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Introduction: Although training in hypoxia has been suggested to improve sea level and altitude performance, most studies have only evaluated its effect on maximal aerobic capacity in either normoxia or hypoxia. The present study evaluated the effect of a live low-train high training regimen on both normoxic and hypoxic endurance performance and aerobic capacity. **Methods:** There were 18 male subjects who performed 20 training sessions in either a normoxic ($F_IO_2 = 0.21$) or hypoxic ($F_IO_2 = 0.12$) environment. Both the Control ($N = 9$) and Hypoxic ($N = 9$) group subjects trained at an intensity that maintained their heart rate at a level corresponding to that elicited at 50% of peak power output attained in normoxia or hypoxia, respectively. Before, during, upon completion, and 10 d after the protocol, subjects' aerobic capacity (VO_{2peak}) and endurance performance (80% of VO_{2peak}) were determined under normoxic and hypoxic conditions. **Results:** Mean $\pm$ SD normoxic VO_{2peak} increased significantly only in the Control group from 45.7 $\pm$ 6.1 to 53.9 $\pm$ 3.9 (ml $\cdot$ kg^{-1} $\cdot$ min^{-1}), whereas hypoxic VO_{2peak} did not improve in either group. The Control group exhibited significant improvements in normoxic, but not hypoxic peak power output (PPO) and time to exhaustion, whereas the Hypoxic group only exhibited improvements in normoxic time to exhaustion. During each testing period, we also assessed pulmonary function, selected hematological variables, and anthropometry. There were no significant changes in these variables in either group after the training protocol. **Conclusion:** The hypoxic training regimen used in the present study had no significant effect on altitude and sea level performance.

Keywords: endurance training, hypoxia, peak power output, time to exhaustion, peak oxygen uptake.

THE ABILITY OF HYPOXIC training in improving sea level and altitude aerobic performance depends on whether the hypoxic "dose," or rather the extent and duration of the hypoxic stimulus, is sufficient to initiate physiological changes that would be manifest in improved performance (36). Several different hypoxic training protocols have been developed over the last few decades (11). One such training regimen advocates daily training sessions in a hypoxic environment, and has been termed "live low-train high" (LL-TH). It is based on the principle of the establishment of substantial reductions in the partial pressure of oxygen in muscle cells, induced by concomitant hypoxia and exercise, thus stimulating specific signaling pathways mediated mainly by the hypoxia-inducible factor, and resulting in performance-relevant changes within the muscle (14). The main allure of the LL-TH is the potential concomitant benefits of both training and hypoxic acclimatiza-

tion, whereas the main drawback of this protocol is the inability to perform high-intensity training (23).

Nevertheless, results regarding the effect of the LL-TH training regimen on exercise performance remain equivocal. Studies on moderately trained subjects or trained subjects (14) using normobaric hypoxia have reported both improvements in altitude (3,12) and sea level performance (1,25), as well as no effect on hypoxic (29,32) and normoxic performance (18,24). Bakkman et al. (2) have even suggested that LL-TH may be disadvantageous, especially for muscle oxidative function, compared to the same training in normoxia.

Since the main aim of the LL-TH protocol is to enhance sea-level performance, very few studies have included the evaluation of both sea-level and altitude performance following the hypoxic training protocol. For athletes participating in winter sports, hypoxic training may be valuable in the preparation for summer training camps at altitudes where snow-covered terrains are available. Altitude acclimatization conducted prior to attendance at an altitude training camp may improve the quality of the training at altitude. Among the few studies using trained athletes that have incorporated testing of hypoxic performance following the LL-TH protocol (31,32), the results are also not consistent. Whereas Terrados et al. (31) observed improvements in total work performed following hypoxic training, others have not (32). Similarly, very few studies using untrained subjects (3,7,12) performed tests under both hypoxic and normoxic conditions and controlled the subjects' training. This is especially important since the strict control of training permits elucidation of the separate effects of training and hypoxia per se.

The differences in the hypoxic doses used by the various studies, namely level of simulated altitude, total duration of the daily exposure and number of exposures,

make it difficult to compare the results of different studies that have used the LL-TH regimen (23) and may also be the source of some of the discrepancies. Further studies investigating performance outcomes also in hypoxia are clearly warranted, to elucidate the potential benefits of hypoxic training on altitude performance (14,27). Although the results of the studies on both trained and untrained subjects are not very compelling, it seems that LL-TH may be used effectively, even by elite athletes, for altitude competition preparation (35). In addition, the possible beneficial effects of the LL-TH protocol for hypoxic performance could provide the rationale for using it as a preparation tool for soldiers and pilots performing demanding missions at altitude (4).

Since some studies have shown potential benefits and the optimal dose has not yet been identified, we decided to test a specific LL-TH regimen that we hypothesized, according to previously tested protocols, would provide benefits. The aim of the present study was, therefore, to evaluate the effect of a specific LL-TH regimen in moderately active subjects on sea level and altitude performance and aerobic capacity with a carefully controlled and identical training of all subjects. We hypothesized that as a consequence of training in normobaric hypoxia, improvements in hypoxic performance will be greater than after normobaric normoxic training.

METHODS

Subjects

There were 18 healthy, young, and moderately active men who participated in this study. The participants had no previous endurance training history and were randomly assigned to either the Hypoxic ($N = 9$; age: 20.1 ± 3.0 yr; stature: 182.8 ± 4.3 cm; body mass: 77.4 ± 8.7 kg; BMI: 23.1 ± 2.3 kg $\cdot$ cm^{-2}; body fat: $11.7 \pm 4.1\%$) or Control ($N = 9$; age: 22.1 ± 4.0 yr; stature: 179.3 ± 5.0 cm; body mass: 72.9 ± 9.7 kg; BMI: 22.6 ± 2.4 kg $\cdot$ cm^{-2}; body fat: $10.4 \pm 3.0\%$) group ($P < 0.05$). Subjects gave their informed consent prior to the study, which was approved by the Institutional ethics committee, and was performed in accordance with the guidelines of the Helsinki Declaration.

Experimental Design

The training protocol (**Fig. 1**) consisted of 20 training sessions on a cycle ergometer over a period of 4 wk (5 sessions per week). Each training session included a 5-min warm-up at 20% of normoxic peak power (PPO), followed by a 60-min bout of exercise, and a 5-min recovery period. The exercise intensity corresponded to the heart rate (HR) achieved at 50% PPO, determined on a previous occasion from tests of maximal aerobic capacity (Vo$_{2peak}$) in normoxia and hypoxia. Thus, the Control group trained at 50% of their normoxic PPO, whereas the Hypoxic group trained at 50% of their hypoxic PPO. The PPO was calculated as the last completed work rate plus the fraction of the time spent in the final, noncompleted work rate multiplied by 30 (21). The Control group performed all their training sessions in normo-

baric normoxia, at 300 m above sea level (altitude of the laboratory). The Hypoxic group performed their training in a climatic chamber (IZR d.o.o., Skofja Loka, Slovenia) at the Jozef Stefan Institute, which maintained the air temperature and humidity at 25°C and 50%, respectively. The normobaric hypoxic environment ($F_IO_2 = 0.12$) within the climatic chamber was maintained with a Vacuum Pressure Swing Adsorption system (b-Cat, Tiel, The Netherlands) that delivered oxygen-depleted air to the chamber. Samples of the chamber air were regularly analyzed for oxygen and carbon dioxide (CO_2) content, and the delivery of the oxygen-depleted air was regulated according to the results of the gas analysis. In the event that the oxygen level achieved the preset fraction of oxygen, delivery of oxygen-depleted air was discontinued. In the event that the oxygen level dropped below the preset level, or the CO_2 concentration increased by 0.5%, a large industrial-type fan was activated, drawing normoxic air from the external environment into the chamber. Since there was a constant flow of gas into the chamber, a relief valve prevented any undue pressure fluctuations within the chamber. During the training, all subjects' HR was monitored continuously (Hosand system, Verbania, Italy) and the external workload was adjusted to maintain the target HR. The HR monitoring software enabled the establishment of an individual training HR interval for each subject. The individual interval was set within ± 4 bpm of the targeted HR measured during the first Vo$_{2peak}$ test attained in normoxia (Control group) or hypoxia (Hypoxic group), respectively. Whenever the subject's heart rate was outside the set interval, a visual signal prompted the researcher to either increase or decrease the work rate accordingly. During each training session, subjects provided ratings of perceived exertion on a Borg scale (0–10) separately for the limb and central sensations.

Hypoxic and normoxic aerobic capacity and endurance performance was evaluated before (Pre), in the middle (Mid), at the end (Post), and 10 d after (After) the LL-TH training program (Fig. 1). Subjects were requested not to participate in any physical activity at least 2 d before and not to drink caffeinated beverages at least 4 h prior to the performance tests. Each test period (Pre, Mid, Post, After) consisted of 2 d of testing separated by a rest day. During the test days, two performance tests were conducted on each day: one incremental (Vo$_{2peak}$) and one constant power (CP) test to exhaustion in either normoxic or hypoxic conditions (Fig. 1). The order of the hypoxic and normoxic tests was randomized and counterbalanced. Anthropometric measurements, hematological examinations, and pulmonary function tests were conducted on separate days before the exercise test days (Fig. 1).

Procedures

Anthropometry and pulmonary function: Measurements of body mass, body stature, and skinfolds were performed in the Pre and Post testing periods only. Body fat was calculated from nine skin fold measurements
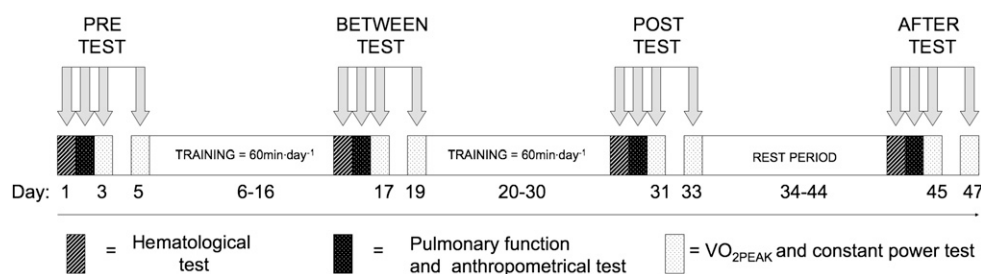
Fig. 1. Schematical presentation of the testing and training schedule.

[triceps, subscapular, chest, suprailiac, abdominal, thigh (mid, above, below) and inguinal] according to the equation of Jackson and Pollock (16). The pulmonary function tests were performed at each testing period (Fig. 1) and included: forced vital capacity, forced expiratory volume in 1 s (FEV_1), peak expiratory flow, slow vital capacity, and maximum voluntary ventilation. The tests were performed according to the criteria published by Miller et al. (26). The pneumotachograph (Cardiovit AT-2plus, Schiller, Baar, Switzerland) was calibrated before each test with a 3-L syringe. Each test was performed three consecutive times and the highest of the three acceptable values was used for the following analysis.

Hematological tests: Blood samples were drawn from the antecubital vein on the morning of the first exercise test day in each of the four testing periods (Fig. 1). The subjects fasted overnight prior to the procedure. The samples were analyzed by a hematological laboratory (Adria laboratories d.o.o., Ljubljana, Slovenia) using the cythochemical impedance method (Pentra120; Horiba ABX Diagnostics, Montpellier, France) for hemogram (coefficient of variation: < 2%), and the turbo-bidiametrical method (Hitachi 912; Roche Diagnostics, Basel, Switzerland) for transferrin analysis. Blood samples were analyzed for red blood cell count (RBC), hemoglobin (Hgb), hematocrit (Hct), transferrin, and ferritin.

Exercise testing: All exercise performance tests were performed in the same sea-level laboratory (Valdoltra Orthopedic Hospital, Ankaran, Slovenia) under equivalent environmental conditions. The normoxic ($F_IO_2 = 0.21$) and hypoxic ($F_IO_2 = 0.12$), corresponding to altitude of 4500 m) Vo_{2peak} tests consisted of an incremental-load exercise to exhaustion performed on an electrically braked cycle-ergometer (ERG 900S, Schiller, Baar, Switzerland). The protocol commenced with a 10-min rest period and a 2-min warm-up at a work rate of 60 W. The tests in hypoxia included a 5-min rest period in normoxia and a 5-min rest period in hypoxia prior to the 2-min warm-up. Thereafter, the load was increased each minute by 30 W. Vo_{2peak} was taken as the average of the 60-s peak Vo_2 values prior to termination of the test. Attainment of Vo_{2peak} was confirmed on the basis of the following criteria: respiratory exchange ratio > 1.1, plateau in Vo_2 during the last 15 s of the trial, and inability to maintain the cycling cadence at the required level of 60 rpm.

The normoxic and hypoxic CP tests were performed at an intensity corresponding to 80% of normoxic Vo_{2peak}. The protocol consisted of a 2-min rest period (either in normoxia or hypoxia, depending on the test),

a 2-min warm-up at a work rate of 60 W, and then cycling to exhaustion with the predetermined workload. Time to exhaustion was determined by the number of seconds the subject was able to sustain the pedaling cadence of 60 rpm. During the Vo_{2peak} tests, the Vo_2 and ventilation (V_E) were recorded at 10-s intervals with a metabolic cart (CS-200, Schiller, Baar, Switzerland). Subjects breathed through a low-resistance two-way valve (Model 2, 700 T-Shape, Hans Rudolph, Inc., Shawnee, KS). In the normoxic condition they inspired room air and in the hypoxic condition they inspired a premixed humidified breathing mixture (12% O_2, 88% N_2) from a 200-L Douglas bag. Flow and volume calibrations of the metabolic cart were conducted with a 3-L plexiglas syringe prior to each test session. The gas analyzers were calibrated with two standard calibration gas mixtures. During the tests we also continuously monitored HR (Vantage NVTM, Polar Electro, Kempele, Finland) and arterial oxygen saturation (S_pO_2) using a pulse oximetry device (Nellcor, BCI 3301, Boulder, CO) with ± 2 units accuracy across the range of 70–100% (22). If necessary, we pre-warmed the finger used for the measurement of S_pO_2 with warm water.

Statistical Analysis

Differences between group means in the two conditions over the training period were analyzed with a 3-way ANOVA [group (Control and Hypoxic) × condition (normoxia-hypoxia) × testing period (Pre, Mid, Post, After)]. A Tukey post hoc test was used to compare the specific differences. The significance level was set at 5%. Due to technical problems (malfunction of the gas sensor cell within the metabolic cart) encountered in four of the hypoxic Pre Vo_{2peak} tests, we used a regression model to predict the missing Pre Vo_2 hypoxic values from the power output-Vo_{2peak} relationship produced from the normoxic and available hypoxic tests. Due to this same technical problem, the Mid Vo_{2peak} results are also omitted from the analysis. All statistical analyses were performed using Statistica 5.0 (StatSoft, Inc., Tulsa, OK).

RESULTS

The average power output, HR, and S_pO_2 values during each week for the Hypoxic and Control groups are presented in **Table I**. The power output of training increased by ~15 W in the Control and by ~10 W in the Hypoxic group, and was significantly lower in the Hypoxic group at all times. HR remained fairly constant

TABLE I. AVERAGE WEEKLY POWER OUTPUT (PO_{mean}), HEART RATE (HR), PERIPHERAL (RPE_{local}) AND CENTRAL (RPE_{cen}) RATE OF PERCIEVED EXERTION, AND ARTERIAL OXYGEN SATURATION (S_pO_2) OF THE CONTROL AND HYPOXIC GROUPS DURING THE TRAINING SESSIONS.

	Control Group				Hypoxic Group			
	1st week	2nd week	3rd week	4th week	1st week	2nd week	3rd week	4th week
PO_{mean} (W)	152 ± 18	158 ± 17	159 ± 16	167 ± 21*	114 ± 22#	118 ± 25#	122 ± 23#	124 ± 19#*
HR (bpm)	146 ± 9	140 ± 7*	140 ± 7*	144 ± 8	151 ± 8#	148 ± 8#	145 ± 9#*	145 ± 9*
RPE_{local}	2.2 ± 0.8	2.2 ± 0.9	2.3 ± 1.2	2.6 ± 1.4	5.2 ± 1.2#	5.4 ± 1.1#	6.2 ± 2.2#*	5.5 ± 1.1#
RPE_{cen}	1.7 ± 0.9	1.8 ± 0.7	2.0 ± 1	2.0 ± 1.3	4.7 ± 0.9#	4.8 ± 0.8#	5.6 ± 1.1#*	4.7 ± 0.9#
S_pO_2	--	--	--	--	79 ± 3	78 ± 3	78 ± 4	80 ± 5

Values are mean ± SD.
* Significant differences within groups between week averages ($P < 0.05$).
# Differences in weekly averages between groups ($P < 0.05$).

(± 3%) during the training in both groups, although there were some significant fluctuations in both groups during the second, third, and fourth training week. However, rates of perceived exertion were significantly higher in the Hypoxic group compared to the Control group during all training sessions (Table I). For the Hypoxic group, the values of S_pO_2 during the training remained similar throughout the training period (Table I).

No significant changes were noted in subjects' anthropometry after the LL-TH protocol. The values of the pulmonary function tests in both groups ranged between 5.5 ± 1.2 L and 5.9 ± 0.6 L for forced vital capacity; 4.6 ± 0.9 L and 4.9 ± 0.5 L for FEV_1; 5.0 ± 0.8 L and 5.9 ± 1.1 L for slow vital capacity; and between 183 ± 3 to 201 ± 27 L for maximum voluntary ventilation. There were no differences between or within groups at any testing period. Similarly, no significant differences in any of the hematological variables were noted between the Control and Hypoxic groups Pre, Mid, Post, or After the LL-TH (**Table II**).

Hypoxic Vo_{2peak}, PPO, and S_pO_2 values were lower than the normoxic values in all testing periods (**Fig. 2** and **3**). The Control group improved their Post and After normoxic Vo_{2peak} compared to the Pre, but hypoxic Vo_{2peak} remained unchanged. There were no hypoxic training-induced changes observed in the normoxic and hypoxic Vo_{2peak} of the Hypoxic group (Fig. 2). PPO improved After LL-TH only for the Control group in normoxia (Fig. 3). No changes were observed in hypoxic PPO Pre, Post, or After the LL-TH protocol for both groups.

V_E, HR, and S_pO_2 at normoxic and hypoxic PPO are presented in Fig. 3. There were no significant differences in either normoxic or hypoxic V_E values at PPO over the training period or between groups. The maximal exercise HR in normoxia decreased significantly at the Post and After testing in the Hypoxic group, but not in the Control group. Although S_pO_2 was lower in hypoxia compared to normoxia, no significant differences between groups or Pre, Post, or After LL-TH were observed.

Mean (SD) endurance performance times are presented in **Fig. 4**. Time to exhaustion at a work rate of 80% normoxic PPO was significantly lower in the hypoxic compared to the normoxic condition in both groups during all testing periods (Fig. 4). Time to exhaustion improved in both Control (Post, $P < 0.01$) and Hypoxic (Post and After, $P < 0.05$) groups, but only under normoxic conditions. No significant differences were observed in the hypoxic condition between groups, or over the testing periods.

The CP tests in hypoxia were performed at the same absolute power output as in the normoxic condition (at 80% PPO Pre Normoxic) and, therefore, at a significantly higher relative workload. The relative power outputs of the hypoxic CP tests, compared to hypoxic Vo_{2peak} tests, remained constant at all testing periods and were similar in both groups for the Hypoxic (97 ± 4%, 96 ± 9%, 95 ± 12%, and 94 ± 9%) and for the Control (96 ± 7%, 97 ± 10%, 99 ± 14% and 99 ± 9%) groups at Pre, Mid, Post, and After testing, respectively.

DISCUSSION

Comparison of the results of the Control and Hypoxic groups revealed that LL-TH did not induce any signifi-

TABLE II. CHANGES IN ERYTHROCYTES (Rbc), HEMOGLOBIN (Hgb), HEMATOCRIT (Hct), TRANSFERRIN, AND FERRITIN AT ALL TESTINGS.

	Control Group				Hypoxic Group			
	Pre	Mid	Post	After	Pre	Mid	Post	After
Rbc ($10^{12} \cdot L^{-1}$)	5.0 ± 0.3	5.0 ± 0.3	4.8 ± 0.2	5.0 ± 0.2	5.0 ± 0.3	4.9 ± 0.3	5.1 ± 0.2	5.0 ± 0.4
Hgb ($g \cdot L^{-1}$)	150 ± 10	151 ± 13	141 ± 12	145 ± 12	142 ± 9	142 ± 10	142 ± 10	146 ± 10
Hct (%)	0.45 ± 0.0	0.46 ± 0.03	0.44 ± 0.03	0.45 ± 0.03	0.45 ± 0.03	0.44 ± 0.03	0.45 ± 0.03	0.45 ± 0.03
Transferrin ($g \cdot L^{-1}$)	2.6 ± 0.3	2.5 ± 0.3	2.6 ± 0.3	2.6 ± 0.4	2.9 ± 0.4	2.7 ± 0.3	2.7 ± 0.3	2.7 ± 0.3
Ferritin ($ng \cdot ml^{-1}$)	91.8 ± 63	85.8 ± 68	76.9 ± 44	68.5 ± 42.6	77.2 ± 26.6	76.8 ± 9.1	86.8 ± 29.4	71.8 ± 21.0

Values are mean ± SD. No significant differences were shown in both groups ($P < 0.05$).
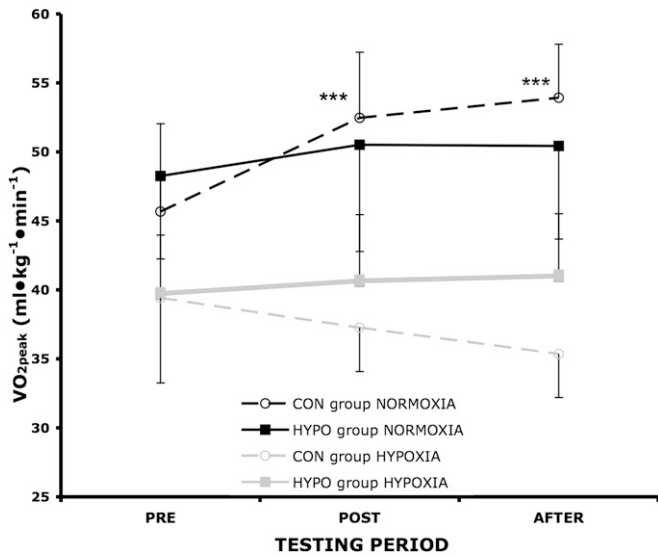Pre: before testing; Mid: during testing; Post: immediately after testing; and After: 10 d after testing.

Fig. 2. Peak oxygen uptake (Vo_{2peak}) (mean ± SD) before (Pre), upon completion (Post), and 10 d after the training (After) of both the Control (CON) and Hypoxic (HYPO) groups in the normoxic and hypoxic conditions. *** $P < 0.01$.

cant improvements in either normoxic or hypoxic Vo_{2peak} and endurance performance. In view of the lack of changes in the hematological and ventilatory responses after LL-TH, it is not surprising that normoxic and hypoxic performance were not enhanced. Similar to the other studies investigating the LL-TH regimen using untrained subjects (14), we did not find any changes in Hgb, Hct, RBC, or other hematological parameters. Our training program consisting of 20 1-h sessions over 4 wk in normobaric hypoxia at a simulated altitude of 4500 m did not alter the selected hematological variables, most probably due to the insufficient hypoxic dose. Rodriguez et al. (28) and Hendriksen and Meeuwsen (13), using 30- and 60-min longer exposures, respectively, to hypobaric hypoxia (4000–5000 m) showed that 9 to 10 exposures over 3 wk induced significant increases in Hct, Hgb, and RBC concentrations. We can, therefore, speculate that in the present study both the level of simulated altitude and the duration of the exposure to altitude were insufficient to induce beneficial hematological changes. Moreover, we can also hypothesize that hypobaric hypoxia may be more efficient compared to normobaric hypoxia, also in
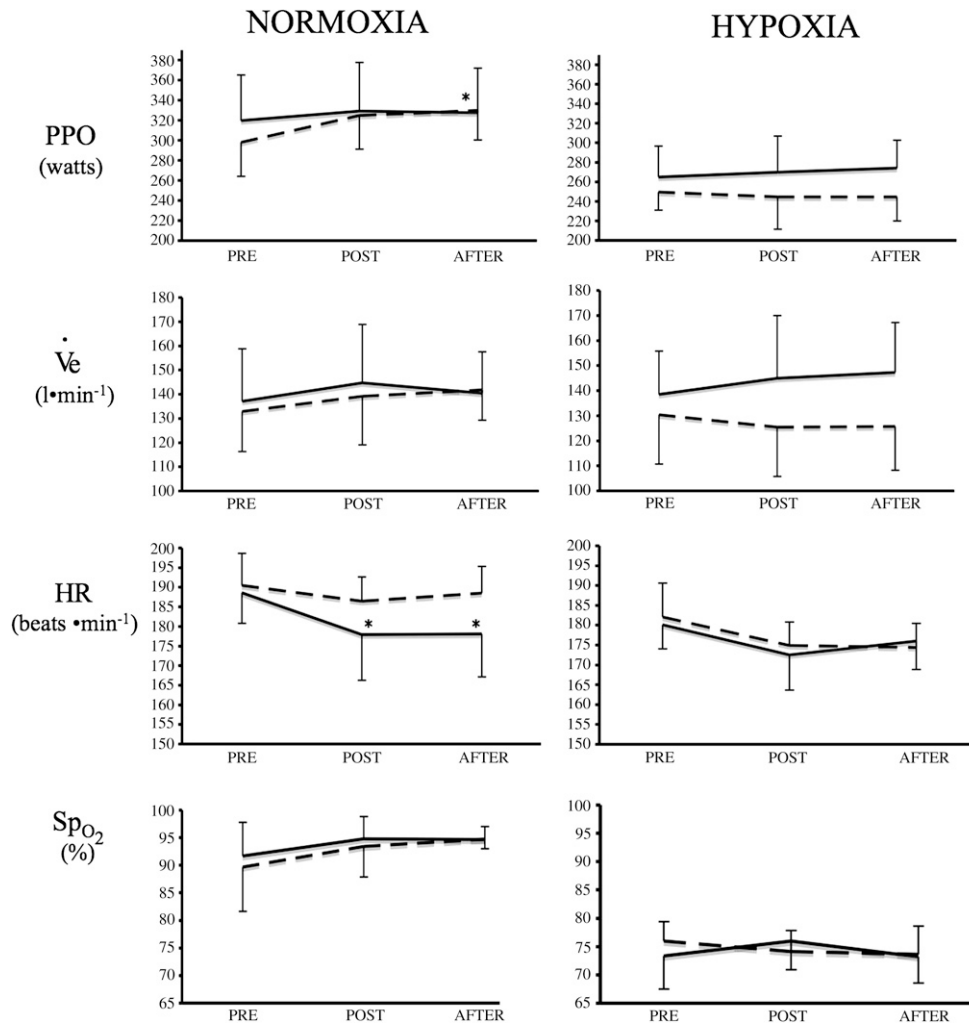

Fig. 3. Maximal values of power output (PPO), minute ventilation (V_E), heart rate (HR), and arterial oxygen saturation (S_pO2) (mean ± SD) during Vo_{2peak} tests in normoxic (left panel) and hypoxic (right panel) conditions Pre, Post, and After the LL-TH protocol. Solid line = the hypoxic group; dashed line = the Control group. * $P < 0.05$; significant differences from PRE testing.
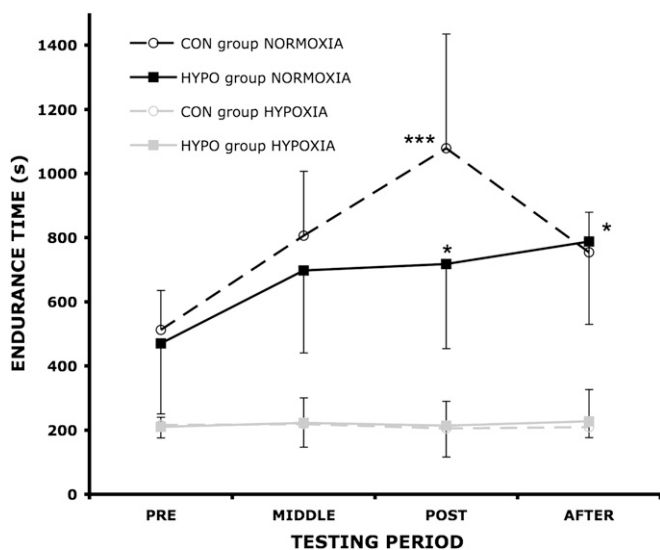
Fig. 4. Time to exhaustion in endurance performances (mean ± SD) Pre, Middle, Post, and After the LL-TH protocol of both the Control (CON) and Hypoxic (HYPO) groups in the normoxic and hypoxic conditions. * $P < 0.05$; *** $P < 0.01$.

terms of simulated erythropoiesis, as differences in physiological responses to both have already been shown (6).

The improvements reported by Geiser et al. (12) in sea-level performance following LL-TH in untrained subjects are not confirmed by the LL-TH protocol used in the present study. The differences in the performance results are most likely due to the higher training intensity (80% Vo_{2peak}) and longer protocol duration (6 wk) employed by Geiser et al. Both groups in the present study trained at the same relative work intensity, corresponding to 50% of normoxic PPO for the Control group and 50% of hypoxic PPO for the Hypoxic group. Thus, the Hypoxic group trained at an absolute work rate that was approximately 18–20 W lower. The improvements in normoxic and hypoxic performance observed in the Control group are due to training per se, whereas the fact that no difference was observed in the normoxic performance of the Hypoxic group probably reflects the lower level of absolute training intensity. Although some studies on untrained subjects (9,24) have reported no differences in performance following normoxic and hypoxic training conducted at the same absolute work rate, it has been demonstrated that the absolute workload (33) seems to be more important in terms of the acute muscular adaptation.

Studies investigating the effect of hypoxic training on exercise performance have, therefore, designed the training protocol to include regular exercises conducted at either the same absolute (24,31) or the same relative (1,2) work rate. Although the former might be more relevant in terms of maintaining or improving muscle strength, the latter allows the elucidation of the cardiorespiratory responses (strain) to exercise. The main changes reported in muscle tissue following LL-TH training in trained and untrained subjects include the up-regulation of hypoxia-inducible factor, increases in vascular endothelial growth factor (15), and increased

capillary density, resulting in greater muscle buffering capacity and lactate tolerance, improved muscle efficiency, and improvements in oxygen transfer (14). We can speculate that these changes were not induced in our Hypoxic group since we did not observe any significant changes in the performance tests.

Our results are in agreement with the findings of previous studies performed in hypobaric hypoxia on untrained athletes (8,18). These studies did not show any improvements in altitude aerobic capacity and reported similar changes in normoxic aerobic capacity in both experimental and hypoxic training groups, indicating that it was the training per se which caused the improvements in sea-level performance. However, Katayama et al. (17,19) reported that seven hypoxic exposures with or without training lasting 1.5 h at altitudes greater than 4000 m caused an increase in the S_pO_2 and maximal aerobic capacity. This improvement has been attributed solely to the increased exercise ventilatory response and not the hematological changes. Since we did not observe any significant changes in the exercise ventilatory response following LL-TH at 4500 m, this fact could also explain why no significant changes were found in the exercise tests of the hypoxic group compared to the control group (Fig. 3). Although the shorter duration of each training session in our study could be a viable explanation, the already mentioned study of Geiser et al. (12) showed that benefits can also be expected using only 30-min training sessions.

Interestingly, the only significant change in the LL-TH group during the normoxic Vo_{2peak} tests was a significant decrease of peak HR in the Post and After tests. Although such an effect is usually not expected, it has also been shown to appear in trained athletes following normoxic endurance training, most likely due to a decrease in the sympathetic drive (34). This phenomena has also been associated with overtraining (20). When incorporating a hypoxic training regimen in an athlete's training schedule, care must be taken that the sum of the basic (normoxic) and hypoxic training does not induce overtraining, as observed by Ventura et al. (32). Overtraining was most likely not a significant contributing factor to the results of the present study, since we did not observe any reductions in ferritin levels that may be indicative of the initial phase of overtraining-induced reduction in performance following LL-TH (10).

As noted by Chapman et al. (5), individual variation in the response to acute and chronic exposures to hypoxia also needs to be considered. Analyzing the individual responses of the subjects participating in the present study, we observed variability in subjects' responses to hypoxic training. In particular, Post hypoxic performance improved (6–35%) in only four subjects in the Hypoxic group, compared to their Pre hypoxic performance. However, the difference between the "responders" and "non-responders" in the Hypoxic group could not be attributed to the differences in any of the measured hematological variables.

We also have to address the limitations of our study. Since the aim of the study was to test a specific LL-TH

dose in untrained subjects, our results have to be interpreted in that manner. The absence of a blinded design and a low exercise training intensity are possible limitations of our study. However, it is extremely difficult to perform high-intensity training sessions and especially to blind subjects when exercising in severe hypoxia.

Despite some reports (1,30,31) of the benefits of the LL-TH training protocol to altitude performance, the results of the present study demonstrate no improvement in either altitude or sea-level performance. Most likely the hypoxic dose used was below the critical dose required to result in hypoxia-induced improvements in performance. Since the exercise training was conducted at a simulated altitude of 4500 m, it is unlikely that an insufficient altitude was the cause of the difference. Most probably it was the duration and the frequency of the exposures that would need to be modified to achieve the reported benefits.

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