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Human Kinetics
BRIEF REPORT

No Influence of Acute Moderate Normobaric Hypoxia on Performance and Blood Lactate Concentration Responses to Repeated Wingates

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Background: Training in hypoxia versus normoxia often induces larger physiological adaptations, while this does not always translate into additional performance benefits. A possible explanation is a reduced oxygen flux, negatively affecting training intensity and/or volume (decreasing training stimulus). Repeated Wingates (RW) in normoxia is an efficient training strategy for improving both physiological parameters and exercise capacity. However, it remains unclear whether the addition of hypoxia has a detrimental effect on RW performance. ***Purpose:*** To test the hypothesis that acute moderate hypoxia exposure has no detrimental effect on RW, while both metabolic and perceptual responses would be slightly higher. ***Methods:*** On separate days, 7 male university sprinters performed 3 × 30-s Wingate efforts with 4.5-min passive recovery in either hypoxia (FiO_2: 0.145) or normoxia (FiO_2: 0.209). Arterial oxygen saturation was assessed before the first Wingate effort, while blood lactate concentration and ratings of perceived exertion were measured after each bout. ***Results:*** Mean ($P = .92$) and peak ($P = .63$) power outputs, total work ($P = .98$), and the percentage decrement score ($P = .25$) were similar between conditions. Arterial oxygen saturation was significantly lower in hypoxia versus normoxia (92.0% [2.8%] vs 98.1% [0.4%], $P < .01$), whereas blood lactate concentration ($P = .78$) and ratings of perceived exertion ($P = .51$) did not differ between conditions. ***Conclusion:*** In sprinters, acute exposure to moderate hypoxia had no detrimental effect on RW performance and associated metabolic and perceptual responses.

Keywords: exercise performance, exercise physiology, intermittent sprint

"Live low train high" altitude/hypoxic training methods such as intermittent hypoxic training, in which athletes live near sea level (normoxia) but train at submaximal intensities under simulated hypoxia are popular.[1,2] Exercising under hypoxic conditions likely represents a more powerful stimulus to upregulate muscle factors (eg, mitochondrial biogenesis, oxidative, and glycolytic enzymes) than equivalent training in normoxia. However, it remains controversial whether intermittent hypoxic training also provides additional performance benefits.[3] A potential reason is that intermittent hypoxic training is accompanied by reduced oxygen flux resulting from lower oxygen availability, in turn negatively impacting training intensity and/or volume (decreasing training stimulus).[3]

Repeated 30-s Wingates (RW) is a form of sprint interval training involving repetition of "all-out" efforts.[4] RW training can provide activation and improvement of both aerobic and anaerobic pathways, which may benefit athletes engaged in high-intensity activities (ie, team sports athletes, track cyclists, and sprint runners).[4,5] Interestingly, adding hypoxic exposure to RW training induced significant increase in anaerobic enzyme activity compared with equivalent training in normoxia.[6] Previous studies also indicated that acute hypoxia has no effect on an "isolated" Wingate sprint performance.[7–9] However, it remains unclear whether acute hypoxia has a detrimental effect on RW performance. To date, only one study examined the effects of acute moderate and severe hypoxia (inspired fraction of oxygen or FiO_2 of 0.164 and 0.136, respectively) versus normoxia on RW performance (4 × 30-s Wingate efforts with 4-min recovery).[10] In this previous study, blood lactate concentration (BLa) was only measured before and after completion of the RW protocol.[10] BLa is often measured to reflect glycogen breakdown, which is glycolytic energy contribution during the course of the actual exercise session. It is therefore important to describe the time course and magnitude of changes in BLa during a typical RW session, and how hypoxic exposure may alter these responses.

Therefore, our intention was to test the hypothesis that acute moderate hypoxia versus normoxia had no detrimental effect on RW performance, while both metabolic and perceptual responses would be slightly higher.

Methods

The sample size was estimated using a power analysis software G*Power (version 3.1.9.2; Bonn University, Bonn, Germany) based on the mean effect ($d = 3.18$) of the within-condition decrement to peak and mean power output during repeated 30-s Wingate efforts.[10] The power analysis resulted in a calculated total sample size of 4 participants. However, 7 male university sprinters (age 19.6 [0.8] y, weight 67.0 [7.0] kg, height 173.5 [6.7] cm, 100-m personal best sprint time: 11.42 [0.34] s, weekly training volume: 8–12 h/wk) were recruited in this study to ensure a robust data set was collected. All participants volunteered to participate after they provided written informed consent. They were born and living near sea level and had not been exposed to hypoxic environments in the past 6 months. The study was conducted according to the Declaration of Helsinki, with the protocol approved by the state research ethics committee at the University of Tokyo (no. 430-2).

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Experimental Trials

This study used a single-blinded, randomized-order, crossover test design. The overview of exercise protocol is displayed in Figure 1. On separate visits, participants performed 3 Wingate efforts (30-s "all-out" cycling bouts) with 4.5-min passive recovery in either hypoxia (FiO_2: 0.145 equivalent to ~3000 m above sea level) or normoxia (FiO_2: 0.209). Participants were instructed to refrain from taking any supplement/energy drinks (ie, caffeine and creatine) and performing heavy exercise 24 h before the tests. All tests were separated by 2–5 d and performed at the same time of day to eliminate the effects of diurnal variations.

Measures

Participants were fitted with a facemask fastened with a Velcro headset connected via plastic tubing to a hypoxic generator (YHS-B05; YKS, Nara, Japan) to simulate normobaric hypoxia. Participants started to inhale the hypoxic or normoxic air from the beginning of the warm-up until the termination of the last Wingate effort when mask was removed. Testing was conducted on a competitive-use road bike connected with a direct-drive cycle trainer (T2800 NEO Smart Trainer; Tacx, Wassenaar, The Netherlands), allowing power output measurement every second. Participants performed a standardized warm-up composed of 10-min low-intensity exercise (100 W, 90 rpm) followed by 2- to 3 × 6-s maximal sprints and finally a 5-min rest period before the first Wingate sprint. During 6-s sprints, participants confirmed their power outputs after each sprint and selected optimal gearing for the Wingates. Peak power output, mean power output, total work for the 3 sprint bouts, and the percentage decrement score were determined.[11] Arterial oxygen saturation was measured by pulse oximetry (BO-750BT; NISSEI, Tokyo, Japan) from fingertip 1 min before the first Wingate effort. A capillary blood sample taken from fingertip was analyzed for BLa with the Lactate Pro 2 (ARKRAY, Kyoto, Japan) portable analyzer immediately before the first Wingate effort and 3 min after each of the 3 exercise bouts. Participants rated their ratings of perceived exertion (RPE; 6–20 Borg scale) 1 min after each repetition.

Statistical Analysis

Differences were analyzed using paired *t* tests or 2-way repeated-measures analysis of variance. Bonferroni correction was used for post hoc pairwise comparison to identify locations of significant effects. All values are expressed as mean (SD). Statistical significance was set at $P < .05$. The magnitude of changes in variables is expressed as standardized effect size (Cohen *d*).

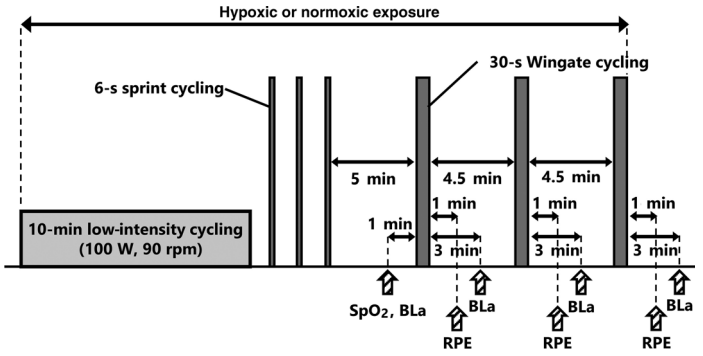

Figure 1 — Overview of experimental trial. BLa indicates blood lactate concentration; RPE, ratings of perceived exertion.

Results

Arterial oxygen saturation was significantly lower in hypoxia versus normoxia (92.0% [2.8%] vs 98.1% [0.4%], $P < .01$, $d = 3.10$). Mean power output (−10.7% [5.3]% for bouts 1–2 and −9.3% [5.2%] for bouts 2–3) and peak power output (−4.5% [6.5%] for bouts 1–2 and −14.2% [8.5%] for bouts 2–3) progressively decreased across repetitions (both $P < .001$), independently of conditions ($P = .92$ and $P = .63$, respectively, Figure 2A and 2B). Total work (670.9 [46.7] vs 670.5 [68.1] kJ/kg, $P = .98$, $d = 0.01$) and percentage decrement score (−10.8% [3.2%] vs −9.6% [3.8%], $P = .25$, $d = 0.35$) did not differ between normoxia and hypoxia. BLa and RPE increased after each sprint repetition (both $P < .001$), irrespective of the condition (Figure 3A and 3B).

Discussion

Our main finding is that acute reductions in oxygen availability had no detrimental effect on markers of RW performance, with also similar BLa and RPE values between normoxic and hypoxic conditions. One previous study reported a slight, yet significant, decrease in single Wingate performance under severe hypoxia (FiO_2: 0.104).[8] This observation was obtained in a group of sprinters who are typically less resistant to fatigue compared with their endurance-trained counterparts. In this study, which also tested sprinters but used moderate hypoxia (FiO_2: 0.145), we failed to report significant difference between conditions not just for the first but for all 3 Wingate efforts. In support, one previous study reported that RW performance (4 × 30-s Wingate efforts with 4-min recovery) was maintained under acute moderate and severe hypoxia (FiO_2: 0.164 and 0.136) compared with normoxia.[10] This lack of a difference for RW performance between normoxic and hypoxic conditions may, at least partially, relate to rest duration between efforts. That said, performances of repeated 400-m sprints with either 1-, 2-, or 5-min recovery were not significantly different under moderate hypoxia compared with normoxia, so that further studies are needed to support this suggestion.[12] When sprinting repeatedly, performance may not differ during the first few repetitions between hypoxia and normoxia, while larger performance decrements become visible under hypoxia when a larger number of maximal efforts are completed.[13] It cannot be ruled out that a larger fatigability would have been observed in this study if more than 3 Wingate efforts were completed, if between-bouts recovery was shorter, and/or if severer hypoxia levels were tested.

As expected, hypoxic exposure in this study (FiO_2: 0.145) caused a significant but rather modest decrease in arterial oxygen saturation (92.0% [2.8%]), closely matching values of ~92% reported using similar conditions (FiO_2: 0.144).[6] In our study, we did not measure muscle oxygenation trends, for instance, by using near-infrared spectroscopy, per se. In one study, exposure to moderate hypoxia (FiO_2: 0.164) versus normoxia significantly accentuated tissue oxygen desaturation levels in the vastus lateralis muscle during an isolated Wingate sprint.[10] Therefore, it is difficult to accept or reject the hypothesis that oxygen-deprived environments induce larger muscle deoxygenation levels during RW. Furthermore, we failed to observe any difference in BLa between conditions, despite progressively higher values across repetitions. McLellan et al[7] reported that muscle lactate concentration after an isolated Wingate sprint in severe hypoxia (FiO_2: 0.108) was about 2.5 times greater than in normoxic condition, whereas BLa did not increase between condition.[7] This highlights that BLa levels do not always relate to

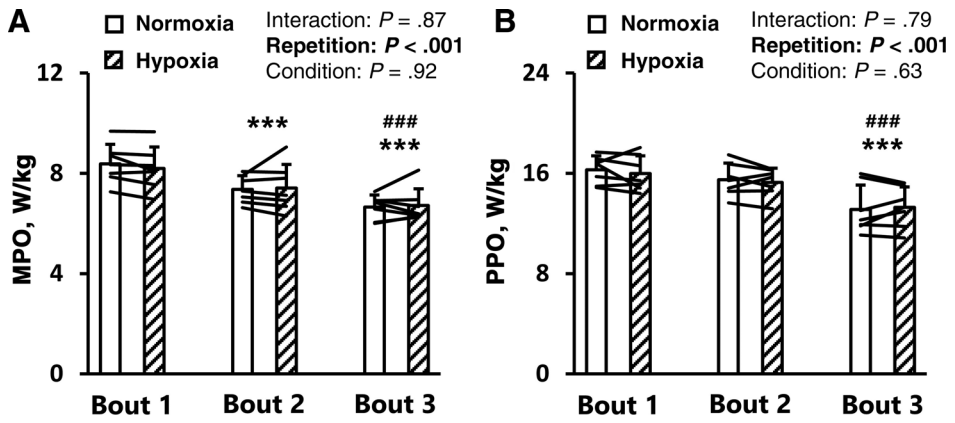

Figure 2 — (A) MPO and (B) PPO. ANOVA significant results are presented in bold. $^{***}P < .001$ versus Bout 1 and $^{###}P < .001$ versus Bout 2. MPO indicates mean power output; PPO peak power output.

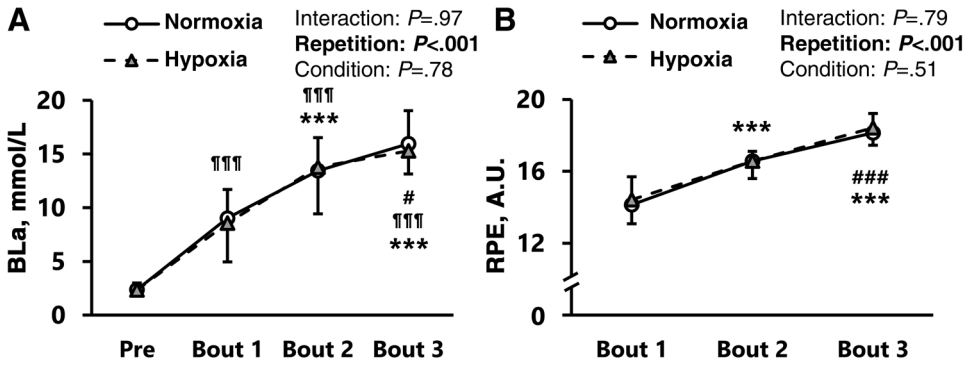

Figure 3 — (A) BLa and (B) RPE before and after each of the 3 sprint bouts. ANOVA significant results are presented in bold. $^{¶¶¶}P < .001$ versus Pre, $^{***}P < .001$ versus Bout 1, $^{#}P < .05$ versus Bout 2, and $^{###}P < .001$ versus Bout 2. BLa indicates blood lactate concentration; RPE, ratings of perceived exertion.

muscle lactate concentration. Although BLa was similar between conditions, we cannot exclude that elevations in muscle lactate concentration may have been larger in our hypoxic condition. Another possible explanation for a lack of apparent difference in BLa between conditions was severity of hypoxia. One previous study using repeated sprint exercise (10 × 6-s "all-out" sprint) under various environmental conditions (FiO_2: 0.12, 0.13, 0.14, 0.15, and 0.21) reported incremental elevations in BLa with decreasing oxygen availability.[14] It remains to be determined how exercise characteristics (work–rest ratios) and hypoxic severities can be manipulated so that muscle lactate concentration response, in turn dictating the nature of physiological adaptations (eg, mitochondrial biogenesis), could be maximized.[15] Moreover, no significant difference was observed for RPE values between the conditions. This observation differs from submaximal and/or longer exercise findings, whereby elevated RPE values are usually reported in oxygen-deprived environments.[13] RW in hypoxia, under the circumstance of this study, is not more demanding perceptually compared with normoxia.

Practical Applications

When oxygen availability is decreased (lower arterial oxygen saturation), similar performance metrics are achieved between moderate hypoxia and normoxia, with also similar BLa and RPE responses. The proposed hypoxic RW session including three 30-s "all-out" exercise bouts may be useful in sprinters because it does not impede the training stimulus and/or exacerbate metabolic/perceptual stress.

Conclusion

Repetition of 3 Wingate efforts in hypoxia had no negative consequences on performance, which also preserved BLa and RPE responses compared with normoxia in competitive sprinters.

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