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Original Article

Impact of progressive hypoxic exposure, with or without exercise, on muscle and peripheral oxygen saturation in athletes

MEILING ZHENG[1], APIWAN MANIMMANAKORN[2*], QINSHAN HUANG[3], WEERAPON SANGARTIT[4], NUTTASET MANIMMANAKORN[5], GUANG YANG[6], BENJAMAS PRATHANEE[7], MICHAEL JOHN HAMLIN[8]

[1] Exercise and Sport Sciences Program, Multidisciplinary Program, Graduate School, Khon Kaen University, THAILAND
[1,2,3] Tianfu School of Tsinghua University High School, CHINA
[2,4] Department of Physiology, Faculty of Medicine, Khon Kaen University, THAILAND
[3] College of Sports and Arts, JiangXi University of Science and Technology, CHINA
[5] Department of Rehabilitation Medicine, Faculty of Medicine, Khon Kaen University, THAILAND
[6] Henan Kaifeng College of Science Technology and Communication, School of Physical Education, CHINA
[7] Mekong Health Science Research Institute, Department Otorhinolaryngology, Faculty of Medicine, Khon Kaen University, THAILAND
[8] Department of Tourism, Sport and Society, Lincoln University, NEW ZEALAND

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Abstract:
Introduction: Hypoxic exercise is increasingly used to enhance training adaptations and mitigate injury risk. However, it is unclear whether peripheral capillary oxygen saturation (SpO2) accurately reflects muscle oxygen saturation (SmO2) in proportion during rest and resistance exercise. Objective: To examine acute SmO2 and SpO2 responses to different levels of hypoxia during rest and low-load resistance exercise (LRE) in athletes. Methods: Eighteen male team-sport athletes (20.44 ± 1.25 years) participated in the study. In a randomized crossover design, participants were exposed to five fractions of inspired oxygen (FiO2: 21.0%, 17.2%, 16.2%, 14.8%, 13.6%) under resting (HP) and exercise (HE) conditions, with a 3-day washout period. The exercise protocol involved knee extensions performed at 50% of the one-repetition maximum (50% 1RM). Key physiological metrics, including SmO_2, SpO2, heart rate (HR), and total hemoglobin (tHb), were continuously monitored. Results: During exercise, SmO_2 declined progressively as FiO_2 decreased, with significant reductions observed across all hypoxic levels compared to normoxia ($p < 0.05$). SpO_2 decreased significantly at FiO_2 levels of 16.2%, 14.8%, and 13.6% (all $p < 0.05$), with the most pronounced drop at 13.6%. A strong positive correlation between muscle and peripheral oxygen saturation changes was found during exercise ($r = 0.974$, $p = 0.005$), but not at rest ($r = 0.763$, $p = 0.133$). Interestingly, during exercise, the percentage changes in HE SmO_2 and HE SpO_2 across sequential hypoxic level transitions (Δlevel 1 to Δlevel 5), the reduction in HE SmO_2 was significantly greater than that in HE SpO_2 (paired t-tests: all $P \leq 0.010$). Discussion: This discrepancy is likely driven by localized physiological mechanisms, such as contraction-induced mechanical occlusion impeding regional blood flow and the slower temporal response of pulse oximetry. While SpO2 provides a proxy for systemic oxygen delivery, the localized metabolic demand and vascular autoregulation within the active skeletal muscle dictate intramuscular oxygen availability, thereby decoupling local deoxygenation from systemic trends. Conclusions: SpO_2 reductions are neither equivalent to nor proportional to SmO_2 during exercise. Systemic oxygenation alone fails to accurately reflect the severity of localized muscle deoxygenation under hypoxic conditions. Therefore, optimizing hypoxic training requires monitoring both systemic and local oxygenation to ensure accurate assessment of muscular hypoxic stress.
Keywords: Normobaric hypoxia; Pulse oximetry; Knee extension; Near-infrared spectroscopy; Muscle deoxygenation

Introduction

Extensive research demonstrates that hypoxic training, particularly when combined with low-load resistance exercise (LRE), serves as a potent stimulus for enhancing muscular hypertrophy and maximal strength (Benavente et al., 2023; La-bantao et al., 2025; Manimmanakorn et al., 2013). These hypertrophic adaptations are primarily attributed to the synergistic effect of mechanical tension and exacerbated metabolic stress, which are particularly pronounced under normobaric hypoxic conditions with a fraction of inspired oxygen (FiO_2) ranging between 13.6% and 15.8% (Namboonlue et al., 2020; Thuwakum et al., 2017). In both clinical and athletic performance settings, peripheral capillary oxygen saturation (SpO_2) is the most frequently utilized metric to quantify systemic physiological stress and titrate the hypoxic dose (Goods et al., 2014). However, while SpO_2

Corresponding Author: APIWAN MANIMMANAKORN, E-mail: mapiwa@kku.ac.th

provides a non-invasive reflection of arterial hemoglobin oxygenation, it remains an indirect measure that may fail to capture the complex oxygen dynamics occurring within the active musculature or muscle oxygenation (SmO_2).

For team-sport athletes, where success in skills such as short-distance running, agility, and jump height is underpinned by muscular strength and mass, focusing on muscle oxygen saturation (SmO_2) provides a more direct measurement of the hypoxic stress within the working muscle (Perrey & Ferrari, 2018). Monitoring SmO_2 via Near-Infrared Spectroscopy (NIRS) is a well-established technique for assessing the balance between oxygen delivery and utilization in the microvasculature, reflected by changes in oxy- and deoxy-hemoglobin concentrations (Ferrari et al., 2004). Indeed, comparative studies have shown that transcutaneous NIRS detects the onset of hypoxia significantly earlier (approximately 5.3 seconds) compared to pulse oximetry (approximately 14.9 seconds), highlighting its superior sensitivity to rapid oxygenation changes (Cheung et al., 2022). This is particularly relevant during high-intensity efforts, where NIRS provides valuable insights into localized muscle deoxygenation patterns that systemic measures cannot capture (Simpson et al., 2025). By measuring the oxygenation state of heme groups in both hemoglobin and myoglobin (Perrey & Ferrari, 2018), NIRS provides a direct window into the metabolic milieu that drives downstream adaptations. Despite this high resolution, the adoption of NIRS in applied sports science is often hindered by the high cost of equipment, technical requirements for data interpretation, and sensitivity to sensor placement. Conversely, SpO_2 via pulse oximetry offers a low-cost, highly portable alternative. Consequently, establishing whether SpO_2 can serve as a valid proxy for SmO_2 during various states of exertion is a question of significant practical and translational value. The physiological rationale for a potential dissociation between SpO_2 and SmO_2 is rooted in the multifaceted nature of the circulatory response to hypoxia. Unlike the systemic arterial pool, the microcirculatory environment of active skeletal muscle is governed by local regulatory mechanisms, including metabolic vasodilation, pressure autoregulation, and Bohr effect mediated shifts in the oxyhemoglobin dissociation curve (Calbet et al., 2009; Faiss et al., 2013). During resistance exercise, intramuscular pressure can further impede local perfusion, potentially decoupling local deoxygenation kinetics from systemic arterial saturation. This dissociation is evident even when systemic cardiovascular strain is matched, as exercising under acute hypoxia at the same heart rate leads to significantly lower muscle oxygenation (StO_2) and greater metabolic stress compared to normoxia (Park et al., 2022). Furthermore, recent evidence confirms the existence of distinct critical oxygenation plateaus in locomotor muscles under both normoxic and hypoxic conditions, underscoring the unique local kinetics of muscle oxygen saturation that function independently of systemic regulation (Kowalski et al., 2026). This dissociation is of critical importance to the researcher and coach alike, as localized muscle hypoxia is the primary catalyst for the stabilization of hypoxia-inducible factor-1α (HIF-1α). HIF-1α acts as a master transcriptional regulator, coordinating the expression of genes involved in angiogenesis, glycolysis, and erythropoiesis (Hoppeler et al., 2008; Lundby & Robach, 2015). If SpO_2 underestimates the degree of local deoxygenation, the intended "metabolic trigger" for these adaptations may be miscalculated.

Despite the growing popularity of hypoxic LRE, the dose-response relationship between graded levels of hypoxia and local muscle deoxygenation remains poorly characterized in athletic populations. Current literature has yet to provide a comprehensive mapping of how varying FiO_2 levels influence the concurrent relationship between systemic and local oxygenation across the transition from rest to active resistance exercise. Although recent methodologies have attempted to standardize the hypoxic dose during high-load resistance exercise by using a saturation clamp to maintain a target SpO_2 (Jiang et al., 2024), it remains unverified whether these systemic targets accurately reflect the actual severity of local muscle deoxygenation. Establishing the limits of SpO_2 as a surrogate for SmO_2 would provide coaches and clinicians with the evidence-based confidence required to optimize hypoxic training prescriptions. Therefore, this study investigated acute responses and the relationship between systemic oxygen saturation (SpO_2) and local muscle oxygenation (SmO_2) across incremental levels of normobaric hypoxia during rest and low-load resistance exercise in team-sport athletes.

Material & methods

Participants

The participants of this study were 18 male team sport athletes (mean age: 20.44 ± 1.25 years; height: 176.89 ± 7.84 cm; weight: 69.83 ± 17.22 kg; BMI: 22.22 ± 2.83 kg/m²; 1RM: 99.39 ± 11.58 lb). All participants were team-sport athletes (including basketball and football) with more than two years of training and competition experience. Participants were recruited during their off-season transition period. All participants provided written informed consent, and the study was approved by the local University Human Research Ethics Committee (Approval No. HE651425). Participants were instructed to quit smoking, avoid consuming alcohol and caffeinated beverages, and refrain from high-intensity exercise within 24 hours prior to the test. Inclusion criteria were: good health with no history of serious medical conditions; no exposure to high-altitude environments exceeding 1,000 meters above sea level within the 6 months prior to the experiment; and no participation in any structured resistance training programs during the 3 months preceding the experiment

(consistent with their off-season detraining status). Exclusion criteria included: history of severe acute mountain sickness; hypertension, diabetes, cardiovascular or pulmonary diseases; and musculoskeletal disorders.

Experimental design

This study employed a quantitative, correlational design with a single-group, two-phase crossover trial. All participants visited the laboratory twice (Figure 1).

During the first visit, the participants familiarized themselves with the entire experimental procedure. Then, participants inhaled gases with five different oxygen concentration levels via a hypoxic generator (ATS-HP-Hyperoxic; Altitude Technology Solutions Co., Ltd., Australia) while in a seated rest state. Level 1: FiO_2 was 21% (normoxic air, equivalent to sea level, 0 m altitude). Level 2: FiO_2 was 17.2% (simulating approximately 1,700 m). Level 3: FiO_2 was 16.2% (simulating approximately 2,200 m). Level 4: FiO_2 was 14.8% (simulating approximately 2,900 m). Level 5: FiO_2 was 13.6% (simulating approximately 3,400 m). Simultaneously, a near-infrared spectroscopy (NIRS) device (Moxy Muscle Oxygen Monitor, model Moxy-3; Fortiori Design LLC, Hutchinson, MN, USA) was fixed on the rectus femoris muscle of the non-dominant leg of the participants to monitor muscle oxygenation status. Participants inhaled 1 minute of normoxic gas between each oxygen concentration. The data of HR, SpO_2, tHb, and SmO_2 were recorded 1 minute after athletes inhaled each specific concentration of gas. All participants underwent a 1RM test at the first visit day after 30 minutes' rest.

It is important to acknowledge that the 60-second equilibration period employed during the resting condition was shorter than the conventional 3–5 minutes typically required for achieving complete steady-state pulmonary gas exchange following an FiO_2 alteration (Gardner, 1980). This abbreviated window was adopted to (a) minimize cumulative hypoxic dose and participant discomfort across five sequential exposures within a single session, and (b) capture acute, standardized trends in SpO_2 and SmO_2 responses under controlled conditions, consistent with methodological approaches used in prior acute hypoxic exercise studies (Nell et al., 2020; Scott et al., 2017). The use of abbreviated rest intervals in our protocol was informed by established methodologies in low-load resistance exercise combined with hypoxic training in athletes, which typically employ 30–60-second rests between sets or conditions (La-bantao et al., 2025; Manimmanakorn et al., 2013).

The second visit took place 72 hours (3 days) after the first visit. Before the test, the participants warmed up for 10 minutes which included cycling on a bicycle ergometer at 50-55 revolutions per minute (rpm) for 7 minutes, followed by 3 minutes of stretching exercises targeting the leg muscles. Following the warm-up, at each of the five oxygen concentration levels (consistent with the first visit: FiO_2 = 21%, 17.2%, 16.2%, 14.8%, and 13.6%), participants first remained seated for 30 seconds and then performed one set of knee extension resistance exercise (50% 1RM, 15 repetitions). Participants were instructed to train at a constant speed, with each repetition taking approximately 2 seconds. The range of motion of the knee joint for each exercise was 90° to 0° (0° at full extension). Participants inhaled normoxic air and remained seated for 1 minute between different oxygen concentration levels to ensure their heart rates returned to baseline. All variables, including HR, SpO_2, tHb, and SmO_2, were measured at every level of oxygen concentration. During the experiment, participants did not report any discomfort.

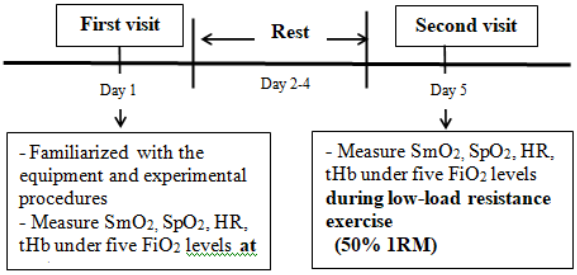

Fig.1 Timeline of study.

Measurements

One-repetition maximum measurement

The one-repetition maximum (1RM) of knee extension was determined using the 10-repetition maximum (10RM) protocol and the formula proposed by Brzycki (Brzycki, 1993) [e.g., 1RM = weight lifted/(1.0278 – 0.0278 × repetitions)]. Before the test, the participants underwent a 10-minute warm-up, which included 7 minutes of power cycling on a Monark cycle at 30–50 rpm with self-selected resistance, followed by 3 minutes of stretching exercises. After the standardized warm-up, an estimated 50% 1RM, as estimated by the participants, was used as the initial load. If a participant was able to complete 10 repetitions at the current weight, the researcher asked the participant to stop and rest for 3 minutes. Thereafter, the researchers gradually increased the weight by 15–20 pounds until the participant was unable to complete 10 repetitions, and then recorded this weight and the number of repetitions completed.

Muscle oxygen saturation and total hemoglobin measurements

Local muscle oxygenation was continuously monitored using a wireless near-infrared spectroscopy (NIRS) device (Moxy Monitor Gen 4, Fortiori Design LLC, USA). The device employs spatially resolved spectroscopy, emitting light at four wavelengths (680, 720, 760, and 800 nm) and calculating the tissue saturation index (reported as SmO_2) and relative total hemoglobin (tHb) concentration. The probe was longitudinally secured on the prepared skin over the rectus femoris muscle belly of the non-dominant leg using adhesive tape and an opaque elastic bandage to prevent motion artifact and light intrusion. Data acquisition followed brief, standardized exposures to each hypoxic level. At rest, participants were exposed to each FiO_2 for 1 minute. The mean SmO_2 and tHb from the final 10 seconds of each resting minute were immediately recorded to represent the acute steady state. During the low-load resistance exercise (LRE) protocol, participants first rested for 30 seconds at the target FiO_2 before performing a 30-second set of 15 repetitions, then immediately recorded all parameters.

Peripheral capillary oxygen saturation and heart rate measurements

SpO_2 and HR were monitored continuously using a wireless finger pulse oximeter (JPD-500D, Jumper Medical Equipment Co., Ltd., China) placed on the fingertip of the middle finger on the same side as the NIRS-monitored leg. To account for the documented latency of pulse oximetry readings behind rapid physiological changes (León-Valladares et al., 2024), data analysis focused on the post-stimulus period. For each hypoxic exposure or exercise set, all parameters were immediately recorded.

Data analysis

All statistical analyses were performed using SPSS (version 28.0; IBM Corp., USA). Continuous variables were expressed as mean ± standard deviation (SD). The normality of all data was assessed using the Shapiro–Wilk test. When the data were normally distributed, one-way repeated measures ANOVA was used to compare differences in each variable across multiple oxygen concentration levels. When a significant main effect was found, Bonferroni post hoc tests were applied for pairwise comparisons. Paired-sample t-tests were used to examine differences between rest and exercise under the same oxygen condition. Pearson's correlation analysis was conducted to evaluate the relationship between SmO_2 and SpO_2. A p-value less than 0.05 was considered statistically significant.

Results

The changes of muscle oxygen saturation (SmO_2), peripheral capillary oxygen saturation (SpO_2), total hemoglobin (tHb), and heart rate (HR) are presented in Table 1.

In the HP (resting) condition, SmO_2, tHb, and HR did not differ significantly between FiO_2 levels (all $p > 0.05$). However, SpO_2 showed significant differences across the five FiO_2 levels ($p = 0.001$). Post-hoc pairwise comparisons revealed that SpO_2 decreased significantly at FiO_2 levels of 16.2%, 14.8%, and 13.6% compared with normoxia ($FiO_2 = 21\%$; $p = 0.026$, $p = 0.013$, and $p = 0.001$, respectively).

In the HE (exercise) condition, SmO_2, SpO_2, and HR all showed significant differences across FiO_2 levels ($p = 0.009$, $p = 0.001$, and $p = 0.041$, respectively). Specifically, SmO_2 declined progressively with each reduction in FiO_2, with significant decreases observed at all hypoxic levels compared to normoxia (post-hoc pairwise: $p < 0.05$ for 17.2%, 16.2%, 14.8%, and 13.6%, respectively; $p = 0.005$ for the most severe level, 13.6%). SpO_2 decreased significantly at FiO_2 values of 16.2%, 14.8%, and 13.6% ($p = 0.024$, $p = 0.009$, and $p < 0.001$, respectively) during exercise. Additionally, SpO_2 differed significantly between FiO_2 levels of 17.2% and 13.6% ($p = 0.002$). Compared with rest, SmO_2 was significantly lower and HR was significantly higher across all FiO_2 levels during HE (all $p < 0.001$), whereas SpO_2 was significantly lower only at the most severe hypoxia ($FiO_2 = 13.6\%$; $p = 0.028$).

Table 1. The SmO_2, SpO_2, tHb and HR levels under HP and HE conditions.

Parameters	Conditions	21% FiO_2	17.2% FiO_2	16.2% FiO_2	14.8% FiO_2	13.6% FiO_2	P-Values (overall differences)
SmO_2 (%)	HP	62.81 ± 7.79	62.33 ± 8.89	62.07 ± 8.96	61.27 ± 9.07	58.79 ± 9.52	0.758
	HE	45.38 ± 9.89*	38.87 ± 9.46*	37.33 ± 9.74*	36.07 ± 9.87*	32.67 ± 9.13*[a]	0.009
SpO_2 (%)	HP	98.59 ± 0.87	97.50 ± 1.63	97.20 ± 1.15[a]	97.07 ± 1.33[a]	96.71 ± 1.20[a]	0.001
	HE	98.18 ± 1.07	97.44 ± 1.09	97.00 ± 0.85[a]	96.86 ± 1.03[a]	95.93 ± 1.21*[ab]	0.001
tHb (g/dL)	HP	12.48 ± 0.29	12.44 ± 0.31	12.49 ± 0.33	12.48 ± 0.29	12.49 ± 0.29	0.990
	HE	12.49 ± 0.35	12.53 ± 0.35	12.44 ± 0.43	12.44 ± 0.43	12.59 ± 0.37	0.720
HR (bpm)	HP	68.07 ± 8.49	69.31 ± 8.90	72.53 ± 9.33	73.00 ± 9.92	75.86 ± 8.45	0.163
	HE	85.71 ± 8.12*	87.50 ± 9.68*	89.21 ± 10.80*	91.31 ± 10.87*	97.07 ± 10.99*[a]	0.041

*Data are means ± SD. Note: g/dL = grams per deciliter, bpm = beats per minute; * Significant difference between HP and HE conditions ($P < 0.05$), [a] = Significant difference compared to 21% FiO_2 ($P < 0.05$), [b] = Significant difference compared to 17.2% FiO_2 ($P < 0.05$).*

Table 2 summarizes the magnitude of changes in SmO_2 and SpO_2 from normoxia (FiO_2 = 21%) to severe hypoxia (FiO_2 = 13.6%) under both conditions. Within the HE conditions, the magnitude of SmO_2 decline was significantly larger than that of SpO_2 (P = 0.001), whereas no significant difference was observed between SmO_2 and SpO_2 changes under HP (P = 0.106).

Table 2. The changes in SmO_2 and SpO_2 from normoxia (FiO_2 = 21%) to severe hypoxia (FiO_2 = 13.6%) under HP and HE Conditions.

Variables	Conditions	Baseline (21% FiO_2)	Final level (13.6% FiO_2)	Changes	P-Values
SmO_2 (%)	HP	62.81 ± 7.79	58.79 ± 9.52	-3.75 ± 3.86	0.106
SpO_2 (%)	HP	98.59 ± 0.87	96.71 ± 1.20	-1.62 ± 1.04	
SmO_2 (%)	HE	45.38 ± 9.89	32.67 ± 9.13	-15.00 ± 6.54	0.001
SpO_2 (%)	HE	98.18 ± 1.07	95.93 ± 1.21	-2.00 ± 1.35#	

Significant p < 0.05 within groups (SmO_2 and SpO_2)

Figure 2 presents the scatter plots of the correlation between SmO_2 and SpO_2 under HP (A) and HE (B) conditions. Each point represents the average values collected from all participants across the five FiO_2 levels. Pearson's correlation analysis was conducted to evaluate the relationship between SmO_2 and SpO_2 across the five FiO_2 levels. Under HE conditions, a strong positive correlation was observed between the two variables (r = 0.974, P = 0.005). Under HP conditions, however, no significant correlation was found (r = 0.763, P = 0.133).

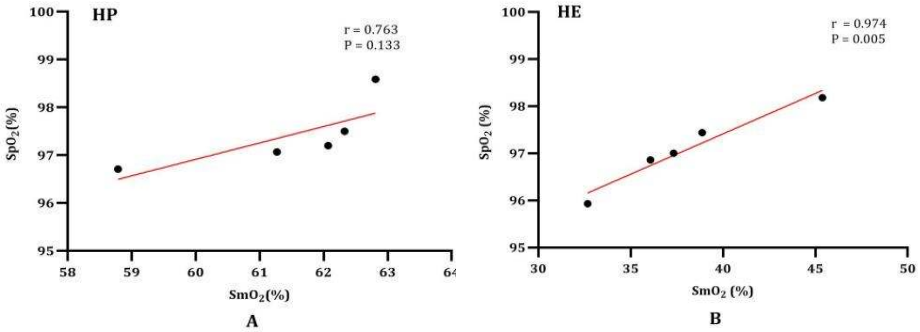

Fig.2 The correlation between SmO_2 and SpO_2 levels under HP (A) and HE (B) conditions.

Figure 3 presents the percentage changes in HE-SmO_2 and HE-SpO_2 across sequential hypoxic level transitions (Δlevel 1 to Δlevel 5). At each transition, the reduction in HE-SmO_2 was significantly greater than that in HE-SpO_2 (paired t-tests, Δlevel 1 to Δlevel 5: P < 0.001, P < 0.001, P = 0.010, P = 0.005, P = 0.006, respectively). These results demonstrate that muscle deoxygenation far exceeds peripheral oxygen desaturation at every hypoxic step, reinforcing that SpO_2 cannot serve as a proxy for SmO_2 during exercise.

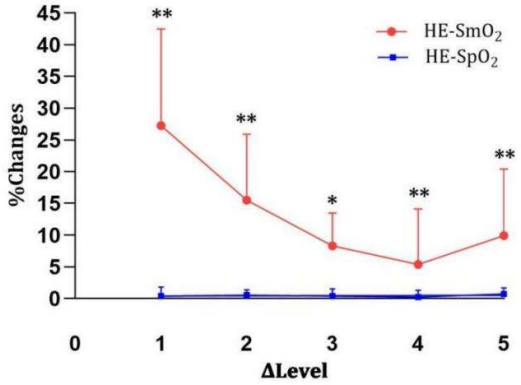

Fig.3 Percentage changes in SmO_2 and SpO_2 during hypoxic exercise (HE) across sequential hypoxic level transitions. Δlevel 1: FiO_2 21% from rest to exercise, Δlevel 2: FiO_2 21% to 17.2%, Δlevel 3: FiO_2 17.2% to 16.2%, Δlevel 4: FiO_2 16.2% to 14.8%, Δlevel 5: FiO_2 14.8% to 13.6%. *P < 0.05, ** P < 0.01, for HE-SmO_2 vs. HE-SpO_2 at each Δlevel (paired t-test).

Discussion

This study investigated the acute effects of progressively reduced FiO_2 on both systemic (SpO_2) and localized muscle oxygenation (SmO_2) in team-sport athletes during rest and low-load resistance exercise. The main findings indicate that during rest, SmO_2 remained stable across all hypoxic levels, while SpO_2 decreased in a dose-dependent manner, with significant reductions observed at $FiO_2 \leq 16.2\%$. During low-load resistance exercise, SmO_2 declined progressively across all hypoxic levels ($p < 0.05$), with the most pronounced decrease occurring at $FiO_2 = 13.6\%$ (from 45.38 ± 9.89% to 32.67 ± 9.13%). Meanwhile, SpO_2 again showed a dose-dependent decrease from $FiO_2 \leq 16.2\%$. Notably, a strong positive correlation existed between changes in SmO_2 and SpO_2 during exercise ($r = 0.974$, $p = 0.005$), but no significant correlation was observed at rest ($r = 0.763$, $p = 0.133$). However, despite this strong correlation during exercise, the magnitude of reduction in SpO_2 (−2.00%) was far smaller than that in SmO_2 (−15.00%), indicating that systemic oxygenation does not accurately reflect the extent of local muscle deoxygenation. These results highlight a relative dissociation between systemic and muscular oxygenation under acute hypoxia, particularly during resistance exercise, and suggest that SpO_2 alone cannot serve as a reliable surrogate for SmO_2.

As demonstrated in Table 1, under resting conditions, only SpO_2 exhibited a gradual decline across progressive hypoxic levels. Conversely, during exercise, SmO_2 and SpO_2 all progressively decreased as hypoxia intensified. These findings indicate that metabolic oxygen demand in working muscles escalates rapidly during exertion. Furthermore, the concurrent decline in HR and SpO_2 aligns with established physiological compensatory mechanisms. In contrast, total hemoglobin volume (tHb) showed no significant changes across any of the hypoxic conditions.

Regarding the resting conditions, the stability of SmO_2 observed in this study may be attributed to a lower physiological stress level imposed by hypoxia on the working muscles. Scott et al. (2017) reported that under mild-to-moderate hypoxia (SpO_2: 90–94%), compensatory mechanisms such as enhanced oxygen extraction and localized vasodilation maintain muscle oxygenation despite reduced arterial oxygen content. This stability contrasts with the progressive decline in SpO_2 that began at $FiO_2 = 16.2\%$, highlighting distinct regulatory pathways between systemic and localized oxygen transport. The dose-dependent decline in SpO_2 reflects the expected physiological response to reduced inspiratory oxygen partial pressure (Perrey & Ferrari, 2018). During low-load resistance exercise (LRE), the additional metabolic demand further exacerbates SmO_2 and SpO_2 decline particularly at $FiO_2 = 13.6\%$, due to increased oxygen consumption and transient ventilation-perfusion mismatch (Girard et al., 2017).

At rest, heart rate (HR) remained stable across all hypoxic levels. This finding is consistent with previous observations in trained athletes (Giles et al., 2016; Oliveira et al., 2017) and suggests that the hypoxic stimuli did not exceed their response threshold. Conversely, during low-load resistance exercise (LRE), HR significantly increased at the most severe hypoxic level ($FiO_2 = 13.6\%$) compared to normoxia ($FiO_2 = 21\%$). This indicates that in trained team-sport athletes, HR is not a sensitive marker of hypoxic stress under resting or mild-to-moderate hypoxic conditions. This lack of sensitivity is likely mediated by training-induced autonomic adaptations, such as elevated parasympathetic tone and plasma volume expansion (Buchheit et al., 2010; Gorini Pereira et al., 2024). Nevertheless, when exercise is compounded by severe hypoxia, the combined metabolic and cardiorespiratory strain triggers a compensatory increase in HR to offset reduced arterial oxygen content and sustain systemic oxygen delivery (Scott et al., 2017). Moreover, Durand et al. (2020) demonstrated that exercise-induced hypoxemia in athletes is associated with increased pulmonary vascular resistance and decreased vascular compliance, further contributing to the dissociation between systemic and local oxygenation.

Notably, tHb, as a surrogate indicator of local blood volume, remains unchanged under all conditions. This demonstrates that acute hypoxia does not alter microvascular perfusion in the rectus femoris, emphasizing the role of oxygen extraction efficiency rather than blood volume shifts in preserving SmO_2 (Calbet et al., 2009; Scott et al., 2017). The absence of change in tHb is consistent with the view that acute hypoxic exposure primarily optimizes oxygen diffusion and metabolic adaptation rather than increasing capillary density (Esaki et al., 2005).

As shown in Figure 2, a very strong positive correlation was observed between SmO_2 and SpO_2 during the LRE condition ($r = 0.974$, $p = 0.005$). Theoretically, this aligns with the macroscopic trend that a decrease in SpO_2 reflects reduced systemic arterial oxygen content, which inevitably limits the oxygen supply and reserves available to peripheral muscle tissues. However, despite this strong positive correlation during exercise, the magnitude of the reductions differed drastically; the percentage decrease in SpO_2 did not mirror the extensive decline seen in SmO_2 (SpO_2: -2.00%; SmO_2: -15.00%), as detailed in Table 2. This discrepancy demonstrates that while SpO_2 and SmO_2 share a positive correlation, SpO_2 cannot serve as a reliable surrogate for quantifying the true magnitude of localized muscle deoxygenation.

In addition, as illustrated in Figure 3, the percentage reduction in SmO_2 between sequential hypoxic levels during exercise exhibited a non-linear trajectory compared to SpO_2. These results indicate that during exertion, muscle deoxygenation decreases far more profoundly across all hypoxic doses than peripheral oxygen saturation. This sharp decline in SmO_2 is heavily driven by the mechanical occlusion of intramuscular blood

flow induced by repetitive muscle contractions, whereas systemic SpO_2 remains relatively stable or exhibits only minor fluctuations. Solsona et al. (2021) similarly observed that during sprint interval exercises under hypoxia, SpO_2 decreased significantly while muscle oxygen extraction and oxyhemoglobin availability were substantially altered, underscoring the independent regulation of peripheral and muscular oxygenation. Interestingly, the relative changes in both parameters were disproportionate across the specific hypoxic conditions, with the most pronounced mismatches occurring at levels 1, 2, and 5 (Figure 3). This divergence underscores the dominant role of localized auto-regulatory mechanisms in intramuscular blood circulation. Furthermore, individual physiological variations, muscle fiber type composition, and exercise technique likely contribute substantially to the observed variability in SmO_2 during hypoxic exercise.

The disproportionate decline between SpO_2 and SmO_2 during exercise can be explained by three interdependent physiological mechanisms. First, temporal response characteristics fundamentally differ between the two technologies. Near-infrared spectroscopy (NIRS) captures real-time changes in microvascular oxy- and deoxy-hemoglobin concentrations with millisecond resolution, directly reflecting the dynamic balance between O_2 delivery and mitochondrial consumption within the active myocyte (Grassi & Quaresima, 2016; Perrey & Ferrari, 2018). In contrast, pulse oximetry measures systemic arterial hemoglobin saturation, which exhibits a 10–30 second physiological lag due to pulmonary transit time, circulatory delay, and peripheral signal averaging (Nitzan et al., 2014). During repetitive dynamic contractions lasting only 30 seconds, this temporal mismatch means that SpO_2 readings cannot track the rapid, phasic deoxygenation events occurring within the working muscle. Second, mechanical occlusion during contraction imposes a powerful local confound. During knee extension at 50% 1RM, intramuscular pressure can exceed 150–200 mmHg, transiently exceeding arteriolar perfusion pressure and causing intermittent, contraction-induced vascular compression (Crenshaw et al., 2000; Fliss et al., 2025). This creates a repeated "ischemia–reperfusion" cycle within the rectus femoris, decoupling local O_2 dynamics from systemic arterial O_2 content. Third, local metabolic vasodilation and autoregulatory feedback loops, including adenosine accumulation, nitric oxide release, and shear stress-mediated responses, act independently of systemic sympathetic drive to maintain microvascular perfusion despite moderate reductions in SpO_2 (Calbet et al., 2009; Joyner & Casey, 2014). These local mechanisms collectively explain why SmO_2 declines far more steeply than SpO_2 during hypoxic exercise, and why the two parameters cannot be used interchangeably. The present results align with those of Nell et al. (2020), who reported no correlation between SmO_2 and SpO_2 during handgrip exercise under severe hypoxia. Furthermore, a study by Vasquez-Bonilla et al. (2021) supports this independence of systemic and local oxygenation, demonstrating that the hypoxic decline in SpO_2 during repeated sprints does not invariably mirror or affect SmO_2 dynamics.

In summary, these results reinforce the concept that systemic oxygenation alone fails to capture the true severity of localized muscle hypoxia during exercise, even though it may provide a reasonable approximation at rest. This divergence during exertion is primarily driven by local confounding factors including mechanical occlusion from elevated intramuscular pressure (Solsona et al., 2025) and metabolic vasodilation (Joyner & Casey, 2014) which collectively decouple microvascular oxygenation from systemic arterial saturation. From a practical standpoint, these findings indicate that relying solely on SpO_2 to titrate hypoxic training intensity would substantially underestimate peripheral muscular stress, potentially leading to suboptimal training prescriptions and inaccurate workload monitoring.

This study has several limitations that should be acknowledged. First, SmO_2 measurements were restricted to the rectus femoris, limiting the generalizability of the data to other muscle groups (e.g., vastus lateralis, gastrocnemius) or deeper tissues with different fiber-type compositions and perfusion characteristics. Second, the specific NIRS device used (Moxy Monitor Gen 4) does not separate absolute oxyhemoglobin from deoxyhemoglobin values, which restricts deeper mechanistic insights into the specific pathways of muscle oxygen utilization and localized blood flow. Third, subcutaneous adipose tissue thickness (ATT) over the rectus femoris was not measured. Given that ATT directly influences NIRS light penetration depth and absolute SmO_2 values, individual variability in adiposity may have introduced systematic bias; we recommend that future studies incorporate ultrasound or skinfold caliper measurements to correct for this variable (Perrey & Ferrari, 2018). Fourth, the 60-second equilibration period employed during the resting hypoxic exposures was shorter than the 3–5 minutes typically required for full pulmonary steady-state; consequently, the resting SmO_2 and SpO_2 values reported here represent acute, transitional responses rather than true steady-state adaptations, and our conclusions regarding resting conditions should be interpreted with this caveat.

Conclusions

This study demonstrates that local muscle oxygen saturation (SmO_2) and systemic peripheral oxygen saturation (SpO_2) exhibit divergent responses to graded hypoxia during low-load resistance exercise. While SmO_2 declined progressively across all hypoxic levels, SpO_2 decreased significantly only at $FiO_2 \leq 16.2\%$. Despite a strong correlation between the two variables during exercise, the magnitude of the SmO_2 reduction was highly disproportionate to that of SpO_2. Rather than directly mirroring systemic arterial oxygen content,

localized muscle deoxygenation appears to be governed primarily by intrinsic regulatory mechanisms, including contraction-induced mechanical occlusion and metabolic vasodilation.

Practically, these findings highlight a critical limitation in using pulse oximetry alone. Relying exclusively on SpO_2 to titrate hypoxic training intensity will systematically underestimate the true severity of muscular hypoxic stress. To optimize training prescriptions and accurately monitor workloads, practitioners and coaches should incorporate localized monitoring tools, such as Near-Infrared Spectroscopy (NIRS), particularly when implementing low-load resistance exercise under hypoxic conditions. Future research should aim to validate these findings using multi-site NIRS monitoring across different muscle groups. Incorporating subcutaneous adipose tissue thickness measurements will also be essential to improve the precision of SmO_2 readings in diverse athletic populations. Furthermore, longitudinal studies are warranted to explore how this dissociation between systemic and local oxygenation evolves across different training phases. Ultimately, integrating both systemic and local oxygenation tracking provides a more comprehensive physiological basis for individualized hypoxic training in applied sports science.

Conflicts of interest

The authors declare no conflicts of interest. The results of this study do not constitute the author's endorsement of this hypoxicator.

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Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the authors utilized artificial intelligence (AI) tools to enhance language clarity and readability. Following the use of these tools, the authors thoroughly reviewed, edited, and verified the content, and assume full responsibility for the final publication.

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