BRIEF REPORT

Differing Levels of Acute Hypoxia Do Not Influence Maximal Anaerobic Power Capacity

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Objective.—The purpose of this study was to determine the effects of different inspired oxygen fractions (FiO_2) on average and peak power capacity during consecutive jumps to assess the effectiveness of a hypoxic explosive-strength program.

Methods.—Eight physically active subjects (aged 33.62 ± 4.07 years; height, 1.77 ± 0.05 m; weight, 74.38 ± 6.86 kg) completed a Bosco jump test, consisting of a series of 15-second “all-out” jumps with 3 minutes of recovery, performed in a normoxia condition (N [$\text{FiO}_2 = 21\%$]) and in two hypoxic conditions: moderate hypoxia (MH [$\text{FiO}_2 16.5\% \text{ O}_2$]) and high hypoxia (HH [$13.5\% \text{ O}_2$]). A force platform provided the average and the maximal power output (W) generated during consecutive jumps. Measurements were also taken of lactate, creatine kinase, arterial oxygen saturation, and perceived exertion using the Borg fatigue scale.

Results.—The average power outputs throughout the entire sets were similar between N (3187 ± 46) and MH (3184 ± 15 ; $P > .05$), but slightly greater with HH (3285 ± 43) compared with N ($P < .05$). Values for lactate during N (7.5 ± 3.0), MH (7.7 ± 4.0), and HH (7.9 ± 3.0 ; $P > .05$), and for creatine kinase (values before, 69.8 ± 15 ; and 24 hours after in N [79.4 ± 15.60], MH [85.2 ± 26.7], and HH [84.3 ± 47.2]; $P > .05$) were similar for all conditions. Only during exercise in hypoxia were moderate and severe hypoxemia induced as the sets increased and FiO_2 was lower ($P < .05$). At the same time, the perceived exertion reported by subjects was substantially higher at HH (8.9 ± 1.1) than at N (7.1 ± 1.9 ; $P < .05$).

Conclusions.—Jumping power output was not negatively affected by mild or high hypoxia in comparison with normoxia during an anaerobic workout despite having higher hypoxemia and a greater perception of exertion.

Key words: intermittent hypoxia training, anaerobic capacity, power output, arterial saturation of oxygen, normobaric hypoxia

Introduction

Explosive-strength (ES) training is a common type of strength training used in sport to improve particularly the specific neural adaptations of muscles.¹ Testing ES performance has also been used traditionally as an indicator of improved neuromuscular characteristics and anaerobic performance.¹ Explosive strength can be determined by measuring the power output generated

during consecutive jumps, according to the well-established Bosco test for anaerobic performance.²

Physical responses of athletes to hypoxia have been extensively studied, but the way in which hypoxia may improve aerobic or anaerobic performance remains inconclusive.³ Although exercise in hypoxia has been shown to be associated with a reduction in maximal oxygen uptake ($\text{VO}_{2\text{max}}$) and flux through skeletal muscle,³ we accept that in activities that have a minimal aerobic component, such as anaerobic performances,⁴ maximal muscle power (ie, Wingate test or Bosco test) and muscle strength are minimally affected by hypoxia.⁵ It is generally assumed that hypoxia exacerbates the reduced capacity for oxygen uptake and

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transport (1% to 2% decrease in $\text{VO}_{2\text{max}}$ for each 1% decrement in oxygen saturation below 95%) leading to diminished aerobic performance. In this regard, Calbet et al⁴ reported a 7% to 16% reduction in aerobic power output during maximal exercises in hypoxia.

However, few reports have addressed decreases in maximal power output during anaerobic exercises under hypoxia to 12% FiO_2 .⁶ Indeed, some studies have recently reinforced this idea, showing significantly higher anaerobic performance after short programs (eg, 12 sessions over 4 weeks) in a regimen of anaerobic exercise when compared with equal sea level training.^{7,8} Under this premise, it could be hypothesized that a workout program combining ES training with hypoxia, which would increase the training stimulus for anaerobic metabolism,³ would lead to greater performance improvement than exercising at the same intensity in normoxia. One prerequisite, however, would be that during such training regimens (anaerobic training), the same absolute work intensity can be maintained in hypoxia as in normoxia.

The present study sought to investigate whether differing levels of inspired FiO_2 modified the capacity to generate power output during an ES workout in comparison with normoxia. We hypothesized that ES could be maintained in mild and severe hypoxia in comparison with normoxia.

Methods

PARTICIPANTS

Eight physically active men (aged 33.6 ± 4.1 years; height, 1.77 ± 0.05 m; body mass, 74.4 ± 6.9 kg) who were nonsmokers and unfamiliar with these workout methods participated in the study. They were informed about the aims of the study, possible risks, and side effects. The study was carried out in accordance with the ethical standards laid down in the 2008 Declaration of Helsinki, and it was approved by the Bioethics Committee of the University of Innsbruck.

EXPERIMENTAL DESIGN

A series of 6 consecutive jumps, lasting for 15 seconds with an intervening rest period of 3 minutes, was performed under different FiO_2 conditions: normoxia (N), $\text{FiO}_2 = 20.93\%$; moderate hypoxia (MH), $\text{FiO}_2 = 16.5\%$ O_2 (equivalent to 2500 m above sea level); and high hypoxia (HH), $\text{FiO}_2 = 13\%$ O_2 (equivalent to 4000 m altitude). These sorts of workouts (ie, plyometric training) have been found to stimulate the stretch-shortening cycle and to increase anaerobic power output.² At 48 hours before the start of the study, participants were allowed to practice the movement

pattern of the countermovement jump. A countermovement jump is where the jumper begins from an upright standing position, makes an initial downward movement by flexing at the knees and hips, and then immediately extends the hips and knees again to jump vertically up from the ground. The movement utilizes the stretch-shortening cycle, where the muscles are pre-stretched before being shortened in the desired direction.

Subjects were advised not to consume any kind of food or drink during the 90-minute period before the test. They were not allowed to perform any physical activity 24 hours before the first session or during the study. The duration of the 3-session test under the different experimental conditions did not exceed 35 minutes. Sessions were performed in a random order at the same time of day, separated by at least 72 hours. Participants were unaware of the simulated altitude at which they exercised. Before each test, subjects performed the same warm-up protocol consisting of 10 minutes of running at 65% of the maximal individual heart rate and stretching for 5 more minutes. During the exercise tests, all subjects wore the same model of shoe (Adidas weightlifting training). Furthermore, a modified CR10 Borg fatigue scale was used as a sensitive method to measure the perceived exertion and fatigue of the subjects immediately after completion of the workout and again at 24 hours afterward. The Borg fatigue scale is a category-ratio scale for most subjective magnitudes that can be used to measure perceived exertion and pain.

PROCEDURES

A hypoxicator (b-cat HA6500M, Tien, the Netherlands) was used to produce simulated altitude conditions by control of oxygen content. Hypoxia was applied by face mask, and the degree of hypoxia was controlled with Draeger Multi-Warm II (Sd 8313300, Lübeck, Germany). Fingertip capillary blood samples were obtained from each subject only 4 minutes after finishing the exercise to determine blood lactate concentrations (Biosen C-Line, EFK Diagnostik, Frankfurt, Germany). At 24 hours before and after the training sessions, creatine kinase (CK) activity was determined (Refloton Sprint, Boehringer, Mannheim, Germany), providing a basal value (24 hours before) and a measurement of the state of muscle tissue damage after the workout.⁹ A force platform device (type 9865 C, Kistler, Wien, Austria) with MLD 3.2 software (Sp Sport Mukel-Leistungs-Diagnose 3.2, Wien, Austria) was used to analyze the maximal and average power generated during the jump tests. The ES performance was expressed in watts (W). During the 30 seconds immediately after each set, the arterial oxygen saturation (Sao_2) was measured with a pulse oxymeter (Onyx II model 9550, Wien, Austria).

Figure 1. Oxygen saturation (SaO_2) at different altitude conditions: normoxia; simulated moderate altitude (16.5% F_{IO_2}); and simulated high altitude (13.5% F_{IO_2}). Statistically significant differences among groups are as follows: (a) normoxia vs moderate altitude; (b) normoxia vs high altitude; and (c) moderate vs high altitude. Significance level was considered for $P < .05$. Solid circles, 550 m; open circles, 2500 m; triangles, 4000 m.

STATISTICAL ANALYSIS

Data analyses were performed by means of the statistical software Sigmaplot (version 11, SYSTAT Software, San Jose, CA). After testing for normal distribution (Shapiro-Wilk), paired Student's t tests were used to compare values of CK, lactate, Borg fatigue scale, and SaO_2 . Power output data from the jumps were analyzed using analysis of variance with the post hoc Holm-Sidak test. The significance level was set at $P < .05$. Data are presented as mean $\pm$ SD.

Results

CREATINE KINASE, LACTATE, AND ARTERIAL OXYGEN SATURATION VALUES

Neither the CK values at 24 hours after exercise (N, 79.4 ± 15.6 U/L; MH, 85.1 ± 26.6 U/L; and HH, 84.2 ± 47.1 U/L; $P > .05$) nor the maximal lactate concentration at the completion of each workout (N, 7.5 ± 3.0 mmol/L; MH, 7.6 ± 4.0 mmol/L; and HH, 7.9 ± 2.9 mmol/L; $P > .05$) differed among hypoxia and normoxia conditions in comparison with CK prevalences (69.8 ± 15). Disregarding the hypoxia level, SaO_2 values decreased from the first to the last set but were higher at N ($98.6\% \pm 0.7\%$ to $96.7\% \pm 1.9\%$) when compared with MH ($94.7\% \pm 2.3\%$ to $90.7\% \pm 3.8\%$) and HH ($95\% \pm 2.9\%$ to $81.5\% \pm 4.6\%$). Statistically significant differences were found for the average of all sets when comparing N ($97\% \pm 1.1\%$) with MH ($91.3\% \pm 3\%$; $P < .002$) and HH ($85.1\% \pm 4.6\%$; $P < .01$).

Comparing both conditions of hypoxia, statistically significant differences were found only from fourth set to the final one (Figure 1).

EXERCISE-RELATED MEASUREMENTS

The average power generated during the entire workout (6 sets) indicates the mean power generated for each subject in each set and did not statistically differ when comparing N and MH (3187 ± 46 W vs 3184 ± 15 W; $P = .82$). However, differences in mean power output were found between N and HH (3187 ± 46 W vs 3285 ± 43 W; $P < .001$). The average power output at the different conditions for the 6 sets of each test is shown in Figure 2. The peak power reached in each set calculated individually shows that the maximal power generated for each subject in each set did not differ among the 3 conditions. The average of the peak power generated during the 6 sets was similar for N (3196 ± 761.4 W), MH (3228 ± 704 W), and HH (3306 ± 735.7 W). At the same time, as can be seen in Figure 3, no significant differences were detected among the first set and the last set of the exercise test under all conditions. Of note, the values selected from the modified Borg fatigue scale showed statistically significant differences only when the HH level was compared with N (N, 7.1 ± 1.9 ; MH, 8.1 ± 1.4 ; and HH, 8.9 ± 2.1 ; $P < .05$).

Discussion

The main finding of this study was that neither mild hypoxia nor high hypoxia negatively affected the capacity to generate anaerobic power output during sets of

Figure 2. Average power output in watts (W) during each of 6 sets under different conditions: normoxia; simulated moderate altitude (16.5% F_{IO_2}); and simulated high altitude (13.5% F_{IO_2}). Black bars, 550 m; light gray bars, 2500 m; dark gray bars, 4000 m.

Figure 3. Average peak power in watts (W) performed by subjects during first set (black bars) and last set (gray bars) under different normobaric conditions: normoxia (N); simulated moderate altitude (MA [16.5% Fio_2]); and simulated high altitude (HA [13.5% Fio_2]).

consecutive jumps in comparison with normoxia. However, subjects exercising in hypoxia reached a greater hypoxemia and perceived exertion as Fio_2 decreased. At the same time, measurements of capillary lactate and creatine kinase did not show significant differences among conditions.

Subjects were able to maintain both average and peak power output during exercise in hypoxia throughout the sets (Figures 2 and 3). These results confirm and extend the observations of previous studies applying anaerobic exercise in hypoxic conditions (Wingate test,^{4,5} running,¹⁰ jumping,⁷ and repeated sprint performance^{6,8}). The efficiency of training in hypoxia should make it possible to maintain the same absolute workload as during exercise in normoxia. All-out exercises in hypoxia conditions largely demand anaerobic energy sources to compensate for the reduction in aerobic ATP production to maintain exercise intensity. Apparently, the reduction in aerobic ATP production during anaerobic exercises in hypoxia is largely compensated through the anaerobic metabolism to maintain the exercise intensity. Thus, Friedmann et al¹⁰ reported significant decreases in O_2 uptake during supramaximal exercise in moderate hypoxia (15% O_2).

Previous studies have speculated that a reduction in the oxygen availability during high-intensity intermittent exercise in hypoxia results in a higher accumulation of blood lactate⁴ due to the greater participation of anaerobic metabolism. In our study, however, after maximal effort at different Fio_2 conditions, participants achieved a similar lactate value, approximately 7 mmol/L (see results). These values were lower than reported in similar studies that tested anaerobic performance but over a period that was twice as long (15 seconds here, vs

30 to 40 seconds).^{5,10} It could be speculated that the 15-second sets would largely affect O_2 stores and CK concentration, and the recovery time of 3 minutes between sets could be sufficient to restore these stores. Conversely, CK level depends on the intensity and type of exercise performed, higher values are related to greater skeletal muscle damage, and the CK level remains elevated 24 hours after intense exercise.⁹ We did not find changes in CK levels under the different test conditions (N and H) 24 hours after exercise, although there was an increasing trend. The short duration of the workout and the single training performed could be one explanation for the minimal increase in CK levels obtained. That does not occur when there is accumulated fatigue from continuous training without complete recovery from fatigue at the muscle level or after endurance events such as a marathon or triathlon.⁹

Another notable finding of this study was that, despite the significant declines observed in Sao_2 during exercise in hypoxia in comparison with normoxia (Figure 1), subjects were able to maintain anaerobic power output throughout the sets. These results concur with previous research showing a pronounced decrease in Sao_2 (~83%) after 30 seconds of anaerobic exercise in hypoxia but not in normoxia (~97%).⁵ In this regard, Calbet et al⁴ reported that larger decreases in Sao_2 during exercise in hypoxia could demonstrate greater anaerobic energy production in hypoxia. Conversely, higher hypoxemia was associated with greater perceived exertion during exercise in hypoxia (~25%) than in normoxia.

It could be speculated that in already well trained athletes, who require a specific, supramaximal exercise stimulus, the addition of hypoxia to an anaerobic exercise regimen could lead to muscular and metabolic stimulus that cannot be attained with training alone. This issue has been recently investigated through short programs of high-intensity intermittent training in hypoxia showing a greater anaerobic performance in comparison with the same protocol performed in normoxia.^{7,8}

Conclusion

The present study showed that average and peak power output during ES training was not negatively affected by different hypoxia levels, but it did slightly increase at high hypoxia. Performing such a training regimen could improve the adaptations of anaerobic metabolism, as has recently been reported. Additionally, it may be speculated that lower training volumes could be enough to obtain similar adaptations when compared with training in normoxia. However, further studies are needed to confirm these conclusions.

As for practical applications, many sports demand high anaerobic power outputs, and improving this

capacity is highly desirable for these athletes. Therefore, this type of training could be helpful for already well trained athletes to increase their training stimulus, and consequently, their training adaptation.

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