

Article

Effects of High-Intensity Interval Training in Normobaric Hypoxia on Anaerobic Performance in Young Untrained Men Across Different Hypoxic Exposure Models

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Featured Application

This study may support coaches, sport scientists and exercise physiologists in designing short-term high-intensity interval training interventions by evaluating whether normobaric hypoxia benefits anaerobic performance.

Abstract

Background: Normobaric hypoxia is increasingly used as an additional environmental stimulus in training programs; however, its practical role in improving anaerobic performance remains unclear. This study analyzed how a four-week high-intensity interval training (HIIT) program performed under different hypoxic exposure models affected selected anaerobic performance indices in young men. **Methods:** Forty-five healthy, non-elite physically active men completed the study. Participants were assigned to one of four groups: control, normoxic training, live low–train high group, and live high–train low group. The training intervention consisted of 12 HIIT sessions performed over four weeks. Anaerobic performance was assessed before and after the intervention using a 20-s maximal cycling sprint test. **Results:** No significant group $\times$ time interactions were found for any variables obtained from the 20-s maximal cycling sprint test, including peak power (PP; $p = 0.109$, $\eta^2 = 0.136$), relative peak power (rel_PP; $p = 0.641$, $\eta^2 = 0.040$), relative mean power (rel_MP; $p = 0.438$, $\eta^2 = 0.063$), relative total work (rel_TW; $p = 0.515$, $\eta^2 = 0.054$), fatigue index (FI; $p = 0.231$, $\eta^2 = 0.099$), and time to peak power (tPP; $p = 0.673$, $\eta^2 = 0.036$). **Conclusions:** The findings suggest that, under the applied conditions, normobaric hypoxia does not provide a sufficient additional stimulus to enhance anaerobic performance adaptations. From an applied exercise physiology perspective, the specificity and structure of the training stimulus are critical when designing short-term HIIT interventions. They may be more important than hypoxic exposure itself for improving short-duration maximal cycling sprint performance.

Keywords: normobaric hypoxia; high-intensity interval training; anaerobic performance



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1. Introduction

Athletes and coaches—in both individual and team sports—continually seek effective strategies to enhance athletic performance. One of the most widely used environmental

approaches is training under hypoxic conditions. Interest in hypoxic training increased markedly after the 1968 Olympic Games in Mexico City, which were held at an altitude above 2000 m [1]. Since then, this approach has become an established component of training programs for many athletes [2,3]. Normobaric hypoxia technology enables precise control over hypoxic dose and exposure time in laboratories. Under hypobaric conditions, the oxygen fraction in the air remains relatively constant. However, reduced barometric pressure decreases the partial pressure of inspired oxygen, which may limit oxygen availability for the blood and tissues [4]. In normobaric hypoxia, a similar effect is achieved by reducing the fraction of inspired oxygen (FiO_2) while maintaining barometric pressure corresponding to normobaric conditions. This is most commonly achieved using nitrogen dilution or oxygen filtration technology [5]. Two main models have been developed: LHTL (live high–train low), which involves passive hypoxic exposure combined with training in normoxia, and LLTH (live low–train high). The latter comprises exercise performed in hypoxia while residing at a low altitude [6]. Currently, particular attention is being paid to the LLTH model combined with high-intensity interval training (HIIT). This approach enables the application of a strong physiological stimulus under controlled conditions of reduced oxygen availability [7].

The effects of hypoxia on exercise performance may result not only from hematological adaptations [1], but also from metabolic and neuromuscular changes that are particularly relevant to high-intensity exercise [5]. Reduced oxygen availability during exercise may attenuate the contribution of oxidative phosphorylation. As a compensatory response, reliance on anaerobic ATP resynthesis increases [8]. Consequently, exercise performed under hypoxic conditions may promote the accumulation of blood lactate and H^+ ions. It can also increase metabolic stress and accelerate the development of peripheral fatigue [8,9]. This mechanism may require the additional recruitment of motor units to maintain mechanical power output [9]. Moreover, earlier fatigue of oxidative type I fibers may favor greater involvement of type II fibers, which play a key role in power generation during anaerobic exercise [10].

Hypoxia may also induce peripheral and molecular adaptations in skeletal muscle. Previous studies have shown that exposure to hypoxia can stimulate mitochondrial biogenesis [11], alter the activity of oxidative and glycolytic enzymes [12,13], increase the expression of monocarboxylate transporters involved in lactate and H^+ transport [14], and support angiogenesis [15]. However, it is still unclear whether these changes lead to measurable improvements in anaerobic performance. In the context of anaerobic exercise, adaptations related to glycolytic metabolism and the regulation of the intramuscular environment may be particularly important. These include buffering capacity and metabolite transport, which may serve to maintain high-intensity exercise under conditions of increasing metabolic stress [16,17]. Nevertheless, the best-documented effects of hypoxia are mainly related to the aerobic component of performance. Such modifications encompass hematological adaptations, improved oxygen transport [1], and enhanced oxygen utilization by tissues [5], factors that may be more significant for aerobic performance than short-duration anaerobic efforts [1,5–7].

High-intensity exercise, in which ATP demand exceeds the capacity for oxidative ATP resynthesis, is characteristic of sports that require short sprints and repeated maximal-effort bouts [18]. In intermittent sports, fluctuations in energy demand translate to performance being dependent on the effective use of both aerobic and anaerobic metabolism [19]. During very short maximal and supramaximal efforts lasting approximately 6–10 s, the phosphagen system (ATP–PCr) plays a dominant role, whereas the contribution of anaerobic glycolysis increases as exercise duration is extended to approximately 30–90 s [19]. In this context, anaerobic performance reflects the maximal capacity to resynthesize ATP through anaerobic

pathways during a single maximal effort [20]. Therefore, indices obtained from short maximal anaerobic tests depend primarily on the ability to generate power through the ATP–PCr system and glycolysis, alongside neuromuscular factors [19].

Power is determined by both force and velocity; therefore, changes in power may result from metabolic adaptations as well as from the ability to rapidly generate force and recruit fast-twitch muscle fibers [5]. Given the findings, hypoxic conditions—particularly when applied during high-intensity exercise—may theoretically provide an additional stimulus for adaptations related to selected indices of anaerobic performance. It should be emphasized, however, that in exercise with a dominant contribution of anaerobic metabolism, the role of oxygen is not limited solely to its direct involvement in ATP resynthesis during the effort itself. Oxygen availability may be particularly important during recovery between successive high-intensity efforts. This is especially true for phosphocreatine resynthesis, restoration of intramuscular homeostasis, and the recovery of the ability to generate high power output [21]. In this context, the potential relevance of hypoxia to anaerobic performance may be related not only to the physiological response during a single maximal effort, but also to adaptations associated with repeated-effort tolerance and recovery between efforts [22]. Nonetheless, the available evidence remains inconclusive, and the observed effects appear to depend on the protocol type, recovery duration, hypoxic dose, and the specific outcomes assessed [23].

Previous studies on the effects of hypoxia on anaerobic performance indicators are limited and inconclusive [24]. Some studies have reported improvements in selected anaerobic indicators after hypoxic training, particularly when sprint-based protocols or maximal-effort exercises were used [7,25–27]. In contrast, no additional benefits of hypoxia have been shown in other studies when compared with the same training performed under normoxic conditions [27–29]. Moreover, the majority of previous studies have focused on active hypoxic training models, whereas less is known about anaerobic performance responses after passive hypoxic exposure combined with normoxic training [24,30–32]. Therefore, direct comparisons of different normobaric hypoxic exposure models using the same HIIT protocol and anaerobic performance indicators remain limited.

The novelty of the present study lies in the assessment of anaerobic performance responses to the same four-week HIIT protocol performed under normoxic conditions and under two different models of normobaric hypoxic exposure: LLTH and LHTL. This approach allowed us to assess whether the mode of applying the hypoxic stimulus—actively during training or passively as nocturnal exposure—was associated with different responses in the analyzed anaerobic performance indicators.

Considering these gaps, the aim of the present study was to evaluate the impact of a four-week HIIT program performed under different models of normobaric hypoxic exposure (LLTH and LHTL). Specifically, we investigated the subsequent effects of these protocols on selected anaerobic performance indicators obtained from a 20-s maximal cycling sprint test.

The following hypotheses were formulated:

Hypothesis 1. *Normobaric hypoxia, compared to the normoxic condition, would result in greater changes in the analyzed anaerobic performance indicators following the same interval training protocol.*

Hypothesis 2. *The LLTH model, in which exercise was carried out under hypoxic conditions, is associated with greater changes in the analyzed anaerobic performance indicators than the LHTL model, with the latter protocol combining passive nocturnal hypoxic exposure and normoxic training.*

2. Materials and Methods

2.1. Participant Characteristics

A total of 48 young, healthy men were recruited for the study (age: 22.4 ± 2.0 years; height: 179.6 ± 6.3 cm; body mass: 76.8 ± 11.5 kg; body fat: $18.9 \pm 5.9\%$; BMI: 23.8 ± 2.8 kg/m²). The participants did not engage in systematic sports training; however, they were also not completely sedentary. The majority of subjects comprised physical education students who regularly participated in practical classes. They demonstrated a moderate level of physical activity assessed using the IPAQ questionnaire (MET-min/week: 2752.61 ± 1698.75). Additionally, as part of the pre-intervention assessment, participants performed an incremental exercise test on a cycle ergometer, during which VO_2max was determined to characterize baseline fitness level. The absolute VO_2max for the whole sample was 3.36 ± 0.58 L/min. The inclusion and exclusion criteria are presented in Table 1.

Table 1. Inclusion and exclusion criteria.

Inclusion Criteria	Exclusion Criteria
→ Male sex	→ Smoking
→ Age 18–29 years	→ Alcohol or psychoactive substance abuse
→ No regular structured physical training or participation in competitive sport	→ Regular physical training in the months preceding the study or during its duration
→ Medical certificate confirming no contraindications to performing physical exercise	→ Changes in dietary habits during the study
→ No exposure to high-altitude hypoxia within 3 months prior to the study	→ Use of pharmacological agents or supplements that could affect physical performance
→ Written informed consent to participate in the study	→ History of altitude sickness
	→ Cardiovascular, respiratory or metabolic diseases

During the study, three participants withdrew from the intervention groups due to illness or poor general well-being. Ultimately, 45 participants were included in the analysis. The detailed recruitment process is shown in Figure 1.

2.2. Study Protocol

For logistical reasons, the study was not randomized. Participants were allocated sequentially to one of four groups:

1. LLTH (live low–train high): training under normobaric hypoxic conditions, corresponding to an altitude of 3000 m ($\text{FiO}_2 = 14.4\%$), while living in normoxic conditions (~ 200 m, $\text{FiO}_2 = 20.95\%$).
2. LHTL (live high–train low): training under normoxic conditions (~ 200 m, $\text{FiO}_2 = 20.95\%$) combined with passive exposure to normobaric hypoxia, corresponding to an altitude of 3000 m ($\text{FiO}_2 = 14.4\%$). The exposure included a total of 12 overnight sessions, each lasting 12 h.
3. LLTL (live low–train low): training and living under normoxic conditions (~ 200 m, $\text{FiO}_2 = 20.95\%$).
4. C (control group): no training intervention.

Due to the nature of the intervention, blinding was not applied.

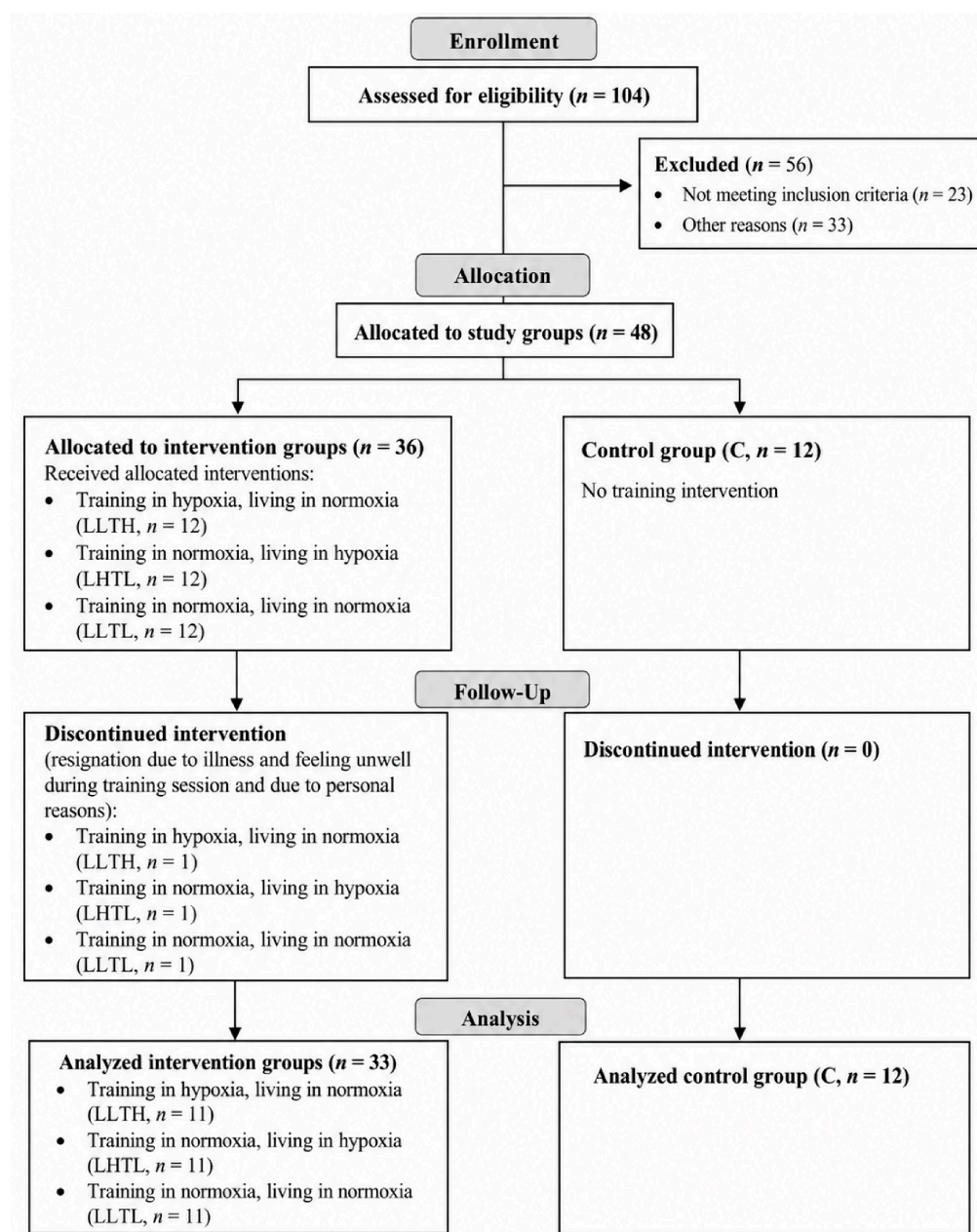


Figure 1. Participant flow diagram. LLTH—live low–train high; LHTL—live high–train low; LLTL—live low–train low; C—control group.

Before the training intervention, all participants underwent somatic measurements, an incremental exercise test on a cycle ergometer, and the 20-s maximal cycling sprint test. Training loads for each group were determined based on the incremental exercise test performed on a cycle ergometer in conditions corresponding to the planned training environment—either hypoxia or normoxia. After a seven-day period following the exercise tests, a four-week training intervention was conducted. Approximately 7–10 days after completion of the intervention, somatic measurements and performance tests were repeated (Figure 2).

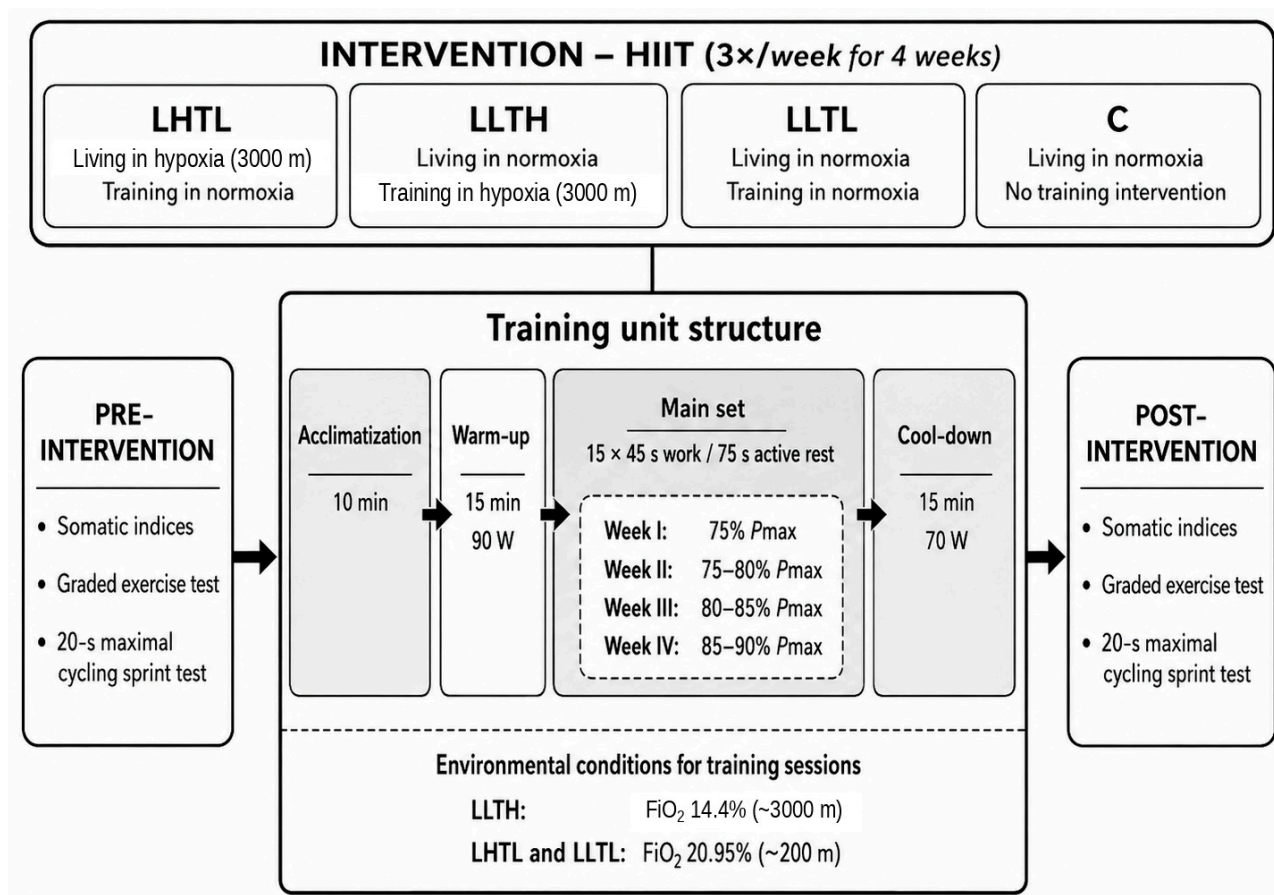


Figure 2. Study protocol and structure of HIIT intervention. HIIT—high-intensity interval training; LHTL—live high–train low; LLTH—live low–train high; LLTL—live low–train low; C—control group; FiO₂—fraction of inspired oxygen; P_{max}—peak power.

Before the experiment, all participants were informed about the aim of the study, the applied procedures, possible side effects, the voluntary nature of participation, and the possibility of withdrawing from the study at any stage without providing justification. The study was carried out in accordance with the 1964 Declaration of Helsinki (with its later amendments). Written informed consent was required for participation in the project. The study protocol was approved by the Bioethics Committee of the Regional Medical Chamber in Kraków (No. 125/KBL/OIL/2023) and was registered at ClinicalTrials.gov (identifier: NCT06204731). The study was conducted under the supervision of qualified medical personnel and took place at the Central Scientific Research Laboratory of the University of Physical Education in Kraków (PN-EN ISO 9001:2015). Before the start of the study, each participant underwent medical screening, which confirmed the absence of contraindications to physical activity or exposure to normobaric hypoxia. The participants were instructed not to change their dietary habits or physical activity levels during the study period, and to refrain from consuming supplements or pharmacological agents that could affect physical performance. Dietary intake was monitored using dietary records completed before and after the intervention.

2.3. Somatic Measurements

Somatic measurements were taken in the morning before the exercise tests. The measurements included body height (BH), body mass (BM), and body composition variables: fat-free mass (FFM), fat mass (FM), percentage body fat (%F), and total body water (TBW). Body height (BH) was measured using the Martin anthropometer (Seritex, New York, NY,

USA), to the nearest 0.1 cm, while body mass (BM) was determined using the Sartorius F 1505-DZA electronic scale (Göttingen, Germany), to the nearest 1 g. Body height and body mass were used to calculate the Quetelet II index, expressed as body mass index (BMI). Body composition was assessed using bioelectrical impedance analysis with the AKERN BIA 101 analyzer (Akern S.r.l., Pontassieve, Italy).

2.4. Evaluation of Anaerobic Capacity and Determination of Training Workloads

20-s Maximal Cycling Sprint Test

In order to establish maximal anaerobic power, participants performed a 20-s maximal cycling sprint test. The test was performed on a cycle ergometer (Monark 875E, Vansbro, Sweden) with a load corresponding to 8.3% of the participant's body mass. The use of a 20-s protocol was intended to reduce the relative contribution of the aerobic component, which is present in the classical 30-s Wingate test, especially in the final phase of the effort [33]. A similar 20-s maximal cycle ergometer test has previously been used to assess anaerobic power in physically active men [34]. The main test was preceded by a 5-min intensive warm-up with a 1.0 kg load and pedaling cadence of 60 rpm, including two 5-s accelerations in the 2nd and 4th minutes. After 2 min of rest, participants started from a stationary position and were instructed to reach maximal pedaling cadence as quickly as possible and then also maintain it for as long as possible.

The following variables were determined during the test: relative total work (rel_TW), peak power (PP) expressed in absolute and relative values (rel_PP), relative mean power (rel_MP), time to peak power (tPP), and fatigue index (FI). All relative values were calculated by normalizing the corresponding absolute values to body mass.

Graded exercise test

To determine individual training workloads under hypoxic and normoxic conditions, participants performed an incremental exercise test on a cycle ergometer (ER900 D-72475 BIT2, Jaeger, Friedberg, Germany). While the LHTL and LLTL cohorts completed the test in normoxia, the LLTH group underwent testing inside a thermoclimatic chamber simulating normobaric hypoxia at an altitude of 3000 m. A ramp protocol was applied. The test began with a 2-min warm-up on the cycle ergometer at a pedaling cadence of 60 rpm and intensity of 110 W. Thereafter, the workload was increased by 30 W every 2 min. The test was continued until volitional exhaustion.

Respiratory variables—including oxygen uptake (VO_2), carbon dioxide production (VCO_2), minute ventilation (VE), fractional concentrations of CO_2 (%FECO₂) and O_2 (%FEO₂) in expired air, and ventilatory equivalents for oxygen (VE/VO_2) and carbon dioxide (VE/VCO_2)—were measured breath-by-breath using a metabolic gas analyzer (MetaLyzer 3R, Cortex, Leipzig, Germany).

2.5. Training Program

Training intervention

Participants in the LHTL, LLTH, and LLTL groups trained three times per week for four weeks. Training sessions were performed on cycle ergometers (Wattbike, Nottingham, UK). Each training session consisted of a 15-min warm-up, followed by 30 min of interval training and 15 min of recovery. The warm-up and final recovery period included 10 min of continuous cycling at 70–90 W and 5 min of stretching.

The interval training comprised 15 repetitions of 45 s exercises, separated by 75 s of active recovery. Exercise intensity was individually prescribed based on the $P_{\max}$ level obtained during the pre-intervention graded exercise test. Training workload was progressively increased across the intervention: week I: 75% $P_{\max}$; week II: 75–80% $P_{\max}$; week III: 80–85% $P_{\max}$ and week IV: 85–90% $P_{\max}$. Active recovery between repetitions was

performed at a workload of 90 W. Training workloads were individualized for each participant based on Pmax determined under the same environmental conditions as the planned intervention—specifically—normoxia for the LHTL and LLTL groups and normobaric hypoxia for the LLTH group. The structure of the training session is presented in Figure 2.

Passive exposure to normobaric hypoxia

On training days, the participants in the LHTL group additionally underwent overnight stays in a thermoclimatic hypoxic chamber three times per week for four weeks. This resulted in a total of 12 overnight sessions, each lasting 12 h, in normobaric hypoxic conditions simulating an altitude of 3000 m.

Environmental conditions

Training sessions and overnight hypoxic exposures were carried out in a thermoclimatic hypoxic chamber (Hypoxico, Bickenbach, Germany) operating on nitrogen-free technology. In this system, the hypoxia generator produces hypoxic air with the following composition: O₂, 14.4%; CO₂, 0.03%; and N₂, 78%. This air is then supplied into the chamber, where its composition is continuously monitored to maintain stable conditions.

All training protocols were performed under the same environmental conditions, with relative humidity sustained at $40 \pm 1\%$ and ambient temperature at $21 \pm 0.5\text{ }^{\circ}\text{C}$. Air movement was below $0.5\text{ m}\cdot\text{s}^{-1}$.

2.6. Statistical Analysis

Before initiating the study, the required sample size was calculated using G*Power software (version 3.1.9.7, Germany). The calculation was based on repeated-measures analysis of variance (RM-ANOVA) for the group $\times$ time interaction, assuming a medium effect size ($f = 0.25$), a significance level of $\alpha = 0.05$, statistical power of $1 - \beta = 0.80$, four study groups, two measurements, and a correlation between repeated measurements of 0.50. The minimum required sample size was 48 participants, corresponding to 12 participants in each group.

Numerical data are presented as means and standard deviations. Prior to analysis, the assumptions for parametric tests were verified. The homogeneity of variance was assessed using Levene's test, and the normality of distributions was evaluated using the Shapiro–Wilk test. The assumption of sphericity was not applicable due to only two repeated measures being included (PRE and POST). Differences between groups, changes over time (PRE vs. POST), and differences in response to the intervention were analyzed using RM-ANOVA. The main effects of group and time, as well as the time $\times$ group interaction, were examined. F-statistics were reported with degrees of freedom of (3, 41) for the group effect and the time $\times$ group interaction, and (1, 41) for the time effect. Effect size was reported as partial eta-squared (η^2) and interpreted as small (0.01), moderate (0.06) or large (0.14) [29]. When significant ANOVA effects were found, post hoc analyses were performed using Tukey's test. Within-group PRE vs. POST comparisons were conducted using paired-sample *t*-tests, and *p*-values were adjusted using the Holm method. Hedges' *g* was reported as the effect size for PRE–POST changes. In accordance with the adopted classification, effect sizes were interpreted as small (0.2), moderate (0.5) or large (0.8) [35]. A priori comparisons of differences in changes ($\Delta = \text{POST} - \text{PRE}$) between the LLTH and LHTL models, as well as between the LLTL and control groups, were also planned. The Holm method was additionally applied to adjust *p*-values for these planned comparisons. Standardized effect sizes (Hedges' *g*) were reported for the planned comparisons. Effect sizes were described to support interpretation regarding the size of observed effects beyond statistical significance testing. The level of statistical significance was set at $p < 0.05$.

3. Results

3.1. Somatic Parameters

Body mass and body composition parameters were analyzed as supplementary variables to support the interpretation of relative indices obtained from the 20-s maximal cycling sprint test (W/kg and J/kg). No significant changes in BM, BMI, FM, or FFM were observed in any of the groups following the intervention (all $p > 0.05$; Table 2). Although a significant time $\times$ group interaction was found, post hoc analyses did not show significant PRE–POST changes within any group. Descriptively, the LLTH group demonstrated only a decreasing trend in %F ($p = 0.052$; ES = -0.84) and an increase in FFM ($p = 0.078$; ES = 0.77). Nonetheless, these changes did not reach the level of statistical significance. An opposite trend was observed in the control group (Table 2).

Table 2. Changes in body composition parameters after intervention and effects of group, time, and time $\times$ group interaction.

Variable	Comparison	PRE	POST	Effect Group F/ p/ ηp^2	Effect Time F/ p/ ηp^2	Interaction F/ p/ ηp^2	Post-hoc p	ES
BM [kg]	C	75.9 $\pm$ 9.7	74.5 $\pm$ 9.2	259.61	0.22	4.25	0.277	−0.52
	LLTL	73.6 $\pm$ 11.4	74.3 $\pm$ 10.7	<0.001	0.640	0.010	0.298	0.42
	LHTL	76.0 $\pm$ 6.1	76.6 $\pm$ 6.4	0.948	0.005	0.229	0.277	0.54
	LLTH	81.9 $\pm$ 16.4	81.5 $\pm$ 15.9				0.298	−0.36
BMI [kg/m ²]	C	23.9 $\pm$ 2.7	23.5 $\pm$ 2.5	144.08	0.19	4.21	0.264	−0.51
	LLTL	23.2 $\pm$ 2.6	23.4 $\pm$ 2.4	<0.001	0.667	0.011	0.279	0.43
	LHTL	23.2 $\pm$ 1.5	23.4 $\pm$ 1.7	0.910	0.004	0.227	0.264	0.55
	LLTH	24.8 $\pm$ 4.1	24.7 $\pm$ 3.9				0.279	−0.35
FM [%]	C	16.0 $\pm$ 5.2	18.2 $\pm$ 4.1	22.61	0.13	4.90	0.354	0.46
	LLTL	17.8 $\pm$ 4.7	17.8 $\pm$ 3.3	<0.001	0.720	0.005	0.976	0.01
	LHTL	18.8 $\pm$ 3.3	18.2 $\pm$ 3.3	0.612	0.003	0.255	0.547	−0.31
	LLTH	23.3 $\pm$ 7.6	20.8 $\pm$ 8.5				0.052	−0.84
FFM [kg]	C	63.6 $\pm$ 8.0	61.2 $\pm$ 6.9	15.00	0.27	6.00	0.179	−0.56
	LLTL	60.1 $\pm$ 6.6	60.8 $\pm$ 7.0	<0.001	0.605	0.002	0.196	0.37
	LHTL	61.6 $\pm$ 4.5	62.5 $\pm$ 3.5	0.511	0.006	0.295	0.179	0.56
	LLTH	61.8 $\pm$ 6.6	63.5 $\pm$ 7.2				0.078	0.77

Data are presented as mean $\pm$ SD. PRE—pre-intervention measurement; POST—post-intervention measurement; C—control group; LLTL—live low—train low; LHTL—live high—train low; LLTH—live low—train high; BM—body mass; BMI—body mass index; FM—fat mass; FFM—fat-free mass; F—F-statistic; p—p-value; ηp^2 —partial eta-squared; ES—effect size. Bold values indicate statistical significance at $p < 0.05$.

3.2. Anaerobic Performance

Analysis of the 20-s maximal cycling sprint test parameters showed no significant improvement in any of the assessed indices in any group or any significant between-group differences in response to the intervention (Table 3).

Table 3. The 20-s maximal cycling sprint test indices of anaerobic performance before and after four-week HIIT intervention according to group, including effects of group, time, and time $\times$ group interaction.

Variable	Comparison	Pre	Post	Effect: Group F <i>p</i> ηp^2	Effect: Time F <i>p</i> ηp^2	Interaction F <i>p</i> ηp^2
tPP [s]	C	5.24 $\pm$ 0.94	5.12 $\pm$ 0.91			
	LLTL	6.03 $\pm$ 1.35	5.78 $\pm$ 1.36	2.58	1.94	0.52
	LHTL	5.12 $\pm$ 1.05	4.78 $\pm$ 0.88	0.066	0.171	0.673
	LLTH	4.98 $\pm$ 0.62	5.03 $\pm$ 0.50	0.159	0.045	0.036
FI [%]	C	17.36 $\pm$ 5.04	18.71 $\pm$ 5.44			
	LLTL	13.59 $\pm$ 4.42	13.92 $\pm$ 3.36	3.26	0.26	1.49
	LHTL	17.26 $\pm$ 3.78	17.79 $\pm$ 4.36	0.031	0.614	0.231
	LLTH	20.05 $\pm$ 6.05	18.69 $\pm$ 4.50	0.192	0.006	0.099
rel_PP [W/kg]	C	12.12 $\pm$ 0.85	12.14 $\pm$ 0.76			
	LLTL	11.72 $\pm$ 0.55	11.93 $\pm$ 0.72	0.59	0.75	0.57
	LHTL	12.25 $\pm$ 0.65	12.31 $\pm$ 0.63	0.625	0.392	0.641
	LLTH	12.19 $\pm$ 1.46	12.14 $\pm$ 1.04	0.041	0.018	0.040
PP [W]	C	923.17 $\pm$ 161.67	906.86 $\pm$ 140.44			
	LLTL	864.12 $\pm$ 119.16	888.28 $\pm$ 112.27	1.29	0.26	2.15
	LHTL	934.74 $\pm$ 100.31	948.88 $\pm$ 109.97	0.289	0.613	0.109
	LLTH	985.65 $\pm$ 155.86	978.49 $\pm$ 137.84	0.087	0.006	0.136
rel_MP [W/kg]	C	9.98 $\pm$ 0.73	9.91 $\pm$ 0.70			
	LLTL	9.94 $\pm$ 0.46	10.12 $\pm$ 0.47	0.33	0.75	0.92
	LHTL	10.15 $\pm$ 0.66	10.13 $\pm$ 0.52	0.800	0.393	0.438
	LLTH	9.79 $\pm$ 1.22	9.92 $\pm$ 0.80	0.024	0.018	0.063
rel_TW [J/kg]	C	198.88 $\pm$ 14.05	198.24 $\pm$ 13.94			
	LLTL	198.78 $\pm$ 9.26	202.50 $\pm$ 9.45	0.35	1.05	0.77
	LHTL	203.06 $\pm$ 13.10	202.65 $\pm$ 10.47	0.789	0.312	0.515
	LLTH	195.90 $\pm$ 24.37	198.44 $\pm$ 16.04	0.025	0.025	0.054

Data are presented as mean $\pm$ SD. PRE—pre-intervention measurement; POST—post intervention measurement; C—control group; LLTL—live low—train low; LHTL—live high—train low; LLTH—live low—train high; tPP—time to peak power; FI—fatigue index; rel_PP—relative peak power; PP—peak power; rel_MP—relative mean power; rel_TW—relative total work; F—F-statistic; *p*—*p*-value; ηp^2 —partial eta-squared.

In the planned comparisons, no significant differences were found between the LLTH and LHTL models for the analyzed variables (Table 4). In the LLTL vs. Control comparison, a significant difference was only observed for the change in PP [W] (*p* = 0.040; ES = 1.037; Table 4).

Table 4. Planned a priori comparisons of changes (Δ = POST – PRE) in variables obtained from 20-s maximal cycling sprint test between intervention models: LLTH vs. LHTL, and LLTL vs. Control.

Variable	Comparison	Δ Group 1 (Mean $\pm$ SD)	Δ Grupa 2 (Mean $\pm$ SD)	<i>p</i>	ES
tPP [s]	LLTH vs. LHTL	0.05 $\pm$ 0.41	−0.34 $\pm$ 0.71	0.268	0.648
	LLTL vs. Control	−0.25 $\pm$ 1.18	−0.12 $\pm$ 0.65	0.746	−0.136
FI [%]	LLTH vs. LHTL	−1.4 $\pm$ 3.4	0.5 $\pm$ 3.9	0.472	−0.502
	LLTL vs. Control	0.3 $\pm$ 2.8	1.4 $\pm$ 2.4	0.472	−0.385
rel_PP [W/kg]	LLTH vs. LHTL	−0.05 $\pm$ 0.66	0.06 $\pm$ 0.38	0.629	−0.202
	LLTL vs. Control	0.21 $\pm$ 0.47	0.03 $\pm$ 0.37	0.620	0.425
PP [W]	LLTH vs. LHTL	−7 $\pm$ 59	14 $\pm$ 34	0.313	−0.428
	LLTL vs. Control	24 $\pm$ 42	−16 $\pm$ 33	0.040	1.037
rel_MP [W/kg]	LLTH vs. LHTL	0.13 $\pm$ 0.59	−0.02 $\pm$ 0.40	0.499	0.283
	LLTL vs. Control	0.19 $\pm$ 0.36	−0.07 $\pm$ 0.27	0.149	0.770
TW [J/kg]	LLTH vs. LHTL	2.5 $\pm$ 11.8	−0.4 $\pm$ 8.0	0.503	0.281
	LLTL vs. Control	3.7 $\pm$ 7.2	−0.6 $\pm$ 4.7	0.212	0.700

Data are presented as mean $\pm$ SD. Δ —change between POST and PRE values; C—control group; LLTL—live low–train low; LHTL—live high–train low; LLTH—live low–train high; tPP—time to peak power; FI—fatigue index; rel_PP—relative peak power; PP—peak power; rel_MP—relative mean power; rel_TW—relative total work; *p*—*p*-value adjusted using Holm method; ES—effect size. Bold values indicate statistical significance at *p* < 0.05.

4. Discussion

The aim of the present study was to examine the effect of an analogous high-intensity interval training program performed using different models of hypoxic training (LLTH and LHTL) on anaerobic performance. Depending on group allocation, participants trained under normoxic or normobaric hypoxic conditions, while the LHTL group additionally underwent passive exposure to normobaric hypoxia. The results indicate that, regardless of the training model, four weeks of HIIT under the studied conditions did not lead to significant changes in sprint-derived anaerobic performance variables. The lack of significant differences between the LLTL group—performing HIIT and living in normoxic conditions—and the control group suggests that the applied HIIT protocol alone did not induce measurable improvements in anaerobic performance among the studied population. Moreover, the additional physiological stimulus provided by normobaric hypoxia—either as hypoxic training or passive hypoxic exposure—was not associated with further improvements in these variables.

The main finding of the present study was the lack of significant changes in sprint-derived anaerobic performance variables, regardless of the training conditions applied. One possible explanation for this result is the nature of the training protocol. In the HIIT protocol, participants performed submaximal exercise, characterized by high intensity but without all-out repetitions. It is plausible that the high-intensity training (maximal or supramaximal), such as sprint interval training (SIT) utilizing 20–30 s Wingate bouts, may more effectively enhance anaerobic capacity over a brief intervention period due to its close alignment with the specific demands of the test. Such findings were reported by Vera-Ibañez et al., who confirmed an increase in peak power after conducting a four-week HIIT protocol based on Wingate sprints [36]. Other studies have also reported improvements in anaerobic performance after four weeks of training using supramaximal-intensity intervals [37,38]. It has been suggested that increasing sprint duration increases the contribution of aerobic energy metabolism [39]. Therefore, classic HIIT performed as exercise

lasting longer than 30 s at near-maximal intensity—approximately 90–95% HR_{max}—may stimulate aerobic adaptations to a greater extent while having a limited effect on anaerobic adaptations assessed using the 20–30 s maximal test. This may partially explain why anaerobic performance variables remained unchanged, regardless of the applied training conditions. This consistency with the protocol's characteristics may be attributed to the limited specificity of the training stimulus relative to the 20-s maximal effort.

The significance of training stimulus specificity is also supported by comparisons regarding the total duration of active work reported in previous studies. In the present study, a single training session included 15 repetitions of 45 s, resulting in a total of 11 min 15 s of active work per session. Across the four-week intervention period, which included 12 training sessions, the total duration of active work was 135 min. Milioni et al. [40] similarly reported no improvements in anaerobic performance indices following a four-week protocol based on 10 repetitions of 1 min of work, corresponding to 10 min of active exercise per session and 110 min across 11 training sessions. In contrast, studies based on short maximal or near-maximal efforts reported improvements in anaerobic parameters despite a substantially shorter total duration of active work [41–44]. Zhang et al. [41] applied two HIIT variants in which the duration of active work in a single session was 6 min. These included a 2:1 protocol with 20 s of work and 10 s of recovery, and a 3:1 protocol with 30 s of work and 10 s of recovery. This protocol accumulated 72 min of active work distributed across 12 sessions over a six-week period. The authors reported improvements in PP and PP/kg. Gutknecht et al. [45] used six sessions of repeated-sprint training, comprising three sets of eight 6-s maximal sprints. This corresponded to only 2 min 24 s of active work per session and a total of 14 min 24 s of active work across the whole intervention. Despite this low training volume, the authors observed significant improvements in PP and MP in the Wingate test. Other studies based on repeated maximal or near-maximal efforts also reported favorable changes in the anaerobic component, despite a relatively short total duration of active work across the intervention [42–44]. Therefore, in the context of improving Wingate test parameters, the specificity of the training stimulus seems to be more important than the total duration of active work alone. This specificity includes short sprints, high power output, repeated maximal efforts and short recovery periods. Consistent with this perspective, the lack of performance changes across all cohorts in the present study appears reasonable. The limited specificity of the training protocol relative to the demands of the Wingate test represents one plausible explanation for this lack of change.

The absence of improvement in anaerobic performance variables in both the normoxic training group and the groups exposed to hypoxia suggests that, under the applied conditions, the addition of normobaric hypoxia did not provide a measurable benefit for anaerobic performance. This finding should be interpreted within the context of inconsistent evidence regarding the effects of different hypoxic models on anaerobic performance. To the best of our knowledge, the current investigation is among the few to analyze anaerobic performance responses to interval training executed under different models of hypoxic exposure while maintaining an identical training protocol. Based on the available evidence, it may be suggested that active LLTH-based approaches have greater potential to improve anaerobic performance than passive hypoxic exposure. However, the findings are often conflicting and appear to depend on the type of protocol applied [24]. Due to the logistical challenges associated with the LHTL model, many studies have focused on the LLTH approach [24,30]. Nevertheless, even within this model, evidence related to anaerobic performance remains limited. In the review by Feng et al. on the LLTH model, only 29 studies reported anaerobic performance outcomes [24]. The results of the meta-analysis indicated that active forms of hypoxic training, including RSH, ISH, s-IHT,

and I-IHT, may be more effective in improving anaerobic performance than training under normoxic conditions. However, for some variants, the key conclusions were drawn from only one study, which limits the reliability of these findings. Improvements in anaerobic parameters after four weeks of interval training in hypoxia have been reported, among others, by Maciejczyk et al., who observed an increase in peak power during the Wingate test [26,46]. At the same time, several authors have not demonstrated additional benefits of hypoxic training compared with the same training performed under normoxic conditions for aerobic and anaerobic performance parameters. This included VO_2max , PP and MP in the Wingate test, repeated sprint ability (RSA), critical power (CP), and high-intensity work capacity (HIWC) [27,28,47–49]. Moreover, potential improvements appear to occur mainly in protocols that are sprint-specific in nature [29,50]. Therefore, it may be suggested that the more demanding the interval performed under hypoxic conditions, the more likely it is that performance improvements may be associated with the additional hypoxic stress.

Evidence regarding the benefits of passive hypoxic exposure is even less conclusive, as this model is usually associated primarily with aerobic and hematological adaptations rather than with direct improvement in the ability to generate anaerobic power. Studies focused on passive hypoxic exposure suggest that exposure to hypoxic conditions alone, regardless of exposure duration [31] or applied altitude [32], may not translate into improved anaerobic performance indices. Using a double-blind, placebo-controlled crossover study design, Bejder et al. implemented a six-week normobaric LHTL intervention with progressive altitude exposure (2500 to 3500 m above sea level, 8 h/day). Despite this protocol, they did not observe improvement in 30-s repeated sprint ability compared to the control condition [32]. In the present study, the LLTL group did not show improvements in anaerobic performance variables after HIIT performed under normoxic conditions alone. In this context, the lack of improvement in the hypoxia-exposed groups, regardless of whether hypoxia was applied during training or as passive exposure, should be interpreted in relation to the applied HIIT protocol. These findings suggest that under the conditions in which the present study was carried out, hypoxic exposure alone was not associated with measurable improvements in these variables.

Another factor that may influence the training response is the dose of hypoxia, which includes both the severity of hypoxia and the duration of exposure. The present study was conducted at a simulated altitude of approximately 3000 m ($\text{FiO}_2 \sim 14.5\%$), which represents a moderate hypoxic stimulus but is sufficient to induce a physiological response [51]. It is possible that a greater level of hypoxia could have affected the results. Warnier et al. assessed the effects of a six-week, 30-s sprint training protocol performed at sea level and at simulated altitudes of 2000 m (FiO_2 16.7%), 3000 m (FiO_2 14.5%) and 4000 m (FiO_2 13.0%) on anaerobic performance measured using the Wingate test. After the intervention, improvement in PP was observed only at 4000 m, whereas training at lower altitudes did not provide additional benefits [7]. The authors suggested that the observed increase in PP may not have resulted from changes in energy pathways, but rather from modifications in muscle structure or the recruitment of fast-twitch fibers. At the same time, Karayigit et al. [52] showed that both moderate (3000 m; FiO_2 14%) and severe (4000 m; FiO_2 12%) normobaric hypoxia reduced muscular endurance, with no changes in maximal muscle strength. This indicates that a greater hypoxic stimulus may limit the ability to sustain high-intensity exercise and, therefore, may not support adaptations related to power generation during short maximal efforts. Thus, the effect of hypoxic dose on anaerobic performance indices may plausibly depend on the balance between a potentially beneficial neuromuscular stimulus, such as type II fiber recruitment, and a simultaneous reduction in exercise tolerance, as well as the ability to maintain high power output during training.

In light of these findings, our results may suggest that the applied normobaric hypoxia alone, regardless of the exposure model, may not have provided a sufficient additional stimulus to induce measurable changes in anaerobic performance. At the same time, the available evidence remains too limited and inconsistent to determine whether hypoxia can effectively support anaerobic adaptations. Therefore, the specificity of the training stimulus—particularly the inclusion of short, repeated maximal or near-maximal efforts—may be an important factor when designing interventions aimed at improving anaerobic performance.

It should be noted that the mechanistic explanations discussed in this section are based on previous literature and were not directly assessed in the present study. Thus, they should be interpreted as possible explanations rather than direct findings of this study.

4.1. Strengths of the Study

An important strength of the present study is its four-group design, which allowed the anaerobic performance response to the same HIIT protocol to be assessed under normoxic conditions and involving two different models of hypoxic exposure. The use of a hypoxic thermoclimatic chamber enabled precise generation and continuous monitoring of specific environmental conditions. In the current trial, hypoxic exposure was applied under stable temperature and humidity conditions. In addition, the same training protocol was used in all groups, including an analogous number of sessions, session structure, and intensity monitoring. This reduced the risk that differences between groups would result from variances in training volume rather than from the environmental conditions under which the training was conducted.

4.2. Study Limitations

The present study has several limitations that should be considered when interpreting the findings. First, although the required sample size was estimated a priori, the final number of participants in each group was relatively small. This may have limited the possibility to detect subtle adaptive changes.

The present study was focused on the effects of training carried out using different models of hypoxic exposure, without directly analyzing the mechanisms responsible for the observed responses. Therefore, mechanistic explanations of the findings should be treated as hypothetical and based on previous literature. Mechanisms that may potentially influence strength, power or muscle fiber shortening velocity, including physiological, biochemical and molecular adaptations within the skeletal muscle as well as non-hematological effects, should be investigated in future studies.

Another limitation is the use of the 20-s Wingate test protocol. Although it allows assessment of short-duration maximal power output, it may limit direct comparability with studies using the standard 30-s Wingate test.

In addition, the participants were not competitive athletes and mostly comprised physical education students. This was reflected in a moderate to relatively high baseline level of physical activity, which could have reduced the potential for measurable changes over a short intervention period. The four-week protocol, combined with HIIT that did not include maximal or supramaximal efforts, may have been an insufficient stimulus to improve anaerobic performance in the studied population.

The present study included only young men, which limits the generalizability of the findings to women, other age groups and competitive athletes. Moreover, the hypoxic dose used in the LHTL model, consisting of 12 overnight exposures of approximately 12 h each, may have been inadequate to induce measurable changes in the analyzed indices of anaerobic performance.

Although dietary intake was evaluated using dietary records completed before and after the intervention, the participants' diets were not standardized. In addition, sleep and general well-being were not objectively monitored during the four-week intervention, which may have influenced individual training responses.

Finally, no follow-up measurements were performed after the intervention, which prevents assessment regarding the persistence of possible training adaptations. The present findings also refer to normobaric hypoxic conditions and may not be directly transferable to training adaptations occurring in a hypobaric environment.

5. Conclusions

The present study provides information on the effects of HIIT performed using different models of hypoxic training on anaerobic performance in young men. The four-week HIIT protocol conducted both under conditions of normoxia and normobaric hypoxia—corresponding to an altitude of approximately 3000 m above sea level—did not lead to significant changes in anaerobic performance assessed using the 20-s maximal cycling sprint test. Under the conditions of the applied protocol, the use of normobaric hypoxia, either as training in hypoxia or as passive overnight exposure consisting of 12 sessions of approximately 12 h each, was not associated with improvements in the analyzed indices of anaerobic performance.

These findings should be interpreted in relation to the specific intervention, population and assessed outcomes. From a practical perspective, the results may suggest that adding normobaric hypoxia to a short-term HIIT protocol does not necessarily provide additional benefits for anaerobic performance variables when the training stimulus is not closely matched to the demands of short maximal sprint efforts. Protocols based on maximal or supramaximal sprint efforts, longer intervention periods or different hypoxic doses may produce varying responses and require further investigation. Therefore, the present findings contribute to the evidence suggesting that the effects of hypoxic HIIT on anaerobic performance are not universal and may depend on the structure and specificity of the training protocol.

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