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Effects of Acute Hypoxia on Psycho-Physiological Response and Muscle Oxygenation during Incremental Running Exercise

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ABSTRACT

Pramkratok W, Yimlamai T. Effects of Acute Hypoxia on Psycho-Physiological Response and Muscle Oxygenation during Incremental Running Exercise. **JEPonline** 2021;24(3):44-54. The purpose of this study was to determine the effects of acute hypoxia exposure on physiological and perceptual responses, and muscle oxygenation during maximal incremental running exercise. Seven male elite rugby sevens players volunteered to participate in this study. In a randomized crossover design, the subjects completed two incremental running exercise tests either under normoxia ($F_{iO_2} = 20.9\%$) or after a 3-hr exposure to hypoxia ($F_{iO_2} = 14.5\%$). During both exercise conditions, pulmonary gas exchange was measured using a portable Metamax3B, rating of perceived exertion (RPE) using Borg's (6-20) scale, and muscle oxygenation at the quadriceps using a portable near-infrared spectroscopy. The results indicated that VO_{2peak} , VE_{peak} , HR_{peak} , vVO_{2peak} , and time to exhaustion were significantly lower ($P < 0.05$), while the Δ tissue oxyhemoglobin and Δ total hemoglobin were significantly higher ($P < 0.05$) in acute hypoxia than in normoxia. There was no significant difference in RPE scale during maximal exercise between the conditions ($P > 0.05$). In addition, there was a strong positive correlation between RPE and time to exhaustion ($r = 0.930$) only in normoxia but not in hypoxia, with no correlations observed in other variables in both conditions ($P < 0.05$). The present finding suggests that despite hypoxia-induced higher physiological and metabolic stress during maximal exercise, perceptual response did not differ between the conditions. These findings suggest that RPE scales may be used as a viable method for monitoring and prescribing exercise intensity during exercise, independent of hypoxia.

Key Words: Incremental Running Exercise, Muscle Oxygenation, Normobaric Hypoxia, Psycho-Physiological Response

INTRODUCTION

Monitoring training load during every training session is crucial for athletes to optimize adaptations and maximize exercise performance. Several methods have been proposed to monitor the training load (16). Rating of perceived exertion (RPE) is one of the most common methods used for regulating exercise intensity in sports science and exercise science, given that it is simple and directly related to subjective assessment of exercise intensity (17,27).

RPE is an indicator of psycho-physiological state, which centrally integrates perceptual, peripheral, experiential, and environmental sensory cues (7,18). Although the precise mechanism(s) responsible for RPE is still debated, central processing of the RPE and its role in setting exercise intensity have been intensely investigated (21). A previous study reported that perceived exertion resulted from the complex integration of afferent feedback to the central nervous system (CNS) that arises from peripheral organs during exercise such as skeletal muscles, heart, and lungs (13). Additionally, RPE has been used to predict maximal or peak oxygen uptake and endurance performance (11,22). Typically, perception of exertion can be investigated by using the psychophysical scale (6). Two most common psychophysical scales include the RPE scale and the category ratio scale (CR) (10). Despite its ecological validity and simplicity, RPE can be influenced by demanding activities and training environments (8,12).

Nowadays, altitude training is a common method employed among endurance athletes in order to improve aerobic and endurance performance (29). However, acute hypoxia exposure is well known to impair exercise performance via a reduction in arterial blood oxygen saturation (SaO_2) and a rise in peripheral markers of muscle fatigue (14,21,25-26). As a result, matched absolute fixed-exercise intensities under hypoxia may lead to greater physiological stress when compared to normoxia (20). Indeed, previous studies noted that RPE was higher in hypoxia than in normoxia during fixed-intensity interval runs and repeated-sprint cycling (8,10). Thus, self-paced or perceptual-regulated exercise intensity may confer an advantage over absolute fixed-intensity exercise in term of monitoring and prescribing intensity of exercise. This is because it may assist in overcoming the over-excessive physiological stress observed during exercise in hypoxia versus normoxia (20).

Although the traditional pulmonary gas exchange (breath-by-breath) system has been developed to determine the relative work intensity and the adequacy of oxygen transport (4), it is very useful to have an indirect method for measuring tissue oxygenation that simultaneously enables the evaluation of muscle metabolism during exercise. Near-infrared spectroscopy (NIRS) is a non-invasive technique to measure skeletal muscle oxygenation and hemodynamic during exercise. The changes in NIRS signal reflect the dynamic balance between oxygen transport and consumption in muscle tissues (20-21,28). A decrease in muscle oxygenation would indicate an increase in muscle utilization relative to muscle oxygen transport. Specifically, muscle oxygenation was reported to be progressively decreased during the incremental exercise until exhaustion (5). There was also a strong relationship between the lactate (ventilatory) threshold and the rapid reduction in muscle oxygenation and the leveling off phase in muscle oxygenation and the maximal oxygen extraction in local muscles (15). However, to date, there is limited study to determine if the relationship between percentual response and change in muscle oxygenation exists during incremental exercise under both normoxia and acute hypoxia.

Therefore, the purpose of this study was to compare the effects of acute hypoxic exposure versus normoxia on psycho-physiological responses and muscle oxygenation during incremental exercise and to determine their relationship in both conditions. We hypothesized that acute hypoxia would increase physiological stress and perceptual response while decrease muscle oxygenation level during incremental exercise compared to normoxia. Further, there is a strong negative relationship between perceptual response and muscle oxygenation during incremental exercise at exhaustion.

METHODS

Subjects

Seven male rugby players (age, 24.14 ± 4.38 yrs; height, 179.29 ± 3.95 cm; weight, 80.37 ± 5.65 kg; body fat, $18.84 \pm 4.86\%$; $\text{VO}_{2\text{peak}}$, 47.09 ± 2.67 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) volunteered to participate in this study. All subjects were members of the Thailand rugby sevens national team. They were not exposed to altitude ($>1,500$ m) at least 6 months before the start of the study. The subjects were informed of the benefits, procedures and risks of the study, and they gave their informed consent before participating. The protocol of the study was approved by the ethics committee at Chulalongkorn University, and was in compliance with the guidelines of the Helsinki Declaration.

Procedures

The subjects were required to complete two incremental running exercise tests either at normoxia ($F_{\text{I}}\text{O}_2 = 20.9\%$) or after spending 3 hrs at rest in normobaric hypoxia ($F_{\text{I}}\text{O}_2 = 14.5\%$), in a randomized crossover design. Before the experiment, the subjects were familiarized with the procedures, equipment, and the incremental running test. On the testing day, the subjects reported to a laboratory between 9 and 10 a.m., and the baseline measurements including $\text{VO}_{2\text{peak}}$, heart rate (HR), SaO_2 , RPE, blood lactate, as well as skeletal muscle oxygenation were determined. Measurements were repeated again after 7 days at a simulated hypoxic room (ATS-5KHP 750 SYSTEM, altitude training systems, NSW, Australia). All experiments were carried out at the same time of the day with similar environmental conditions (temperature ~ 24 to 25°C ; relative humidity ~ 40 to 42% ; and barometric pressure ~ 756 to 759 mmHg). The subjects were instructed to avoid any kind of strenuous activities 24 hrs before the measurements were taken. They were also asked to maintain their normal diet, refrain from caffeine intake, and get a light meal 2 to 3 hrs before the measurements (Figure 1).

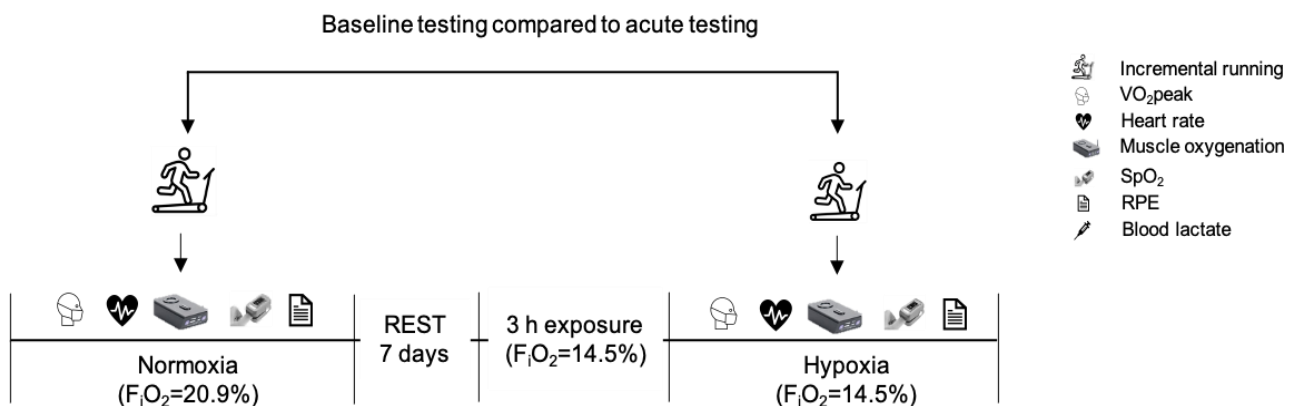


Figure 1. The Testing Procedures.

Measurements

Body Composition

Body composition (i.e., height, body weight, and percentage body fat) was measured using a body composition analyzer with ultrasonic height measurement (ioi353, Jawon Medical, Korea).

Incremental Running Exercise Test

The subjects completed an incremental running exercise test on a motorized treadmill (H/p/cosmos, Mercury, Germany) to measure VO_2peak under hypoxia and normoxia. The test began with running at an initial speed of $10 \text{ km}\cdot\text{hr}^{-1}$ with a constant 1% incline for 1 min. Then, the speed was increased by $0.5 \text{ km}\cdot\text{hr}^{-1}$ every minute until volitional exhaustion (9). Respiratory gas exchange was measured breath-by-breath using a portable Cortex Metamax3B (CORTEX, Biophysik GmbH, Germany). Before each test, gas volume was calibrated using a 3-L syringe, and a gas analyzer was calibrated using a standard gas mixture of 15% O_2 and 5% CO_2 .

HR was continuously monitored using a Polar heart rate monitor (POLAR H7; Polar Electro Oy, Kampele, Finland). SaO_2 was measured by an Oximeter (Nonin pulse oximeters, Nonin Medical Inc., Plymouth, USA) using a finger probe prior to and immediately after incremental exercise. VO_2peak was defined as the highest 30-sec average measured prior to termination of the test. The lowest running speed corresponding to VO_2peak was defined as the $v\text{VO}_2\text{peak}$. Attainment of VO_2peak was confirmed on the basis of the following criteria: (a) respiratory exchange ratio (RER) >1.10 ; and (b) blood lactate concentration (Lactate) after exercise cessation $>8 \text{ mmol}\cdot\text{L}^{-1}$. Verbal encouragement was given for each subject during the test.

Rating of Perceived Exertion (RPE)

All subjects were thoroughly briefed on the use of the RPE scale before testing. RPE was measured every minute by a Borg's 6 ("no exertion at all") – 20 ("maximal exertion") numeric scale. The subjects were instructed to pay close attention to how difficult the exercise felt, combining total exertion, fatigue, and physical stress in hypoxia, without considering one particular factor, such as leg pain and shortness of breath (21). The validity and benefits of using RPE have been previously described (19).

Blood Lactate Measurement

A capillary blood sample ($25 \mu\text{L}$) was taken from the subjects' fingertip before the test and at 3 min after the incremental running test (9). The right-hand middle finger was dried and cleaned with an alcohol wipe prior to using a sterile lancet (Accu-Check Safe-T-Pro Plus, Birmingham, United Kingdom). The first drop of blood was disregarded and then two or three drops of blood were collected into a $75\text{-}\mu\text{L}$ capillary glass tube. A $5 \mu\text{L}$ blood sample was pipetted and immediately injected into a blood lactate analyzer (LM5; Analox, United Kingdom) for blood lactate concentration analysis. The blood lactate analyzer was calibrated before each measurement.

Muscle Oxygenation

A portable near-infrared spectroscopy (Portamon, Artinis, Medical System, Gelderland, The Netherlands) was used to measure changes in muscle oxygenation of the left vastus lateralis during the incremental running exercise test. The device used the modified Beer–Lambert law and SRS methods to calculate the absolute concentration of tissue oxyhemoglobin (O_2Hb),

deoxyhemoglobin (HHb), total hemoglobin (tHb), and tissue saturation index (TSI). Before testing, the optical sensor and detector were placed on the mid-belly of the vastus lateralis muscle. To ensure that the optical sensor and detector did not move relative to the skin, the device was secured with sports adhesive tape and wrapped with a black elastic bandage around the leg to prevent contaminations from ambient light. The NIRS signal was transferred to a personal computer via Bluetooth™ technology for data acquisition (10 Hz, DPF = 4), analog-to-digital conversion, and subsequent analysis using a software program (Oxysoft 172 3.0.95 version 1511) with a moving 3 second average (23). TSI was expressed in (%), while O₂Hb, HHb and tHb were expressed in micromolar (μM). The ΔTSI, ΔO₂Hb, ΔHHb and ΔtHb were calculated by subtracting the actual value near maximal exercise from its baseline value before the incremental running test. The reliability of NIRS data was confirmed by Intraclass Correlation Coefficient (ICC) Alpha value of 0.97 to 0.98 (28).

Statistical Analyses

All statistical analyses were conducted using SPSS 23.0 (SPSS Inc., Chicago, IL., USA). Data were expressed as means ± SD. Normality of data were confirmed by the Shapiro-Wilk test. Dependent sample *t*-test was used to determine the mean difference between normoxia and hypoxia, and the Wilcoxon signed rank test was used for a non-parametric statistical analysis. Pearson product moment correlation coefficient was used to determine the relationship between conditions. The level of significance was set at *P*<0.05 for all analyses.

RESULTS

Physiological, Perceptual and Muscle Oxygenation Variables

Table 1 showed changes in physiological, perceptual, and muscle oxygenation during incremental running test to exhaustion during normoxia and acute hypoxia. VO_{2peak}, VE_{peak}, HR_{peak}, vVO_{2peak}, and time to exhaustion were significantly decreased, while the muscle oxygenation, ΔO₂Hb and ΔtHb, were significantly higher during acute hypoxia compared to normoxia. Interestingly, however, there were no significant differences in RPE, blood lactate, ΔTSI, and ΔHHb observed between two conditions.

Table 1. Physiological, Perceptual and Muscle Oxygenation Variables during Incremental Running Test during Normoxia and Acute Hypoxic Exposure.

Variables	Normoxia (F _i O ₂ = 20.9%) Mean ± SD (n = 7)	Acute Hypoxia (F _i O ₂ = 14.5%) Mean ± SD (n = 7)	P value
Physiological Responses:			
VO_{2peak} (mL·kg ⁻¹ ·min ⁻¹)	47.09 ± 2.67	36.35 ± 2.01*	0.000
VE_{peak} (L·min ⁻¹)	114.60 ± 17.20	108.21 ± 17.02*	0.016
HR_{peak} (beats·min ⁻¹)	186.29 ± 2.98	169.29 ± 9.11*	0.002
SaO₂ (%)	88.00 ± 4.58	80.86 ± 0.90*	0.007
Lactate (mmol·L ⁻¹)	11.49 ± 1.05	12.27 ± 0.96	0.107
vVO_{2peak} (km·hr ⁻¹)	14.36 ± 0.56	12.57 ± 0.35*	0.001
Time to exhaustion (min)	10.17 ± 0.89	5.96 ± 0.58*	0.000

Perceptual Response:			
RPE (6 to 20)	18.14 ± 0.69	18.29 ± 0.49	0.356
NIRS:			
Δ TSI (%)	-21.75 ± 7.05	-18.10 ± 5.14	0.217
Δ O₂Hb (μM)	-11.28 ± 4.70	-5.54 ± 3.58*	0.003
Δ HHb (μM)	15.21 ± 3.16	14.19 ± 5.19	0.633
Δ tHb (μM)	3.93 ± 2.12	8.65 ± 3.35*	0.024

Data were expressed in mean ± SD

*Significant difference between conditions at $P < 0.05$

Correlation between Physiological, Perceptual, and Muscle Oxygenation Variables during Maximal Exercise under Acute Hypoxia and Normoxia

Table 2 shows the correlations between physiological and perceptual responses and changes in muscle oxygenation at maximal exercise under normoxic and acute hypoxic conditions. Interestingly, there was a positive correlation between RPE and time to exhaustion only in normoxic condition but not in hypoxic condition. No other significant correlations were found between RPE, cardio-respiratory and muscle oxygenation variables (TSI, O₂Hb, HHb, tHb) in both conditions.

Table 2. Correlations between Physiological and Perceptual Responses and Muscle Oxygenation at Maximal Exercise in Normoxia and Acute Hypoxia.

	RPE	P value	ΔTSI	P value	ΔO₂Hb	P value	ΔHHb	P value	ΔtHb	P value
Normoxia:										
VO₂peak (mL·kg ⁻¹ ·min ⁻¹)	-0.297	0.517	0.011	0.982	-0.309	0.500	0.111	0.813	-0.519	0.233
VEpeak (L·min ⁻¹)	0.392	0.385	-0.592	0.161	-0.454	0.306	0.666	0.103	-0.016	0.974
HRpeak (beats·min ⁻¹)	-0.428	0.338	-0.656	0.110	-0.689	0.087	0.559	0.193	-0.694	0.083
SaO₂ (%)	0.474	0.282	-0.502	0.251	-0.287	0.533	0.476	0.281	0.072	0.878
Lactate (mmol·L ⁻¹)	0.487	0.267	0.623	0.135	0.301	0.512	-0.533	0.218	-0.128	0.785
vVO₂peak (km·hr ⁻¹)	0.713	0.072	0.277	0.548	0.080	0.864	0.013	0.978	0.197	0.673
Time to exhaustion (min)	0.930*	0.002	0.049	0.916	-0.169	0.717	0.223	0.631	0.043	0.927
RPE (6-20)	-	-	0.104	0.824	-0.257	0.579	0.206	0.657	-0.262	0.571

Hypoxia:										
VO₂peak (mL·kg ⁻¹ ·min ⁻¹)	-0.561	0.190	0.018	0.970	0.184	0.693	-0.279	0.544	-0.236	0.610
VEpeak (L·min ⁻¹)	-0.021	0.965	0.115	0.806	-0.195	0.676	-0.364	0.423	-0.714	0.071
HRpeak (beats·min ⁻¹)	-0.697	0.082	-0.244	0.598	-0.061	0.897	0.064	0.891	0.034	0.942
SaO₂ (%)	-0.271	0.556	-0.530	0.221	-0.484	0.271	0.531	0.220	0.305	0.506
Lactate (mmol·L ⁻¹)	0.555	0.196	-0.544	0.207	-0.636	0.125	0.629	0.130	0.294	0.522
vVO₂peak (km·hr ⁻¹)	0.354	0.437	0.213	0.647	-0.101	0.830	-0.379	0.401	-0.695	0.083
Time to exhaustion (min)	0.460	0.299	-0.478	0.278	-0.708	0.075	0.296	0.519	-0.299	0.515
RPE (6-20)	-	-	-0.223	0.631	-0.473	0.284	0.303	0.509	-0.037	0.937

*Statistical significance at P<0.05

DISCUSSION

The main findings of this study were: (a) acute hypoxic exposure resulted in significant reductions in performance (i.e., vVO₂peak and time to exhaustion) and physiological responses (i.e., VO₂peak, VEpeak, and HRpeak), but higher in ΔO₂Hb and ΔtHb during incremental running exercise compared to normoxia; (b) perceptual response based on RPE did not differ between the conditions; and (c) there were no correlations found between RPE and other physiological metabolic variables between the conditions, except for time to exhaustion. These findings suggest that perceived exertion may limit exercise performance during incremental exercise to exhaustion rather than physiological and metabolic stress induced by acute hypoxia.

It is generally believed that performance decrements with hypoxia are related to local muscle metabolic and functional alterations, particularly in response to an increased rate of H⁺. In the current study, we reported performance (i.e., vVO₂peak and time to exertion) and physiological stress (i.e., VO₂peak, VEpeak, HRpeak, and SaO₂) was reduced in hypoxia compared to normoxia. These findings are consistent with the previous reports (21,28) showing that hypoxia (F_iO₂ = 14.5%) caused significant reductions in physiological and performance responses compared to normoxia (20). This reduced performance has been purported to rely not only on the feedback signals from both the cardio-respiratory and working muscles (peripheral fatigue), but also the activity of the central nervous system (central fatigue) (2-3).

Interestingly, in the present study, RPE during exercise at exhaustion was similar under acute hypoxia and normoxia, despite differing in cardio-respiratory and metabolic responses observed between conditions. This suggests that the peripheral fatigue may not be a limiting factor of performance impairment under hypoxia. Nonetheless, we found a strong positive correlation between RPE and time to exhaustion. This finding was supported by a previous study (22) demonstrating that exercise performance is limited by perceived exertion.

With respect to muscle oxygenation, the present study demonstrated ΔTSI and ΔHHb , a proxy measure of oxygen extraction, at exhaustion did not differ between conditions. This finding was in line with the findings from several studies (21,24,28) that demonstrated the muscle's capacity of oxygen extraction is preserved under a wide range of oxygenation. Thus, it is unlikely that oxygen extraction by the skeletal muscles would limit exercise performance during maximal exercise. Moreover, we found that while the ΔO_2Hb value was decreased negatively, the ΔtHb value was increased at exhaustion in acute hypoxia compared to normoxia. This finding supports the notion that moderate hypoxia exhibited a decrease in SaO_2 and impaired oxygen delivery to the working muscle. This in turn led to a compensatory increase in blood flow for matching oxygen delivery and extraction to meet the metabolic demand during exercise (21). However, we found no correlations between RPE and all the muscle oxygenation variables measured in this study. This finding affirms that skeletal muscle oxygenation is not a major determinant of RPE.

Apart from peripheral fatigue, the central nervous system (CNS) may play a role in limiting exercise performance under hypoxia. This is indicated in that the CNS drive was altered with hypoxia (1-2). However, the exact mechanism underlying how this occurs is not completely understood and beyond the scope of this study. Nonetheless, it is possible that the accumulation of metabolites such as H^+ , due to hypoxia, may trigger sensory feedback to the CNS via group III and IV afferent fibers (2). Additionally, evidence has accumulated that indicates that it may involve the changes in cerebral oxygenation. During exercise in hypoxia, cerebral oxygenation has been reported to decrease compared with the resting level (28). Unfortunately, the cerebral blood flow and oxygenation was not measured in this study. Further studies are needed to solve this issue.

Limitations in this Study

This study has its limitations. Since only male elite rugby sevens players were participated in this study, the results are not generalized to female players and other athletic populations. Different types of athletes may have different perceptual responses, therefore the results should be interpreted with cautions.

CONCLUSIONS

Our findings indicate that despite significant reductions in performance and physiological responses at maximal exercise in acute hypoxia, the perceptual response did not differ from normoxia. In addition, rating of perception of exertion did not appear to be related to changes in muscle oxygenation in both conditions. These findings provide information that perceived exertion based on RPE scales may be used as a viable method for monitoring and prescribing exercise intensity during exercise and during a training session independent of hypoxia.

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