Repeated sprint training in hypoxia delays fatigue during 30-sec all-out sprint and reduces blood lactate concentrations after exercise in trained cyclists: a case study

Naoya Takei1*, Katsuyuki Kakinoki2 and Hideo Hatta1

1 Department of Sports Sciences, The University of Tokyo, 3-8-1 Komaba, Meguro, Tokyo 153-8902, Japan
2 Blue Wych Limited Company, 2-2-28-1005 Mizuhiki, Atsugi City, Kanagawa 228-0015, Japan

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Abstract  Repeated sprint training in hypoxia (RSH) is a potential training strategy to improve short-term high-intensity sprint ability and evoke adaptation of lactate metabolism. Therefore, the purpose of the present study was to examine the effects of RSH on Wingate sprint performance and lactate metabolism. Eight university cyclists performed 6 sessions of RSH (3 × 30-sec sprint with 5-min recovery, FiO2: 14.5%) over 6 consecutive days. Two days before (pre-test) and 7-9 days after (post-test) the training intervention, subjects performed Wingate tests as performance tests. We took blood samples before and 0.5, 1, 2, 3, 4, 5, 7, 10 min after the Wingate test to evaluate physiological adaptations. Mean power outputs were unchanged after the intervention (p = 0.094, d = 0.23). Whereas, the fatigue index was significantly improved (p = 0.025, d = 0.43). According to time course change in power outputs during the Wingate test, power outputs at 26 s, 29 s and 30 s were significantly improved after the intervention (p = 0.029, 0.001 and 0.001; d = 0.54, 0.74 and 0.74, respectively). Area under the curve (AUC) of blood lactate concentration was significantly lowered after the intervention (p = 0.048, d = 0.46). Six sessions of RSH over 6 consecutive days delayed fatigue during the Wingate test. AUC of blood lactate concentration was lowered after the intervention, indicating that glycogen breakdown was reduced (glycogen sparing effect) and/or lactate oxidation was increased during and after the Wingate test when the same work was performed. The effects of glycogen sparing and increased lactate oxidation would provide a competitive advantage to athletes performing multiple sprints.

Keywords: exercise performance, exercise physiology, metabolism, glycogen sparing

Introduction

Over the past few decades, many athletes have utilized altitude/hypoxic training such as ‘live high-train high’ or ‘live high-train low’ methods to enhance their sea-level exercise performance3–5. However, these methods forced athletes to stay at altitude or hypoxic residence separated from their normal daily lives, because long time hypoxic exposure (at least >12 h/day) is required to invoke positive physiological adaptations (e.g. hematological adaptation)3. Recently, intermittent hypoxic training (IHT), which possibly invokes non-hematological adaptation, has been attracting great attention from scientists and coaches. IHT is a newly established hypoxic training method in which athletes can stay at residence near sea-level and are trained with brief simulated hypoxic exposures (minutes to a few hours). Repeated sprint training in hypoxia (RSH) is considered a form of IHT and defined as repetition of several short (≤30-s) ‘all-out’ exercise bouts in hypoxia, interspersed with incomplete recoveries5–6. Compared to repeated sprint training in normoxia (RSN), some studies reported RSH provides additional training effects during the 10-sec repeated sprint test (i.e. delaying fatigue during repeated sprints)5,6. According to MacDougall et al. (1998), RSN improved repeated 30-sec sprint performance and increased phosphofructokinase activity (PFK, a key enzyme of glycolysis)3. Moreover, Puype et al. (2013) reported that RSH markedly increased PFK activity by a greater extent than RSN6. Therefore, RSH, compared to RSN, is considered a potential training strategy which dramatically invokes physiological adaptations and brings additional benefits in performance for competitive athletes. Although the repeated “long” sprint (30 s) is also a familiar method to improve exercise performance in the exercise science community, most of the RSH studies were conducted using repeated “short” sprint (4-15 s)6. Therefore, there is a lack of evidence and knowledge about repeated long sprint in hypoxia. In this
study, we examined the effects of a repeated 30-s sprint on exercise performance and lactate metabolism.

From a practical point of view, the number and duration of training interventions should not be so large in order to maintain an athlete’s training plan. Faiss et al. (2015) reported that 6 sessions of RSH improved exercise performance, compared to RSN7. Moreover, Kasai et al. (2019) reported that 6 consecutive days of RSH improved exercise performance (improved repeated 10-s sprint ability) and evoked physiological adaptation (increased phosphocreatine [PCr] content)9. Therefore, 6 consecutive days (6 sessions) of RSH seems to be enough to improve exercise performance and evoke physiological adaptations.

The present study was a case study to investigate whether 6 consecutive days of RSH enhanced single sprint performance (30-sec) and invokes some adaptation of lactate metabolism.

Materials and Methods

Subjects. Eight university trained cyclists (6 males and 2 females, height: 172.3 ± 8.6 cm, weight: 58.8 ± 8.3 kg, age: 21.0 ± 1.0 years) participated in this study. Subjects usually trained 2-4 hours/day and 5 times/week. All were born and living at sea-level, and had not been exposed to hypoxia in the past 12 months. The nature and risk of the study was fully explained to all subjects before obtaining written informed consent. This study was performed in accordance with the ethical standards of the Helsinki Declaration and was approved by the Research Ethics Committee at The University of Tokyo (No. 430-2).

Design. This study was conducted in a non-blinded manner. Subjects performed 6 sessions of RSH over 6 consecutive days. RSH consisted of 3 repetitions of 30-sec all-out sprint with 5-min passive recovery. As pre- (2 days before the first RSH) and post- (7-9 days after the final RSH) performance tests, subjects performed single 30-sec all-out sprints (Wingate test). From practical points of view, subjects performed post tests 7-9 days after the last training session in this study, because most athletes tend to reduce training volume and/or intensity 1-2 weeks before a competition for tapering and peaking. Subjects had kept performing normal cycling training (low to moderate intensity) during the intervention, but refrained from any form of high intensity exercise.

Methodology. The subjects performed pre- and post-tests, breathing sea-level room air (FiO2: 20.9%). They performed RSH breathing normobaric-hypoxic air (FiO2: 14.5%) delivered through a facemask and hose during the training and its warm-up. The normobaric-hypoxic air was generated by the hypoxic training system (YHS-B05, YKS, Japan), using the oxygen-filtration technique. The training and tests were performed using subjects’ own competitive-use road bikes and a cycle trainer (Jet Fluid pro trainer, CycleOps, U.S.A.). Power outputs were recorded by a power meter (PowerTap Hubs, CycleOps, U.S.A.) sampling at 1 Hz. Peak power output (PPO), mean power output (MPO) and fatigue index (FI) were measured for performance variables. FI was calculated as a percentage of power-drop between highest 5-s average power output and lowest 5-s average power output by reference to Inbar et al. (1996)9. Blood lactate concentration was measured by portable lactate analyzer (Lactate Pro 2, Arkray, Japan). All tests were performed at the same time of day to minimize the effects of diurnal variations. The subjects finished a light meal 3-4 hours before the tests. We did not record the meal subjects had, but they were instructed to have usual balanced meal and nobody had conducted special diets which biased some of the three major nutrients, like a low-carbohydrate and high-fat diet called a ketogenic diet. Before the Wingate tests, subjects performed 10-min low-intensity exercise (100 W, 90 rpm) and a few times of 6-sec sprints for warm-up and self-selecting optimal gearing for Wingate tests. The Wingate tests were performed with verbal encouragement. Blood samples were taken from finger tips 1 min before and 0.5, 1, 2, 3, 4, 5, 7 and 10 min after the Wingate tests. Area under the curve (AUC) of blood lactate concentration was calculated from Tai’s formula10. The subjects performed the training (11.5 min) and its warm-up (= 15 min) under normobaric hypoxic conditions (hypoxic exposure time: = 26.5 min/session).

Statistical Analysis. Differences were analyzed using paired t-tests or two-way ANOVA. Bonferroni correction was used for post hoc comparison. All values were expressed as mean ± standard deviation. Statistical significance was set at p < 0.05. The magnitude of changes in variables were expressed as standardized effect size (ES, Cohen’s d).

Results

Table 1 shows all performance and metabolic variables measured in pre- and post-tests. MPO and PPO were not improved after the training intervention (p > 0.05). FI was significantly improved after the intervention (p = 0.025, d = 0.43). Fig. 1 shows time course changes in power outputs over the 30-sec all-out sprint (Wingate test). Power outputs at 26 s, 29 s and 30 s were significantly improved after the intervention (p = 0.029, 0.001 and 0.001; d = 0.54, 0.74 and 0.74, respectively). Fig. 2 shows time course changes in blood lactate concentration before and after the Wingate test. Blood lactate concentrations were not significantly different after the intervention, at any time points including peak points (p > 0.05). AUC of blood lactate concentration was significantly decreased after the intervention (p = 0.048, d = 0.46).
In the present study, MPO and PPO during single 30-sec all-out sprints were not improved after the training intervention. However, the improvement in MPO was close to significant and a small effect size was observed (Table 1, $p = 0.094$, $d = 0.31$). Some RSH studies using repeated short sprint reported significant and larger improvements in MPO and PPO. Exercise performance tests in these studies were repeated short sprints, while we used a single Wingate sprint as a performance test in this study. Therefore, we could not compare these results directly by reason of different methods of evaluation. In this study, we used a training protocol with a short period (6 days) and small number of sessions (6 sessions), by reason of practicality, and failed to see significant improvement in...
MPO and PPO. Some studies, using the same period and number of sessions, reported improvements in exercise performance; but there is the possibility that this period and number of sessions is the lower limit for improving performance. It is also possible that statistical power was insufficient due to the small sample size of the present study (n = 8). In this study, we recruited competitive subjects during pre-race training (4 weeks before the target race). Because it is hard to reproduce the same conditions, we could not recruit additional subjects to improve statistical power. Further studies with a larger sample size and sufficient statistical power are needed. We observed decreased fatigue during the single 30-sec all-out sprints. According to Fig. 1, power outputs at 26 s, 29 s and 30 s were significantly improved (p < 0.05) and improvement in power outputs at 14 s, 15 s, 23 s and 28 s were close to significant (p = 0.098, 0.056, 0.070 and 0.097, respectively). Also, FI was significantly improved after the intervention (p = 0.025, d = 0.43). Previous studies reported that RSH delayed fatigue during multiple 10-sec repeated sprints with 20-sec recovery. As far as we know, the present study reported for the first time that RSH delayed fatigue during single 30-sec all-out sprints.

Interestingly, AUC of blood lactate concentration was decreased after the intervention, indicating RSH reduced glycogen breakdown during the single all-out 30-sec sprint or increased lactate oxidation by mitochondria during and after the sprint. The present study suggests that subjects performed the same work during the sprint despite less glycogen breakdown (‘glycogen sparing’ during sprint). According to previous studies, an excessive decrease in muscle glycogen concentration impaired repeated sprint ability (RSA). Therefore, the ‘glycogen sparing’ effect of RSH in the present study possibly improves RSA reducing glycogen use during sprint and preventing an excessive decrease in muscle glycogen. It was also suggested that there was an increase in lactate oxidation during and after the Wingate sprint. Energy production by mitochondrial oxidation plays an important role in PCr re-synthesis after exercise and quick PCr re-synthesis may contribute to improvement in RSA. Moreover, increased energy production by increased lactate oxidation in this study may play an important role in RSA because energy production by glycogen breakdown would be decreased in later part of repeated sprints. Therefore, the increased lactate oxidation observed in this study possibly improves RSA, if subjects perform additional sprints.

In contrast, the previous studies reported that RSN increased PFK activity and post-exercise blood lactate concentration. Moreover, Puype et al. (2013) reported that PFK activity was markedly increased after 6 weeks of RSH (4-9 × 30-sec sprint with 4.5-min recovery, 3 times/week, FiO₂: 14.5%), indicating RSH would increase lactate breakdown during sprints. Therefore, decreased lactate concentration in the present study may be contrary to the results of these previous studies, even though there wasn’t any control. However, increasing PFK activity does not always result in acceleration of glycolysis during exercise. In the study by Puype et al. (2013), for example, blood lactate concentrations 3 min after the first and last 30-sec all-out sprints were not significantly different after the training intervention in spite of increasing PFK activity. According to a pilot study by Gatterer et al. (2018), blood lactate concentration 2-3 min after the single 30-sec all-out sprint was 17% lower after 3 weeks of RSH (4 × 30-sec sprint with 5-min recovery, 3 times/week, FiO₂: 17.1%). In the study by Gatterer et al. (2018), however, we should note that statistical significance was not observed possibly due to the small sample size (n = 5). The present study reported similar results to the study by Gatterer et al. (2018). It showed blood lactate concentration 3 min after single 30-sec all-out sprint was 13% lower, and this decrease was close to significant (p = 0.053) after 6 consecutive days of RSH (3 × 30-sec sprint with 5-min recovery, FiO₂: 14.5%). Although the hypoxic condition and length of the training period differed from the pilot study by Gatterer et al. (2018), the present study utilized a similar training model in terms of repetitions of sprint (3 or 4 repetitions of 30-sec sprint) and reported analogous results in terms of lactate metabolism. The effects of RSH on lactate metabolism may differ if the training variables (i.e. repetitions of sprint) are manipulated. Further studies investigating the effects of RSH on lactate metabolism using different training protocols are needed.

Practical Applications

The present study shows that RSH delayed fatigue during single 30-sec all-out sprints, and possibly reduced glycogen breakdown (glycogen sparing) and increased lactate oxidation by mitochondria. The effects of glycogen sparing and/or increased lactate oxidation may increase RSA and provide a competitive advantage for athletes who need to perform multiple sprints during competitions. The RSH protocol used in the present study would be easy to implement into athletic training protocols due to its short period and duration of training (over 6 days and ≈ 26.5 min/day).

Conclusions

Six consecutive days of RSH delayed fatigue during single 30-sec all-out sprints in trained cyclists. RSH reduced blood lactate concentration after the sprint, indicating reduced glycogen breakdown and/or increased lactate oxidation during and after the sprint.

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Conflict of Interests

K. Kakinoki, an employee of Blue Wych Limited Company, voluntarily participated in the present study.

References