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Combined Effects of Normobaric Hypoxia and Resistance Training Intensity on Anaerobic Performance, Lactate Kinetics, and Neuromuscular Function in Trained Ice Hockey Players: A randomized trial

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Combined Effects of Normobaric Hypoxia and Resistance Training Intensity on Anaerobic Performance, Lactate Kinetics, and Neuromuscular Function in Trained Ice Hockey Players: A randomized trial

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Abstract

Background

Ice hockey requires a specific combination of physical attributes, including explosive power, speed, agility, and specific metabolic capacities. This study aimed to determine whether hypoxic conditions during resistance training at high and low intensity enhance anaerobic power, lactate kinetics, and neuromuscular function compared to normoxic conditions in highly trained ice hockey players.

Methods

In this randomized, four-arm, parallel-group trial, forty male ice hockey athletes (aged 18–25 years) were randomly allocated to four groups and performed either normoxic or normobaric hypoxic resistance training at high (80% of one repetition maximum – 1RM) or low intensity (30% of 1RM) twice a week for six weeks. Pre and post-tests were done for one repetition maximum leg press, anaerobic performance (30s Wingate test), lactate kinetics, and muscle

contractility by tensiomyography. Data were analysed using 2 × 4 (Time × Group) mixed-design repeated-measures ANOVA with Bonferroni-corrected post hoc comparisons.

Results

All groups improved their 1RM leg press with no differences between groups. Peak power increased, and time to peak power during the Wingate test decreased after high-intensity resistance training in normoxia (N-H) and high-intensity resistance training in hypoxia (H-H) ($p< 0.001$). The H-H and N-H groups showed a higher power decline in repeated Wingate ($p< 0.001$). The low load hypoxia training (H-L) improved lactate clearance, increased HCO3act decline, and decreased pH decline ($p<0.001$). Vastus lateralis contraction time increased in the N-H group ($p=0.021$), and contraction displacement decreased in H-H.

Conclusions

Crucially, hypoxic resistance training does not uniformly enhance all performance outcomes; rather, it produces specific, intensity-dependent adaptations. The intensity of resistance training elicits a superior training response to hypoxia, enhancing power in ice hockey players.

Trial registration

ClinicalTrials.gov, NCT06930027, registered retrospectively on 08 April 2025.

Keywords: Resistance training, anaerobic condition, exercise, hypoxia

Background

Ice hockey requires a specific combination of physical attributes, including explosive power, speed, agility, and specific metabolic capacities [1, 2]. The intermittent nature of the sport, characterized by short bouts of maximal or near-maximal exertion interspersed with recovery periods, poses unique physiological demands [3, 4]. Given ice hockey's considerable anaerobic requirements, training strategies aimed at enhancing peak power, reducing fatigue, and improving lactate clearance during repeated high-intensity efforts are critically important [2, 5].

Resistance training is a key component of athletic preparation in intermittent sports, promoting improvements in muscular strength, power, and anaerobic performance [6, 7]. Adjusting variables such as training load, volume, and rest intervals enables coaches to elicit targeted muscular adaptations [8]. Typically, high-load, low-repetition protocols are utilized to maximize strength gains [9], whereas lower-load, high-repetition schemes induce greater metabolic stress, promoting muscular endurance and hypertrophic responses through distinct physiological pathways [10]. Parallel developments in sport science have led to investigations into hypoxic exposure as a potential strategy to enhance training adaptations [11]. Aerobic adaptations to hypoxic exposure, such as increased erythropoiesis, enhanced capillarization, mitochondrial biogenesis, and improved oxidative metabolism, are well-documented [12-14]. However, the specific impact of hypoxic training on anaerobic and particularly strength-related adaptations remains speculative and less clearly established, especially in ice hockey.

Resistance training combined with hypoxic exposure (hypoxic resistance training, HRT) can potentially induce unique physiological adaptations compared to normoxic conditions. Hypoxic conditions during resistance exercise may intensify metabolic stress by promoting reliance on anaerobic glycolysis, resulting in elevated metabolite accumulation, including lactate, hydrogen ions, and inorganic phosphate [15, 16]. Additionally, hypoxia increases sympathetic nervous system activation, elevating plasma concentrations of noradrenaline and adrenaline relative to normoxic exercise at similar intensities [17]. Such metabolic disturbances are likely to facilitate adaptations beneficial to anaerobic performance [18]. Moreover, hypoxia may alter motor unit recruitment patterns, accelerating the activation of high-threshold motor units that predominantly innervate fast-twitch fibers, essential for explosive actions [19, 20]. Furthermore, combined mechanical tension and hypoxia-induced metabolic stress may synergistically activate molecular signaling pathways involved in muscle hypertrophy and angiogenesis, which can support anaerobic performance [16, 21].

Empirical evidence from a previous study [22] demonstrated superior muscular strength gains following six weeks of moderate-load (70% 1RM) resistance training under systemic hypoxia compared to normoxia. Another finding indicated that enhanced muscle endurance and hypertrophic responses occurred in trained female athletes who underwent five weeks of low-load (20% 1RM) resistance training under hypoxic conditions, compared to their normoxic counterparts [23]. Despite compelling theoretical mechanisms, empirical research examining hypoxic resistance training has yielded mixed outcomes. While some studies report significant enhancements in muscular strength, hypertrophy, and power from hypoxic versus normoxic training [22, 23], others indicate minimal or comparable benefits [19]. Such discrepancies may result from methodological variability, including differences in hypoxic severity, resistance training parameters (intensity, volume, exercise type), and participant characteristics. Specifically, elite ice hockey athletes routinely engage in strength-oriented heavy resistance training supplemented by anaerobic conditioning, yet it remains unclear how an additional hypoxic stimulus might modulate these targeted adaptations.

The current study employs an extensive battery of assessments pre- and post a six-week intervention to explore the efficacy and physiological mechanisms of hypoxic resistance training on performance parameters relevant to ice hockey, including strength, anaerobic capacity, buffering capabilities, and muscle contractility. The primary objective was to determine whether hypoxic conditions during resistance training enhance anaerobic power, lactate kinetics, and neuromuscular function compared to normoxic conditions in highly trained ice hockey players. A secondary hypothesis anticipates an interaction effect between training intensity and oxygen environment, predicting that low-load training in hypoxia will produce adaptations equivalent to high-load training in normoxia.

Methods

Study design

This study followed the CONSORT guidelines and employed a six-week, randomized, open-label, parallel-group design with a 2 × 2 factorial structure to evaluate the independent and combined effects of oxygen condition (normoxia vs. normobaric hypoxia) and resistance-training intensity (low vs. high) on anaerobic performance, lactate kinetics, and muscle contractility in ice hockey athletes. Following baseline testing, forty participants were randomly assigned via computer-generated sequence into one of four groups (n = 10 each): low-intensity resistance training in normoxia (N-L), high-intensity resistance training in normoxia (N-H), low-intensity resistance training in hypoxia (H-L) and high-intensity resistance training in hypoxia (H-H). Each athlete completed two supervised leg-press sessions per week (twelve total), with mid-intervention load progression, standardized warm-up, controlled tempo, and cooldown. Participants were blinded to their environmental condition throughout the intervention: all training sessions were conducted inside a normobaric environmental chamber, and the chamber's FiO_2 display was concealed from view. Athletes were informed only that they would train under varying "oxygen conditions" but received no feedback as to whether they were in the hypoxic (~13.5% FiO_2) or normoxic (~20.9% FiO_2) environment, ensuring that expectations did not influence performance or perceptual responses. One-repetition maximum (1RM) testing for the leg-press was performed 48 hours prior to the baseline assessment session. Pre- and post-intervention assessments—including Wingate anaerobic tests for upper and lower limb, serial capillary lactate sampling (0, 4, 8 min), and tensiomyography measurements of the vastus lateralis and triceps brachii—were conducted within one week before and after the training period. The inclusion of upper-limb testing controlled for nonspecific training adaptations, ensuring observed lower-limb changes were attributable to the leg press intervention. Testers of 1RM, Wingate, lactate, and TMG were unaware of group assignment. The trial was preregistered at ClinicalTrials.gov (NCT06930027).

Subjects

A G*Power 3.1 a priori calculation (f = 0.25 [$\eta_p^2 = 0.06$], $\alpha = 0.05$, $1–\beta = 0.80$; 2×4 mixed-ANOVA; r = 0.50; $\varepsilon = 1.00$) indicated a required sample of 36 (n = 9 per group). To allow for potential dropouts, forty male ice hockey athletes (aged 18–25 years; training history ≥ 5 years) were enrolled (Figure 1). All athletes were free of musculoskeletal injury for ≥ 6 months, had no hypoxic training or altitude exposure in the preceding six months, and routinely engaged in in-season strength training. Exclusion criteria included any injury or condition limiting resistance exercise within 3 months, cardiovascular/metabolic/respiratory/neuromuscular disorders, concurrent research interventions, altitude-related illness history, or use of performance-modulating substances within four weeks of enrollment. Written informed consent was obtained per the Declaration of Helsinki, and the protocol was approved by the institutional ethics committee (Bioethics Commission of the Academy of Physical Education in Katowice, 4-XI/2024). Two participants withdrew for personal reasons at the beginning of the intervention and were immediately replaced by two additional eligible athletes. The remaining forty subjects were randomly allocated via computer-generated sequence (randomizer.org) into four groups (n = 10 each).

Figure 1. CONSORT flow diagram.

1RM testing

During the familiarization session, leg-press one-repetition maximum (1RM) was determined on a Keiser Leg Press A420 pneumatic device (Keiser, Fresno, CA, USA). Following a general warm-up (5 min cycling at ~70 rpm against 1 $W \cdot kg^{-1}$) and dynamic drills (two sets of five bodyweight squats, five lunges per leg, five push-ups, and five resistance-band standing rows per arm), participants performed three preparatory sets on the leg press: 15 reps at 20% estimated 1RM, 10 reps at 40%, and 5 reps at 60%. The first maximal attempt was then set at ~80% of the estimated 1RM; subsequent loads were increased in 10–20 kg increments until

volitional failure, with a 3 min passive rest between successful lifts. Throughout testing, subjects were seated with knees flexed approximately at a 90° angle and feet flat on the footplates, and instructed to execute each concentric phase "as fast as possible." The highest load lifted with correct technique was recorded as the participant's 1RM.

Training program

The training intervention comprised 12 supervised leg-press sessions over 6 weeks, delivered twice weekly under either normoxic ($FiO_2 \approx 20.9\%$) or simulated hypoxic (~3 500 m; $FiO_2 \approx 13.5\ \%$) conditions according to group allocation (Table 1). Each session began with a five-minute warm-up on a cycle ergometer at 100 W, followed by a single preparatory set on the leg-press at approximately 50 % of that day's planned load. Thereafter, high-intensity groups (N-H, H-H) performed four sets of ten repetitions, while low-intensity groups (N-L, H-L) executed four sets using a 30/15/15/15 repetition scheme. Load was set at 70 % of the individual's 1RM for weeks 1–3 and increased to 80 % for weeks 4–6 in the high-intensity arms, whereas low-intensity arms progressed from 20 % to 30 % 1RM over the same periods. All repetitions were performed with a controlled two-second eccentric and one-second concentric tempo; rest intervals were standardized at two minutes between sets for high-intensity sessions and one minute for low-intensity sessions. Investigators adjusted loads weekly based on performance feedback to maintain target intensities and provided continuous supervision to ensure technique, adherence, and safety, and each session concluded with a five-minute cooldown at 50 W.

Table 1. Training program parameters for every group

Arm	Condition (Altitude; FiO_2)	Load Weeks 1–3	Load Weeks 4–6	Repetition Scheme
N-L	Normoxia (0 m; FiO_2 20.9 %)	20 %	30 %	30 / 15 / 15 / 15

N-H	Normoxia (0 m; FiO_2 20.9 %)	70 %	80 %	4 × 10
H-L	Hypoxia (~ 3 500 m; FiO_2 13.5 %)	20 %	30 %	30 / 15 / 15 / 15
H-H	Hypoxia (~ 3 500 m; FiO_2 13.5 %)	70 %	80 %	4 × 10

N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia.

Anaerobic performance assessment

Anaerobic performance was assessed using a repeated Wingate protocol. After a standardized warm-up (5 min cycling at 50 W plus two 5 s submaximal sprints), participants completed two 30 s maximal efforts on an arm-crank ergometer (Brachumera Sport, Lode, Groningen Netherlands) at a resistance of 7.5 % body mass, with 5 min of passive recovery between sprints. Following a 2 h passive rest period, they then performed two 30 s maximal sprints on a Lode Excalibur cycle ergometer (Lode BV, Netherlands) using the same relative load and recovery scheme. Power output was sampled at 1 Hz throughout each trial, and for every sprint we calculated peak power (highest 5 s mean), mean power (30 s average), time to peak power [s] and inter-bouts peak power and mean power decline [%].

Lactate and blood-gas analysis

Capillary blood samples were collected at baseline, 4- and 8-min post-exercise for both upper- and lower-limb protocols. Using a sterile, single-use lancet, approximately 20 µL of blood was drawn from the non-dominant fingertip into heparinized capillary tubes (75 µL capacity) by capillary action. A 0.5 µL aliquot was immediately analyzed by Biosen C-line Clinic (EKF-diagnostic GmbH, Germany) and the analyzer was calibrated before each session. All assays were performed at 22–24 °C and completed within ~15 s; any error code or physiologically implausible value prompted immediate re-sampling and re-analysis. Late-phase lactate clearance rate (8’- 4’) was quantified using the formula:

193 $$\textit{Clearance Rate per Minute (\%)} = \ln(LA4'') - \ln(LA8'') / \Delta t \times 100$$

194 By applying a natural-logarithmic transformation to lactate concentrations, the inherently
195 exponential decay of lactate — governed by non-linear processes such as enzymatic
196 metabolism and membrane transport [24] —is linearized.

197 Concomitant capillary blood-gas measurements were made at 0 min and 4 min post-exercise.
198 A separate 20 µL sample was collected and analyzed on a GEM Premier 3500 blood-gas
199 analyzer (Instrumentation Laboratory, USA) with daily calibration and quality controls per
200 manufacturer guidelines providing pH, partial pressure of CO_2 (pCO_2)), bicarbonate
201 concentration [HCO_3^-act]), and arterial oxygen saturation (SpO_2).

202 ***Contractile properties assessment***

203 Tensiomyographic evaluation of muscle contractile properties was performed on the vastus
204 lateralis and triceps brachii using a TMG-S2 device (TMG-BMC, Ljubljana, Slovenia). With
205 participants supine—knee flexed to ~30° for the quadriceps site and elbow extended to ~10°
206 for the triceps—bipolar self-adhesive electrodes (5×5 cm) were placed 2–3 cm proximal and
207 distal to the sensor, which was positioned perpendicular to the muscle belly at maximal girth;
208 all landmarks were marked in indelible ink to ensure repeatability. Single 1 ms square-wave
209 pulses were delivered sequentially at 50, 75, and 100 mA, with 10 s rest between stimuli to
210 prevent fatigue. From each displacement–time curve, maximal radial displacement (Dm), delay
211 time (Td; stimulus to 10 % of Dm), contraction time (Tc; 10 % to 90 % of Dm), sustain time
212 (Ts; duration above 50 % of Dm), and half-relaxation time (Tr; 90 % to 50 % of Dm) were
213 extracted. Two trials per muscle were recorded, and the trial exhibiting the greatest Dm was
214 used for analysis, providing a detailed profile of neuromuscular responsiveness and contractile
215 kinetics.

216 ***Statistical analysis***

All data are presented as mean ± SD with 95%CI. Normality of distribution was assessed by the Shapiro–Wilk test and homogeneity of variances by Levene's test. For each dependent variable, a 2 × 4 mixed-design repeated-measures ANOVA was performed with Time (pre vs. post) as the within-subjects factor and Group (N-L, N-H, H-L, H-H) as the between-subjects factor. Partial eta squared (η_p^2) was used to quantify ANOVA effect sizes, with benchmarks of 0.01 (small), 0.06 (medium), and 0.14 (large). Significant Time × Group interactions were followed up with Bonferroni-corrected pairwise comparisons. Within-subject changes were characterized by Cohen's d_nz (mean change / SD of change; 0.2 = small, 0.5 = medium, 0.8 = large), and between-group contrasts by Cohen's *d* (pooled SD; same benchmarks). Between-group contrasts at each time point were examined with independent-samples t-tests, also Bonferroni-adjusted. Statistical significance was set at $\alpha = 0.05$ for all analyses. All statistical analyses were performed using Jamovi (version 2.3.21; The Jamovi Project, Sydney, Australia).

Results

Baseline anthropometric and body-composition variables for each experimental group are summarized in Table 2. Body mass, height, muscle mass, and fat mass did not differ significantly between groups prior to the intervention.

Table 2. Essential characteristics of experimental groups

Group	Body mass [kg]	Height [cm]	Muscle mass [kg]	Fat mass [%]
N-L	83.45 ± 12.26	179.60 ± 7.87	41.49 ± 2.87	13.17 ± 8.29
N-H	82.31 ± 12.18	177.08 ± 7.08	40.79 ± 1.20	12.87 ± 6.75
H-H	81.83 ± 10.40	178.70 ± 8.91	40.02 ± 1.67	10.56 ± 5.48
H-L	77.17 ± 11.52	179.13 ± 7.34	41.61 ± 1.68	11.77 ± 4.65

Results presented as means with standard deviation. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia.

Anaerobic performance

Anaerobic performance outcomes, including peak and mean power, time to peak power, and one-repetition maximum (1RM) for leg press, are presented for each group at pre- and post-intervention in Table 3. Values are expressed as mean ± SD with 95% confidence intervals.

Table 3. Descriptive statistics of experimental groups' performance indicators

Group	Peak power [W] (M ± SD; 95 % CI)				Mean power [W] (M ± SD; 95 % CI)				Time to peak power [s] (M ± SD; 95 % CI)		1RM [kg] (M ± SD; 95 % CI)	
	W1		W2		W1		W2		W1		Leg press	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST
H-H	1015.5 ± 97.9; (945.5–1085.5)	1141.6 ± 96.7; (1072.4–1210.8)	929.9 ± 89.6; (917.5–942.4)	1014.2 ± 85.9; (999.9–1028.4)	650.2 ± 75.4 (598.1–702.3)	655.7 ± 77.8 (602.5–708.9)	674 ± 70.6 (624–725)	679.4 ± 73.1 (626.7–731.3)	2.48 ± 0.19; (2.34–2.61)	2.30 ± 0.24; (2.13–2.47)	316.0 ± 33.8 (291.8–340.2)	355.5 ± 37.6 (328.6–382.4)
H-L	1014.4 ± 116.0; (931.4–1097.4)	1044.9 ± 128.0; (953.4–1136.5)	932.6 ± 106.7; (927.1–938.1)	948.1 ± 116.1; (938.9–957.4)	640.8 ± 85.9 (578.6–703.0)	661.5 ± 88.2 (599.1–723.9)	671 ± 80.6 (613–728)	702.3 ± 84.7 (641.4–763.2)	2.47 ± 0.18; (2.34–2.60)	2.53 ± 0.20; (2.39–2.68)	309.5 ± 31.0 (287.3–331.7)	327.7 ± 32.8 (304.3–351.2)
N-H	1019.0 ± 112.8; (938.3–1099.6)	1141.5 ± 148.2; (1035.5–1247.6)	937.9 ± 103.8; (931.3–944.6)	1016.4 ± 132.0; (1005.8–1027.0)	610.4 ± 120.1 (528.2–692.6)	620.1 ± 122.3 (537.0–703.2)	635 ± 115.4 (552–717)	643.8 ± 117.0 (559.3–728.3)	2.44 ± 0.22; (2.28–2.60)	2.26 ± 0.34; (2.01–2.50)	304.8 ± 30.3 (283.1–326.5)	340.7 ± 31.1 (318.4–363.0)
N-L	1028.6 ± 88.8; (965.1–1092.2)	1017.7 ± 109.4; (939.4–1095.9)	941.4 ± 81.3; (933.1–949.8)	932.6 ± 100.3; (924.8–940.3)	689.5 ± 215.7 (537.9–841.1)	694.8 ± 218.1 (543.0–846.6)	708 ± 208.9 (558–857)	713.2 ± 212.4 (561.1–865.3)	2.45 ± 0.29; (2.24–2.65)	2.41 ± 0.33; (2.17–2.65)	299.4 ± 27.4 (279.8–319.0)	317.2 ± 29.1 (296.4–338.0)

Results presented as means with standard deviation. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia; W1 – first wingate bout; W2 – second wingate bout

For 1RM variable ANOVA analysis revealed a significant main effect of Time, $F(1,36) = 960.60$, $p < 0.001$, $\eta p^2 = 0.964$; no main effect of Group, $F(3,36) = 1.29$, $p = 0.293$; and a significant Time × Group interaction, $F(3,36) = 40.50$, $p < 0.001$, $\eta p^2 = 0.772$ (Table 3). Bonferroni-corrected paired comparisons indicated significant pre-to-post increases in all groups—H-H ($\Delta = +39.46$, $p < 0.001$, $dz = 3.60$), N-H ($\Delta = +35.91$, $p < 0.001$, $dz = 3.28$), H-

L (Δ = +18.26, $p < 0.001$, d = 1.67), and N-L (Δ = +17.79, $p < 0.001$, dz = 1.62). No between-group differences were observed at either the pre- or post-test (all $p > 0.05$).

Figure 2. Leg press 1RM change, N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of Peak power showed a significant main effect of Time, $F(1, 36) = 17.95$, $p < 0.001$, $\eta p^2 = 0.333$; no main effect of Group, $F(3, 36) = 0.73$, $p = 0.541$; and a significant Time × Group interaction, $F(3, 36) = 6.34$, $p = 0.009$, $\eta p^2 = 0.271$ (Table 3). Bonferroni-corrected paired comparisons showed significant pre-to-post increases in two conditions—H-H (Δ = +126.1, $p = 0.011$, dz = 0.64) and N-H (Δ = +122.59, $p = 0.015$, dz = 0.66)—whereas the H-L (Δ = +30.56, $p > 0.05$) and N-L (Δ = +26.00, $p > 0.05$) groups showed non-significant changes. No between-group differences were observed at either the pre- or post-test (all $p > 0.05$).

Figure 3. Wingate peak power change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of Time to peak power revealed a significant main effect of Time, $F(1,36) = 11.34$, $p = 0.002$, $\eta_p^2 = 0.239$; no main effect of Group, $F(3,36) = 0.40$, $p = 0.753$, $\eta_p^2 = 0.032$; and a significant Time × Group interaction, $F(3,36) = 5.11$, $p = 0.005$, $\eta_p^2 = 0.299$ (Table 3). Bonferroni-corrected post-hoc comparisons showed a significant pre-to-post decrease only in the N-H group (Δ = –0.248, $p = 0.009$, dz = 1.50). The H-H group also decreased (%Δ = –0.230), a change that approached significance ($p = 0.063$), whereas H-L and N-L did not change (both $p > 0.05$). No between-group differences were observed at either time point (all $p > 0.05$).

Figure 4. Wingate time to peak power change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Repeated-measures ANOVA on inter-bout Wingate peak power decline [%] revealed a significant main effect of Time, $F(1,36) = 42.35$, $p < 0.001$, $\eta_p^2 = 0.541$, and a significant Time × Group interaction, $F(3,36) = 7.63$, $p < 0.001$, $\eta_p^2 = 0.389$; and main effect of Group, $F(3,36) = 4.51$, $p = 0.009$, $\eta_p^2 = 0.273$ (Figure 5). Bonferroni-corrected paired comparisons showed that the H-H (%Δ = +2.74, $p < 0.001$, dz = 1.13) and N-H (%Δ = +2.54, $p = 0.002$, dz = 1.08) groups exhibited significantly greater declines in peak power from pre- to post-test, indicating increased fatigue across bouts. In contrast, H-L (%Δ = +0.84, $p = 1.000$) and N-L (%Δ = –0.06, $p = 1.000$) groups did not show significant changes. At post-test, the H-H group had a significantly higher power decline compared to N-L (%Δ = +2.80, $p = 0.001$, d = 0.96), and the N-H group also showed a higher decline compared to N-L (%Δ = +2.60, $p = 0.004$, d = 0.89); no other between-group differences were significant. No significant changes were found for inter-bout mean power decline [%].

Figure 5. Wingate peak power decline change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Metabolic changes

Metabolic parameters—lactate concentration at 4 min (LA_4) and 8 min (LA_8), and lactate clearance rate (LCR)—for each group at pre- and post-intervention are summarized in Table 4. Values are presented as mean ± SD with 95 % confidence intervals.

Table 4. Descriptive statistics of experimental groups' lactate kinetics

Group	LA_4 [mmol·L^{-1}] (M ± SD; 95 % CI)		LA_8 [mmol·L^{-1}] (M ± SD; 95 % CI)		LCR [%] (M ± SD; 95 % CI)	
	PRE	POST	PRE	POST	PRE	POST
H-H	15.50 ± 2.01; (14.06 – 16.94)	16.60 ± 2.11; (15.09 – 18.11)	11.64 ± 1.51 (10.56–12.71)	11.98 ± 1.52 (10.89–13.07)	7.17 ± 8.22 (1.29–13.05)	8.16 ± 6.33 (3.63–12.69)
H-L	14.89 ± 2.46; (13.13 – 16.65)	15.17 ± 2.33; (13.50 – 16.83)	11.98 ± 1.98 (10.57–13.40)	10.40 ± 1.60 (9.26–11.54)	5.43 ± 3.60 (2.86–8.00)	9.44 ± 3.61 (6.86–12.02)
N-H	15.13 ± 2.77; (13.14 – 17.11)	16.03 ± 2.42; (14.30 – 17.76)	11.30 ± 2.07 (9.82–12.78)	11.78 ± 1.78 (10.51–13.05)	7.29 ± 5.03 (3.69–10.88)	7.70 ± 4.09 (4.78–10.63)
N-L	15.08 ± 2.28; (13.44 – 16.71)	15.27 ± 2.06; (13.80 – 16.74)	11.74 ± 1.77 (10.47–13.01)	11.06 ± 1.49 (9.99–12.12)	6.26 ± 5.17 (2.57–9.96)	8.07 ± 3.92 (5.27–10.88)

297 Results presented as means with standard deviation. N-L - low-intensity resistance training in
298 normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance
299 training in hypoxia and H-H - high-intensity resistance training in hypoxia, La – lactate, LCR
300 – lactate clearance rate
301 For peak lactate (4') variable ANOVA analysis revealed a significant main effect of Time, $F(1,$
302 $36) = 36.47$, $p < 0.001$, $\eta p^2 = 0.496$; no main effect of Group, $F(3, 36) = 0.4$, $p = 0.754$; and a
303 significant Time × Group interaction, $F(3, 36) = 4.71$, $p = 0.007$, $\eta p^2 = 0.282$ (Table 4).
304 Bonferroni-corrected paired comparisons showed significant pre-to-post increases in two
305 groups—H-H ($\Delta = +1.10$, $p = 0.001$, $d = 0.7$) and N-H ($\Delta = +0.90$, $p = 0.003$, $dz = 0.7$)—
306 whereas H-L and N-L did not change reliably ($p > 0.05$). No between-group differences were
307 observed at either time point (all $p > 0.05$).
308 Figure 6. Peak lactate change, N-L - low-intensity resistance training in normoxia, N-H - high-
309 intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and
310 H-H - high-intensity resistance training in hypoxia;
311 Analysis of Late-phase lactate clearance per minute rate showed a significant main effect of
312 Time, $F(1,36) = 20.96$, $p < 0.001$, $\eta_p^2 = 0.368$; no main effect of Group, $F(3,36) = 0.02$, $p =$
313 0.997; and a significant Time × Group interaction, $F(3,36) = 3.99$, $p = 0.015$, $\eta_p^2 = 0.250$ (Table
314 4; Figure 7). Bonferroni-corrected simple-main-effect tests revealed that only the H-L group
315 showed a significant pre-to-post increase ($\%\Delta = –4.008$, $p < 0.001$, $dz = 1.61$), whereas the H-
316 H ($\%\Delta = –0.986$), N-H ($\Delta = –0.530$), and N-L ($\Delta = –0.901$) groups did not change significantly
317 (all $p = 1.000$). No between-group differences were significant at either time point (all $p > .05$).
318 Figure 7. Late phase clearance rate change
319 Blood gas parameters—arterial pH and bicarbonate (HCO_3^-) measured at rest (0') and 4' post-
320 exercise, and oxygen saturation (SaO_2) at rest and 4' post-exercise—are summarized for each
321 group in Table 5. Values are presented as mean ± SD with 95 % confidence intervals.

Table 5. Descriptive statistics of experimental groups' blood gas analysis

Group	pH (M ± SD; 95 % CI)				HCO3-act [mmol/L] (M ± SD; 95 % CI)				SaO2 [%] (M ± SD; 95 % CI)	
	0'		4'		0'		4'		4'	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST
H-H	7.39 ± 0.02; (7.38–7.40)	7.39 ± 0.02; (7.38–7.40)	7.20 ± 0.03; (7.18–7.22)	7.18 ± 0.04; (7.15–7.21)	23.8 ± 0.48; (23.0–24.6)	23.7 ± 0.65; (22.4–24.9)	19.0 ± 2.20; (15.0–23.0)	19.1 ± 2.50; (14.1–24.1)	94.5 ± 0.46; (94.1–94.9)	94.2 ± 0.68; (93.7–94.7)
H-L	7.40 ± 0.02; (7.39–7.41)	7.39 ± 0.02; (7.38–7.40)	7.23 ± 0.03; (7.21–7.25)	7.18 ± 0.04; (7.15–7.21)	24.2 ± 0.55; (23.1–25.3)	23.8 ± 0.38; (23.0–24.6)	19.5 ± 1.40; (17.0–22.0)	19.4 ± 1.70; (17.1–21.7)	94.6 ± 0.44; (94.3–94.9)	94.3 ± 0.60; (93.9–94.7)
N-H	7.38 ± 0.02; (7.37–7.39)	7.38 ± 0.02; (7.37–7.39)	7.16 ± 0.03; (7.14–7.18)	7.16 ± 0.03; (7.14–7.18)	24.0 ± 0.60; (23.0–25.0)	23.8 ± 0.25; (23.3–24.3)	19.7 ± 1.50; (18.1–21.3)	19.0 ± 1.40; (17.8–20.2)	94.3 ± 0.50; (93.8–94.8)	94.4 ± 0.55; (93.9–94.9)
N-L	7.39 ± 0.02; (7.38–7.40)	7.40 ± 0.02; (7.39–7.41)	7.19 ± 0.03; (7.17–7.21)	7.20 ± 0.03; (7.18–7.22)	24.1 ± 0.70; (22.7–25.5)	23.6 ± 0.42; (22.7–24.5)	19.8 ± 2.50; (16.8–22.8)	19.5 ± 2.90; (16.3–22.7)	94.7 ± 0.40; (94.4–95.0)	94.8 ± 0.45; (94.4–95.2)

Results presented as means with standard deviation. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia

Analysis of pH decline [%] revealed a significant main effect of Time, $F(1,36) = 21.70$, $p < 0.001$, $\eta_p^2 = 0.376$; no main effect of Group, $F(3,36) = 2.82$, $p = 0.053$, $\eta_p^2 = 0.190$; and a significant Time × Group interaction, $F(3,36) = 11.00$, $p < 0.001$, $\eta_p^2 = 0.479$ (Figure 8). Bonferroni-corrected simple main-effect tests showed that only the H-L group decreased from pre- to post-testing (%Δ = –0.425, $p = 0.035$, dz = 0.58), whereas H-H, N-H, and N-L did not change (all $p > 0.05$). Between-group comparisons at post-test indicated that N-H scored higher than H-L (%Δ = +0.561, $p = 0.005$, d = 1.60), with no other pairwise differences reaching significance.

Figure 8. pH decline change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of HCO3act %decline revealed a significant main effect of Time, $F(1, 36) = 5.25$, $p = 0.028$, $\eta p^2 = 0.127$; no main effect of Group, $F(3, 36) = 1.15$, $p = 0.341$; and a significant Time × Group interaction, $F(3, 36) = 4.55$, $p = 0.008$, $\eta p^2 = 0.275$ (Figure 9). Bonferroni-corrected simple-main-effect tests showed that only the H-L group decreased from pre- to post-

test (%Δ = -2.98, p = 0.023, dz = 0.60); remaining groups did not change (p > 0.05). No between-group differences were found at either time point (all p > 0.05).

Figure 9. HCO3act decline change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

No significant effects were found for pre-post 4' SaO2.

Contractile properties

Contractile properties assessed by tensiomyography—including contraction time (Tc), maximal displacement (Dm), half-relaxation time (Tr), delay time (Td), and sustain time (Ts)—for each group at pre- and post-intervention are presented in Table 6. Values are expressed as mean ± SD with 95 % confidence intervals.

Table 6. Descriptive statistics of experimental groups' tensiomyography outcomes

Group	Tc [ms] (M ± SD; 95 % CI)		Dm [mm] (M ± SD; 95 % CI)		Tr [ms] (M ± SD; 95 % CI)		Td [ms] (M ± SD; 95 % CI)		Ts [ms] (M ± SD; 95 % CI)	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST
H-H	28.6 ± 1.95; (27.3; 30.0)	28.7 ± 1.93; (27.3; 30.1)	7.70 ± 0.57; (7.29; 8.11)	7.18 ± 0.36; (6.92; 7.43)	72.4 ± 6.3; (66.2; 78.6)	75.1 ± 5.9; (69.5; 80.7)	21.8 ± 2.7; (19.1–24.5)	23.2 ± 2.9; (20.2–26.2)	131.8 ± 14.7; (117.4–146.2)	128.4 ± 15.1; (124.0–152.8)
H-L	30.50 ± 3.67; (27.9; 33.1)	30.45 ± 3.84; (27.7; 33.2)	7.17 ± 0.56; (6.77; 7.57)	7.35 ± 0.35; (7.10; 7.60)	65.2 ± 4.8; (60.6; 69.8)	66.3 ± 4.6; (61.9; 70.7)	19.8 ± 2.1; (17.6–22.0)	20.7 ± 2.3; (18.1–23.3)	122.4 ± 9.6; (112.8-132.0)	121.7 ± 10.2; (111.6–131.8)
N-H	32.60 ± 2.75; (30.6; 34.6)	33.45 ± 3.14; (31.2; 35.7)	7.61 ± 0.23; (7.44; 7.77)	7.42 ± 0.09; (7.35; 7.48)	67.8 ± 4.2; (63.8; 71.8)	68.9 ± 4.0; (65.1; 72.7)	23.9 ± 2.8; (20.6–27.2)	24.1 ± 2.6; (21.0–27.2)	139.7 ± 11.4; (128.3–151.1)	138.2 ± 11.8; (126.5–150.0)
N-L	31.15 ± 3.96; (28.3; 34.0)	31.20 ± 3.90; (28.4; 34.0)	7.39 ± 0.30; (7.18; 7.60)	7.59 ± 0.13; (7.50; 7.68)	69.5 ± 5.3; (64.0; 75.0)	70.2 ± 5.1; (65.0; 75.4)	22.4 ± 2.9; (19.0–25.8)	21.9 ± 3.2; (18.1–25.7)	131.5 ± 12.6; (119.2–143.8)	132.8 ± 13.2; (120.7–144.9)

Results presented as means with standard deviation. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia, Dm – maximal displacement, Tc – contraction time, Tr – relaxation time, Td – delay time, Ts – sustain time.

Analysis of Tc revealed no significant main effect of Time, $F(1,36) = 3.75$, $p = 0.061$; a significant main effect of Group, $F(3,36) = 3.09$, $p = 0.039$, $\eta p^2 = 0.205$; and a significant Time × Group interaction, $F(3,36) = 3.01$, $p = 0.043$, $\eta p^2 = 0.201$ (Figure 10). Bonferroni-corrected

paired comparisons showed a significant pre-to-post increase only in the N-H group ($\Delta = +0.8$, $p = 0.021$, $dz = 0.59$), whereas the other groups did not change (all $p > 0.05$). No significant between-group differences were observed at either the pre or post-test (all $p > 0.05$).

Figure 10. Time of contraction change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of Dm revealed no main effect of Group, $F(1.38) = 0.67$, $p = 0.264$; NO significant main effect of Time, $F(1, 83) = 6.21$, $p = 0.184$; and a significant Time × Group interaction, $F(3, 36) = 5.43$, $p = 0.001$ $\eta_p^2 = 0.401$ (Figure 11). Bonferroni-corrected paired comparisons of the simple Time effect within each group showed a significant decrease only in H-H ($\Delta = -0.52$, $p = 0.004$, $dz = 0.69$). Between-group contrasts at post-test indicated that N-L scored higher than H-H ($\Delta = +0.42$, $p = 0.029$; $d = 0.52$), while no other pairwise differences reached significance (all $p > 0.05$).

Figure 11. Muscle displacement change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

For the remaining TMG variables, no significant main effects or interactions were observed (all $p > 0.05$).

Discussion

This study is among the first to investigate how normobaric hypoxia and resistance-training intensity jointly affect performance metrics in elite ice-hockey players. Our results reveal a nuanced interplay between oxygen availability and training load, yielding distinct physiological adaptations. Crucially, hypoxic resistance training does not uniformly enhance all performance outcomes; rather, it produces specific, intensity-dependent adaptations.

Strength and neuromuscular adaptations

All training groups improved leg press strength after 6 weeks, but training intensity was the dominant driver of strength gains. High-intensity resistance training (70–80% 1RM) in normoxia (N-H) produced significant 1RM increases, as expected from heavy load training in well-trained athletes. Notably, adding hypoxia to high-load training (H-H) did not further amplify maximal strength gains compared to normoxic training. This aligns with prior studies in both untrained and trained populations showing no significant additive effect of hypoxic exposure on 1RM strength when training load is already high [19, 25]. A recent meta-analysis likewise found overall similar 1RM improvements for resistance training in hypoxia vs. normoxia (SMD ~0.13), with no clear advantage of hypoxia on maximal strength in trained subjects [19]. These findings suggest that for developing maximal force, traditional heavy training provides the primary stimulus, and systemic hypoxia offers little extra benefit in well-trained individuals – likely because heavy loads already fully recruit high-threshold motor units and induce substantial neuromuscular adaptation [26].

The hypoxic low-load group (H-L) exhibited only modest increases in maximal strength, whereas the normoxic low-load group (N-L) showed minimal change. This pattern suggests that the added metabolic stress of hypoxia cannot fully compensate for the deficit in mechanical load at low intensities. While previous studies in untrained populations reported that low-load training under hypoxia can induce some strength gains relative to no training [23], our findings align with the broader evidence that robust neuromuscular adaptations require sufficiently heavy loading. Hypoxic exposure alone, despite enhancing metabolic stress and hypertrophic signaling, does not replicate the neural recruitment and connective tissue stimulus provided by high-load lifting. Accordingly, our data concur with recommendations that greater training loads are essential to reap the full benefits of hypoxic training [8]. From a practical perspective, low-load hypoxic resistance exercise may still serve as a useful adjunct when heavy training is contraindicated or impossible. In scenarios such as injury rehabilitation

or in-season periods of reduced loading, exercising under hypoxia could help athletes preserve muscle function and partial strength gains while minimizing musculoskeletal strain. However, this approach should be viewed as a temporary compromise rather than a replacement for high-load training. Ultimately, for maximal strength development, traditional heavy-load protocols remain irreplaceable, with hypoxic training providing only a supplementary stimulus when full mechanical loading cannot be applied.

Beyond maximal strength, we observed neuromuscular functional changes via TMG. All groups showed some adaptation in muscle contractile properties, but training intensity again had greater effects than hypoxia. High-intensity training (especially H-H) tended to increase peak rate of force development and induce small shifts in TMG indices consistent with muscle hypertrophy (e.g. reduced muscle belly displacement, indicating greater muscle stiffness). Low-intensity normoxic training caused minimal changes in contractile parameters, but low-load hypoxic training did produce moderate improvements, presumably by engaging fast-twitch fibers through metabolic fatigue. Although between-group differences in TMG measures were not large, the pattern suggests hypoxia can enhance neuromuscular function under light loads. Interestingly, a prior study on circuit training in hypoxia noted similar neural adaptation (H-reflex, EMG) in hypoxic vs normoxic conditions for resistance-trained athletes, reinforcing that hypoxia does not hinder neural gains [25]. In studied population, H-H training preserved explosive neuromuscular performance as well as N-H training did. There was no evidence that hypoxia impaired neural drive; if anything, the heightened effort in hypoxia could increase neural stress acutely. Indeed, short-term hypoxic training has been shown not to dampen neural function or reflex responses in trained lifters [25]. In sum, the interaction between hypoxia and training intensity for strength-related adaptations appears synergistic only at low loads. Hypoxia can compensate for low mechanical tension by boosting neural activation

and metabolic stimuli, but when loads are heavy, the ceiling for neural recruitment is already reached, yielding comparable outcomes in hypoxia vs normoxia.

Anaerobic performance and lactate kinetics

A key finding was that anaerobic performance (Wingate test) and lactate kinetics diverged across the four interventions. Wingate peak power—an index of alactic/anaerobic capacity—increased in every group except the low-intensity normoxia (N-L) condition, with the largest gains occurring under a strong anaerobic stimulus (high load, hypoxia, or both). High-intensity normoxic training (N-H) boosted peak power, likely via enhanced muscle strength and neuromuscular coordination during the 30-s sprint. Adding hypoxia amplified these effects: the high-load hypoxia group (H-H) showed the greatest improvement, indicating an additive benefit of low O_2 and heavy loads. Even the low-load hypoxia group (H-L) achieved modest gains, whereas N-L did not—underscoring hypoxia's role in potentiating otherwise insufficient stimuli. These results align with emerging evidence that hypoxic training enhances single-bout anaerobic output; for example, four weeks of FiO_2 ~14–15% interval training raised Wingate peak power—a change not seen in normoxia [27].

Hypoxic exposure appears to stimulate greater anaerobic enzyme activity and fast-twitch fiber engagement, bolstering the phosphagen and glycolytic contribution to all-out efforts [12, 28, 29]. In our study, the H-H condition – effectively a "train high (intensity) & high (altitude)" approach – provided both maximal mechanical and metabolic stress, yielding superior peak power gains. In contrast, purely high-load normoxic training improved power mainly via strength gains, and purely hypoxic low-load training did so via metabolic adaptations; combining both stimuli (H-H) offered no compromise in power development. Importantly, none of the hypoxic protocols detracted from power output. Concerns that training in low O_2 might blunt high-power performance (due to acute fatigue or reduced training quality) were not borne out – partipicants did not exhibit any decrement in post-training peak

power in hypoxic groups, consistent with reports that adding hypoxia does not hinder gains in sprint or power measures in athletes mentioned before.

Mean power and power decline during the Wingate (indicators of anaerobic endurance) showed a slightly different pattern. Here, training intensity alone was less effective; the N-H group's improvements in sustaining power over 30 s were modest. Intriguingly, the hypoxia-trained groups demonstrated better lactate tolerance and power maintenance. The H-H and H-L conditions both led to faster post-exercise lactate clearance and a smaller drop-off in power in the later stages of the Wingate, suggesting enhanced anaerobic endurance. Mechanistically, chronic hypoxic training likely induced higher glycolytic flux and acid stress in each session, stimulating adaptations in buffering capacity and lactate handling [12]. Research indicates that intermittent hypoxic training augments glycolytic enzyme levels [30] and muscle buffer capacity [31], which can delay fatigue in maximal exercise. Repeated exposure to hypoxia in our protocol would have increased muscle and blood lactate during training sessions - as seen acutely in IHRT studies [32] in turn triggering adaptive increases in lactate transporters and buffering compounds. Although we did not directly measure muscle buffer capacity, the significantly lower drop in blood lactate after Wingate in hypoxia-trained players (particularly H-H) supports this notion. There is similar evidence by [12] where post wingate ΔpH values after hypoxia training program has proportional decline. Our findings also agree with [33] who reported improved repeated-sprint fatigue resistance after hypoxic training, attributable to increased buffering and anaerobic energy contribution.

Practically, hypoxic resistance workouts may modestly mimic sprint-interval training by enhancing an athlete's capacity for sustained high-intensity efforts and rapid recovery. For intermittent-sport athletes (e.g., hockey forwards performing repeated shifts), improved lactate clearance and power maintenance in hypoxia-trained groups could translate into superior repeat-sprint ability on ice. Notably, low-intensity normoxic training (N-L) alone failed to

484 significantly affect anaerobic power or lactate kinetics—underscoring that sufficient load,
485 hypoxic stress, or both are required to provoke anaerobic adaptations. In summary, combining
486 hypoxia with resistance exercise intensifies the metabolic stimulus, boosting peak anaerobic
487 power and the mechanisms of lactate removal and tolerance.

488 ***Hypoxia–intensity interactions***

489 When lifting heavy loads (≥70% 1RM), the stimulus for myofibrillar adaptation is
490 already maximal in normoxia – all motor units are engaged and mechanical tension on fibers
491 is high. Hypoxia in this context adds metabolic strain but does not markedly increase motor
492 unit recruitment (since it's already near-maximal) nor mechanical load, so the incremental
493 gains in strength or hypertrophy are small [25]. This explains why H-H did not significantly
494 outperform N-H in pure strength metrics. However, even in high-load training, hypoxia can
495 steer adaptations toward the metabolic side: e.g. slightly more hypertrophy of type II fibers or
496 improved muscular endurance, as hinted by our finding of H-H showing the greatest lactate
497 clearance improvement.

498 On the other hand, at low loads, normoxic training provides mainly an endurance
499 stimulus with little hypertrophic effect – many high-threshold fibers remain untaxed due to the
500 load being below their recruitment threshold. Introducing hypoxia in low-load training changes
501 this calculus by causing premature fatigue of active fibers and forcing the nervous system to
502 recruit larger motor units to maintain force output [32]. Effectively, hypoxia raises the relative
503 intensity of effort for a given load. The end result is that H-L training can induce muscle fiber
504 adaptations somewhat akin to higher-load training (as evidenced by the 11–15% strength gains
505 in athletes with low-load hypoxic training) [23].

506 In simplistic terms, hypoxia acted as an "intensity amplifier" for low-load training, but
507 as an "additional strain" for high-load training. There may also be a ceiling to tolerable
508 metabolic stress – in H-H sessions, athletes were training near their strength limits and in low

O_2, which is extremely demanding. Although our athletes completed all sessions, it is conceivable that hypoxia could heighten fatigue to the point of limiting training volume or quality for some individuals. Our design controlled for volume (equal sets/reps in all groups), so H-H athletes ultimately achieved similar volume as N-H, just at higher perceived effort. This suggests well-trained athletes can tolerate combined hypoxic + high-load stimuli without detriment, but coaches should monitor recovery carefully if employing such methods.

Overall, the interaction between hypoxia and intensity was complementary up to a point. We found no evidence of negative interference; rather, hypoxia mostly potentiated adaptations, especially for submaximal loads. Where no extra benefit was seen (strength 1RM in heavy training), it is likely because normoxic training already elicited near-maximal adaptation. The current literature echoes this interpretation: several studies report no "double counting" of gains when hypoxia is added to a robust resistance program, yet they acknowledge hypoxia's efficacy in scenarios of reduced load or volume. Coaches should therefore consider the specific goal: if maximal strength/power is the sole aim, traditional heavy training at sea-level suffices. But if one seeks to gain ancillary adaptations (e.g. hypertrophy, local muscular endurance) or must use lighter loads, hypoxic sessions can provide an extra adaptive boost.

Limitations

Despite its methodological strengths, this study has several limitations. First, the modest sample size (n = 40; 10 per group), though powered for primary outcomes, may have limited detection of subtle between-group interactions and reduces generalizability—larger cohorts could clarify non-significant trends such as strength differences. Second, the six-week intervention, sufficient for neuromuscular adaptations, may have been too brief to elicit full hypertrophic or enzymatic changes; longer follow-ups would determine whether hypoxic benefits persist or normoxic training eventually matches them. Third, our exclusively young, highly trained male ice-hockey players limit applicability to other populations (e.g., untrained

individuals or female athletes), whose responses to hypoxia and load may differ. Fourth, we tested only leg-press exercise at ~3,500 m equivalent hypoxia, leaving uncertain whether findings extend to other movements, sports-specific activities, or alternative hypoxic doses. Fifth, although we report performance and lactate kinetics, we did not assess molecular or histological markers (e.g., HIF-1α expression), so mechanistic inferences remain speculative; future work should include direct measures of cellular pathways. Finally, validated laboratory tests captured key performance metrics but lacked on-ice, sport-specific assessments, which would strengthen practical relevance. Future research should investigate long-term adaptations and incorporate sports-specific performance metrics such as skating speed, number of shifts won, and on-ice recovery.

Conclusions

Crucially, hypoxic resistance training does not uniformly enhance all performance outcomes; rather, it produces specific, intensity-dependent adaptations. The intensity of resistance training elicits a superior training response to hypoxia, enhancing power in ice hockey players.

References

Abbreviations

1RM One-repetition maximum

ANOVA Analysis of variance

CI Confidence interval

CONSORT Consolidated Standards of Reporting Trials

Dm Maximal radial displacement

EMG Electromyography

FiO_2 Fraction of inspired oxygen

H-H High-intensity resistance training in hypoxia

H-L Low-intensity resistance training in hypoxia

HCO_3^-act Actual bicarbonate concentration

HIF-1α Hypoxia-inducible factor 1-alpha

HRT Hypoxic resistance training

IHRT Intermittent hypoxic resistance training

LA_4, LA_8 Blood lactate concentration 4 and 8 min post-exercise

LCR Lactate clearance rate

N-H High-intensity resistance training in normoxia

N-L Low-intensity resistance training in normoxia

pCO_2 Partial pressure of carbon dioxide

SaO_2 Arterial oxygen saturation

SD Standard deviation

SMD Standardized mean difference

Tc Contraction time

Td Delay time

TMG Tensiomyography

Tr Half-relaxation time

Ts Sustain time

W1, W2 First and second Wingate bout

Declarations

Ethics approval and consent to participate

The study was approved by the Bioethics Commission of the Academy of Physical Education in Katowice (approval no. 4-XI/2024) and was performed in accordance with the Declaration of Helsinki. All participants provided written informed consent prior to participation.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

AT, PS and RR conceived and designed the study; AT, KS and MŻ collected the data; AT and PS analysed and interpreted the data; AT and PS drafted the manuscript; RR supervised the project. All authors read and approved the final manuscript.

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Figures

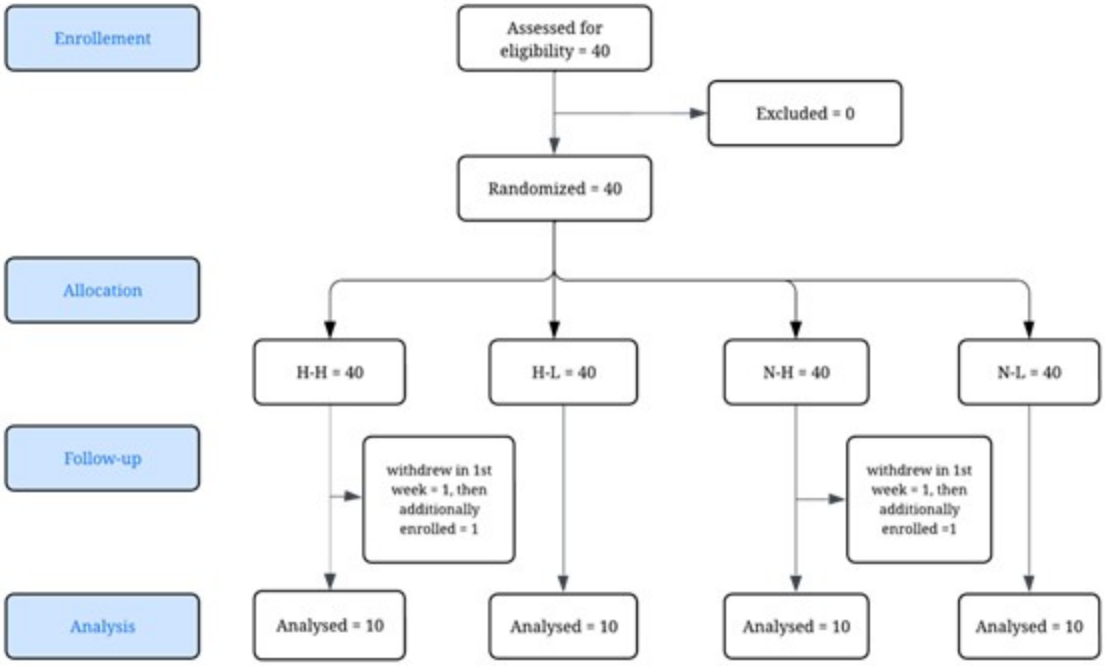


Figure 1

CONSORT flow diagram.

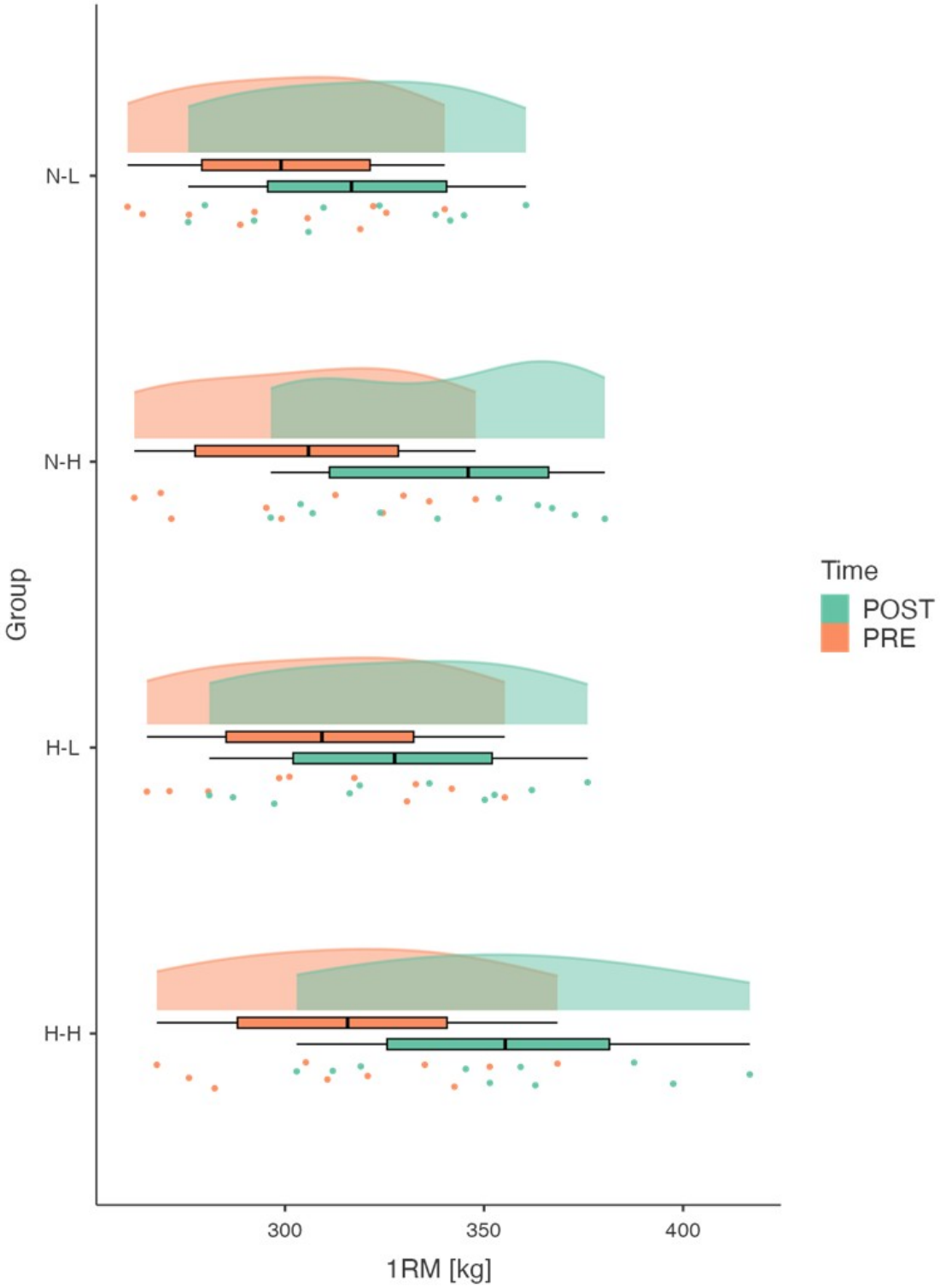


Figure 2

Leg press 1RM change, N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of Peak power showed a significant main effect of Time, $F(1, 36) = 17.95$, $p < 0.001$, $\eta p^2 = 0.333$; no main effect of Group, $F(3, 36) = 0.73$, $p = 0.541$; and a significant Time × Group interaction, $F(3, 36) = 6.34$, $p = 0.009$, $\eta p^2 = 0.271$ (Table 3). Bonferroni-corrected paired comparisons showed significant pre-to-post increases in two conditions—H-H ($\Delta = +126.1$, $p = 0.011$, $dz = 0.64$) and N-H ($\Delta = +122.59$, $p = 0.015$, $dz = 0.66$)—whereas the H-L ($\Delta = +30.56$, $p > 0.05$) and N-L ($\Delta = +26.00$, $p > 0.05$) groups showed non-significant changes. No between-group differences were observed at either the pre- or post-test (all $p > 0.05$).

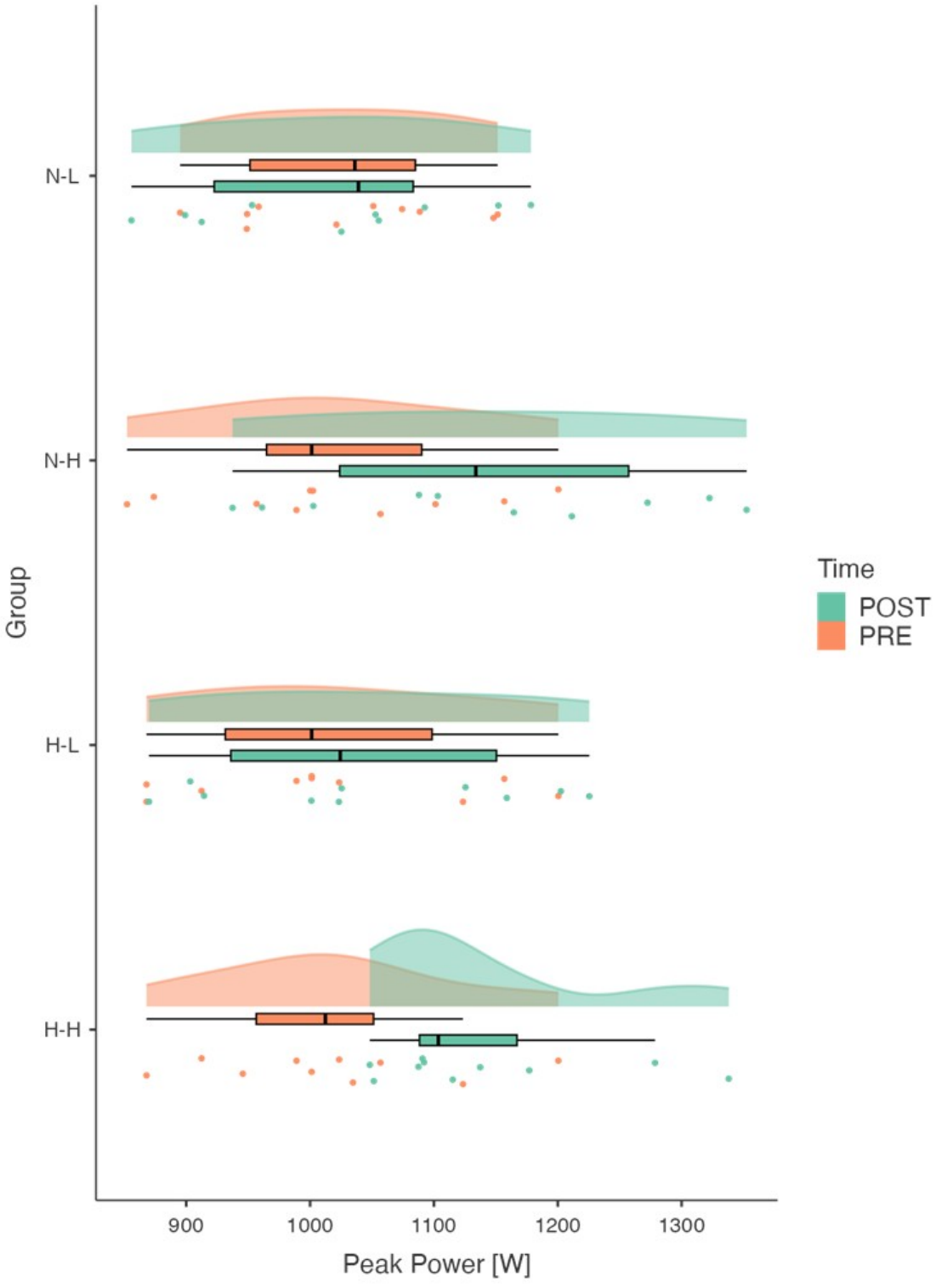


Figure 3

Wingate peak power change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of Time to peak power revealed a significant main effect of Time, $F(1,36) = 11.34$, $p = 0.002$, $\eta_p^2 = 0.239$; no main effect of Group, $F(3,36) = 0.40$, $p = 0.753$, $\eta_p^2 = 0.032$; and a significant Time × Group interaction, $F(3,36) = 5.11$, $p = 0.005$, $\eta_p^2 = 0.299$ (Table 3). Bonferroni-corrected post-hoc comparisons showed a significant pre-to-post decrease only in the N-H group ($\Delta = -0.248$, $p = 0.009$, $dz = 1.50$). The H-H group also decreased ($\%\Delta = -0.230$), a change that approached significance ($p = 0.063$), whereas H-L and N-L did not change (both $p > 0.05$). No between-group differences were observed at either time point (all $p > 0.05$).

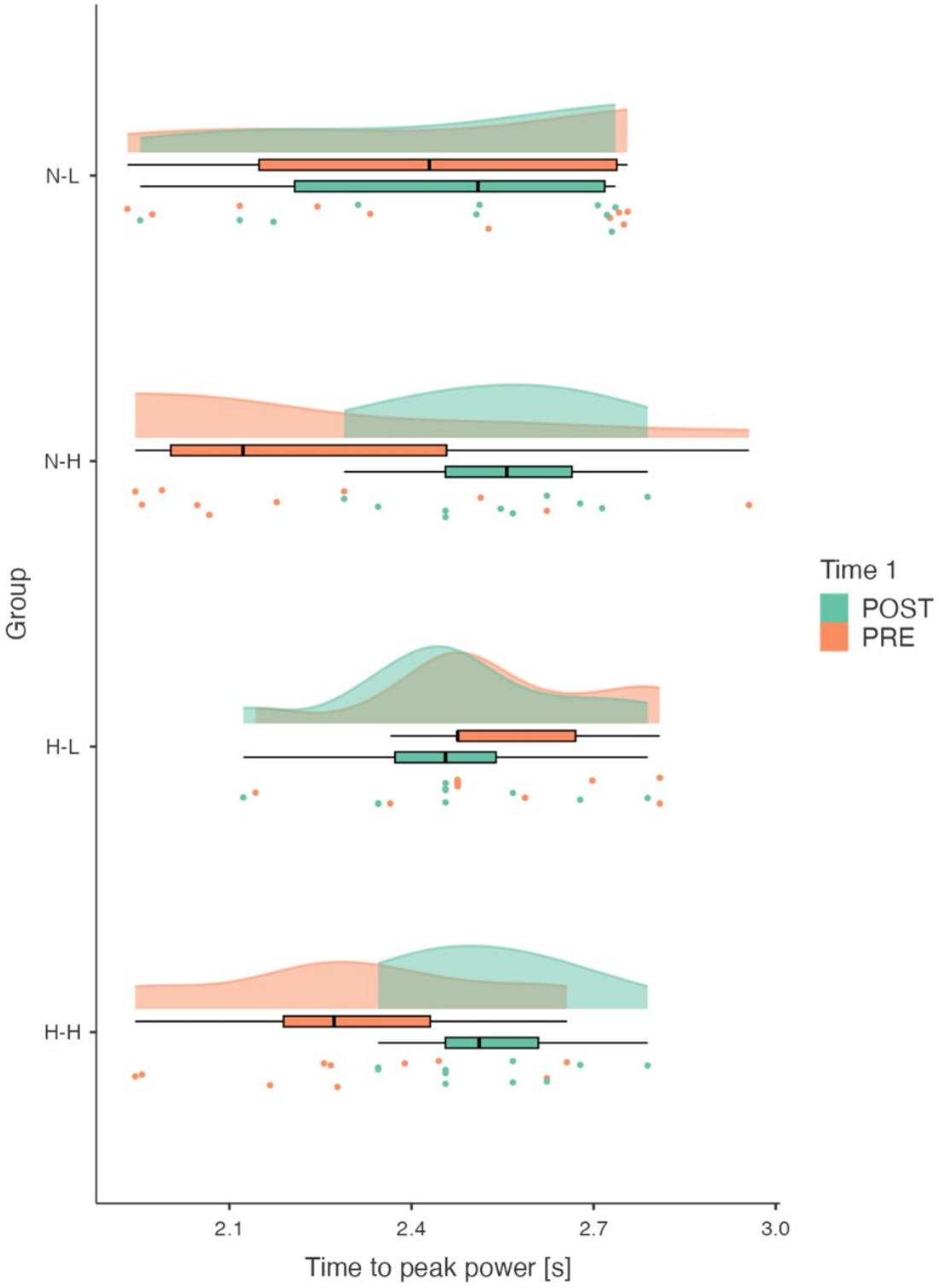


Figure 4

Wingate time to peak power change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Repeated-measures ANOVA on inter-bout Wingate peak power decline [%] revealed a significant main effect of Time, $F(1,36) = 42.35$, $p < 0.001$, $\eta_p^2 = 0.541$, and a significant Time × Group interaction, $F(3,36) = 7.63$, $p < 0.001$, $\eta_p^2 = 0.389$; and main effect of Group, $F(3,36) = 4.51$, $p = 0.009$, $\eta_p^2 = 0.273$ (Figure 5). Bonferroni-corrected paired comparisons showed that the H-H (%Δ = +2.74, $p < 0.001$, dz = 1.13) and N-H (%Δ = +2.54, $p = 0.002$, dz = 1.08) groups exhibited significantly greater declines in peak power from pre- to post-test, indicating increased fatigue across bouts. In contrast, H-L (%Δ = +0.84, $p = 1.000$) and N-L (%Δ = –0.06, $p = 1.000$) groups did not show significant changes. At post-test, the H-H group had a significantly higher power decline compared to N-L (%Δ = +2.80, $p = 0.001$, d = 0.96), and the N-H group also showed a higher decline compared to N-L (%Δ = +2.60, $p = 0.004$, d = 0.89); no other between-group differences were significant. No significant changes were found for inter-bout mean power decline [%].

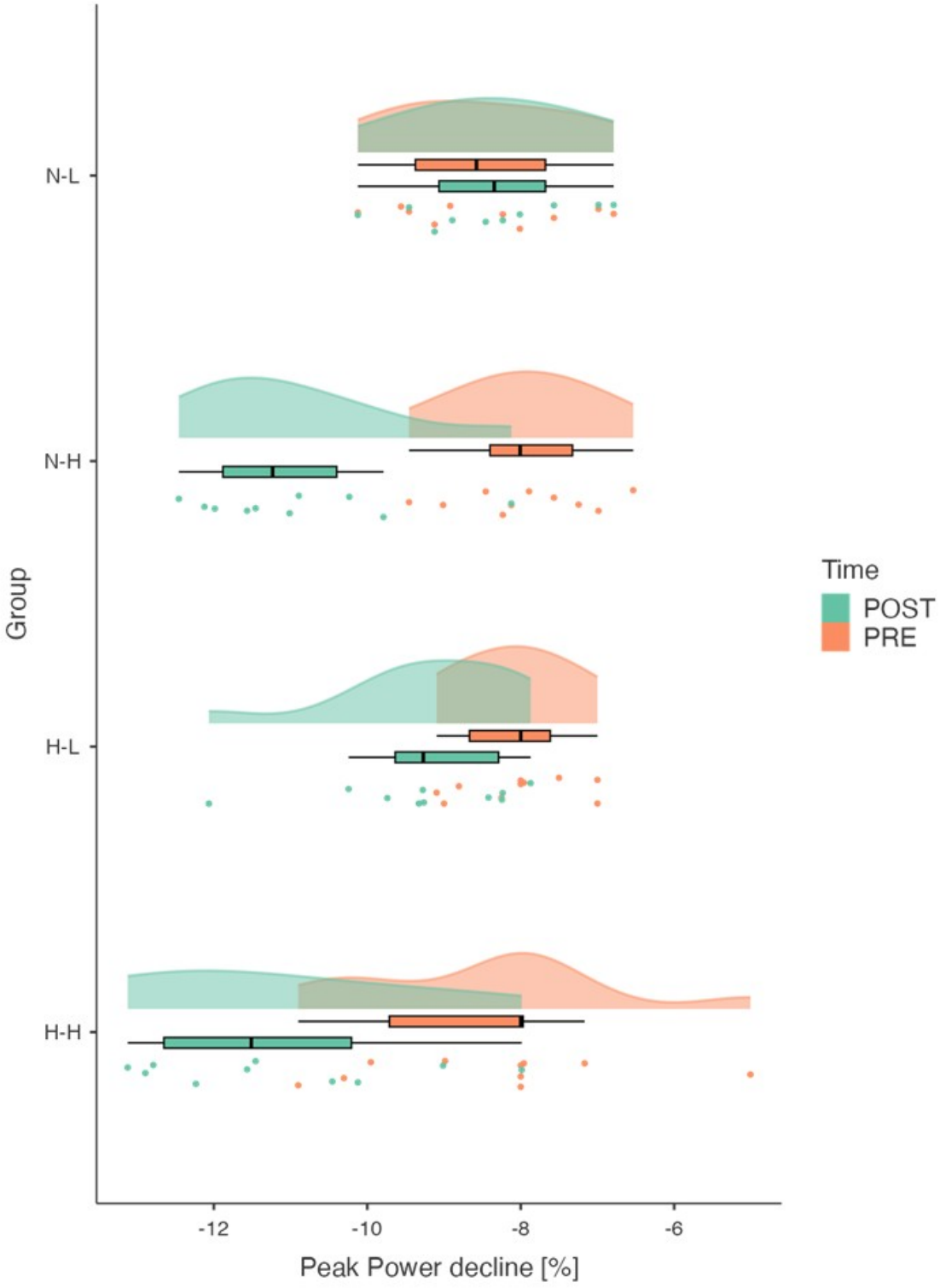


Figure 5

Wingate peak power decline change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Metabolic changes

Metabolic parameters—lactate concentration at 4 min (LA_4) and 8 min (LA_8), and lactate clearance rate (LCR)—for each group at pre- and post-intervention are summarized in Table 4. Values are presented as mean ± SD with 95 % confidence intervals.

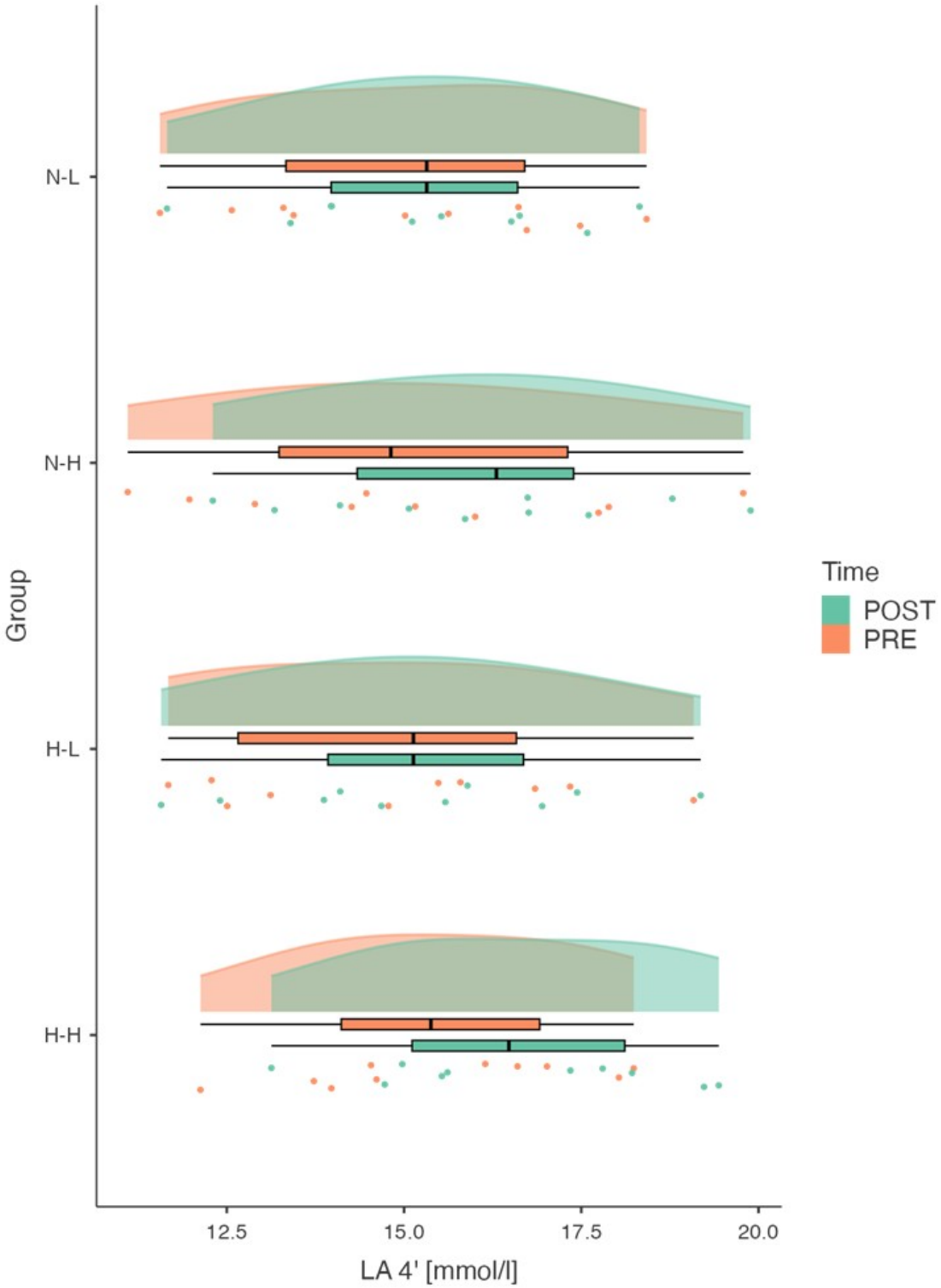


Figure 6

Peak lactate change, N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of Late-phase lactate clearance per minute rate showed a significant main effect of Time, $F(1,36) = 20.96$, $p < 0.001$, $\eta_p^2 = 0.368$; no main effect of Group, $F(3,36) = 0.02$, $p = 0.997$; and a significant Time × Group interaction, $F(3,36) = 3.99$, $p = 0.015$, $\eta_p^2 = 0.250$ (Table 4; Figure 7). Bonferroni-corrected simple-main-effect tests revealed that only the H-L group showed a significant pre-to-post increase (%Δ = −4.008, $p < 0.001$, dz = 1.61), whereas the H-H (%Δ = −0.986), N-H (Δ = −0.530), and N-L (Δ = −0.901) groups did not change significantly (all $p = 1.000$). No between-group differences were significant at either time point (all $p > .05$).

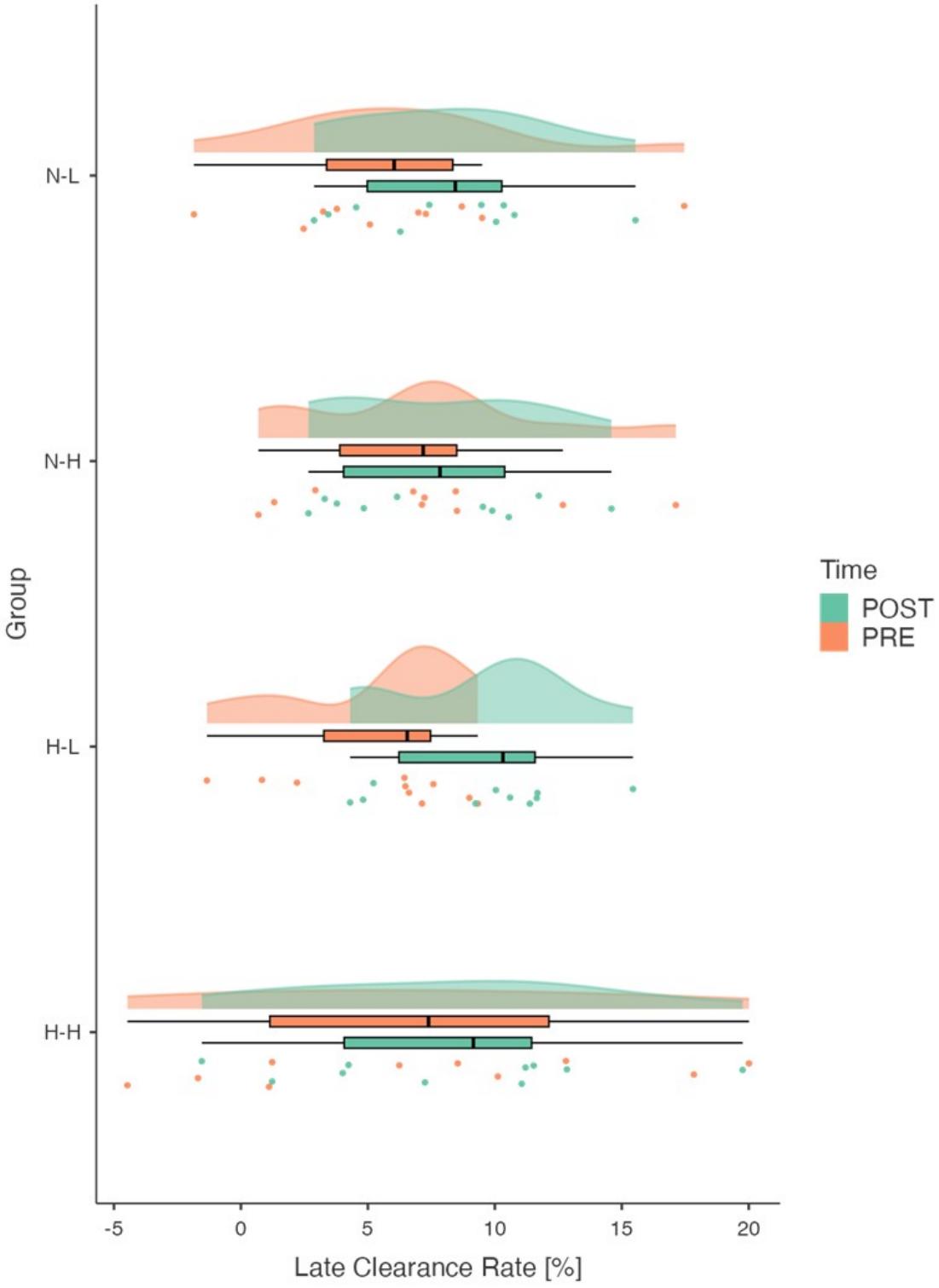


Figure 7

Late phase clearance rate change

Blood gas parameters—arterial pH and bicarbonate (HCO_3^-) measured at rest (0') and 4' post-exercise, and oxygen saturation (SaO_2) at rest and 4' post-exercise—are summarized for each group in Table 5. Values are presented as mean ± SD with 95 % confidence intervals.

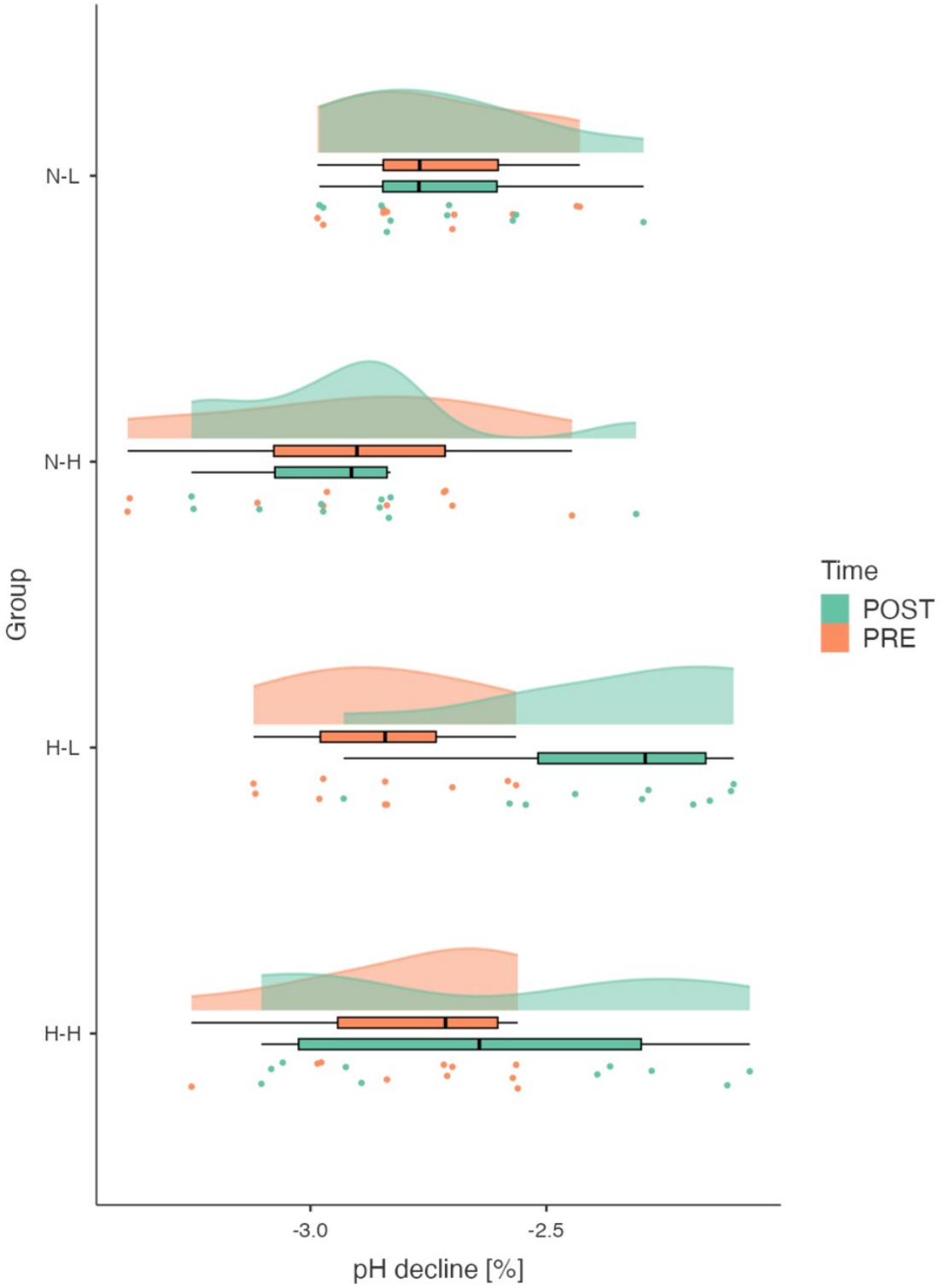


Figure 8

pH decline change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of HCO3act %decline revealed a significant main effect of Time, $F(1, 36) = 5.25$, $p = 0.028$, $\eta p^2 = 0.127$; no main effect of Group, $F(3, 36) = 1.15$, $p = 0.341$; and a significant Time × Group interaction, $F(3, 36) = 4.55$, $p = 0.008$, $\eta p^2 = 0.275$ (Figure 9). Bonferroni-corrected simple-main-effect tests showed that only the H-L group decreased from pre- to post-test (%Δ = -2.98, $p = 0.023$, $dz = 0.60$); remaining groups did not change ($p > 0.05$). No between-group differences were found at either time point (all $p > 0.05$).

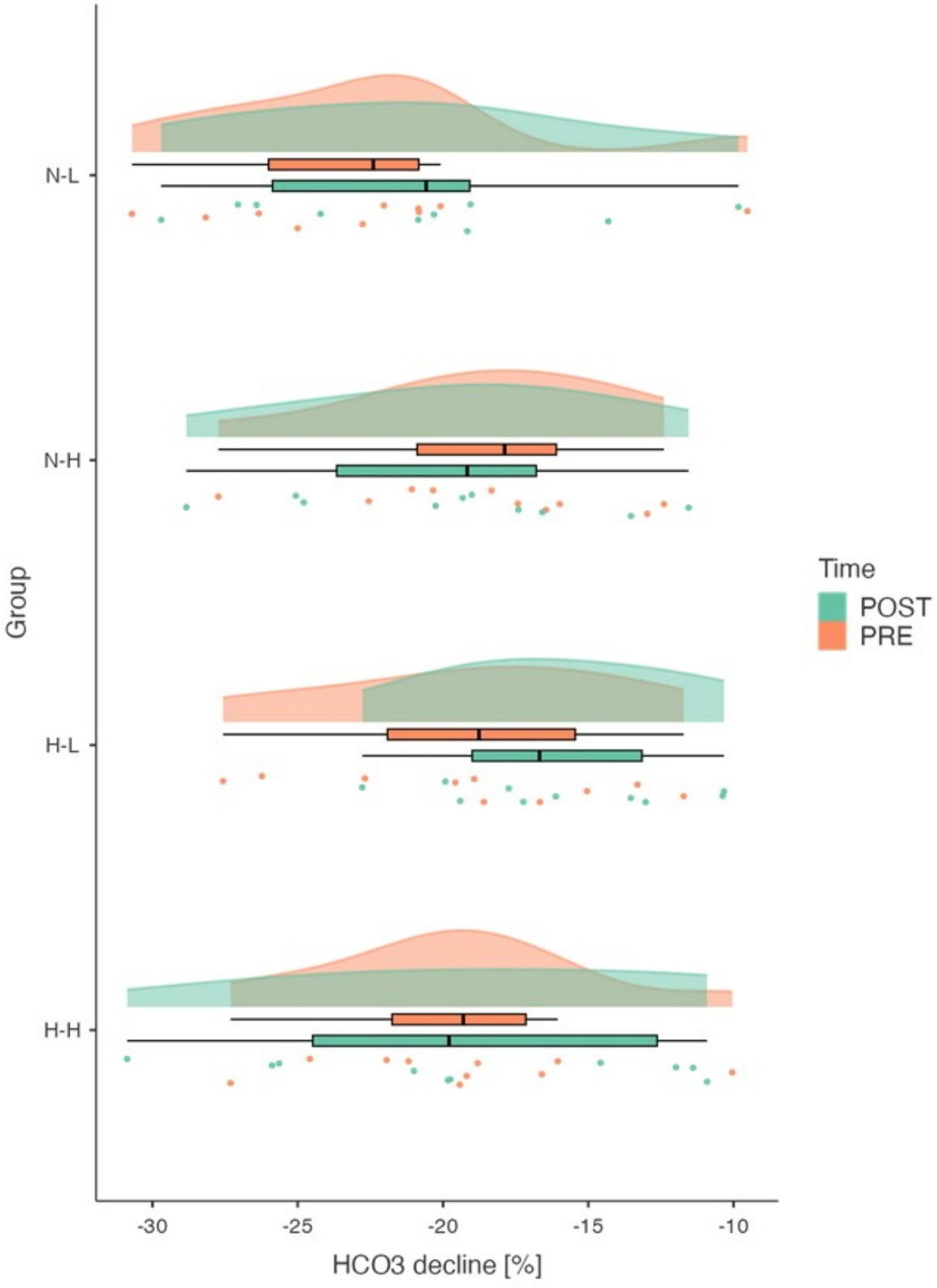


Figure 9

HCO3act decline change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

No significant effects were found for pre-post 4’ SaO2.

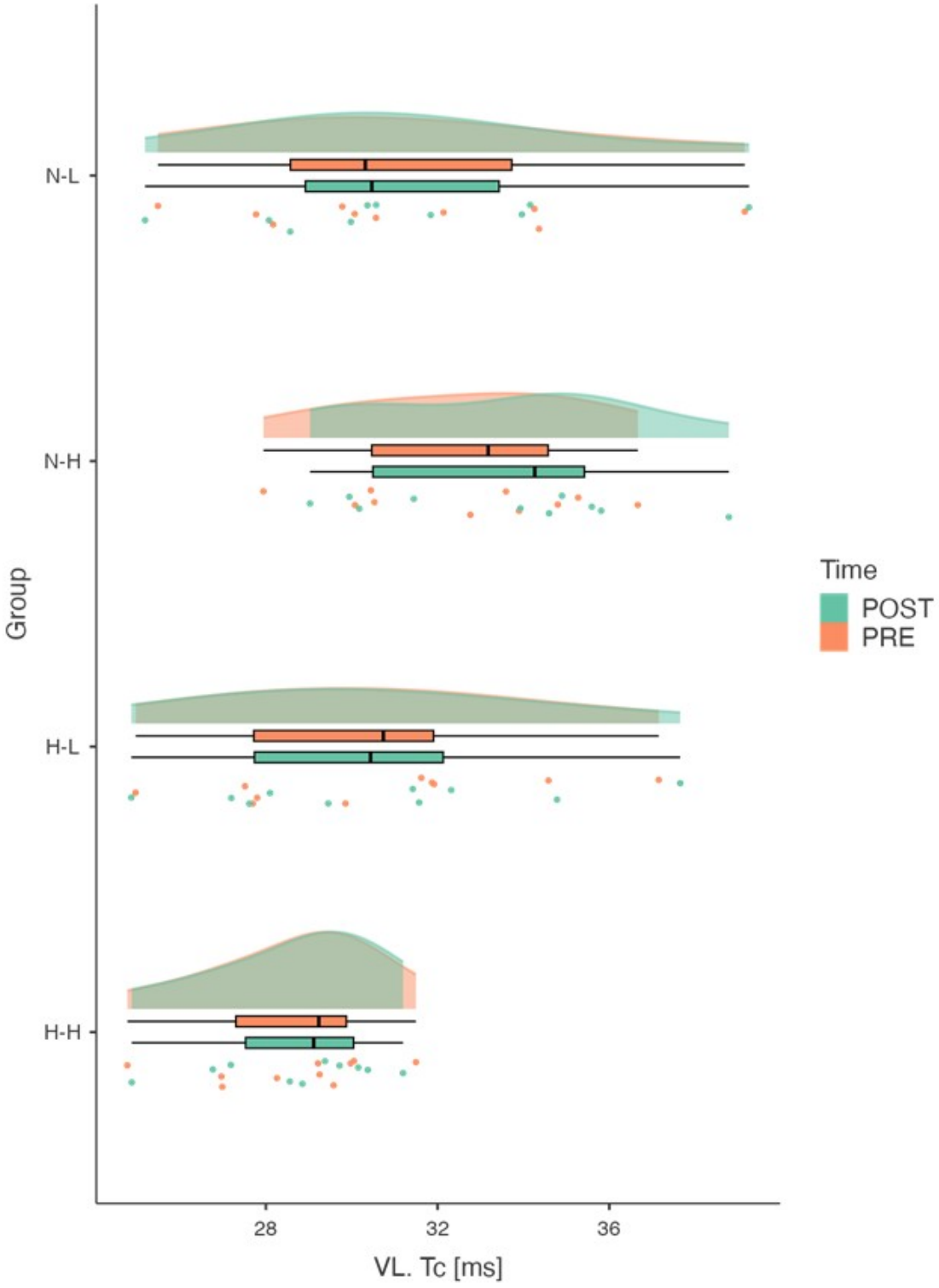


Figure 10

Time of contraction change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

Analysis of Dm revealed no main effect of Group, $F(1.38) = 0.67$, $p = 0.264$; NO significant main effect of Time, $F(1, 83) = 6.21$, $p = 0.184$; and a significant Time × Group interaction, $F(3, 36) = 5.43$, $p = 0.001$ $\eta_p^2 = 0.401$ (Figure 11). Bonferroni-corrected paired comparisons of the simple Time effect within each group showed a significant decrease only in H-H ($\Delta = -0.52$, $p = 0.004$, $dz = 0.69$). Between-group contrasts at post-test indicated that N-L scored higher than H-H ($\Delta = +0.42$, $p = 0.029$; $d = 0.52$), while no other pairwise differences reached significance (all $p > 0.05$).

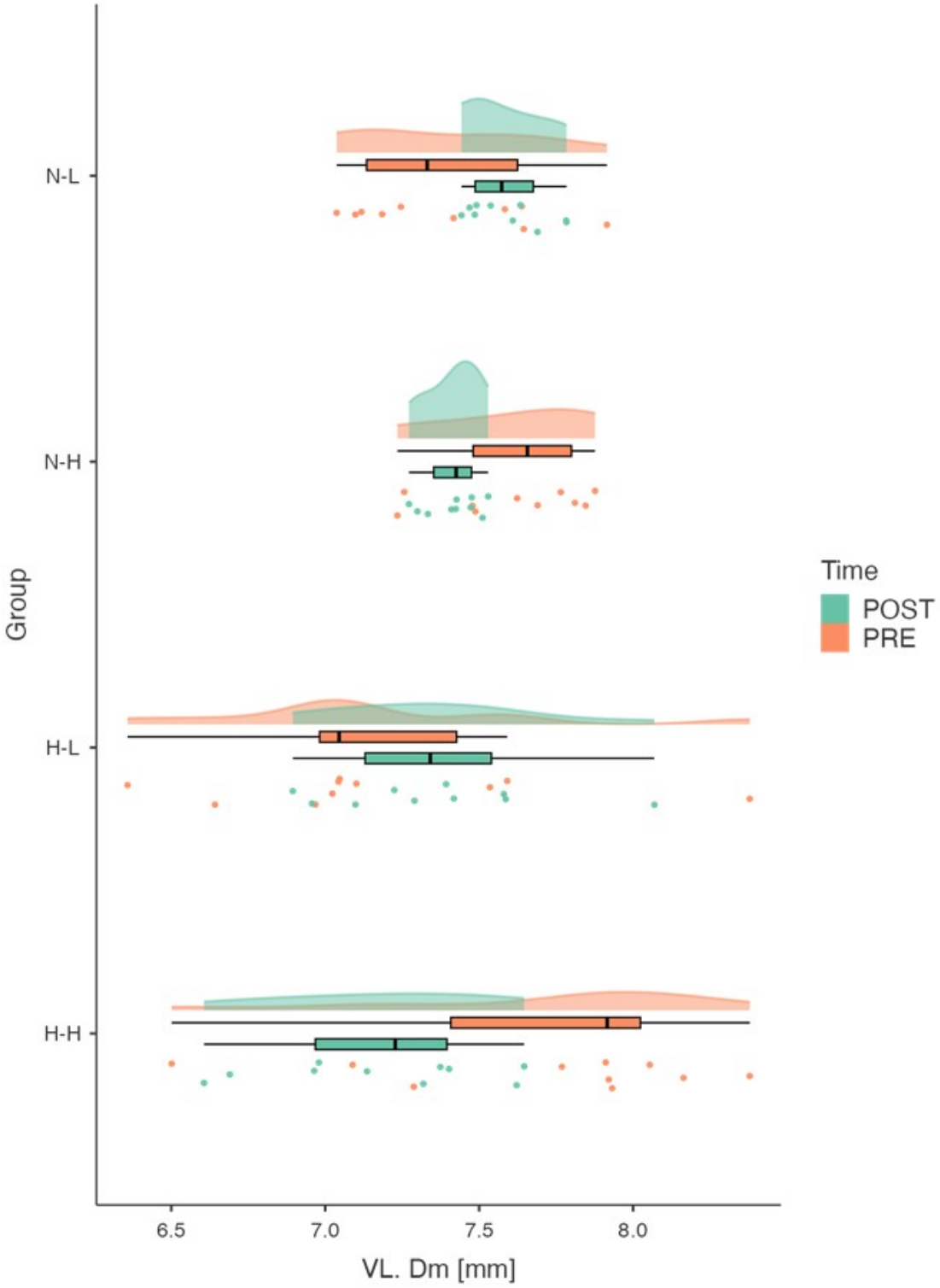


Figure 11

Muscle displacement change. N-L - low-intensity resistance training in normoxia, N-H - high-intensity resistance training in normoxia, H-L - low-intensity resistance training in hypoxia and H-H - high-intensity resistance training in hypoxia;

For the remaining TMG variables, no significant main effects or interactions were observed (all $p > 0.05$).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- CoverletterBMC.docx
- Dataset.xlsx
- CONSORTChecklistHypoxia.docx