studies (Scott et al. 2015a; Kon et al. 2012; Alvarez-Herms et al. 2015b) have examined the utility of RT under hypoxia (RTH) to enhance muscular performance. For instance, strength exercises performed in hypoxic conditions have shown to increase intramuscular metabolic stress ($FiO_2 = 13\%$) (Kon et al. 2012), enhance hypertrophic signaling and muscle hypertrophy (FiO_2 14.4%) (Kon et al. 2014), as well as increase the concentration of anabolic hormones (Kon et al. 2012). Furthermore, studies have observed that one moderate-load resistance (i.e., 3 sets of 10 repetitions at 60% of 1RM) and hypoxic training session ($FiO_2 = 16\%$) can increase muscle activation (Scott et al. 2016) but does not affect maximal anaerobic power capacity (Alvarez-Herms et al. 2015a). In addition, exercising in hypoxia is known to induce greater respiratory and cardiovascular responses and increases sympathetic activation (Reeves et al. 1992).

The application of RTH in sports has been shown to improve muscle strength (↑ 15% of 3-s maximal voluntary contraction; ↑ 18% the area under 30-s force curve), muscle size (↑ 6% cross-sectional-area) and muscle endurance (↑ 23% the number of repetitions at 20% 1RM) in netball athletes after 5 weeks of training at 20% 1RM and at 80% SaO_2 (Manimmanakorn et al. 2013). Nonetheless, there are several disadvantages with RT (and consequently with RTH) that include: (a) the lengthy time required to complete a training session that consists of many exercise sets with reasonable inter-set rest durations, (b) moderate cardiovascular benefits when compared to other forms of training (e.g., aerobic training), and (c) minimal loss of body fat after a period of training (Alcaraz et al. 2011).

To address the excess time devoted to RT, a "novel" high-intensity resistance circuit training (HRC) was presented and showed positive effects on muscular hypertrophy, strength, and power performance while decreasing fat mass, due to the higher total metabolic and cardiovascular demand incurred either during the training session or during the post-training recovery phase (Alcaraz et al. 2008, 2011; Romero-Arenas et al. 2013). Thus, HRC training produces similar positive effects on physical performance and body composition as RT methods but with the advantage of a much shorter training session (~30–40 min) (Alcaraz et al. 2011). Therefore, the addition of systemic hypoxia to HRC is as an interesting strategy to improve athletic performance and further metabolic adaptations using a lower exercise volume and shorter session duration when compared to RTH. However, it is still unclear how the level of hypoxia can impact one's ability to perform an HRC training session. Therefore, in the perspective to develop future HRC training programs in hypoxia, the aim of this study was to determine if an HRC training session under hypoxia produces greater acute effects on physical performance than on blood gases, blood metabolites, and blood electrolyte responses. Our hypothesis was that an HRC training session under high and moderate hypoxic conditions produces negative acute effects on strength and power, with greater blood lactate concentration, and blood electrolytes changes compared to normoxia.

Methods

Design

This study used a comparative, double-blind, randomized crossover design to test the effect of high and moderate hypoxia on metabolic and acid–base balance, blood oxygenation, electrolyte, and half-squat performance acute responses to a HRC training session. Subjects performed a HRC protocol under three conditions of O_2 availability, each on separate occasions in a random order: (1) normoxia (NORM; fraction of inspired oxygen (FiO_2) = 0.21; ~0 m altitude); (2) moderate hypoxia (MH; $FiO_2 = 0.16$; ~2.100 m altitude); and (3) high hypoxia (HH; $FiO_2 = 0.13$; ~3.800 m altitude). During each session (exercise and recovery), subjects wore a mask that was connected to a hypoxic generator (GO2 Altitude hypoxicator, Biomedtech, Australia), which controlled the availability of oxygen. All subjects were blinded to the level of FiO_2 for each trial. No specific familiarization trials were conducted as all participants had the previous experience with HRC training. All

HRC sessions were well tolerated by the subjects, and no one reported any side effects.

Subjects

Twelve healthy, nonsmoking, male subjects (age: 25.1 ± 4.8 years; height: 174.6 ± 5.3 cm; weight: 70.3 ± 6.8 kg; fat mass: 12.1 ± 1.8%; bench press 6RM: 57.1 ± 12.8 kg; half-squat 6RM: 95.9 ± 21.6 kg) participated in this study. The subjects were physically active and experienced with resistance training as they performed resistance exercise on average three times per week in the 4 years prior to the study. Subjects did not have any musculoskeletal disorder and reported not having been exposed to moderate or high altitude in the 3 months prior to the study. All subjects gave signed, informed consent and the study was approved by the University's Institutional Science Ethics Committee.

Procedures

Subjects came to the laboratory a total of four times during a 3-week period, each visit was separated by at least 72 h of recovery under natural conditions (normoxia). In the first visit, body composition was assessed using a segmental multifrequency bioimpedance analyzer (Tanita BC-601, Tanita Corp., Tokyo, Japan) and the load for each subject's 6 repetition maximum (6-RM) for each of the six exercises of the HRC protocol was determined. Three days later, subjects performed the HRC protocol under one of the environmental conditions. The third and fourth training sessions consisted of the same HRC protocol but under the remaining experimental conditions. The order of the conditions for each HRC training session was randomized, and each subject performed the protocol at the same time of day for each visit. In addition, subjects were asked to maintain their habitual diet and hydration status and not to ingest caffeine or alcohol at least 24 h before each testing session nor to perform an exhaustive training bout in the 48 h preceding each visit.

6RM testing

The 6-RM was used to measure muscle strength in each of the following six exercises: bench press, deadlift, biceps flexion (preacher curl), half-squat, triceps extension, and ankle extension (calf raise). Prior to testing, subjects warmed-up on a stationary bicycle for 5 min at 75 W. Afterwards, subjects performed ten repetitions at 50% of the perceived 1-RM, followed by active stretching. Next, standard procedures were used to determine each subject's 6-RM loads for each of the exercises (ACSM 2009; Alcaraz et al. 2008).

Experimental trials of high-resistance circuit sessions (HRC)

Subjects started with a general warm-up, which involved sub-maximal cycling on a stationary bike for 5 min at 75 W while maintaining 75–100 rpm. This was followed by 5 min of active stretching of all major muscle groups. Subjects then performed a specific warm-up, which consisted of three sets of three exercises (bench press, deadlift, and elbow flexion), using the following sequence: ten repetitions at 50% of 6-RM, 1-min rest, eight repetitions at 75% of 6-RM, 2-min rest, and repetitions to failure with a 6-RM load. The 6-RM load was adjusted by ±2.5% if a subject performed ±1 repetitions or by ±5% if a subject performed ±2 repetitions (ACSM 2009). Afterwards, subjects rested for 3-min prior to starting the HRC session. During the last minute of the resting period, subjects were asked to put on the mask and start breathing in the hypoxic air.

In each HRC training session, there were two short circuits (blocks) of three sets, with three different exercises in each set. Resting periods were passive and lasted for 35 s between exercises (which was sufficient time to move safely from one exercise to the next), 3-min between sets within a block, and 5-min between blocks. Subjects lifted loads where only 6 repetitions could be performed (6-RM, ~85–90% of 1-RM). Block 1 was composed of three sets of bench press, deadlift and elbow flexion (preacher curl). Block 2 was comprised three sets of half-squat, triceps extension (French press), and ankle extension (calf raise). Block 1 always proceeded before Block 2 in each HRC training session (Fig. 1). To standardize the protocol, the eccentric phase of each exercise was performed over 3 s (controlled by digital metronome), whereas the concentric phase was performed at maximum velocity (Alcaraz et al. 2008, 2011). These single- and multi-joint exercises were chosen to work both major and minor muscle groups, which were based on ACSM (2009) recommendations. All sessions were supervised by an experienced lifter to ensure

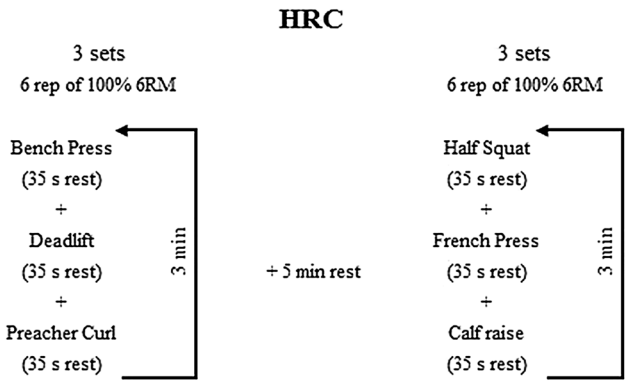

Fig. 1 High-resistance circuit training (HRC) protocol

that volitional fatigue was achieved safely and rest periods were strictly controlled. A linear position transducer (Chronojump, Barcelona, Spain) was attached to the bar and used to measure force and power during each set of the half-squat exercise. The half-squat exercise was chosen only to measure peak force and power, as it activates higher muscle mass than the other exercises and also provides the appropriate conditions to position the encoder for accurate measurement. The rating of perceived exertion (RPE; 6–20 scale) was also obtained immediately following each set.

Finally, finger prick blood extractions at rest and at the end of each block were performed on the right hand, while the subjects stood with their arms flexed. A capillary tube of 65 µl was used to collect the blood sample. The following parameters were analyzed to quantify blood gases, metabolites, electrolytes, and acid–base status (ABL 90 Flex, Radiometer, Westlake, USA.): pH, CO_2 partial pressure (pCO_2; mmHg), O_2 partial pressure (pO_2; mmHg), arterial oxygen saturation (SaO_2), bicarbonate (HCO_3; mmol/l), sodium (Na^+; mmol/l), potassium (K^+; mmol/l), calcium (Ca^{2+}; mg/dl), chloride (Cl; mmol/l), lactate (mmol/l), and glucose (Glu; mg/dl) concentrations.

Statistical analysis

The Statistical Package for the Social Sciences (SPSS for Windows; v.20.0) was used for all statistical analyses. Descriptive statistics (mean ± standard deviation) were calculated. The assumption of normality and homoscedasticity was verified using the Shapiro–Wilks W-test prior to using the parametric tests. A two-way, repeated-measures analysis of variance (group x time) with Bonferroni post hoc analysis was used to investigate differences in variables. Statistical significance was set at $p \leq 0.05$.

Results

Figure 2 and Table 1 show that peak power in the first set of half-squat was lower in HH compared to NORM (−23%) and MH (−20%). Similarly, peak force was also reduced in HH compared to NORM and MH (both −20%). Moreover, lower mean force (−13%) and power (−5%) were observed in HH compared to NORM. In the second set of half-squat, peak power and peak force were also lower in HH compared to NORM (−23 and −20% respectively). No differences were observed between NORM and MH in the first two sets of half-squat, and no significant differences in peak force and power during the final set among the different conditions were observed (Fig. 2). No differences in mean force and power during the first and the final sets were observed among the different conditions. Regarding the subject´s perceived exertion, higher RPE values were observed in HH (Basal: 6.0 ± 0.0; end of block 1: 14.2 ± 3.1; end of block 2: 13.82 ± 2.5) compared to both NORM (Basal: 6.0 ± 0.0; end of block 1: 12.1 ± 1.8; end of block 2: 11.6 ± 1.2) and MH (Basal: 6.0 ± 0.0; end of block 1: 12.6 ± 2.0; end of block 2: 11.9 ± 1.9) in the last block ($p \leq 0.05$). No significant differences among the different conditions were observed in RPE at the end of the first block.

Table 2 shows the results of blood gases at the end of each block of HRC training in the three environmental conditions. There were no significant differences in basal values of pO_2 or in SaO_2. However, at the end of the first block, lower pO_2 and SaO_2 were observed in MH (pO_2: −9%; SaO_2: −4%) and HH (pO_2: −11%; SaO_2: −11%) compared to NORM. Similar statistical trends were also observed in the second block, with pO_2 and SaO_2 tending to be lower in HH compared to NORM ($p = 0.057$ and $p = 0.064$, respectively). Furthermore, reduced pCO_2 was shown in HH compared to NORM in the first (−8%) but not in the second block.

No differences in acid–base parameters were observed in basal conditions (Table 3). At the end of the first block, pH was similar among the different conditions, but higher blood lactate and reduced blood HCO_3^- were observed in HH compared to NORM (+37 and −15%, respectively) and MH (+32 and −14%, respectively). At the end of the second block, blood pH and HCO_3^- were lower in HH compared to NORM (−1 and 18%, respectively) and MH (−1 and −16%, respectively), whereas blood lactate was higher compared to NORM (+44%) and MH (+41%).

No differences in blood electrolytes and glucose concentration were shown under basal conditions. However, Na^+ concentration was higher in HH compared to NORM in the first (+1%) and second (+2%) blocks. Furthermore, Cl^- concentration was greater in HH and MH compared to NORM in the last block (+2 and +1%, respectively) (Table 4). No significant differences in Ca^{2+}, K^+, and glucose concentrations were observed among the different conditions at the end of the first and second blocks (Table 4).

Discussion

To our knowledge, this is the first study that investigated the effects of moderate and high systemic hypoxia on physical performance, blood gases, acid–base balance, and blood electrolytes during a HRC training session. The main findings show that: (i) high ($FiO_2 = 0.13$) but not moderate ($FiO_2 = 0.16$) hypoxia decreased muscular performance in the early sets of a HRC training session; (ii) high hypoxia significantly reduced blood oxygenation in the first but not the second block of the HRC training session; (iii) high but

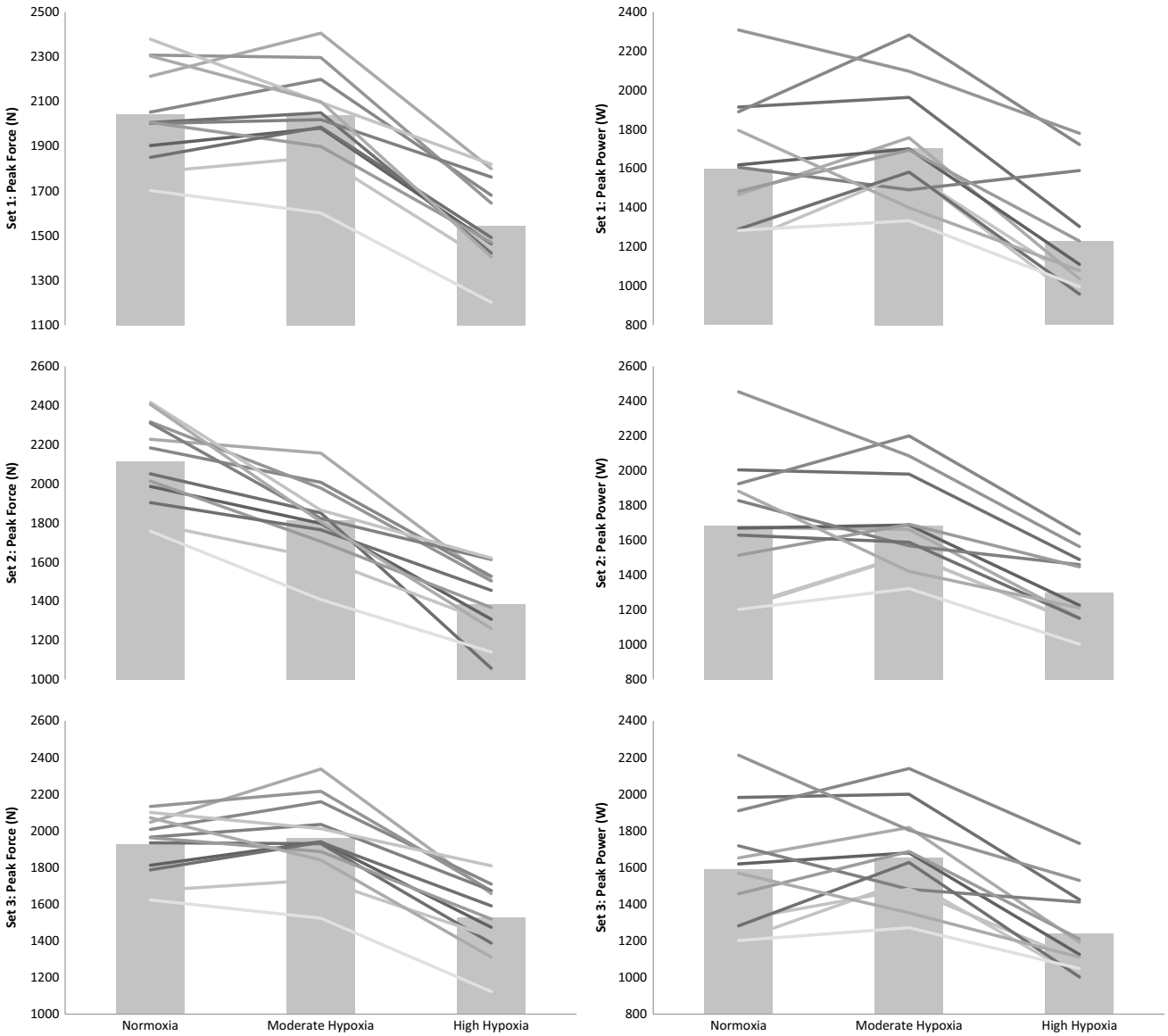

Fig. 2 Individual and mean of peak force and power during each set of half-squat

not moderate hypoxia markedly increased blood lactate and reduced blood HCO_3^- leading to reduced blood pH at the end of the HRC training session; and (iv) high hypoxia induced minor changes to blood electrolytes and blood glucose responses during a HRC training session.

Effect of hypoxia on the performance of a HRC training session

The previous studies have shown that acute exercise performed in hypoxia reduces anaerobic performance (Brosnan et al. 2000; Bowtell et al. 2014). However, other studies have reported no change in peak and mean power between varying conditions of oxygen availability in 5×5 repetitions at 80% 1RM, with 3 min of squat and deadlift (Scott et al. 2015a) and 5×14 repetitions at 50% 1RM with 1 min of rest in bench press and leg press (Kon et al. 2012). We observed a significant decrease in peak force and peak power during the first two sets of the half-squat sequence between HH and NORM conditions and a significant decrease in mean force and power during the second set between HH and NORM. This response is associated to exacerbated perturbations of cellular homeostasis in active muscles. Higher blood lactate concentrations, lower blood pH, and decreased oxygen availability (under HH) suggest increased reliance on glycolysis to maintain ATP supply, indicating a greater anaerobic energy release with acute hypoxia (Scott et al. 2015b). Therefore, when aerobic

Table 1 Mean and peak force and power during each set of half-squat of HRC training under three different conditions

	Peak force (N)			Peak power (w)		
	NORM	MH	HH	NORM	MH	HH
Set 1	2050.3 ± 396.4*	2040.2 ± 316.3$^{\$}$	1705.7 ± 669.8	1597.0 ± 561.2*	1544.7 ± 461.1$^{\$}$	1228.4 ± 603.6
Set 2	2114.0 ± 438.6*	1815.4 ± 682.8	1687.8 ± 845.3	1684.1 ± 561.5*	1387.1 ± 655.9	1298.4 ± 711.6
Set 3	1926.0 ± 713.2	1962.2 ± 717.3	1656.3 ± 882.4	1593.4 ± 680.6	1529.0 ± 629.4	1240.3 ± 726.5
	Mean force (N)			Mean power (w)		
	NORM	MH	HH	NORM	MH	HH
Set 1	1617.9 ± 338.2	1645.43 ± 347.6	1515.3 ± 561.5	751.5 ± 211.9	737.0 ± 220.3	728.0 ± 300.6
Set 2	1625.1 ± 272.0*	1537.4 ± 573.9	1425.6 ± 547.9	625.7 ± 325.2*	599.9 ± 266.6	596.1 ± 328.3
Set 3	1363.6 ± 554.7	1311.7 ± 667.4	1301.9 ± 700.6	721.3 ± 157.0	702.7 ± 266.5	642.1 ± 262.5

Mean ± Standard deviation; NORM = normoxia; MH = 0.16% FiO_2; HH = 0.13% FiO_2

*Significant differences between normoxia and high hypoxia, $^{\$}$Significant differences between moderate and high hypoxia, *$p < 0.05$

Table 2 Blood gases and arterial oxygen saturation (SaO_2) values of HRC training under three different conditions

	pCO_2 (mmHg)			pO_2 (mmHg)			SaO_2 (%)		
	NORM	MH	HH	NORM	MH	HH	NORM	MH	HH
Basal	39.6 ± 2.5	39.6 ± 2.4	39.1 ± 2.8	78.5 ± 7.9	76.6 ± 11.6	77.5 ± 10.4	98.1 ± 0.2	98.1 ± 0.3	98.2 ± 0.4
Block 1	40.5 ± 3.2**	39.6 ± 3.1	37.4 ± 2.7	75.5 ± 13.3**	68.4 ± 4.7††	67.0 ± 12.4	94.4 ± 2.9*	91.0 ± 2.4†††	84.1 ± 5.6$^{\$\$}$
Block 2	35.3 ± 2.8	34.5 ± 2.8	33.8 ± 4.2	74.2 ± 15.0	70.8 ± 18.7	69.8 ± 11.5	93.8 ± 3.3	92.7 ± 6.0	89.9 ± 6.5

Mean ± Standard deviation; NORM = normoxia; MH = 0.16% FiO_2; HH = 0.13% FiO_2; pCO_2 = carbon dioxide pressure; pO_2 = oxygen pressure

*Differences between normoxia and high hypoxia, †Differences between normoxia and moderate hypoxia, $^{\$}$Differences between moderate and high hypoxia, *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$; $^{\dagger\dagger}p < 0.01$; $^{\dagger\dagger\dagger}p < 0.001$; $^{\$\$}p < 0.01$

Table 3 Acid–base values of HRC training under three different conditions

	HCO_3 (mmol/l)			La (mmol/l)			pH		
	NORM	MH	HH	NORM	MH	HH	NORM	MH	HH
Basal	25.9 ± 1.2	26.0 ± 1.1	26.1 ± 1.3	2.3 ± 0.8	2.0 ± 0.7	2.3 ± 0.9	7.42 ± 0.01	7.41 ± 0.06	7.42 ± 0.02
Block 1	19.0 ± 2.9*	18.9 ± 2.2	16.2 ± 2.6$^{\$}$	8.7 ± 2.9*	9.0 ± 3.6	11.9 ± 2.1$^{\$}$	7.33 ± 0.05	7.33 ± 0.04	7.27 ± 0.05
Block 2	19.5 ± 3.5*	19.1 ± 2.7	16.0 ± 2.9$^{\$}$	8.6 ± 3.5*	8.8 ± 3.3	12.4 ± 2.6$^{\$}$	7.35 ± 0.06*	7.35 ± 0.05	7.28 ± 0.07$^{\$}$

Mean (standard deviation); NORM = normoxia; MH = 0.16% FiO_2; HH = 0.13% FiO_2; *La* lactate

*Differences between normoxia and high hypoxia, $^{\$}$Differences between moderate and high hypoxia, *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

metabolism is not capable of meeting ATP demand, the breakdown of phosphocreatine and activation of anaerobic glycolysis can be further elevated to meet the short-term requirements for ATP (Calbet et al. 2003).

Furthermore, an increase in the rate of PCr hydrolysis rate can also occur during hypoxic conditions, resulting in an increase in Pi. Moreover, an increase in intracellular acidosis due to glycolytic pyruvate production results in elevated lactate, which, in turn, can contribute to muscular fatigue (Bowtell et al. 2014). Thus, during hypoxia, there is increased reliance on non-aerobic metabolism to compensate for the limitation in aerobic ATP production (Calbet et al. 2003). In addition, limited oxygen availability and brief rest intervals affect the muscle's ability to maintain the balance between ATP breakdown and ATP production, thereby limiting PCr recovery as well as cellular recovery after each exercise bout (Hogan et al. 1999). Furthermore, increased activities of cellular processes, such as ion pumps, try to achieve homeostasis during rest intervals require ATP, much of which is derived from aerobic glycolysis (Colliander et al. 1988). Thus, lower muscular performance during HH in the first two sets of half-squat exercises is likely due to an inadequate supply of ATP from aerobic and non-aerobic metabolism to meet the demand, as a consequence of limited O_2 availability, with ensuing accumulation of

Table 4 Electrolytes and glucose values of HRC training under three different conditions

	Ca^{2+} (mg/dl)			Na^{+} (mmol/l)		
	NORM	MH	HH	NORM	MH	HH
Basal	5.0 ± 0.2	5.1 ± 0.4	5.2 ± 0.4	142.3 ± 2.4	143.8 ± 2.8	144.0 ± 2.0
Block 1	5.1 ± 0.1	5.1 ± 0.1	5.1 ± 0.1	145.5 ± 2.0**	146.5 ± 1.4	147.5 ± 1.4
Block 2	5.1 ± 0.2	5.1 ± 0.3	5.1 ± 0.3	144.5 ± 2.3*	146.2 ± 2.3	147.1 ± 1.5
	K^{+} (mmol/l)			Cl^{-} (mmol/l)		
	NORM	MH	HH	NORM	MH	HH
Basal	5.2 ± 0.8	4.9 ± 0.6	5.0 ± 0.9	108.6 ± 3.1	107.6 ± 2.3	108.1 ± 1.4
Block 1	5.3 ± 0.8	4.8 ± 0.7	4.9 ± 0.6	108.2 ± 2.2	109.9 ± 1.1	109.9 ± 2.7
Block 2	5.1 ± 0.6	5.2 ± 0.6	4.9 ± 0.4	107.7 ± 1.8*	109.3 ± 1.0†	109.6 ± 2.4

Glucose (mg/dl)

	NORM	MH	HH
Basal	106.6 ± 22.4	106.0 ± 14.1	103.4 ± 6.3
Block 1	99.7 ± 6.0	98.2 ± 7.1	100.1 ± 13.6
Block 2	102.2 ± 6.5	99.1 ± 6.3	107.4 ± 9.2

Mean ± Standard Deviation; NORM = normoxia; MH = 0.16% FiO_2; HH = 0.13% FiO_2

*Differences between normoxia and high hypoxia, †Differences between normoxia and moderate hypoxia, $*p < 0.05$

metabolic products and ionic imbalances that together impair muscle function.

For the third set, peak and mean power and force were not different among the different conditions, suggesting that during the last set, half-squat performance was not only dependent on O_2 availability but more affected by the accumulation of cellular metabolites (i.e. Pi or H^+). The imbalance in cellular electrolytes also limits performance under hypoxic conditions compared to normoxia. These results are in accordance with those obtained recently by Scott et al. (2016). They showed an increase in the concentration of metabolic products that promote higher levels of muscular fatigue, which induces the activation of additional motor units and leads to higher muscle activation during hypoxic resistance exercise. Thus, given that metabolic acidosis inhibits muscle contractility and subsequently promotes the recruitment of additional high-threshold motor units, these results suggest the presence of higher levels of lactate under hypoxic conditions of the current study.

As expected, RPE values were significantly higher at the end of the HH training session than in MH and NORM conditions, suggesting that the HH session was perceived as more difficult than MH and NORM. These results are in accordance with Alvarez-Herms et al. (2015b), who have shown significant differences in RPE score between high hypoxia ($FiO_2 = 13.5\%$) and normoxia during a series of six consecutive jumps (lasting 15 s with rest periods of 3 min). In contrast, Scott et al. (2015a) showed no significant differences in RPE scores during a high-intensity resistance training session (5 set of 5 repetitions at 80% of 1RM with 3-min rest between sets) using the same hypoxic levels as our study. These conflicting results can be explained by the different types of training (traditional vs. high circuit training) and the slight differences in intensity level (80 vs. 85% 1RM). Nevertheless, perceived exertion is a useful variable to confirm the intensity level of the training protocol, as demonstrated by this study.

Effect of hypoxia on blood oxygenation during an HRC training session

Oxygenation levels (pO_2) was higher in NORM compared to MH and HH at the end of the first block. The previous studies are in accordance with our findings showing increased muscle deoxygenation in hypoxic conditions during maximal contractions (Richardson et al. 2006). This observation is in line with the known effect of hypoxia on the acute ventilatory, cardiac, and vascular responses to exercise, all of which contribute to maintain adequate O_2 supply to tissues despite progressive pO_2 reduction (Cerretelli and Samaja 2003). Nevertheless, the lower pO_2 observed in the exercising muscle cells in hypoxia likely acts as a potent signal to trigger specific muscle responses to training (Bowtell et al. 2014). Interestingly, a significant decline in SaO_2 values was observed, which was related to the increased hypoxia level at the end of the first block. Larger decreases in SaO_2 during hypoxia are associated with greater anaerobic energy production (Alvarez-Herms et al. 2015b; Calbet et al. 2003). The previous studies have showed similar findings after anaerobic exercise under hypoxia (Scott et al. 2015a; Alvarez-Herms et al. 2015b). However, in our study, no significant differences in pO_2, pCO_2, and SaO_2 were observed in the second block. This is in agreement with a recent study by Scott et al., (2016), who obtained lower levels of SaO_2 in hypoxia than in normoxia but no difference in muscle oxygenation status between conditions during moderate-load resistance training (3 sets of 10 repetitions at 60% of 1RM; $FiO_2 = 16\%$). According to these authors, these findings can be explained by the type of resistance exercise applied during the training session and the level of hypoxia employed (Scott et al. 2016). Further research to elucidate this point is needed.

Effect of hypoxia on blood acid–base balance during a HRC training session

The previous studies showed no significant differences in lactate values during an explosive strength session (Alvarez-Herms et al. 2015b) under normoxia or hypoxia or after 30–40 s of supramaximal exercise (McLellan et al. 1990). In contrast, our study showed significantly higher blood lactate concentrations at the end of the two blocks with HH compared to MH and NORM. Our results agree with those shown by Calbet et al. (2003) in cyclists using an anaerobic test. A reduction in oxygen availability can explain the higher lactate levels observed during intense exercise and reflect a greater contribution of anaerobic glycolysis to supply ATP (Calbet et al. 2003).

In addition, acid–base balance is an important limiting factor in physical exercise because of its mechanistic role in regulating energy metabolism and ion homeostasis (Juel 2008). During intense muscle activity, an increase in cellular production of lactate and H^+ greatly contributes to acidosis (Juel 2008). The removal of H^+ and lactate via monocarboxylate cotransporters (Juel 1998), as well as the presence of carbonic anhydrase which affects the rate of H^+ and HCO_3^- transport (Zoll et al. 2006), works together to help regulate pH in the muscle. Previously, Buchheit et al. (2012) indicated that acute high-intensity interval training under hypoxia (2400 m of simulated altitude) modifies skeletal muscle acid–base balance response via increases in H^+ fluxes from the muscle to the blood, resulting in a decrease in blood pH and HCO_3^-. Similarly, we found that blood pH was lower in HH compared to NORM and MH (−1% in both cases) at the end of the second block. Moreover, blood HCO_3^- was reduced in HH compared to

NORM and MH at the end of the first and second blocks (−14 to −18%), indicating that an HRC training session under hypoxic conditions produces a higher muscle buffering response to reduce the pH fluctuations and to maintain blood pH near the physiological level (Juel 2008).

Effect of hypoxia on the blood electrolytes response to an HRC training session

Intense exercise increases lactic acid and H^+ concentrations and induces pronounced perturbations in Na^+, K^+, and Cl^- (Sejersted and Sjogaard 2000). These electrolyte changes are linked with fatigue and contribute to the decrease in muscle force and performance (McKenna et al. 2008). Similarly, intense fatiguing contractions have been shown to induce cellular K^+ efflux and Na^+ and Cl^- influx, causing pronounced perturbations in interstitial K^+ and Na^+ concentrations (McKenna et al. 2008). Furthermore, Na^+ and Cl^- ions can affect muscle function and fatigue and can also modulate muscle H^+ via the strong differences in plasma ion (Cairns et al. 2004). In addition, it has been reported that a net Cl influx (from plasma to muscle) occurs during intense large muscle mass exercise, indicating that Cl^- ions are taken up by the muscle (Mckenna et al. 1997). In addition, HCO^-/Cl^- exchange across the erythrocyte (RBC) membrane (chloride shift) transport plays a key role in maintaining electrical balance across the red cell membrane and producing a buffering response of RBC to maintain the pH near the physiological level (Böning et al. 2007). These chloride responses could explain the significantly higher blood Cl^- concentration observed in this study in HH and MH compared to NORM in the last block. Therefore, our results show that Cl^- ions were altered with acute strength training under hypoxia.

Moreover, we observed that blood Na^+ concentration significantly increased in HH compared to NORM at the end of each block. This small elevation in plasma Na^+ has been observed with exercise (Street et al. 2005), which suggests a higher Na^+ release by the contracting muscle causing an increase in plasma Na^+ concentration (Sostaric et al. 2006) under high hypoxic conditions. Thus, higher blood electrolyte concentrations (Cl^- in HH and MH vs. NORM and Na^+ in HH vs. NORM) likely contribute to higher fatigue under hypoxic environment. Although the development of severe fatigue is multifactorial, ionic interactions appear to play an important role in the physiological and performance responses to intense exercise (e.g., HRC training session) (Cairns et al. 2004).

Practical application

This research contributes to the understanding of the acute physiological response of HRC training session under different levels of hypoxia. It provides evidence for its potential applicability to sports that use resistance strength training in their training programs. HRC training sessions performed in hypoxia do not produce the similar acute responses as the same training session performed under normoxic conditions.

These differences must be taken into account when designing and optimizing the training load for short-term adaptations. Therefore, coaches must be careful when designing resistance training sessions under high hypoxic conditions, as it is more stressful than moderate hypoxia or normoxia and can affect the training stimuli or the goal of the training session. The subjects of this study were well-trained athletes, experienced in resistance training. Thus, the findings of this study are more applicable to resistance-trained athletes who aim to enhance strength performance than to other populations who remained to be tested. Nevertheless, due to the high response of glycolysis to HH training, the results of this study apply to other athletes such as team sports players, sprinters, or endurance athletes who may want to optimize their strength training sessions using shorter duration.

Conclusions

The results of this study showed that HRC performed in high, but not moderate hypoxia decreased muscular performance and increased the rating of perceived exertion. HRC under high hypoxic conditions also reduced blood oxygenation, increased blood lactate, and reduced blood HCO_3^- and pH. In addition, high hypoxia induced minor changes to blood electrolyte and blood glucose responses to an HRC training session. Further research is needed to examine neural and endocrine responses, as well as the morphological and strength adaptations to HRC under hypoxic conditions. In addition, more work is needed to clarify if this training method can promote hypertrophic, metabolic, and strength gains.

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