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ORIGINAL ARTICLE

Repeated sprint training in hypoxia and repeated long sprint ability in highly trained sprint runners: A pilot study

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ABSTRACT

Repeated sprint training in hypoxia (RSH) provides additional improvement in repeated "*short*" (<10-s) sprint ability compared to the same training in normoxia. Although team sports require to perform repeated "*short*" (<10-s) sprints during incomplete recovery situations, some sports (e.g., roadcycling) require repeated "*longer*" (>10-s) sprints during the race. However, evidence regarding the effect of RSH on repeated "*longer*" (>10-s) sprint ability is lacking. Ten highly trained sprint runners conducted six sessions of repeated sprint training (2-3 sets of 5 × 10-s cycle sprints) in hypoxia (HYP) or normoxia (NOR). Before (pre-) and after (post-) the training intervention, participants performed repeated "*longer*" (>10-s) sprint tests (5 × 100-m "*all-out*" sprints with 30-s recoveries) in normoxia. Running velocity and blood lactate concentrations were measured for repeated 100-m sprints. No significant difference was observed (p > .05) in repeated sprint ability between the pre- and posttests, independently training group. Blood lactate concentrations were significantly lower post-HYP than pre-HYP or post-NOR. This study revealed that RSH did not provide any additional training benefits for repeated "*longer*" (>10-s) sprints in highly trained participants compared to equivalent training in normoxia. However, RSH induced significantly lower blood lactate responses after repeated "*longer*" (>10-s) sprints.

Keywords: Performance analysis, Hypoxic training, Exercise physiology, Sports performance, Blood lactate, Simulated altitude.

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INTRODUCTION

Team sports (e.g., soccer) require the ability to repeat maximal or near-maximal efforts (i.e., sprints) interspersed with short incomplete recoveries; this ability is defined as repeated sprint ability (RSA) (Girard et al., 2011). Therefore, to improve RSA, team sports athletes often perform repeated sprint exercises as their training, which consist of multiple bouts of short sprint efforts (≤10-s sprint) with incomplete recovery (≤60-s) (Bishop et al., 2011). Recently, the addition of hypoxic exposure to repeated sprint training (i.e., RSH, Repeated sprint training in hypoxia) has become attractive to researchers and practitioners because of the significant additional training effects compared to normoxic equivalent training (Faiss et al., 2013A; Brocherie et al., 2017). For instance, Faiss et al. (2013 B) found that 4 weeks of repeated sprint training (3 sets of 5 × 10 s all-out sprint; work/rest ratio = 1:2, inter-set recovery = 5-min) in hypoxia (FiO_2:0.146) drastically increased (~38% improvement) the number of sprint repetitions to exhaustion (i.e., repeated sprint ability) and induced significant molecular adaptations (upregulation of mRNA for HIF-1α [+55%], carbonic anhydrase III [+35%], and monocarboxylatetransporter-4 [+20%]). To date, RSH is considered one of the best ways to improve sea-level repeated sprint ability, particularly in team sports athletes (Brocherie et al., 2017).

Team sports athletes, who are one of the main targets of RSH, are often required to perform multiple short sprint efforts (≤10 s) interspersed with incomplete recovery during matches (Faiss et al., 2013A; Girard et al., 2011). Therefore, most RSH studies have adopted RSA tests consisting of short-duration (≤10 s) sprints with incomplete recovery (~30 s) (Faiss et al., 2013A; Brocherie et al., 2017). However, in fact, some sports (e.g., road cycling) require longer sprints (>10 s) in incomplete recovery situations. For example, criterium, one of the road cycling events, consists of several laps around a short winding course, where athletes are required to perform >70 sprints during the ~90-minute race (Ebert et al., 2006). While the majority of sprints in the criterium event are short duration (≤10 s), longer duration sprints (11 to 15 s) also account for approximately 15% of all sprints and should not be ignored (Ebert et al., 2006). However, to date, there is a lack of evidence that the effect of RSH on the RSA test consists of longer sprints (>10-s). Therefore, this study was conducted to confirm the hypothesis that RSH can induce additional improvements in the RSA test consists of longer sprints (~10–15-s) with incomplete recovery (30-s).

MATERIAL AND METHODS

Participants

Twelve highly trained sprint runners (Age: 21.8 ± 1.5 year; height: 1.73 ± 0.04 m; weight: 62.3 ± 2.5 kg) volunteered to participate in this study after obtaining their written informed consent. Their personal best records are within 20% of the world-leading performance, categorized as "*highly trained*" (Tier 3), based on established criteria (McKay et al., 2022). During the intervention, two participants were withdrawn due to illness and injury, leaving ten participants for data analysis. None of the participants had been exposed to a hypoxic environment for at least three months prior to the study. This study adhered to the Declaration of Helsinki and was approved by the Research Ethics Committee of the University of Tokyo (No. 891).

Procedures

This study was conducted as a single-blind, randomized, controlled trial with participants blinded to inhaled air conditions (hypoxia or normoxia). This study consists of six training sessions ("*all-out*" sprint cycling) accompanied by pre- and post-tests ("*all-out*" sprint running). All participants were sprint runners familiar with cycling, who regularly performed "*all-out*" sprint cycling as part of their training. During the pre- and post-tests, participants performed 5×100-m "*all-out*" sprints with 30-s recoveries in normoxia (sea-level track) after self-selected warm-up exercises (e.g., walking, jogging, dynamic stretching, and sprinting) similar to their

usual pre-competition routine. The test and training sessions were separated into 3–5 days. Participants were asked to refrain from taking any ergogenic substances (e.g., supplements/energy drinks) for 24 h prior to the test and refrain from heavy physical activity for 48 h prior to the test. All tests were conducted at the same time of the day within participants to mitigate circadian effects. After the pre-test, participants were randomly assigned to either the hypoxic (HYP) or normoxic (NOR) training group based on their mean running velocity during the test. We verified that the mean running velocity during the pretest was similar between the groups (p = .33) (7.22 ± 0.12 vs. 7.04 ± 0.33 m/s, HYP and NOR, respectively). During the training intervention, the participants performed six sessions of repeated sprint training (2-3 sets of 5 × 10-s all-out cycle sprints; recovery: inter-sprint = 30-s, inter-set = 5min) in either hypoxia (FiO_2:0.143) or normoxia (FiO_2:0.209) for 2 weeks (3 times per week). The training sets were initially two sets and then increased to three sets in the fifth and sixth training sessions. Before the repeated sprint training, participants performed a self-selected warm-up exercise (e.g., walking, jogging, dynamic stretching) followed by 3 × 10-s cycle sprints at increased voluntary effort (60, 80, and 100% of maximal voluntary effort). After the warm-up, participants were fitted with a face mask connected by a plastic tube to a hypoxic air generator to simulate normobaric hypoxia (FiO_2:0.143; for HYP) or to an air compressor to provide normoxic air (FiO_2:0.209; for NOR). They kept the mask on during repeated sprint training sessions until the last sprint effort was completed.

Measures

During the pre- and post-tests, the running time and speed of 100 m were determined using two high-speed panning cameras (LUMIX DMC-FZ300, Panasonic, Japan), which were used to film the running participants at 240 Hz, as previously described (Kakehata et al., 2022). The sprint decrement score (Sdec) was calculated according to previous research (Girard et al., 2011). Blood samples were collected from the fingertip to measure blood lactate concentration using a lactate analyser (Lactate Pro 2, Arkray, Japan) immediately before and 3, 5, 7, and 10 min after the 100 m repeated sprints. The area under the curve (AUC) of the blood lactate concentration was calculated as previously described (Tai, 1994). All cycling exercises were performed using an electrically braked cycle ergometer (PowerMax VIII, Konami, Japan), and the workloads were set at 7.5% of each participant's body weight. The handle and seat positions were replicated within participants for each session. A hypoxic generator (Everest Summit II, Hypoxico, USA) or air compressor (SRL0.75DSN, Hitachi, Japan) provided hypoxic (~120 L/min) or normoxic (~100 L/min) air through a plastic tube and face mask.

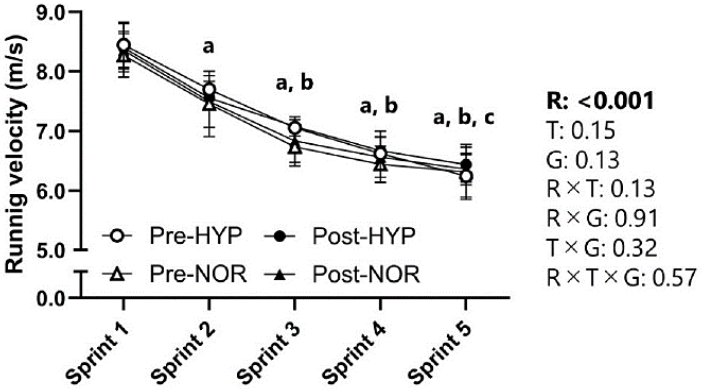
Analysis

Statistical analyses were conducted using Prism (v10.2.2; GraphPad Software, USA). Normality was assessed using the Shapiro-Wilk test. If normality assumptions are violated, the Wilcoxon signed-rank test was applied. Differences were analysed using one-, two- (Training [Pre, Post] × Group [HYP, NOR]), or three-way (Training [Pre, Post] × Group [HYP, NOR] × Repetition [Sprint 1, 2, 3, 4, 5 or Before, 3-min, 5-min, 7-min, 10-min]) repeated measures ANOVA. Tukey's multiple comparison test was used for post-hoc pairwise comparisons to identify significant effects. Mauchly's test of sphericity was employed for all ANOVA results. If these assumptions were violated, a Greenhouse-Geisser correction was applied to adjust the degrees of freedom. All values are expressed as the mean ± SD. Statistical significance was set at $p < .05$.

RESULTS

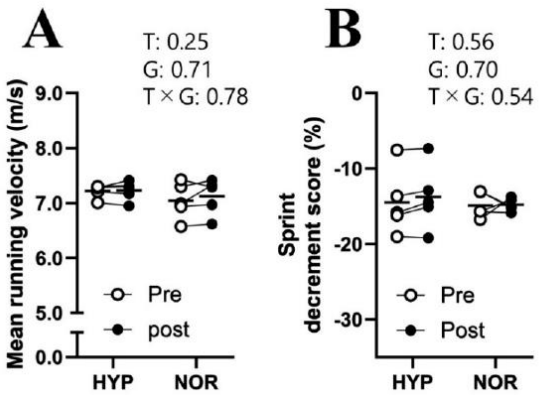
Total workload during six sessions of training did not differ (p = .75) between groups (6.39 ± 0.30 vs. 6.45 ± 0.23 kJ/kg, HYP and NOR respectively). The running velocity significantly decreased across repetitions (p < .001), independently of training (p = .15) or group (p = .13; Figure 1). No significant effect of training (p = .25) or group (p = .71) was observed for the mean running velocity of five bouts of 100-m sprints (Figure 2). There

was also no significant effect of training (p = .56) or group (p = .70) for Sdec (Figure 2). The blood lactate concentration significantly increased across repetitions (p < .001), and a significant effect of training (p < .001) and group (p = .002) were observed (Figure 3). A significant effect of training (p = .010) was identified in the AUC of blood lactate concentration, and post hoc analysis revealed a significant decrease in postHYP compared to pre-HYP (p = .008) and post-NOR (p = .029; Figure 4).



Note. Values were expressed as mean ± SD. Circle and triangle markers indicate the values for the hypoxic and normoxic training groups, respectively. Open markers express pre-test values, whereas closed markers express post-test values. a Significantly different from sprint 1 (p < .001). b Significantly different from sprint 2 (p < .001). c Significantly different from sprint 3 (p < .01). R, repetition; T, Training; G, group; HYP, hypoxic training group; NOR, normoxic training group.

Figure 1. Time course changes in running velocity across 100-m sprint repetitions.



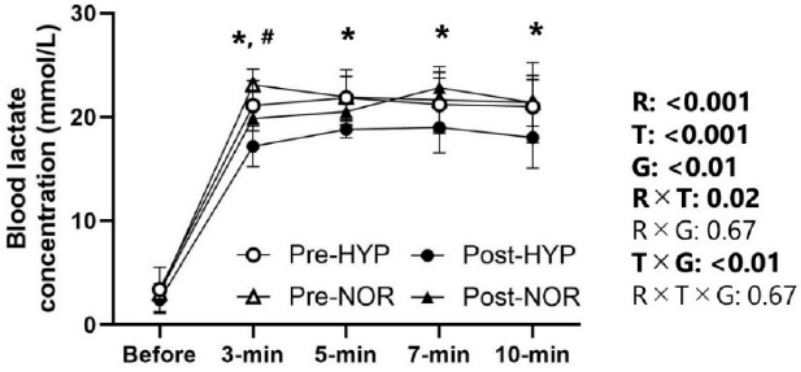
Note. Horizontal lines indicate mean values. Each plot and solid line indicate individual values and before-after changes. Open markers express pre-test values, whereas closed markers express post-test values. T, Training; G, group; HYP, hypoxic training group; NOR, normoxic training group.

Figure 2. Mean running velocity (A) and sprint decrement score (B) over five bouts of 100-m sprints.

DISCUSSION

One of the major findings of this study was that a 2-week RSH training intervention did not improve performance for a repetitive longer duration (>10-s) of sprints in highly trained sprint runners, contrary to the hypothesis of this study. These results suggest that RSH may not be effective in enhancing the ability to repeat longer duration (>10-s) sprints that are commonly observed in road cycling (i.e., criterium) (Ebert et al., 2006). Conversely, previous studies have demonstrated that RSH training intervention provides

significant additional training effects for the RSA test, which involves repeated short duration (≤10-s) sprints (Faiss et al., 2013A, 2013B; Brocherie et al., 2017).



*Note. Values were expressed as mean ± SD. Circle and triangle markers indicate the values for the hypoxic and normoxic training groups, respectively. Open markers express pre-test values, whereas closed markers express post-test values. * Significantly different from Before (p < .001). # Significantly different between the Pre-NOR and Post-HYP groups (p < .05). R, repetition; T, Training; G, group; HYP, hypoxic training group; NOR, normoxic training group.*

Figure 3. Time course changes in blood lactate concentration of five bouts of 100-m sprints.

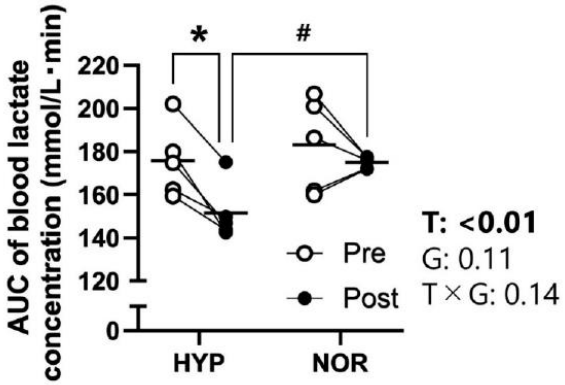


*Figure caption: Horizontal lines indicate mean values. Each plot and lines indicate individual values and before-after changes. Open markers express pre-test values, whereas closed markers express post-test values. * Significantly different from Pre (p < .01). # Significantly different between PostHYP and Post-NOR (p < .05). T, Training; G, group; HYP, hypoxic training group; NOR, normoxic training group.*

Figure 4. Area under the curve of blood lactate concentration for five bouts of 100-m sprints.

These differences can be explained by differences in energy metabolism for different exercise durations. It has been demonstrated that during intense electrical stimulation of biopsied muscle samples, phosphocreatine (PCr) degradation reaches a maximum rate (11 mmol/kg dry muscle) in as little as 1.3 seconds, followed by a rapid decline in the rate of degradation (Gastin, 2001; Greenhaff and Timmons, 1998). In addition, quick restoration of PCr by aerobic energy systems during short incomplete recovery periods is considered important for maintaining performance in repeated short (≤10-s) sprint exercises (Bishop et al., 2011). Thus, PCr breakdown and resynthesis may account for the majority of performance in repeated short duration (≤10-s) of sprints. In contrast, during repeated longer duration sprints (>10 s), the contribution of PCr degradation is relatively reduced, while the relative contribution of glycolytic energy production increased

because the maximum rate of glycolysis reaches ~5 s and then remains high for 10–20 s (Gastin, 2001; Greenhaff and Timmons, 1998). In addition, when performing repeated longer sprints (30-s), the energy contribution from lactate oxidation becomes higher in the latter bouts of the sprints (Parolin et al., 1999). Positive adaptations for PCr metabolism (e.g., increased PCr storage, increased oxygen flux) are the primary adaptations observed in RSH (Fiass et al., 2013 B; Kasai et al., 2019); however, consistent adaptations with respect to lactate metabolism (i.e., lactate production and oxidation) have not been identified by RSH intervention. These differences may lead to different outcomes of RSH on the performance adaptation of repeated short (≤10-s) or long (>10-s) duration sprints.

In this study, post-exercise blood lactate concentrations were significantly lower in post-HYP than pre-HYP and post-NOR. According to previous studies, there are no consistent results on adaptation to lactate metabolism during hypoxic sprint training. Several studies have reported that hypoxic sprint training increases glycolytic enzyme activity (Puype et al., 2013; Suzuki, 2019), but is not necessarily accompanied by increased blood lactate concentration (Puype et al., 2013; Faiss et al., 2013 B). Conversely, several studies have reported that blood lactate concentrations were rather decreased in post-performance tests after hypoxic sprint training interventions (Takei et al., 2020; Gatterer et al., 2018), and these studies support the findings of this study. One possible reason for the lack of consistent results is that blood lactate concentration is only an indirect indicator, determined by the balance between lactate efflux from the working muscle and uptake from the blood vessels into tissues (Brooks, 2018). Thus, the decrease in blood lactate concentration can be explained by both decreased lactate appearance from working muscle and/or increased lactate removal into tissues. However, decreased lactate production might not occur since hypoxic training increases glycolytic enzyme activity (Puype et al., 2013; Suzuki, 2019). In addition, during repeated sprint exercise, lactate production is stimulated only in the first and second sprints, and lactate oxidation predominates in the later sprints (Parolin et al., 1999). Therefore, decreased lactate responses in this study may be due to increased lactate removal from the blood into tissues. To support this notion, a previous study reported that RSH could induce angiogenesis and increase blood flow to skeletal muscles, suggesting a facilitated lactate shuttle system (Faiss et al., 2013 B; Brooks, 2018). Although we did not directly measure lactate metabolism, lactate uptake by the working muscle may be used for oxidative energy production. Future studies are needed to directly investigate lactate metabolism (e.g., muscle biopsy, tracer analysis) after RSH.

CONCLUSION

Six sessions of RSH intervention (2-3 sets of 5 × 10-s all-out cycle sprints) over 2 weeks did not improve RSA consisting of a long duration (~11–15 s) of sprints in highly trained sprint runners. However, RSH could lead to reduced lactate responses after repeated 100-m sprints, indicating increased uptake of blood lactate into tissues (i.e., mitochondria-enriched muscle).

AUTHOR CONTRIBUTIONS

The contributions of this study are as follows: Experimental design: NT and GK. Experimental implementation: NT, GK and HS. Data analysis: NT and GK. Paper composition: NT, GK, HH. This article has been reviewed and approved by all listed authors.

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DISCLOSURE STATEMENT

No potential conflict of interest was reported by the authors.

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