

Research article

Pronounced muscle deoxygenation during supramaximal exercise under simulated hypoxia in sprint athletes

Kazuo Oguri¹✉, Hajime Fujimoto², Hiroyuki Sugimori³, Kei Miyamoto⁴, Toshiki Tachi¹, Sachio Nagasaki⁵, Yoshihiro Kato⁵ and Toshio Matsuoka⁵

¹ Faculty of Management, Shizuoka Sangyo University, Japan, ² International Pacific University, Japan, ³ Faculty of Education, Gifu University, Japan, ⁴ Department of Reconstructive Surgery, Gifu University School of Medicine, Japan, ⁵ Department of Sports Medicine and Sports Science, Gifu University Graduate School of Medicine, Japan.

Abstract

The purpose of this study was to determine whether acute hypoxia alters the deoxygenation level in vastus lateralis muscle during a 30 s Wingate test, and to compare the muscle deoxygenation level between sprint athletes and untrained men. Nine male track sprinters (athletic group, VO_{2max} 62.5 ± 4.1 ml/kg/min) and 9 healthy untrained men (untrained group, VO_{2max} 49.9 ± 5.2 ml·kg⁻¹·min⁻¹) performed a 30 s Wingate test under simulated hypoxic ($F_{I}O_2 = 0.164$ and $P_{I}O_2 = 114$ mmHg) and normoxic conditions. During the exercise, changes in oxygenated hemoglobin (OxyHb) in the vastus lateralis were measured using near infrared continuous wave spectroscopy. Decline in OxyHb, that is muscle deoxygenation, was expressed as percent change from baseline. Percutaneous arterial oxygen saturation (SpO₂), oxygen uptake (VO₂), and ventilation (VE) were measured continuously. In both groups, there was significantly greater muscle deoxygenation, lower SpO₂, lower peakVO₂, and higher peakVE during supramaximal exercise under hypoxia than under normoxia, but no differences in peak and mean power output during the exercise. Under hypoxia, the athletic group experienced significantly greater muscle deoxygenation, lower SpO₂, greater decrement in peakVO₂ and increment in peakVE during the exercise than the untrained group. When the athletic and untrained groups were pooled, the increment of muscle deoxygenation was strongly correlated with lowest SpO₂ in the 30 s Wingate test under hypoxia. These results suggest that acute exposure to hypoxia causes a greater degree of peripheral muscle deoxygenation during supramaximal exercise, especially in sprint athletes, and this physiological response would be explained mainly by lower arterial oxygen saturation.

Key words: NIRcws, muscle deoxygenation, hypoxic, 30s Wingate test, athletes.

Introduction

The performance of supramaximal exercise such as a 30 s Wingate test is not impaired by acute hypoxia. On the other hand, performance under hypoxic conditions alters significantly some physiological responses such as lower peak oxygen uptake (VO₂) (Calbet et al., 2003), lower oxygen saturation, and higher muscle lactate concentration (McLellan et al., 1990). However, little is known regarding the trends of peripheral intramuscular oxidative metabolism during supramaximal exercise under hypoxic conditions.

Near infrared continuous wave spectroscopy

(NIRcws) has been used to non-invasively and continuously evaluate the kinetics of skeletal muscle oxygen saturation during various exercises (Quaresima et al., 2003). Using the NIRcws, some studies have shown that a significantly greater degree of peripheral muscle deoxygenation occurs during various dynamic exercises, including constant-load cycling (Costes et al., 1996; Richardson et al., 1995), incremental cycling (Subudhi et al., 2007), and leg resistance exercise (Oguri et al., 2004) under hypoxic conditions. As one possible explanation for this response during exercises under hypoxia, it is suggested that the reduced arterial oxygen content plus metabolic demand decreases the overall muscle oxygen content (Costes et al. 1996). From the results and discussion of these previous studies, we hypothesized that acute hypoxia would cause a greater degree of peripheral muscle deoxygenation during supramaximal exercise when compared with performance under normoxia, whereas performance during the exercise would be maintained by an increment in anaerobic energy production.

Some studies have indicated a greater decrement in aerobic performance and VO_{2max} in highly trained athletes than untrained subjects under acute hypoxic conditions, and this phenomenon could be explained mainly by a lower arterial oxygen content (Martin and O'Kroy., 1993; Mollard et al., 2007). In trained athletes, a greater oxygen diffusion limitation in the pulmonary capillaries could account for a greater arterial oxygen desaturation than in sedentary subjects (Dempsey et al., 1982). As a consequence of arterial desaturation, arterial oxygen content and oxygen availability for the muscles would decrease. These factors lead us to the hypothesis that athletes would experience a greater degree of muscle deoxygenation during supramaximal exercise under hypoxia when compared with untrained subjects.

Here, we investigated, using NIRcws, the effects of acute hypoxia on the degree of deoxygenation in vastus lateralis muscle during a 30 s Wingate test, and compared muscle deoxygenation levels in sprint athletes and untrained subjects.

Methods

Subjects

Nine male track sprinters whose competitive events are 100m, 200m, and 400m (athletic group) and 9 healthy untrained men (untrained group) participated in the ex-

periments. All members of the athletic group had ≥ 8 years competitive experience and performed at least 5 training sessions per week under supervision of a coach. $\text{VO}_{2\text{max}}$ during a ramp-loaded bicycle exercise (10Watts/min) until exhaustion was measured using breath-by-breath analysis (Oxycon Gamma analyzer, Mijnhardt, Germany). Age, anthropometric, and physical fitness characteristics of the subjects are presented in Table 1. Subjects were informed of the purpose of the trial and all provided written informed consent before enrollment, in accordance with the Helsinki Declaration (WMADH, 2000). The study protocol was approved by the ethics committee of the Gifu University School of Medicine.

Exercise protocol

Each subject repeated 30 s Wingate tests on 2 separate days, spaced at least 1 week apart, one under normoxic ($F_{\text{I}}\text{O}_2 = 0.209$ and $P_{\text{I}}\text{O}_2 = 150$ mmHg, normoxia) and one under hypoxic conditions ($F_{\text{I}}\text{O}_2 = 0.164$ and $P_{\text{I}}\text{O}_2 = 114$ mmHg, hypoxia) for a simulated altitude of approximately 2000 m. The order of these two testing trials (i.e. under hypoxia or under normoxia) was balanced among subjects and was performed in a single-blind manner. To simulate this altitude, we used a normobaric low-oxygen chamber (ALTICUBE, Hypotec, Japan). The laboratory was maintained at 23–24 °C and a relative humidity of 50–60% during all tests. The 30 s Wingate test was performed on a mechanically braked cycle ergometer (POWERMAX-V2, Combi, Japan) with 7.5% body weight resistance for 30 seconds (Bar-Or et al., 1987; McLellan et al., 1990). Beforehand, all subjects experienced the 30 s Wingate test more than once. Prior to the start of the test, subjects were allowed to warm-up by cycling for 5 min at 50W under hypoxic and normoxic conditions. Results of the 30 s Wingate test were expressed as peak and mean power output. Both power values were normalized to body mass.

NIRcws measurement

A commercially available NIRcws monitor (BOM-L1TR, Omegawave, Japan) was used to evaluate the kinetics of peripheral muscle oxygenation during rest (30 s), exercise (30 s), and recovery (30 s). The instrument can emit laser light at wavelengths of 780, 810, and 830 nm, and determine the relative values of oxygenated hemoglobin (OxyHb), deoxygenated hemoglobin (DeoxyHb), and total hemoglobin (TotalHb), based on the Beer-Lambert law (Figure 1). At these wavelengths, the absorption coefficient of hemoglobin is obtained according to the method of Matcher et al. (1995). In the present study, OxyHb, DeoxyHb, and TotalHb are expressed as arbitrary units (a.u.), which do not represent the actual physical volume, as described (Kawaguchi et al., 2001). The basic principle of this measurement has been extensively discussed by Kawaguchi et al. (2001).

The optical probe of the instrument was placed on the skin over the right vastus lateralis muscle (approximately 15 cm above the proximal border of the patella and 5 cm lateral to the midline of the thigh). The distance between the incident and receiving point was 30 mm, and these were fixed with a tape after shielding with a rubber

sheet and vinyl. The NIRcws data were input into a personal computer every second for a sampling frequency of 1 Hz via an A/D transducer.

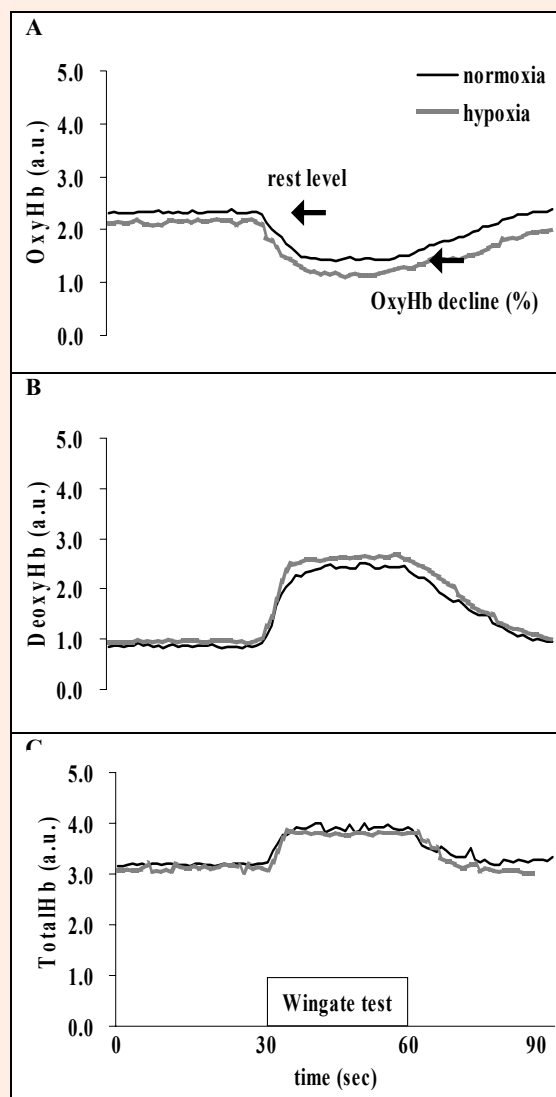


Figure 1. Typical changes in OxyHb (A), DeoxyHb (B), and TotalHb (C) in the right vastus lateralis muscle during rest, Wingate test and recovery in athlete subject under normoxic and hypoxic condition. a.u. =arbitrary unit.

The resting level of OxyHb was defined as the mean value of OxyHb for the last 30 s of the 20 min rest in a sitting posture for each subject. The OxyHb decline (%), that is the muscle deoxygenation level, during the 30 s Wingate test was defined as the decreasing rate from the resting level (100%) to the lowest level of OxyHb during the exercise (Oguri et al., 2004) (Figure 1). DeoxyHb and TotalHb increment (%) during the exercise was defined in a similar manner.

The thickness of femoral subcutaneous fat and skin was determined using B-mode ultrasonography (Toshiba Sonolazer- α , Toshiba Medical Systems, Japan), at a frequency of 5 MHz. For the ultrasonography measurement, the skin at the same site as the NIRcws probe was precisely identified and marked. One experienced technician performed all the ultrasonographic measurements.

Expiratory gas exchange, SpO₂, and blood lactate measurements

Expiratory gas was measured using breath-by-breath analysis (Oxycon Gamma analyzer, Mijnhardt, Germany) continuously during rest, exercise, and recovery at 10 s intervals. The maximum levels of VO₂ and VE during the exercise were defined as the peakVO₂ and peakVE, respectively. Percutaneous arterial oxygen saturation (SpO₂) was measured non-invasively using a forefinger probe connected to a pulse oximeter (NPB-40, Nellcor Puritan Bennett Inc, CA) during rest, exercise, and recovery at 5 s intervals. Capillary blood samples were drawn from the hyperemic ear lobe at rest, 1, 3, and 5 min following the 30 s Wingate test, and blood lactate was immediately determined using portable lactate monitors (Lactate Pro, Arkray, Japan).

Statistical analysis

All data are expressed as means ± standard deviation (SD). The Physiological characteristics of the athletic and untrained groups were compared using unpaired t-tests. A two-way analysis of variance for repeated measures was used to analyze the effects of hypoxia on performance and NIRcws parameters, peakVO₂, peakVE, SpO₂, and blood lactate, and their differences between the athletic and untrained groups. When a significant *F* ratio was obtained, Tukey's post hoc test was used to clarify significant differences in the various pairwise relationships. Pearson's correlation coefficients were used to examine the relationship between lowest SpO₂ at the 30 s Wingate test under hypoxia and ΔOxyHb (Δ represents the difference between hypoxic and normoxic values). These statistical analyses were performed by using the SPSS 11.0 statistical software. The level of statistical significance was set at *p* < 0.05.

Table 1. Physiological characteristics of athletic and untrained group. Data are means (±SD).

Variables	Athletic (n=9)	Untrained (n=9)
Age (years)	22.3 (1.6)	22.3 (1.4)
Height (m)	1.69 (.05)	1.68 (.05)
Weight (kg)	61.0 (8.0)	58.2 (7.4)
BMI (kg·m ⁻²)	21.3 (2.1)	20.7 (1.8)
VO ₂ max (ml·kg ⁻¹ ·min ⁻¹)	62.5 (4.1)	49.9 (5.2) ***
Fat and skin thickness (mm)	3.3 (1.1)	3.4 (1.2)

Fat and skin thickness = subcutaneous fat and skin thickness on surface in the right femoral muscle. *** *p* < 0.001, n.s. = not significant, athletic vs untrained.

Results

VO₂peak was significantly higher in the athletic than in the untrained group (*p* < 0.001), but there were no significant differences in age, height, weight, BMI, and femoral fat and skin thickness between two groups (Table 1). Performance parameters and respiratory gas during a 30-s Wingate test appear in Table 2. Compared with the untrained group, the athletic group had significantly higher absolute and relative peak and mean power output (*p* < 0.05). The absolute and relative peak and mean power output did not differ significantly between performance under normoxia and hypoxia conditions for either group.

The athletic group had significantly higher peakVE and peakVO₂ during the 30 s Wingate test than the untrained group in both environments (*p* < 0.05). Acute hypoxia led to a significantly higher peakVE and significantly lower peakVO₂ during the 30 s Wingate test in both groups (*p* < 0.05). In the athletic group, a significant greater increment in peakVE and decrement in peakVO₂ occurred under hypoxia when compared with the untrained group (*p* < 0.05).

Figure 1 illustrates a typical example of the variation in OxyHb, DeoxyHb, and TotalHb during the 30 s Wingate test in an athletic subject under normoxic and hypoxic conditions. In both environments, all subjects showed a dramatic decrease in OxyHb, increase in DeoxyHb and TotalHb immediately following the start of the 30 s Wingate test, followed by peak level in the latter part of the exercise. These values returned collaterally to their respective resting level after the exercise. Compared with normoxia, there were lower minimum values in OxyHb (1.4 ± 0.2 in normoxia vs. 1.2 ± 0.2 a.u. in hypoxia, *p* = 0.179), higher maximal values in DeoxyHb (2.5 ± 0.5 vs. 2.7 ± 0.6 a.u., *p* = 0.104), and lower maximum values in TotalHb (4.0 ± 0.7 vs. 3.9 ± 0.8 a.u., *p* = 0.458) under hypoxia, but not significantly, in the athletic group. In these parameters of NIRcws, there was no significant difference between the athletic and untrained groups. On the other hand, as shown in Figure 2, OxyHb decline (%) in the vastus lateralis muscle was significantly greater under hypoxic than normoxic conditions in both groups (*p* < 0.05 ~ 0.01). Under hypoxic conditions, the athletic group experienced a significantly greater OxyHb decline (%) than the untrained group (*p* < 0.01). DeoxyHb increment (%) was significantly greater under hypoxic than normoxic conditions (*p* < 0.05 ~ 0.01), and significantly greater in the athletic group than in the untrained group (*p*

Table 2. Performance parameters and respiratory gas during 30-s Wingate test. Data are means (±SD).

Variables	Normoxia		Hypoxia	
	Athletic	Untrained	Athletic	Untrained
Peak power (W)	858 (120) *	581 (71)	854 (120)*	578 (67)
Peak power/weight (W·kg ⁻¹)	14.1 (.7) *	10.3 (.9)	14.0 (1.3)*	10.2 (1.1)
Mean power (W)	604 (89) *	473 (52)	609 (89)*	465 (50)
Mean power/weight (W·kg ⁻¹)	9.9 (.6)	8.6 (.6)	10.0 (.8)*	8.3 (.6)
PeakVE (l·min ⁻¹)	116.7 (10.0) *	99.8 (9.4)	124.4 (9.6)* ¶	102.3 (11.1)¶
ΔpeakVE (%)			6.8 (2.9)*	2.5 (2.2)
PeakVO ₂ (ml·min ⁻¹ ·kg ⁻¹)	57.8 (4.5) *	44.1 (5.3)	53.7 (3.3)*¶	42.5 (5.3)¶
ΔpeakVO ₂ (%)			-6.9 (2.8)*	-3.7 (1.8)

Δ represents the difference between hypoxia and normoxia values.

* *p* < 0.05 compared with untrained, ¶ *p* < 0.05 compared with untrained normoxia.

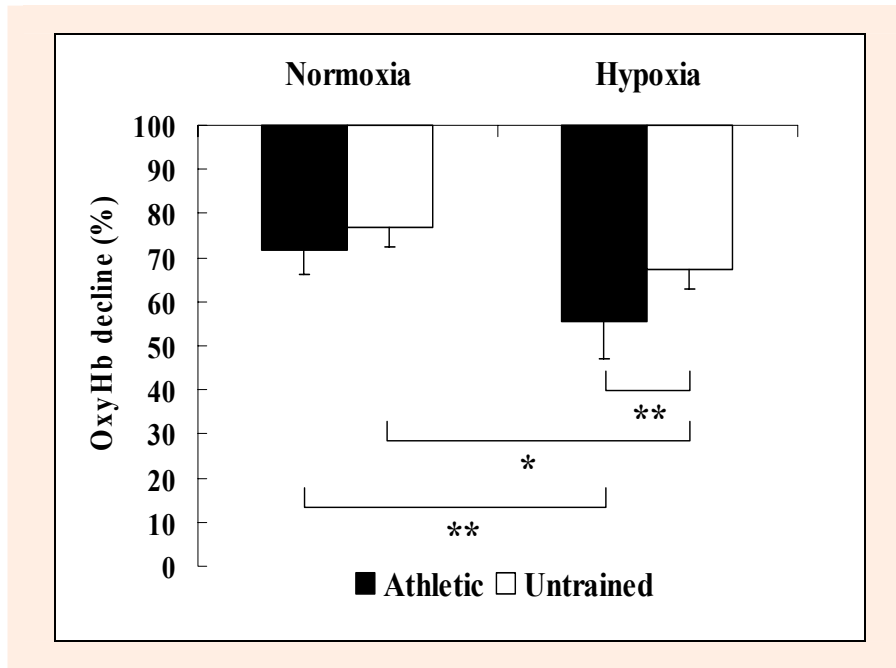


Figure 2. Comparison of OxyHb decline in vastus lateralis muscle during 30 s Wingate test between hypoxia and normoxia in athletic (n=9) and untrained group (n=9). * and ** denote $p < 0.05$ and $p < 0.01$, respectively.

< 0.01). There was no significant difference in TotalHb increment (%) among not only the environments but also the groups.

SpO₂ decreased immediately following the start of the 30 s Wingate test, followed by peak levels in the latter part of the exercise in both groups under both environments (Figure 3). Significant lower SpO₂ during exercise and recovery was observed under hypoxia when compared with that under normoxia in both groups ($p < 0.05$). Under hypoxia, the athletic group had significantly lower

SpO₂ levels during the 30 s Wingate test and recovery compared to the untrained group ($p < 0.05$). As shown in Figure 4, blood lactate increased for the duration of the recovery period in both groups under both environments. There was no difference in blood lactate at rest and after the 30 s Wingate test between hypoxia and normoxia conditions in either group. When the athletic and untrained groups were pooled, there was a high negative correlation between lowest SpO₂ in the 30 s Wingate test under hypoxia and Δ OxyHb (Figure 5).

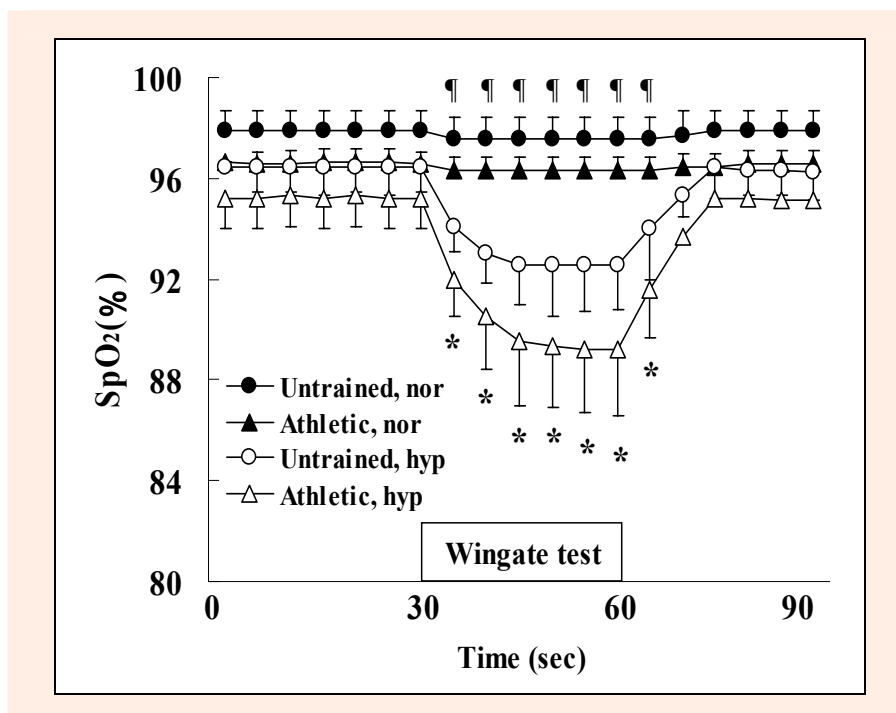


Figure 3. Time course of SpO₂ during rest, the 30 s Wingate test and recovery in hypoxia and normoxia. SpO₂ = percutaneous arterial oxygen saturation. Nor = normoxia. Hyp = hypoxia. * $p < 0.05$ Athletic v.s. Untrained in hypoxia; ¶ $p < 0.05$ hypoxia v.s. normoxia in both groups.

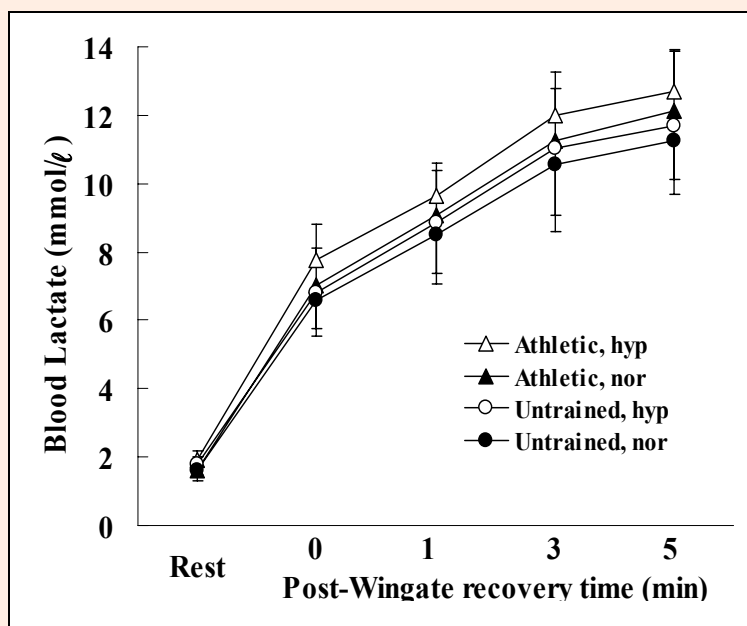


Figure 4. Time course of blood lactate during rest and after the 30 s Wingate tests and recovery in hypoxia and normoxia. Nor = normoxia. Hyp = hypoxia.

Discussion

The present study was designed to investigate the effects of acute normobaric hypoxia on peripheral muscle deoxygenation during the supramaximal exercise in trained track sprinters and healthy untrained non-athletes. As expected, the condition of acute hypoxia caused greater muscle deoxygenation in vastus lateralis muscle during performance of a 30 s Wingate test when compared with performance of the test under the condition of normoxia,

without impairing the anaerobic performance in either group. Additionally, the athletic group, which had higher VO_2max and peak power output, showed a greater degree of muscle deoxygenation during the 30 s Wingate test than the untrained group under hypoxic conditions (Figure 2).

Previously, very few studies have examined the effect of acute hypoxia on the trends in peripheral muscle oxygenation during supramaximal exercise. To the best of our knowledge, Ito et al. (2001) alone have investigated

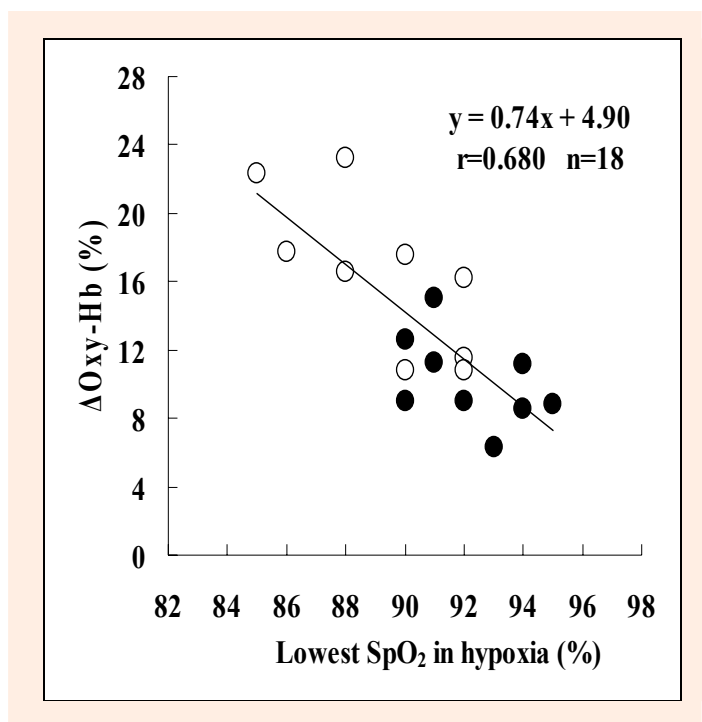


Figure 5. Relationship between lowest SpO₂ at the 30 s Wingate test in hypoxia and ΔOxyHb in all subjects. Δ represents the difference between hypoxia and normoxia values. Open symbols represent athletic group; closed symbols represent untrained group.

the effects of acute hypoxia ($F_{I}O_2 = 0.144$) on peak VO_2 , peakVE, and the vastus lateralis muscle oxygenation measured by NIRcws during and following a 30 s Wingate test in triathletes. As a result, they have reported that there were larger decrement in peak VO_2 , increment in peakVE, and slower recovery rate of muscle oxygenation following a 30 s Wingate test under hypoxia compared with that under normoxia, whereas hypoxia did not impair the performance of the test. Although their result is different from ours in the period which was observed at experiments, both studies exhibit similarities in the obvious difference between hypoxia and normoxia conditions in muscle oxygenation trends. Concerning the effects of hypoxia on the degree of muscle deoxygenation during exercise, some researchers have examined them during other exercises using NIRcws. Costes et al. (1996) have reported that the vastus lateralis muscle oxygenation was 99.3% at rest and decreased slightly to 94.9% during a 30 min steady-state cycling exercise under normoxia, whereas, under hypoxia, muscle oxygenation at rest (91.7%) was significantly lower than under normoxia and decreased dramatically to 82.7% during the exercise. In addition, several studies have shown that acute hypoxia has caused a greater degree of vastus lateralis muscle deoxygenation during constant-load (Richardson et al., 1995), incremental maximal exercise (Subudhi et al., 2007), and parallel squat exercises (Oguri et al., 2004) compared with normoxia. It follows from the present study and previous reports that acute hypoxia would enhance the degree of peripheral muscle deoxygenation during supramaximal exercise compared with normoxia.

Tissue oxygenation, defined as the relative saturation of OxyHb, depends on the balance between oxygen delivery, as reflected by the product of blood flow and arterial oxygen content, and oxygen extraction (Subudhi et al., 2007). As one possible explanation for greater muscle deoxygenation under hypoxia, the reduced arterial oxygen content plus metabolic demand has been reported to decrease the overall muscle oxygen content during exercise under hypoxia, whereas only venous blood is deoxygenated by metabolic demand during exercise under normoxia (Costes et al., 1996). A pulmonary diffusion limitation due to reduced oxygen partial pressure under hypoxia induces arterial oxygen desaturation, finally reducing arterial oxygen content and oxygen availability for the muscles (Dempsey et al. 1982; Raynaud et al. 1986). In the present study, we found that a larger difference in muscle deoxygenation between performance under hypoxic and normoxic conditions was accompanied by lowest SpO₂ in the 30 s Wingate test under hypoxia (Figure 5). These findings suggest that arterial oxygen desaturation caused by pulmonary diffusion limitation would be the dominant factor to explain a pronounced muscle deoxygenation during supramaximal exercise under hypoxic conditions. As another likely explanation for tissue deoxygenation, it is suggested that oxygen extraction could play an important role during exercise under hypoxia. Jensen-Urstad et al. (1995) have reported that not only arterial oxygen saturation but also arteriovenous oxygen difference were lower during exercises under hypoxia than under normoxia. Arteriovenous oxy-

gen difference, as reflected in oxygen extraction, may partly explain a more pronounced muscle deoxygenation under hypoxia. In addition, the above decreased oxygen availability during a hypoxic 30 s Wingate test is consistent with the decrement in peak VO_2 and increment in peakVE, which is supported by the findings of McLellan et al. (1990) and Ito et al. (2001).

There are no reports, as far as we know, to compare the effects of hypoxia on muscle oxygenation trends during supramaximal exercise between athletes and untrained subjects. In athletes, a greater degree of peripheral muscle deoxygenation seems to be caused during hypoxic supramaximal exercise in comparison with sedentary people (Figure 2). Bae et al., (1997) reported that sprinters and non-athletes elicited 95% and 82% of cuff ischaemia deoxygenation respectively during a 30 s Wingate test, and they attributed the greater muscle deoxygenation during the exercise in athletes to their training status. On the other hand, there are several reports to support that higher physical fitness causes the dramatic changes of cardiorespiratory responses during various exercises under hypoxia. Martin and O'Kroy. (1993) and Mollard et al. (2007) have shown that highly trained subjects had a greater decrement in VO_{2max} , maximal heart rate, and ventilation under hypoxia compared with untrained subjects.

In trained subjects, a greater oxygen diffusion limitation in the pulmonary capillaries could account for a greater arterial oxygen desaturation than in sedentary subjects (Dempsey et al., 1982). As a consequence of larger arterial desaturation, arterial oxygen content and oxygen availability for the muscles decrease (Mollard et al., 2007). In the present study, we have found that the athletic group had significantly lower SpO₂ (Figure 3) and larger decrement in muscle oxygenation (Figure 2) during the 30 s Wingate test compared with the untrained group under hypoxia. Our findings are consistent with earlier results, suggesting that pronounced muscle deoxygenation during supramaximal exercise under hypoxia in athletes would be explained mainly by the greater arterial oxygen desaturation. In addition, the high level of tissue oxygen extraction in trained subjects might play an important role in the decrease in oxygen availability for the muscles under acute hypoxia. Tissue oxygen extraction under normoxia is much greater in trained subjects than in untrained subjects. The higher power output during the 30 s Wingate test suggests that the athletic group had a greater tissue oxygen extraction than the untrained group. Because trained athletes would approach, even under normoxia, the physiological upper limit of tissue oxygen extraction, they could no more increase tissue oxygen extraction to compensate for the arterial oxygen desaturation under hypoxia (Mollard et al., 2007). Although we had not measured the arterial oxygen content and tissue oxygen extraction to explain the greater muscle deoxygenation in athletes, this statement is supported by a larger peak VO_2 decrement and peakVE increment under hypoxic conditions in the athletic group.

A limitation of the current study was that cardiovascular parameters such as heart rate, blood flow, stroke volume, and cardiac output during 30 s Wingate tests

were not measured, thereby, making it difficult to discuss rigorously the factors underlying the greater degree of muscle deoxygenation under hypoxia. Furthermore, we expected that blood lactate would increase more during hypoxia following the 30 s Wingate test compared with that under normoxia owing to the enhancement of the anaerobic energy released (Jensen-Urstad et al., 1995). However, blood lactate concentration during the 30 s Wingate test was not altered by hypoxia in any group. McLellan et al. (1990) have reported that despite the higher muscle lactate values that were observed following the hypoxic Wingate test, blood lactate levels were reduced compared with the normoxic test. And they suggest that a decrease in the post-exercise hyperaemia following the hypoxic Wingate test could explain the higher muscle but lower blood lactate concentrations. It may be difficult to determine the effect of hypoxia on lactate kinetics during and following supramaximal exercise using capillary blood samples.

The thickness of subcutaneous fat and skin is the main factor influencing the sensitivity and accuracy of NIRCws (Quaresima et al., 2003). In the present study, because the athletic and untrained groups had a similar thickness of femoral subcutaneous fat and skin (< 4.0 mm) on their vastus lateralis muscle (Table 1), a higher sensitivity and lower error would be worked out (Wang et al., 2001).

Conclusion

The present study demonstrated that acute simulated hypoxia would cause a greater deoxygenation level in vastus lateralis muscle during a 30-s Wingate test, and the degree of muscle deoxygenation under hypoxia is greater in sprint athletes than untrained subjects. The increment of muscle deoxygenation is correlated with lowest SpO₂ at the 30-s Wingate test under hypoxia.

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Key points

- The deoxygenation trends in the vastus lateralis muscle during 30 s Wingate test in track sprinters and untrained men under simulated hypoxic and normoxic conditions was investigated using near infrared spectroscopy.
- Acute hypoxia caused a greater degree of peripheral muscle deoxygenation than normoxia, whereas there were no changes in performance such as power output during 30 s Wingate test.
- Sprint athletes show a greater degree of peripheral muscle deoxygenation during 30 s Wingate test in hypoxia when compared with untrained subjects.
- A larger difference in muscle deoxygenation between hypoxia and normoxia is accompanied by lowest SpO₂ at the 30 s Wingate test in hypoxia.

AUTHORS BIOGRAPHY

**Kazuo OGURI****Employment**

Associate Professor, Faculty of Management, Shizuoka Sangyo University, Japan.

Degree

PhD

Research interests

Muscle metabolism, body composition assessment.

E-mail: oguri@iwata.ssu.ac.jp

Hajime FUJIMOTO**Employment**

International Pacific University, Japan.

Degree

MS

Research interest

Exercise physiology.

E-mail: Fujimoto@gifu-u.ac.jp

Hiroyuki SUGIMORI**Employment**

Associate Professor, Faculty of Education, Gifu University, Japan.

Degree

MS

Research interest

Exercise physiology.

E-mail: sugimori@gifu-u.ac.jp

**Kei MIYAMOTO****Employment**

Assoc. Professor, Department of Reconstructive Surgery for Bone and Joint, Gifu University Graduate School of Medicine, Japan.

Degree

MD, PhD

Research interests

Orthopaedics, biomechanics.

E-mail: kei@bg8.so-net.ne.jp

Toshiki TACHI**Employment**

Lecturer, Faculty of Management, Shizuoka Sangyo University, Japan.

Degree

MS

Research interest

Economy of locomotion, health and fitness.

E-mail: tachi@iwata.ssu.ac.jp

Sachio NAGASAKI**Employment**

Lecturer, Department of Sports Medicine and Sports Science, Gifu University School of Medicine, Japan.

Degree

PhD

Research interests

Neuronal physiology, balance.

E-mail: sachiona@gifu-u.ac.jp

Yoshihiro KATO**Employment**

Department of Sports Medicine and Sports Science, Gifu University School of Medicine, Japan.

Degrees

MD, PhD

Research interests

Cardiology, pediatrics.

E-mail: yoshi-kt@cc.gifu-u.ac.jp

**Toshio MATSUOKA****Employment**

Professor, Department of Sports Medicine and Sports Science, Gifu University School of Medicine, Japan.

Degree

PhD

Research interests

Anaerobic threshold, hypoxic training.

E-mail: matsuoka@cc.gifu-u.ac.jp

✉ **Kazuo Oguri, PhD.**

Faculty of Management, Shizuoka Sangyo University, 1572-1 Owara, Iwata, Shizuoka, 438-0043, Japan.