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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 [Fio_2 = 21%]) and in two hypoxic conditions: moderate hypoxia (MH [Fio_2 16.5% o_2]) and high hypoxia (HH [13.5% 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 (Vo_2max) 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 Vo_2max for each 1% decrement in oxygen saturation below 95%) leading to diminished aerobic performance. In this regard, Calbet et al[4] 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), Fio_2 = 20.93%; moderate hypoxia (MH), Fio_2 = 16.5% o_2 (equivalent to 2500 m above sea level); and high hypoxia (HH), 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 prestretched 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-Warn 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).