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ABSTRACT

PURPOSE: We compared psycho-physiological responses to six repeated-sprint sessions in normobaric hypoxia (RSH) and normoxia (RSN) in team-sport athletes during a 2-wk ‘live high-train low’ training camp. **METHODS:** While residing under normobaric hypoxia (≥ 14 h.d⁻¹; FiO₂ 14.5-14.2%), twenty-three lowland elite field hockey players performed, in addition to their usual training, six sessions (4 × 5 × 5-s maximal sprints; 25 s passive recovery; 5 min rest) under either normobaric hypoxia (FiO₂ ~14.5%) or normoxia (FiO₂ 21%). Sprint 1 and 5 times, physiological strain [heart rate (HR), arterial oxyhemoglobin saturation (SpO₂)] and perceptual responses (overall peripheral discomfort, difficulty breathing and lower-limb discomfort) were monitored. **RESULTS:** During the first session, HR increased across sets (P<0.001), independently of the conditions while SpO₂ was globally lower (P<0.001) for RSH (averaged value: 91.9±1.2%) vs. RSN (96.9±0.6%). Thereafter, SpO₂ and HR remained similar across sessions for each condition. While first sprint time remained similar, last sprint time and fatigue index significantly decreased across sets (P<0.01) and sessions (P<0.05) but not between conditions. Ratings of overall perceived discomfort, difficulty breathing and lower-limb discomfort were higher (P<0.05) in RSH vs. RSN at the first session. During subsequent sessions, overall perceived discomfort (time (P<0.001) and condition (P<0.05) effects), difficulty breathing (time effect; P<0.001) and lower-limb discomfort (condition (P<0.001) and interaction (P<0.05) effects) values decreased to a larger extent in RSH vs. RSN. **CONCLUSION:** Despite higher hypoxia-induced physiological and perceptual strain during the first session, perceptual responses improved thereafter in RSH so as not to differ from RSN. This indicates an effective ‘acclimation’ and tolerance to this innovative training.

Key words: Acclimation, altitude, high-intensity exercise, team sports, perceived peripheral discomfort, ratings of perceived exertion.

Introduction

A growing number of team-sport athletes are using altitude training with the belief that it can promote greater physiological adaptations compared to similar training conducted near sea level, in turn further improving in-game physical performance¹. Time constraints during the yearly competitive calendar render the implementation of ‘traditional’ altitude training (*e.g.*, 3 weeks of ‘live high-train high’ at natural altitude or ‘live high-train low’ in altitude dormitories) challenging. Innovative hypoxic methods such as repeated-sprint training in hypoxia (RSH), defined as the repetition of several short (≤ 30 s) ‘all-out’ exercise bouts in deprived-O₂ environments interspersed with incomplete recoveries (exercise-to-rest ratio $\leq 1:5$)², encounter an expanding popularity in the team-sport community³. By adding hypoxic stimuli to repeated-sprint training (2-6 weeks), larger benefits have been reported on in-game physical performance-related variables [*e.g.*, Yo-Yo intermittent recovery and/or repeated-sprint ability (RSA) tests] when compared to similar training in normoxia (RSN)^{4,5}.

In the available literature, most of RSH studies are laboratory-based through the use of standard hypoxic facilities (*e.g.*, O₂-filtration chambers/tents or breathing hypoxic mixtures with a mask) while sprinting on a cycle ergometer⁶, a treadmill⁵, over-ground using short shuttles⁷ or when treadmill and over-ground runs were combined⁴. At top level, coaches and athletes are naturally looking for sport-specific exercises with a direct relevance (high ecological validity) to their programmes. New technologies such as the mobile inflatable hypoxic marquees⁸ now offer advancements of hypoxic training practical applications, notably with the opportunity to train (*e.g.*, repeated sprinting over ~30 m, small-sided games, resistance training in hypoxia) under field-based hypoxic conditions (*e.g.*, on natural/artificial grass in footballers wearing their football cleats). We have recently demonstrated superior effects of training inside these marquees using a RSH paradigm as compared to RSN, when combined with residing at moderate simulated altitude (≥ 14 h.d⁻¹ at

a simulated altitude of 2800–3000 m), for improving RSA in elite field-hockey players during an in-season 2-wk training camp ⁹.

Determining the day-to-day athletes’ tolerance to the prescribed training load is fundamental for optimal periodization and adaptations (*i.e.*, maximize performance gains and minimize overreaching or injury risks) ¹⁰. In team-sports, heart rate (HR) and rate of perceived exertion (RPE) are common indicators of players’ degree of physical strain ¹¹. Although the RPE is a recognized marker of subjective assessment of exercise intensity, particularly if this is known by the participants ¹², we have recently reported that the perception of peripheral discomfort is independent from the sense of effort during sub-maximal and maximal cycling bouts, despite a hypoxic-induced exaggeration of the physiological responses and higher ratings of perceived difficulty breathing ¹³. Single sprint performance requires, by nature, a maximal ‘sense of effort’ or ‘perception of effort’ (*i.e.*, depending on the definition used by authors ¹⁴), but produces quite moderate perceived discomfort values due to mild sensory inputs from heart, lungs and active muscles ¹⁵. However, when such maximal efforts are repeated (*i.e.*, at a remaining maximal ‘sense of effort’) peripheral discomfort increases substantially ¹⁵. While this highlights the necessity to distinguish and individually report the accompanying perceptions of perceived discomfort (*i.e.*, overall peripheral discomfort, difficulty breathing and lower-limb discomfort), our understanding of responses to RSH training so far is limited to mucosal immune function ¹⁶.

Our aim was therefore to compare changes in sprint performance along with physiological [HR and arterial oxyhemoglobin saturation (SpO₂)] and perceptual responses (RPE, overall peripheral discomfort, difficulty breathing and lower-limb discomfort) to six repeated-sprint sessions in normobaric hypoxia (RSH) and normoxia (RSN) in elite team-sport players, conducted during a 2-wk ‘altitude’ training camp.

Methods

Participants

After being informed of the potential risks and benefits involved, twenty-three lowland elite field hockey players (Table 1) provided their written consent to participate in this study. The experiment was approved by the Anti-Doping Lab Qatar institutional review board (Agreement SCH-ADL-070) and conformed to the current Declaration of Helsinki guidelines. Participants practiced $\sim 7.0\text{-}9.0$ h.wk⁻¹ of training (5-7 weekly sessions) and competitive matches (1-2 matches per week) during the season. Training sessions included aerobic, anaerobic and strength training in isolation or combined with sport-specific training (*i.e.*, technical and tactical skills). All participants were born and raised at altitudes < 1000 m and had not travelled to elevations > 2000 m for more than 48 h in the 6 months prior to the study.

Experimental design/Protocol

This study is part of a large experiment conducted during an in-season competition phase (*i.e.*, December-February, including the winter break) and additional details about the research design can be found elsewhere⁹. Participants were matched for initial fitness level and playing position and randomly assigned to a normobaric hypoxia (RSH, n = 11) or normoxia (RSN, n = 12) group. In addition to their usual field hockey practice, all participants undertook six repeated-sprint training sessions (at least 36 h apart), while residing under normobaric hypoxic conditions (≥ 14 h.d⁻¹ at 2800–3000 m; FiO₂ 14.5-14.2%), during a 2-wk in-season training camp.

Sprint performance was evaluated during RSH/RSN training sessions (Fig. 1). Before and after each of the four sets of each training session (see below), several variables were collected to assess psycho-physiological strain associated with the completion of the

proposed intervention: HR and SpO₂ together with RPE, overall perceived peripheral discomfort, lower-limb discomfort and difficulty breathing.

Specific repeated-sprint training

Each session lasted ~50 min, including a 15-min warm-up [*i.e.*, low-intensity running with athletic (*e.g.*, skipping, high knee runs) and acceleration drills (*e.g.*, shorts bursts of progressive accelerations and maximal 10-m straight-line sprints)], the repeated-sprint training routine and a 10-min recovery phase (*i.e.*, cool down and passive stretching). Specifically, the repeated-sprint training routine included four sets of 5 × 5-s maximal sprints in alternating directions interspersed with 25 s of passive recovery with 5 min of standing rest between sets. With an exercise-to-rest ratio of 1:5, up to six players trained together with an invariant execution order. Practically, each participant departed for sprinting every 5 s, as controlled by audio beeps. Because of space restriction inside the marquee, they had to decelerate on a short distance (*i.e.*, ~10 m). During all training sessions, participants were constantly reminded to assume a standing ready position for 2 s before starting, and were vigorously encouraged to perform ‘all-out’ efforts in trying to reach peak acceleration and to maintain the highest possible running speed for every sprint.

Training sessions were completed on an indoor synthetic grass inside a mobile inflatable simulated hypoxic equipment (Altitude Technology Solutions Pty Ltd, Brisbane, Queensland, Australia), as previously described⁸. Briefly, it comprised a polyvinyl chloride inflatable running lane tunnel (length = 45 m, width = 1.8m and height = 2.5m) and a state-of-the-art hypoxic trailer (55 kW screw compressor), generating over 3000 Lpm of hypoxic air with FiO₂ between 21% and 10% (a simulated altitude up to 5100 m). Temperature and humidity were maintained constant at ~25°C and ~55% relative humidity, owing to the exercise-generated humidity compensated by the dry air supplied inside the marquee. For

RSH, ambient air was mixed with nitrogen (from pressurized tanks) to reduce FiO_2 to ~14.5% in order to simulate an altitude of ~3000 m. While up to six participants trained simultaneously in the marquee (Fig. 1), they were all assigned to different teammates during the six sessions, in order to reinforce motivation. For blinding purposes, participants were told (head coach request) that they were all training under hypoxic conditions but had no accurate information about actual simulated altitude levels. The total hypoxic exposure corresponded exactly to the training duration, *i.e.*, with RSH / RSN participants breathing hypoxic / normoxic air from the time of entrance to exit of the marquee.

Sprinting timing

During training, the straight-line sprints were performed back and forth. Sprint performance (timing) during the first and the last (sprint 1 and 5, respectively) sprint of each of the four sets was measured to the nearest 0.01 s using photocells connected to an electronic timer (Polifemo Radio light, Microgate, Bolzano, Italy). Timing gates were positioned at 0- and 20-m distance intervals, with additional turned-off gates placed at a distance of ~35 m (corresponding to ~5-s duration sprint) in order to make sure participants produced maximal effort (*i.e.*, no deceleration) throughout each sprint bout. Performance fatigability (*i.e.*, percent increase in time between the fastest and slowest sprints) was calculated using the following formula:

$$\text{Fatigue index (FI, \%)} = [(\text{sprint 5} - \text{sprint 1}) / \text{sprint 1}] \times 100^{17}.$$

Physiological responses to exercise

HR and SpO₂

HR and SpO₂ were monitored (*i.e.*, participants were blinded to values), respectively, via a wireless Polar monitoring system (Polar Electro Oy, Kempele, Finland) and non-invasive pulse oximeter using a finger probe (GO2TM Achieve 9570-A, Nonin, Plymouth,

MN, USA). These values were first recorded prior to and immediately upon completion of the standardized warm-up (*i.e.*, 15 min following entry to the mobile hypoxic marquee). Afterwards, physiological responses were collected exactly 1 min and 4 min into the 5-min resting period following each of the four repeated sprints sets.

Perceived Exertion Measures

Rating of perceived exertion and training load

As an index of overall feeling of subjective perceived exertion, session RPE was assessed within 10 min of completing each training session with the 6-20 Borg scale. Training loads (arbitrary units, a.u.) were calculated for all players as total training session duration (min) \times session RPE¹⁸.

Perceived discomfort scales

Based on modified Borg CR10 scales, ratings of overall perceived peripheral discomfort, perceived lower-limb discomfort and perceived difficulty breathing¹³ were recorded (invariant order) at the same time intervals as physiological responses (*i.e.*, 1 min and 4 min after each of the four exercise sets). Beforehand, players were thoroughly instructed that the ‘perceived discomfort’ scales, visible to participants at all times, were used to evaluate their subjective perception of (i) overall peripheral discomfort (*‘How uncomfortable do you feel overall?’*), (ii) specific lower-limb discomfort (*‘How uncomfortable do your legs feel?’*) and (iii) difficulty breathing (*‘How uncomfortable does it feel to breathe?’*) (Fig. 1).

Statistical analysis

Normal distribution was examined using Kolmogorov-Smirnov normality test. Separate two-way analyses of variance (ANOVA), with repeated measures for condition (RSH vs. RSN), time [(session 1, 2, 3, 4, 5 vs. 6) or (set 1, 2, 3 vs. 4)] and possible interaction

between these two factors was conducted. When ANOVA revealed a significant effect, post hoc comparisons were made using the Bonferroni method. Averaged values (mean of the 6 sessions) and percentage changes (relative to session 1) were compared between the two groups using a one-way ANOVA. For each ANOVA, effect size (ES) was calculated (Cohen's *d*) with the following criteria: an ES of < 0.2 is classified as a 'trivial', $0.2-0.4$ as a 'small', $0.5-0.7$ as a 'moderate' and > 0.8 as a 'large' effect. Data are presented as means \pm SD. Statistical analyses were performed using Sigmaplot 11.0 software (Systat Software, San Jose, CA, USA). Statistical significance was set at $P < 0.05$.

Results

Prior to entering the mobile hypoxic marquee, HR and SpO₂ did not differ among conditions ($P = 0.06$ and 0.12 , respectively) and between sessions ($P = 0.15$ and 0.23) (Fig. 2). Following the 15-min warm-up period, there was a hypoxia-induced increase in HR and decrease in SpO₂ values (both $P < 0.001$). Sprint performance outcomes along with psychophysiological responses to RSH and RSN are presented in Figs. 2 and 3 with the corresponding statistical analyses (*i.e.*, averaged data of all sets) displayed in Table 2.

During the first session, SpO₂ remained depressed in RSH ($P < 0.001$) while HR increased across sets ($P < 0.001$), independently of the conditions (Fig. 2). Thereafter, both SpO₂ and HR remained similar across sessions for each condition. While first sprint time was similar between conditions, sets and sessions (Fig. 2 and Table 2), last sprint time and sprint 1-5 fatigue index significantly decreased across sets and sessions (time effect, $P < 0.01$ and < 0.05 , respectively) leading to significant lower averaged sprint 5 time values ($P < 0.001$) in RSH compared with RSN (Fig. 2 and Table 2). At the first session, ratings of overall perceived discomfort, difficulty breathing and lower-limb discomfort were higher ($P < 0.05$) in RSH compared with RSN (Fig. 3 and Table 2), with an effect of time ($P < 0.001$) on

ratings of overall perceived discomfort and difficulty breathing. During subsequent sessions, overall perceived discomfort (time and condition effects; $P < 0.001$ and 0.05 , respectively), difficulty breathing (time effect; $P < 0.001$) and lower-limb discomfort (condition and interaction effects; $P < 0.001$ and 0.05 , respectively) values decreased to a larger extent in RSH *vs.* RSN (Fig. 3 and Table 2).

While session RPE did not differ between conditions at the first session ($P = 0.10$), perceived exertion was significantly lower ($P < 0.05$) in RSH compared with RSN, with a significant different magnitude of change ($P < 0.05$) (Table 2). Statistical analysis revealed no main effect of condition ($P = 0.93$), time ($P = 0.80$) and interaction ($P = 0.18$) for training load (pooled values for the six sessions: 592 ± 68 and 593 ± 69 in RSH and RSN, respectively) monitored during the 2-wk period.

Discussion

The main findings of the present investigation were as follows: (i) despite a higher hypoxia-induced physiological stress (*i.e.*, SpO_2) and higher-perceived ratings, sprint performance responses were comparable for RSH and RSN at the first session; and (ii) as training sessions progressed, perceptual responses improved for RSH, indicative of an effective altitude ‘acclimation’. Effective tolerance to this innovative RSH training was accompanied by an improved repeated sprints performance (*i.e.*, lower averaged sprint 5 time).

Sprint performance

Single sprint performance of short duration (< 10 s) is generally well-preserved under hypoxic (2000-3800 m) *vs.* normoxic conditions¹⁹. In line with these observations, in our study, the first sprint performance was also similar between conditions, sets and sessions. This is presumably due to an increase in the rate of anaerobic energy release, which

compensates for the limited oxidative contribution when sprinting in O₂-deprived environments²⁰. As expected, as sprints were repeated, a significant time effect was found for last sprint time and performance fatigability, which may primarily relate to a decline in adenosine triphosphate resynthesis from phosphocreatine hydrolysis and non-oxidative glycolysis¹⁷. However, whereas hypoxia has been associated with earlier and larger RSA performance decrements^{19, 21}, in our study, no difference was observed between conditions. These discrepant findings may result from the smaller number of sprints repetition within a set and/or the shorter distances monitored (*i.e.*, first and last effort of 5 repetitions over 20 m) *vs.* those actually completed (5-s sprints or ~30 m sprints distance). This methodological choice was guided by the ease of comparisons with our companion paper where a 8 × 20 m RSA test was used during pre- and post-tests⁹. Regardless, the significant improvement in sprint 5 performance over successive sessions confirms that RSH (and to a lesser extent RSN) is an effective time-efficient training strategy to improve RSA performance²². This corroborates previous findings from our research group using isolated-⁶, mixed-⁴ or combined-RSH training⁹.

Physiological and perceptual responses

In the present study, exposure to hypoxia during training (*i.e.*, RSH) induced lower SpO₂ values compared to similar training near sea level in normoxia (*i.e.*, RSN), also in line with available RSH literature^{4, 5, 19}. Lower SpO₂ values are generally accompanied by increased values for HR, minute ventilation and O₂ debt¹⁹. Although HR is not always a valid marker for prolonged anaerobic exercise, in the present study, HR increased with exercise and across sets. When ten trained male team-sport athletes completed 3 sets of repeated sprints (9 × 4 s) on a non-motorized treadmill at sea level and at simulated altitudes of 2000, 3000, and 4000 m, total work across all sets was highest at sea level with also

correspondingly lower mechanical performance at each successive altitude ²¹. In line with our study, however, HR values did not differ between conditions. Potentially different time points for measuring HR values during recovery periods post-exercise may have led to different HR readings between conditions.

In addition to an exacerbated rate of peripheral fatigue development ^{23, 24}, an acute repeated-sprint exercise in hypoxia is known to elevate subjective fatigue sensations compared to normoxia ^{13, 24}. Accordingly, the higher overall session RPE and difficulty breathing perceived in RSH compared with RSN at the first session might relate to the higher ventilation response that is generally observed for any given absolute exercise intensity in hypoxia ²⁵. This was further accompanied by higher leg discomfort observed in RSH compared with RSN at the first session, which possibly reflects the greater accumulation of metabolic by-products (*e.g.*, blood lactate concentration) ²⁶ when repeated sprints were performed under acute normobaric hypoxia ^{17, 19, 21, 23, 24}. Although blood lactate concentration was not measured here, larger values under RSH may have stimulated the sensory feedback from group III/IV muscle afferents to a larger extent ²⁷, as previously observed after arterial occlusion ²⁸. Bearing in mind that these neurons play a twofold role – *i.e.*, limiting motoneuronal output, thereby modulating central fatigue; and/or enhancing circulatory and ventilatory response, thereby preventing peripheral fatigue – quantifying their influence in the regulation of the cardiorespiratory response to RSH deserves further attention. That said, the higher perceptual responses (RPE, overall peripheral discomfort, difficulty breathing and lower-limb discomfort) observed in RSH *vs.* RSN at the first session (acute effect) were attenuated from the second session onwards with essentially similar values in reference to RSN over the remaining five sessions (‘acclimation’ effect). This is contrasting with previous findings demonstrating that normobaric hypoxia and exercise when combined exacerbate perceived levels of exertion ²⁵. This discrepancy may be due to

difference in exercise mode between studies (*i.e.*, repeated sprints *vs.* Wingate-based high-intensity training *vs.* 4–6 × 30 s of ‘all-out’ cycling efforts). Regardless, the attenuated perceptual responses reported in RSH after the initial session are likely due to an improved tolerance to the hypoxic stimulus, possibly via an increased hypoxic ventilatory response mediated by improved chemosensitivity^{29, 30}. One cannot rule out that the prolonged exposure to hypoxia during the nights has improved the chemosensitivity and therefore accelerated the ‘acclimation’ during the RSH sessions.

Despite lower SpO₂ values in RSH *vs.* RSN, ratings of RPE, overall peripheral discomfort and difficulty breathing remained rather low even under hypoxic exposure (*i.e.*, values never exceeding 7 on CR10 scales), which may explain why we failed to report significant difference between the two conditions after the first ‘acclimation’ session. On the one hand, it could be argued that longer sprints, shorter recoveries or combination of both (*i.e.*, more intense exercise-to-rest ratios) and/or the use of more severe hypoxic conditions [also causing larger performance fatigability;²¹] might lead to significant difference between RSH and RSN. On the other hand, this also suggests that a manipulation of such training-related factors (*i.e.*, exercise-to-rest ratios and/or hypoxic levels) is necessary to optimize individual responses to RSH³¹. The increased lower-limb discomfort observed in RSN may be attributed to possible inter-session fatigue appearance. However, this would have also been observed in RSH, unless hypoxic ‘tolerance’ had lessened this phenomenon. Despite being constantly reminded about what perceived lower-limb discomfort actually measured, it remains possible that several players rated the discomfort of their legs with a variety of sensations – *e.g.*, muscle pain, joint and foot pressure, and muscle tension⁷ – experienced during repeated sprinting, not just how uncomfortable their legs felt.

Previous studies have documented that short-term intermittent normobaric/hypobaric hypoxia (*e.g.*, 1-2 h.day⁻¹ over 7-10 consecutive days) leads to ventilatory

‘acclimation/acclimatization’ to hypoxia, characterized by a progressive recovery in physiological functions (*i.e.*, increase in SpO₂ and decrease in HR values) during hypoxic exercise ³². In this study, however, SpO₂ remained stable and the decrease in HR was not significant across sessions notably for RSH, mainly due to high individual variability in response to hypoxic exercise. Pending confirmatory research, it is possible that an increased hypoxic ventilatory response may have been counteracted by other likely RSH-induced physiological mechanisms as hemodynamic ³³ and/or muscular adaptations ³⁴.

Practical applications

We observed that ratings of overall perceived discomfort, difficulty breathing and lower-limb discomfort decreased after the first RSH session to reach comparable values with RSN, while sprint performance achieved with or without hypoxic exposure followed similar trends across sessions. Together with larger performance improvement (*i.e.*, lower averaged sprint 5 time), these positive perceived adaptations for RSH may have played a role in the post-intervention performance gains ⁹. This indicates that the proposed RSH modality (*i.e.*, four sets of 5 × 5-s maximal sprints interspersed with 25 s of passive recovery with 5 min of standing rest between sets at FiO₂ ~14.5%/~3000 m), combined with normobaric hypoxic residence (≥ 14 h.d⁻¹ at FiO₂ 14.5-14.2%; 2800–3000 m), is sufficient in severity, duration and/or frequency to elicit a significant hypoxic ‘acclimation’. This study therefore adds to the recent results of Born et al. ¹⁶, where mucosal immune function was not further impaired with RSH, by showing that players tolerance to RSH is effective after only one session. Since RSH does not negatively alter psycho-physiological responses when compared to RSN, coaches and their athletes are encouraged to implement such time-efficient hypoxic interventions in order to gain a competitive edge ⁹. By closely monitoring training loads and psycho-physiological responses of players in response to each session, this investigation

opens new frontiers to optimize future hypoxic training applications (*e.g.*, appropriate periodization to implement isolated RSH and/or in combination with prolonged residence at simulated altitude) in team sports.

Conclusion

This is the first study examining psycho-physiological responses to repeated-sprint training (6 sessions) in hypoxia and normoxia in elite team-sport players over a 2-wk in-season ‘live high-train low’ training camp. During the first session, hypoxia exposure during training elevated physiological and perceptual responses, whereas sprint performance (timing) was similar. Despite higher hypoxia-induced physiological stress (*e.g.*, lower SpO₂ values), improved tolerance to the hypoxic training (effective hypoxic ‘acclimation’) led to a decrease in perceptual responses (that were not maximal in both groups) in RSH, so as to become essentially similar to those measured in RSN. These positive adaptations could have played a role in the larger RSH-related improvement in repeated sprint performance.

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Figure 1. Illustration of the mobile inflatable simulated hypoxic equipment used for repeated-sprint training sessions. External (A, overall view of the 45-m long marquee) and inside tunnel view with players completing sprints (B) and monitoring their psychophysiological responses (C).

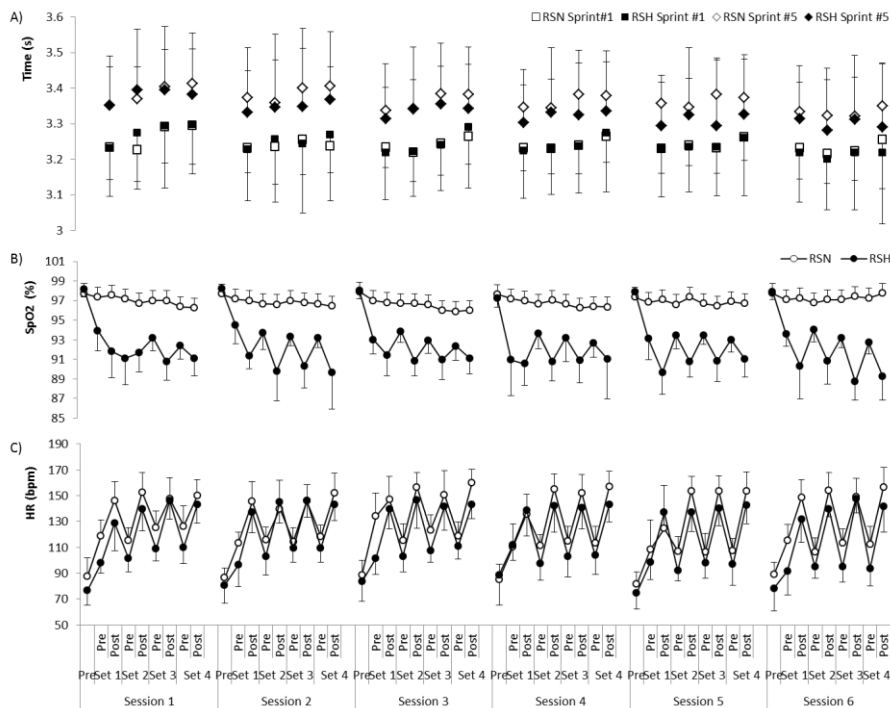


Figure 2. Sprint times (A), arterial oxyhemoglobin saturation (SpO₂; B) and heart rate (HR; C) for the four sets of repeated-sprint exercise performed during each training session. Values are mean ± SD, N = 23. Data is presented for normobaric hypoxia (RSH; FiO₂ ~14.5%) and normoxia (RSN; FiO₂ 21.0%).

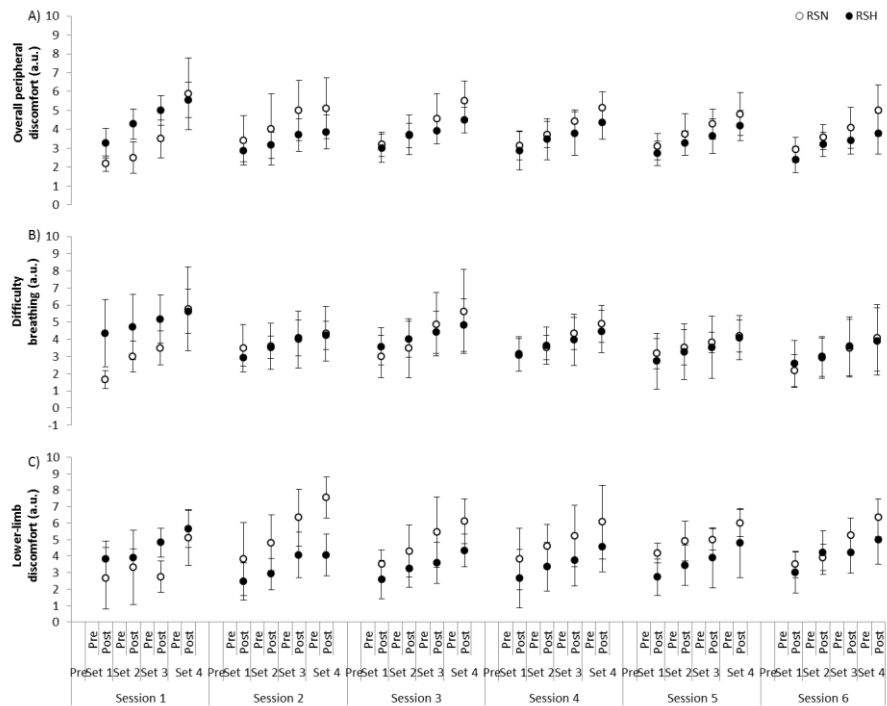


Figure 3. Ratings of overall peripheral discomfort (A), difficulty breathing (B) and lower-limb discomfort (C) for the four sets of repeated-sprints performed during each training session. Values are mean \pm SD, N = 23. Data is presented for normobaric hypoxia (RSH; $FiO_2 \sim 14.5\%$) and normoxia (RSN; $FiO_2 21.0\%$).

Table 1. Subjects’ characteristics.

	RSB (n = 11)	RSN (n = 12)
Age (yr)	27.4 ± 4.6	24.9 ± 4.5
Height (cm)	178.7 ± 8.2	178.9 ± 4.7
Weight (kg)	77.0 ± 7.7	76.9 ± 8.5
BMI (kg.m ⁻²)	24.1 ± 1.6	24.0 ± 1.8
VO _{2max} (ml.min ⁻¹ .kg ⁻¹)	52.7 ± 2.4	52.5 ± 1.8
HR _{max} (beats.min ⁻¹)	189 ± 4	192 ± 3

VO_{2max} = maximal oxygen uptake; HR_{max} = maximal heart rate.

Values are mean ± SD.

Table 2. Sprint performance parameters and psycho-physiological responses (average of all sets) during the 6 sessions of repeated-sprint training in hypoxia (RSH) and in normoxia (RSN).

	Training	Session number						Average	Changes (%)	ANOVA main effects (effect sizes)
		1	2	3	4	5	6			
Sprint performance										
Sprint time 1 (s)	RSH	3.27±0.14	3.25±0.17	3.24±0.13	3.24±0.13	3.24±0.13	3.21±0.15	3.24±0.14	-1.8±3.8	C = 0.99 (0.00); T = 0.15 (0.22); I = 0.95 (0.08)
	RSN	3.26±0.08	3.24±0.08	3.24±0.07	3.24±0.06	3.24±0.06	3.23±0.09	3.24±0.07	-0.5±2.9	
Sprint time 5 (s)	RSH	3.38±0.16	3.35±0.19	3.34±0.16	3.32±0.16	3.31±0.17	3.30±0.16*	3.33±0.16####	-2.4±3.8	C = 0.55 (0.23); T < 0.01 (0.31); I = 0.80 (0.11)
	RSN	3.38±0.08	3.38±0.08	3.36±0.06	3.36±0.06	3.36±0.07	3.33±0.09	3.36±0.07	-1.3±2.9	
FI (%)	RSH	4.6±1.9	4.3±2.7	3.9±3.2	3.4±2.4	3.0±2.4	2.2±3.6*	3.6±2.8	-34.1±133.9	C = 0.16 (0.32); T < 0.05 (0.44); I = 0.99 (0.07)
	RSN	5.6±4.1	5.4±1.2	4.6±2.3	4.5±1.9	4.4±2.5	3.6±3.3*	4.6±2.7	-62.7±127.7	
Physiological responses										
SpO ₂ (%)	RSH	92.0±1.6####	92.0±1.4####	92.0±1.1####	91.9±1.0####	91.9±0.9####	91.7±1.2*,###	91.9±1.2####	-0.9±1.4	C < 0.001 (3.48); T < 0.001 (0.42); I < 0.001 (0.39)
	RSN	97.0±0.5	96.8±0.6	96.5±0.6	96.7±0.4	96.8±0.7	97.3±0.6	96.9±0.6	0.2±0.6	
HR (bpm)	RSH	142±13	144±13	143±10	141±13	139±11	138±14	140±12	0.7±12.9	C = 0.053 (0.53); T = 0.11 (0.35); I = 0.55 (0.23)
	RSN	148±13	145±12	153±14	145±11	143±13	150±13	147±12	0.9±9.8	
Perceptual responses										
RPE (a.u.)	RSH	14.6±0.8	13.3±0.9	14.1±1.0	14.1±1.3	13.8±1.7	13.1±1.3	13.8±1	-10.5±15.4#	C < 0.05 (0.61); T = 0.31 (0.28); I = 0.09 (0.37)
	RSN	14.4±2.6	14.1±1.9	14.3±1.8	14.1±0.9	14.3±1.9	14.8±0.9	14.3±1.7	4.0±17.7	
Overall	RSH	4.5±0.8#	3.4±0.7**	3.8±0.7**	3.6±1.0**	3.5±0.8**	3.2±0.8***	3.7±0.8#	-28.5±22.4#	C < 0.05 (0.55);

	Training	Session number						Average	Changes (%)	ANOVA main effects (effect sizes)
		1	2	3	4	5	6			
peripheral discomfort (a.u.)	RSN	3.5±1.0	4.4±1.6	4.2±1.0	4.1±0.7	4.0±0.9	43.9±0.9	4.0±1.0	-2.7±51.9	T < 0.001 (0.62); I = 0.61 (0.21)
Difficulty breathing (a.u.)	RSH	5.0±1.6	3.7±0.8*	4.2±1.2*	3.8±1.2*	3.4±1.6**	3.3±1.5***	3.9±1.3	-33.8±25.7	C = 0.68 (0.12); T < 0.001 (0.69);
	RSN	3.5±1.2	3.9±1.5	4.3±1.8	4.0±0.9	3.7±0.9	3.2±1.4	3.7±1.3	-26.3±31.4	I = 0.72 (0.18)
Lower-limb discomfort (a.u.)	RSH	4.5±0.9#	3.4±1.2***,###	3.4±1.1**,##	3.6±1.6**,##	3.7±1.6*,#	4.1±1.3	3.8±1.3###	-9.5±34.0#	C < 0.001 (0.84); T = 0.60 (0.21);
	RSN	3.5±1.7	5.6±1.7***	4.8±1.5**	4.9±1.8***	5.0±0.8***	4.8±1.0**	4.8±1.4	26.5±78.0	I < 0.05 (0.41)

Notes: Values are mean ± SD. C, T, I – condition, time and interaction effects, respectively.

* significantly different from session number 1, P < 0.05, ** < 0.01, *** < 0.001. # significantly different from RSN, P < 0.05, ### < 0.001.