A clustered repeated-sprint running protocol for team-sport athletes performed in normobaric hypoxia

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A Clustered Repeated-Sprint Running Protocol for Team-Sport Athletes Performed in Normobaric Hypoxia

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Abstract

The present study compared the performance (peak speed, distance, and acceleration) of ten amateur team-sport athletes during a clustered (i.e., multiple sets) repeated-sprint protocol, (4 sets of 4, 4-s running sprints; i.e., RSR444) in normobaric normoxia (FiO₂ = 0.209; i.e., RSN) with normobaric hypoxia (FiO₂ = 0.140; i.e., RSH). Subjects completed two separate trials (i. RSN, ii. RSH; randomised order) between 48 h and 72 h apart on a non-motorized treadmill. In addition to performance, we examined blood lactate concentration [La⁻] and arterial oxygen saturation (SpO₂) before, during, and after the RSR444. While there were no differences in peak speed or distance during set 1 or set 2, peak speed (p = 0.04 and 0.02, respectively) and distance (p = 0.04 and 0.02, respectively) were greater during set 3 and set 4 of RSN compared with RSH. There was no difference in the average acceleration achieved in set 1 (p = 0.45), set 2 (p = 0.26), or set 3 (p = 0.23) between RSN and RSH; however, the average acceleration was greater in RSN than RSH in set 4 (p < 0.01). Measurements of [La⁻] were higher during RSH than RSN immediately after Sprint 16 (10.2 ± 2.5 vs 8.6 ± 2.6 mM; p = 0.02). Estimations of SpO₂ were lower during RSH than RSN, respectively, immediately prior to the commencement of the test (89.0 ± 2.0 vs 97.2 ± 1.5 %), post Sprint 8 (78.0 ± 6.3 vs 93.8 ± 3.6 %) and post Sprint 16 (75.3 ± 6.3 vs 94.5 ± 2.5 %; all p < 0.01). In summary, the RSR444 is a practical protocol for the implementation of a hypoxic repeated-sprint training intervention into the training schedules of team-sport athletes. However, given the inability of amateur team-sport athletes to maintain performance in hypoxic (FiO₂ = 0.140) conditions, the potential for specific training outcomes (i.e. speed) to be achieved will be compromised, thus suggesting that the RSR444 should be used with caution.

Key words: Acceleration, altitude, football, multiple-set.

Introduction

Intermittent hypoxic training (IHT) has emerged as a popular training method for team-sport athletes and aims to evoke greater adaptations than performance of similar training in normoxia. More recently, a new training method has been investigated by several researchers (Faiss et al., 2013, Galvin et al., 2013, Puype et al., 2013, Brocherie et al., 2015, Goods et al., 2015) that includes the performance of repeated sprints (RS) in hypoxic conditions (RSH). Previous RSH studies have used a variety of protocols, with sprint durations ranging from 5 to 30 s and recovery periods ranging from 15 s to 5 min (Galvin et al., 2013, Faiss et al., 2013, Puype et al., 2013, Goods et al., 2015). While three of these studies demonstrated RSH to evoke superior adaptations in muscle perfusion (Faiss et al., 2013), glycolytic enzyme activity (Puype et al., 2013) and the ability to perform repeated bouts of high-intensity aerobic work, i.e., improved Yo-Yo Intermittent Recovery 1 test performance (Galvin et al., 2013), one study demonstrated no additional benefit (Goods et al., 2015).

The concept of replicating the movements performed during team-sport match play (e.g. number/duration of sprints/recovery periods) is appealing when designing a RS training protocol to improve the RS ability (RSA) of team-sport athletes, however, further research is required regarding the efficacy of utilizing specific RS training for this purpose (Buchheit, 2012). A recent RS training study, which was limited by the lack of a control group, utilized a protocol that was characterized by clusters (i.e., multiple sets) of sprints which were separated by short (20 s) recovery periods, with longer recovery periods (4.5 min) between clusters (Serpiello et al., 2011). The authors suggested that single-set repeated-sprint protocols poorly reflect the demands of team-sports and that multiple sets provide for more accurate assessment of team-sport performance (Serpiello et al., 2011). This is supported by time-motion analysis studies that have described the repeated-sprint activity of team-sports (Spencer et al., 2004, Gabbett and Mulvey, 2008).

Combining the two ideas of clustered repeated-sprint training and RSH, Goods and colleagues (2014) demonstrated that peak power output (PPO) can be maintained at a simulated altitude of 3000 m, but not 4000 m, during three sets of 9 x 4-s sprint efforts, when compared with sea-level (RSN). The impairment of PPO at a simulated altitude of 4000 m would be an important factor to consider when designing a similar RSH training protocol, given the importance of specific muscle contraction speeds when training for sprint speed (Kristensen et al., 2006).

In addition to peak speed/power output and total work (e.g., mean speed or total distance), the rate of acceleration should be considered as an important performance outcome when prescribing a repeated-sprint training program for team-sport athletes (Lockie et al., 2011). Previous studies that have defined and described repeated-sprint activity during international-level team-sport competition, have reported average sprint times during repeated-sprinting in elite men’s hockey (Spencer et al., 2004) and elite women’s soccer (Gabbett and Mulvey, 2008) to be 1.8 s and 2.1 s, respectively. Meanwhile, average sprint times in elite Australian-rules football have

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been reported to be < 3 s (Coutts et al., 2010). The short duration of sprints in various team-sports highlights the importance of acceleration to performance (Lockie et al., 2011), given that players are not likely to reach maximum velocity during sprint efforts (Duthie et al., 2006) and that these short sprints are considered a decisive determinant of match-winning situations (Carling et al., 2006). Furthermore, Osgnach et al. (2010) quantified the metabolic demands of elite soccer and emphasized the importance of quantifying the demands of accelerations (and decelerations), which “constitute a large and crucial fraction of every match” (Osngnach et al., 2010). Hence, it is intuitive that equal, if not greater, importance is placed on improving a team-sport athlete’s ability to accelerate quickly than on improving their peak velocity.

In the present study, we sought to design a protocol that not only replicates repeated-sprint patterns in team-sports (i.e. clusters), but also allows for adequate recovery so as to attenuate reliance on aerobic metabolism and impairment in speed, acceleration, and total work (i.e., distance). Our aim was to examine performance (peak speed, acceleration and total work) and physiological responses (blood lactate and \( S_pO_2 \)) during performance of a RS running (RSR) protocol in order to provide information to be used in designing a RSH training protocol for team-sport athletes.

**Methods**

Ten amateur team-sport athletes (four Australian rules footballers, four rugby union players and two soccer players) volunteered to participate in the present study and gave their written informed consent. All procedures used in the study were approved by the Griffith University Human Research Ethics Committee. The physical characteristics of the group were (mean ± SD): age 22.6 ± 4.7 y, body mass 88.8 ± 7.3 kg, and height 1.83 ± 0.06 m. All participants had competed for a minimum of three consecutive years in their respective sport, as well as completed a minimum of two months (2-3 times per week) of intense training immediately prior to involvement in the study. All participants performed a repeated-sprint running (RSR) test consisting of sixteen (four sets of four) 4-s sprints separated by 26 s (and 2 min 26 s between sets) of passive recovery in a standing position (i.e., RSR444) on two occasions in a commercial normobaric hypoxic chamber (Pro diving Services, Sydney, Australia) while breathing either room air (\( FiO_2 = 0.209 \)) or \( FiO_2 = 0.140 \) (i.e., hypoxia). The hypoxic environment was created via the extraction of oxygen from air which was subsequently pumped into the chamber. Oxygen concentration was monitored using a gas detector (KB-501, Kingsby Electronics,) which utilizes an electrochemical sensor. The \( FiO_2 (0.140) \) was selected due to the ability of highly-trained team-sport athletes to maintain performance during a similar study in our lab (Morrison et al., 2015) involving 10 x 6-s sprints in hypoxic (\( FiO_2 = 0.140 \)) conditions. Relative humidity and temperature were maintained between 45-50% and 19-21°C, respectively. While we acknowledge that a passive recovery does not replicate team-sport movement demands, the protocol was designed to allow the maintenance of speed, acceleration and total work (i.e., distance) as well as the rotation of up to four athletes on one treadmill (thus improving team-sport training efficiency). The study followed a cross-over design with one group (\( n = 5 \)) performing the RSR444 in normoxia first and then in hypoxia between 48 h and 72 h later; the other group (\( n = 5 \)) performed the RSR tests in the reverse order. Testing sessions were performed immediately prior to participants’ training sessions for their respective sports, and replaced specific repeated-sprint training activity for those training sessions. While the authors acknowledge that the second session may have been impacted by the short washout period, the investigation is counterbalanced and the participants would have been performing repeated-sprints regardless of if they completed the testing sessions or not. In addition, all participants indicated prior to commencing each session, via pre-participation questionnaires, that they were not suffering from any soreness. Participants performed the tests at the same time of day and were asked to 1) refrain from consuming alcohol/performing strenuous exercise in the 24 h prior to the tests, 2) refrain from consuming caffeine on the day of the tests, and 3) be consistent with food and fluid intake for both tests. All participants indicated in the pre-participation questionnaires that they were not taking any supplements.

The RSR444 was performed on a non-motorized treadmill (Curve 3.0, Woodway, Waukesha, Wisconsin, USA) with a curved running surface made from sixty vulcanized rubber slats and a drive system consisting of ball-bearings and roller glides. The non-motorized nature of the treadmill allowed the participants to accelerate maximally.

All participants were in the later stages of their pre-season training and were accustomed to performing repeated sprints/high-intensity running. Nonetheless, prior to any testing, participants were required to complete three familiarization sessions on the treadmill. Each session included a 5-min warm-up jog, 4-5 high-intensity (but not all-out) ~4-s efforts with ~1 min rest between them, as well as the completion of 2, 3 and 4 sets of 4 x 4-s sprints (with the same rest periods as during the RSR444) during sessions 1, 2 and 3, respectively. These sessions were designed to 1) allow participants to master the technique required to accelerate/sprint maximally and 2) condition their lower limbs to performing demanding repeated-sprint sequences.

Before the commencement of the RSR444, participants were required to complete a standardized warm-up comprising a 5-min jog on the non-motorized treadmill at ~10 km/h interspersed with three 4-s sprints at ~90-s intervals, and a 10-min period that included dynamic stretching of all major lower limb muscle groups. A 3-min seated recovery period immediately followed the completion of RSR444. The warm-up, RSR444 and recovery period were all undertaken in the environmental condition for that testing day. Prior to each sprint, the chief investigator provided participants with a 10 s and 5 s warning before counting them in “3, 2, 1, go!”. Between sets, participants were provided with 1 min and 30 s warnings as well. Participants were provided with strong verbal...
encouragement during all sprints. No feedback was given regarding speed or distance achieved during each sprint.

Peak speed (m·s⁻¹) and acceleration (m·s⁻²) data were calculated via Pacer Performance System software (Fitness Technology, Adelaide, Australia) which continuously recorded time (s) data and distance (m) data, via a tachometer mounted on the treadmill drum. Data were sampled at a rate of 200 Hz. Each sprint start was judged to be at the moment the treadmill belt recorded a speed of 0.05 m·s⁻¹ or greater and continued to increase. Exactly 4 s of data were then used in the analysis of peak speed, which was calculated by taking the highest value observed in the 4-s sprint period. Acceleration was calculated as the rate of change in velocity during the first 0.5 s of the sprint, as done previously (Serpiello et al., 2011). The distance covered during each sprint was determined and recorded. Seven team-sport athletes, (similarly trained to those in the present study) performed two trials of the RST444 in normoxia (between 48 and 72 h apart) to assess the reliability of measuring speed and acceleration across the RST444. Typical error as a coefficient of variation via log-transformed speed (set 1 = 1.7, set 2 = 2.0, set 3 = 2.1, set 4 = 1.5 %) and acceleration (set 1 = 6.7, set 2 = 6.0, set 3 = 4.7, set 4 = 5.3 %) data were calculated for each set using an Excel spreadsheet for reliability (Hopkins, 2009). To determine the validity of acceleration data, we compared values obtained using the method described in the present study, with values obtained using the method described by Serpiello and colleagues i.e., judging the sprint start to be at a treadmill belt speed of 0.05 m·s⁻¹ vs 1 m·s⁻¹, respectively. While using the method described by Serpiello and colleagues resulted in acceleration values being slightly higher, the pattern of decrease across the sets observed was the same as that observed using the method described in the present study. Capillary blood lactate concentration ([La⁻]) was determined from earlobe blood samples using a handheld analyser (Lactate Pro, Arkray Factory Inc., KDK Corporation, Shiga, Japan). Resting (baseline) measurements were obtained in normoxic conditions prior to the warm-up on both testing days. Subsequent to this, measurements were taken immediately prior to the commencement of the RSR₄₄₄, and after 90 s of recovery following Sprints 8 and 16. Estimations of arterial oxygen saturation (S₉O₂) were also made immediately prior to the start of the RSR₄₄₄, and immediately following Sprints 8 and 16 using a portable pulse oximeter (Octive Tech, 300CSE, Beijing Choice Electronic Technology Co., Ltd Beijing, China).

Fully factorial ANOVA were used to compare performance (speed, distance and acceleration) and metabolic ([La⁻] and S₉O₂) values achieved during each sprint between the two environmental conditions (i.e., normoxia and hypoxia) and across the RSR₄₄₄ protocol. Least squares difference pairwise comparisons were used where significant F values were observed. All data is expressed as mean±standard deviation and significance was accepted at p < 0.05. SPSS Statistics software version 19 was used.

**Results**

While not statistically significant, peak speed, distance, and acceleration were, in every case, approximately 1-2% lower during set 1 of the RSR₄₄₄ compared with set 2 in both the normoxic and hypoxic conditions (Figure 1). Thus, maximal values were typically achieved in set 2 of the RSR₄₄₄. There was no difference in the highest single

![Figure 1. Peak speed (bars) and distance (lines) values averaged across four, 4-s sprints for each set in normoxia and hypoxia achieved in male amateur team-sport athletes. * Distance covered during sprints greater in normoxic conditions (p < 0.05). † Different from normoxia (p < 0.05).](image-url)
peak speed value recorded during RSN compared with RSH (8.05 ± 0.32 vs 8.00 ± 0.39 m·s⁻¹, respectively; p = 0.55). Figure 1 shows the peak speed and distance values averaged across the four, 4-s sprints in each set under both conditions. There were no differences in peak speed (p = 0.88 and 0.05, respectively) or distance (p = 0.35 and 0.41, respectively) achieved during set 1 or set 2 of RSN compared with RSH. However, the average peak speed achieved in set 3 was lower (p = 0.04) and by Sprint 12, single-sprint peak speed was lower (7.67 ± 0.44 vs. 7.78 ± 0.35 m·s⁻¹; p = 0.03) during RSH compared with RSN. Similarly, the average distance achieved during sprints in set 3 was lower (p = 0.04) and by Sprint 11, single-sprint distance was lower (23.07 ± 1.21 vs. 23.97 ± 1.15 m; p = 0.03) during RSH compared with RSN, respectively. There was no difference in the average of acceleration values achieved during sprints in set 1 (p = 0.45), set 2 (p = 0.26), or set 3 (p = 0.23) of the RSR444 between the conditions, but a difference was observed in set 4 during RSH (5.47 ± 1.23 m·s⁻²) compared with normoxia (5.84 ± 1.51 m·s⁻²; p < 0.01). Figure 2 illustrates the similarities in acceleration and peak speed during a single sprint in set 1 of the RSR444 between normoxia and hypoxia (left panel), and the disparity in peak speed, but not acceleration between the two conditions in set 3 of the RSR444 (right panel).

There was no difference in [La⁻] measured at baseline or after Sprint 8 during RSN compared with RSH, respectively (Table 1). However pre-test (i.e., post warm-up) [La⁻] was higher during RSH than RSN, as was [La⁻] measured after Sprint 16. Measurements of SpO₂ were higher at all time-points during RSN compared with RSH (Table 1).

**Discussion**

During performance of a clustered repeated-sprint running protocol (4 sets of 4, 4-s sprints; i.e., RSR₄₄₄) in a hypoxic environment (RSH), amateur team-sport athletes were unable to match the peak speed recorded in normoxia (RSN) during set 3 and set 4. Similarly, total distance covered during RSN could not be replicated during set 3 or set 4 during RSH. Acceleration was more, but not completely, resilient (i.e. only impaired during the final set) to a reduction in oxygen availability and/or the effects of fatigue during the clustered repeated-sprint running protocol in hypoxia. These results provide information that may be useful in designing clustered RSH training protocols, which may be altered depending on the intended training outcomes.

The impairment in peak speed observed during sets 3 and 4 of the hypoxic trial in the present study is in contrast to the findings of our previous study (Morrison et al., 2015) that examined a traditional repeated-sprint running protocol (1 set of 10, 6-s sprints) in hypoxia (FiO₂ = 0.140). Conversely, (Bowtell et al., 2013) reported similar findings to the present study stating that peak speeds during the ten, 6-s sprints was lower in hypoxic conditions. The variability in the construct (i.e., number/duration of sprints and/or recovery periods) of the repeated-sprint protocols among studies may explain the conflicting findings regarding peak speed achieved during

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**Table 1.** Blood lactate concentration [La⁻] and arterial oxygen saturation (SpO₂) measured in team-sport athletes before, during, and immediately after performing the RSR₄₄₄ (4 sets of 4, 4-s sprints) in normoxic (21%) and hypoxic (14%) conditions. Data are means (±SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Condition</th>
<th>Baseline</th>
<th>Pre-test</th>
<th>Post-sprint 8</th>
<th>Post-sprint 16</th>
</tr>
</thead>
<tbody>
<tr>
<td>[La⁻] mmol·L⁻¹</td>
<td>Normoxia</td>
<td>1.0 (.3)</td>
<td>1.7 (1.4)</td>
<td>7.1 (2.0)</td>
<td>8.6 (2.6)</td>
</tr>
<tr>
<td></td>
<td>Hypoxia</td>
<td>1.0 (.2)</td>
<td>2.9 (1.6)*</td>
<td>8.0 (2.2)</td>
<td>10.2 (2.5)*</td>
</tr>
<tr>
<td>SpO₂ (%)</td>
<td>Normoxia</td>
<td>-</td>
<td>97.2 (1.5)</td>
<td>93.8 (3.6)</td>
<td>94.5 (2.5)</td>
</tr>
<tr>
<td></td>
<td>Hypoxia</td>
<td>-</td>
<td>89.0 (2.0) *</td>
<td>78.0 (6.3) *</td>
<td>75.3 (6.3) *</td>
</tr>
</tbody>
</table>

*Different from normoxia (p < 0.05). Baseline measurements were obtained in normoxic conditions before the warm up. Pre-test measurements were performed in the environmental condition, after the warm up and immediately before the commencement of the RSR₄₄₄.
performance in hypoxia compared with normoxia. In addition, the ability of an athlete to match the peak speeds achieved during repeated sprinting in normoxia when in a hypoxic environment could be dependent on their training status. Highly-trained team-sport athletes in the study by Morrison and colleagues (Morrison et al., 2015) are likely to have had the ability to compensate for hypoxia-induced reductions in aerobic energy provision. In contrast, it is possible that participants in the present study were unable to offset the reduced aerobic energy provision with an increase in anaerobic energy contribution. This is an important finding as an inability to match speed during RSH compared with RSN imposes a different training stimulus on the athlete. Therefore, athletes and coaches should be mindful that during RSH, desired contraction speeds may not be replicated, representing an alteration in the neuromuscular load which may affect neuromuscular adaptations (Buchheit and Laursen, 2013a). It should also be noted that despite a reduced running speed (i.e., sets 3 and 4 of RSH in the present study), RSH may result in superior training adaptations compared with RSN, given that training at a lower absolute intensity which elicits greater physiological/metabolic responses can evoke superior adaptations (Mohr et al., 2007). Therefore, a combination of RSH that elicits greater physiological/metabolic adaptations, and RSN, for the maintenance of contraction speeds, could be considered as a viable training intervention. In addition to reduced aerobic energy provision, another factor influencing the ability to maintain peak speed is the initial sprint speed i.e., the faster the first sprint, a larger decrement ensues. Nonetheless, the initial sprint speeds were comparable between the normoxic and hypoxic environments in the present study, thus excluding initial speed as an influence on speed decrement.

Mean speed (Morrison et al., 2015), mean power output (Goods et al., 2014, Brosnan et al., 2000), and mechanical work (Smith and Billaut, 2010) measured in participants during repeated-sprint training has been previously compared between normoxic and hypoxic conditions. In the present study, we examined the distance covered during each sprint running in the RSR444 so as to improve the validity of the feedback for coaches and athletes. Nonetheless, the common purpose of these measurements is to quantify the average or total work performed during each sprint rather than a peak or instantaneous value. Markers of total work may provide additional information regarding the total metabolic demand of the repeated-sprint session. In addition to the importance of matching peak contraction speeds, replicating the metabolic cost of training in normoxia may also be an important consideration, depending on the intended training outcomes. In agreement with previous findings (Goods et al., 2014, Brosnan et al., 2000, Smith and Billaut, 2010), the present study demonstrated that total work performed (i.e., distance covered) was reduced during RSH compared with RSN.

Goods and colleagues (2014) suggested that the decline in mean power output during RSH, compared with RSN, could be explained by the observed decline in oxygen saturation. This is corroborated by Billaut and Smith (2010), who demonstrated that a decline in mechanical output during prolonged repeated sprints in normoxia was related to a decline in $S_O^2$. It is plausible that reduced arterial oxygen saturation could contribute to fatigue during repeated sprints, given that PCR resynthesis (Haseler et al., 1999) and $H^+$ removal (Tomlin and Wenger, 2001) are oxygen dependent processes. A reduction in arterial oxygen saturation might then contribute to attenuated neuromuscular activity, and consequently a decrease in performance (Billaut and Smith, 2010) possibly via reflex inhibition of alpha motoneurons (Garland, 1991). Smith and Billaut (2010) demonstrated that a decrease in arterial oxygen saturation may also contribute to fatigue during RSH via a reduction in cerebral oxygenation. Hence, the decline in average peak speeds and total work performed (average distance covered during sprints) in the present study, could be explained in part by the ~19% difference in $S_O^2$ estimated following the final sprint. Finally, a decline in peak speed and total work during RSH compared with RSN, could also be partly explained by reduced contribution from aerobic metabolism ((Bowtell et al., 2013, Calbet et al., 2003, Balsom et al., 1994), although a compensatory increase in contribution from anaerobic metabolism has previously been reported (Calbet et al., 2003). In the present study, the higher $[La^-]$ measured following the final sprint in RSH, compared with RSN, may be due to an increase in anaerobic glycolysis in response to a reduced oxygen uptake. It should also be noted that this observed increase may have been due to impaired lactate clearance.

Athletes were unable to match acceleration during set 4 in RSH compared with RSN in the present study. Given that peak speed and distance covered were reduced compared with RSN after set 2 during RSH, this suggests that peak acceleration can be maintained for longer when oxygen availability is reduced. Distance covered is dependent on the entire 4-s effort and peak speed is typically achieved at the end of 4-s. In contrast, peak acceleration is typically achieved in the first second of the sprint (see Figure 1). Given that the shorter duration in which peak acceleration is found in comparison with peak speed and maximum distance, acceleration might be less dependent on aerobic energy production, and/or less affected by ionic disturbances that result in fatigue. The ability to match acceleration for more sprint repetitions during RSH, when compared with RSN, provides coaches with a framework with which to prescribe RSH. Indeed, Serpiello and colleagues (2011), demonstrated that clustered repeated-sprint running training in normoxia evoked improvements in acceleration that were up to four times greater than improvements in peak speed.

Training protocols that stimulate a high rate of PCR breakdown and glycolysis as well as demanding a high rate of $H^+$ and K$^+$ removal are likely to result in adaptations to the anaerobic energy systems. In traditional repeated-sprint protocols with inadequate recovery between sprints, the rate of glycolysis can be reduced by 90% with a concomitant increase in aerobic energy production (Gaitanos et al., 1993). Thus, the final sprints of a repeated-sprint protocol with inadequate recovery may result in
reduced performance and an altered training focus. The $RSR_{44}$ not only more closely replicates team-sport repeated-sprint patterns compared with traditional repeated-sprint protocols (i.e., multiple vs single set), it allows for manipulation of sprint and recovery durations in accordance with the desired physiological response/neuromuscular strain (Buchheit and Laursen, 2013b). For example, the prescription of longer recovery periods between sprints and/or sets would increase the emphasis on anaerobic energy production/neuromuscular strain (Buchheit and Laursen, 2013b), with each subsequent sprint attainable. In the present study, although the recovery time between sprints was slightly longer in duration (26 vs 20 s) than that in the study by Serpiello and colleagues (2011), the recovery time between sets was much shorter in duration (146 vs 270 s). Hence, it is probable that an inadequate recovery time between sets in the present study contributed to the decline in peak speed, acceleration (and therefore reduced neuromuscular strain) and distance that was observed in RSH, compared with RSN.

Another important consideration to make when designing RSH training protocols for team-sport athletes is practicality. The $RSR_{44}$ was designed so that four athletes could complete the protocol at one time using the same treadmill. Although the specificity of the protocol to team-sports is reduced by having a passive recovery, such a design is advantageous in that it reduces the time necessary for a squad to complete a training session (many hypoxic chambers are only large enough to fit 4-6 treadmills), whilst also providing the necessary motivation for “all-out” sprinting.

**Conclusion**

In summary, peak speed, total distance, and acceleration during the $RSR_{44}$ were impaired in hypoxia compared with normoxia. These findings were in conjunction with a decrease in $SpO_2$ and an increase in $[La^-]$ in the hypoxic condition. The findings of the present study provide a framework on which to base future research which could aim to determine the efficacy of utilizing a RSR protocol similar to the one in the present study as a hypoxic training intervention. Based on the results of the present study, it is logical that, if improved speed is a desired training outcome, either: 1) a modified version of the $RST_{44}$, which allows for greater rest between sets, is used to allow maintenance of muscle contraction speeds; or 2) the performance of the $RST_{44}$ in hypoxia is utilized in conjunction with normoxic speed training.

**Acknowledgements**

None to report other than that the experiments performed in the present study comply with the current laws of Australia.

**References**


Key points

- The RSR444 is a practical, multiple-set repeated-sprint running protocol designed for team-sport athletes.
- During performance of the RSR444 in hypoxia (FiO₂ = 0.140), amateur team-sport athletes were unable to replicate the peak speed, distance covered or acceleration achieved in the final set(s) during sprints in normoxia.
- A decrease in SpO₂ and an increase in [La⁻] were observed during performance of the RSR444 in hypoxia, compared with normoxia.

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