

Oxygen Saturation, Heart Rate, and Blood Lactate Responses to Acute Simulated Altitude in Sprinters and Middle- and Long-Distance Athletes

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Abstract

Background

Acute hypoxia reduces oxygen availability and may increase physiological strain during supramaximal exercise. This study examined peripheral oxygen saturation (SpO₂), heart rate (HR), and blood lactate responses to graded simulated altitude in Indian sub-elite sprinters and middle- and long-distance (MD/LD) athletes.

Methods

Forty-four male athletes, comprising 22 sprinters and 22 MD/LD athletes, completed a 30-second Wingate test under normoxia and simulated altitudes of 2000 and 3000 m. Hypoxic trials followed 60 minutes of exposure, and trials were separated by at least 72 hours. SpO₂ was measured at rest and immediately post-exercise. HR was assessed at rest, during Wingate exercise, and at three-minute recovery. Blood lactate was measured at rest, immediately post-exercise, and at six recovery points up to 30 minutes. Mixed-design repeated-measures analysis of variance (ANOVA) examined group, altitude, and time or phase effects.

Results

SpO₂ decreased progressively with altitude and fell further after exercise. Altitude, time, and altitude × time effects were significant (all $p < 0.001$), with an altitude effect size of $\eta^2 = 0.991$. No group-related SpO₂ effects were significant. HR increased with altitude and remained above resting values at three-minute recovery. Altitude, phase, and altitude × phase effects were significant (all $p < 0.001$). The overall HR group effect was nonsignificant ($p = 0.053$), but the group × altitude × phase interaction was significant ($p = 0.010$). Lactate reached its highest sampled mean at three minutes and remained elevated at 30 minutes. Sprinters showed higher post-exercise and recovery concentrations. Lactate showed significant group, altitude, and time effects (all $p < 0.001$) and all two-way interactions, but no significant three-way interaction ($p = 0.123$).

Conclusions

Acute simulated altitude increased desaturation, cardiovascular strain, and post-exercise lactate concentrations. Group differences were clearer for lactate than for overall SpO₂ or HR, but cannot be attributed solely to training background or slower lactate clearance. Combined physiological assessment and serial lactate sampling may support more informed monitoring of exercise responses under acute hypoxia.

Categories: Environmental Health, Sports Medicine, Cardiology

Keywords: acute hypoxia, blood lactate, heart rate, peripheral oxygen saturation, simulated altitude, wingate anaerobic test

Introduction

Acute altitude exposure imposes an immediate systemic physiological challenge. The lower inspired oxygen partial pressure reduces arterial oxygenation and limits oxygen delivery to active tissues. In response, ventilation, sympathetic activity, heart rate (HR), and cardiac output (CO) increase to preserve oxygen transport [1,2]. Theunissen et al. [3] showed that acute exposure to 3,842 m caused marked hypoxemia with increased HR and CO. Similar responses have been reported during simulated altitude exercise, where peripheral oxygen saturation (SpO₂) declines progressively as hypoxic severity increases [4]. Thus, intense exercise under acute hypoxia combines high metabolic demand with restricted oxygen availability.

Exercise-induced desaturation becomes more noticeable during supramaximal exercise. Oguri et al. reported a greater fall in SpO₂ and increased muscle deoxygenation during a 30-s Wingate test under simulated hypoxia, despite preserved anaerobic power [5]. Karayigit et al. also demonstrated a dose-dependent reduction in SpO₂ with increasing simulated altitude [6]. Sarikaya et al. observed progressive desaturation

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together with increased HR during exercise [7]. However, HR responses are influenced by exercise intensity and workload prescription. HR may rise when the same external workload is maintained under hypoxia [4], but may remain relatively stable when workload is reduced to match physiological strain [8]. Therefore, SpO₂ and HR together provide a useful indication of systemic oxygen stress and cardiovascular compensation.

Blood lactate provides an additional measure of metabolic strain. Supramaximal exercise rapidly increases glycolytic adenosine triphosphate (ATP) production and lactate appearance. Lactate is now recognized as an important metabolic substrate and signalling molecule rather than simply an anaerobic waste product [9]. Hypoxia may increase glycolytic reliance because oxidative ATP production becomes constrained. Yoshida et al. demonstrated greater lactate release from exercising muscle during acute hypoxia [10]. Maldonado-Rodriguez et al. similarly reported higher post-exercise lactate during hypoxic sprint exercise [11]. In contrast, Oguri et al., Ofner et al., and Yamaguchi et al. found little or no hypoxia-related difference in lactate [5,12,13]. These conflicting findings suggest that lactate responses depend on hypoxic dose, exercise duration, recruited muscle mass, training status, and recovery kinetics.

Training background may further modify these responses. Sprint athletes are adapted for rapid force production and high glycolytic flux, whereas endurance athletes possess greater oxidative capacity and cardiovascular efficiency. Kennedy et al. showed that endurance-trained athletes maintained central hemodynamics more effectively during acute hypoxia [14]. Conversely, Oguri et al. observed greater hypoxia-induced desaturation and muscle deoxygenation in sprint athletes [5]. Athlete level may also influence tolerance, as elite players maintained function better than sub-elite players during acute hypoxia [15].

Despite growing evidence, limited information is available on the combined recovery patterns of SpO₂, HR, and blood lactate after standardized supramaximal exercise across graded simulated altitudes, particularly in Indian sub-elite athletes. Therefore, this study aimed to examine these responses following Wingate exercise under normoxia and simulated 2,000 and 3,000 m altitude, and to compare sprint athletes with middle-/long-distance athletes (MD/LD).

Materials And Methods

This study used a mixed factorial repeated-measures design, with athlete group (sprinters vs. MD vs. LD athletes) as the between-subject factor and simulated altitude and measurement time/phase as within-subject factors. Altitude was experimentally varied within each athlete. We conducted the study in the Department of Exercise Physiology, Sports Authority of India, Netaji Subhas National Institute of Sports (SAI NSNIS), Patiala, India, between March and June 2025. Each participant completed a Wingate anaerobic test under three oxygen conditions: normoxia, simulated 2,000 m altitude, and simulated 3,000 m altitude. We maintained a minimum interval of 72 hours between consecutive experimental trials to reduce the influence of residual fatigue. All participants completed at least one familiarization session before data collection. This session included familiarization with the Wingate test and the procedures used inside the hypoxic chamber.

The final sample consisted of 44 Indian male sub-elite athletes. Participants were classified by primary athletic event as sprinters (n = 22) or middle- and long-distance athletes (MD/LD; n = 22). Eligible participants were aged 20-30 years. They had competed at least at the national level and had a minimum of four to five years of sport-specific training age. All participants were actively engaged in their regular training programs during the study period.

Athletes were excluded if they had experienced a musculoskeletal injury during the preceding six months or reported cardiovascular or respiratory complaints that could influence exercise performance or the physiological response to hypoxia. Eligibility was assessed using medical history and physical examination. Height and body mass were recorded before experimental testing using standardized measuring equipment.

An a priori sample-size calculation was performed using G*Power version 3.1.9.4 (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany). The estimated minimum sample requirement was 40 participants (sample-size sensitivity calculation, using a two-sided independent-groups t test with equal group sizes, $\alpha = 0.05$ and 80% power, indicated a detectable standardized mean difference of Cohen's $d = 0.865$). An additional 10% was included to account for possible attrition or incomplete observations. Accordingly, the target sample was increased to 44 athletes. All 44 participants completed the experimental protocol and were included in the final analysis, with 22 athletes in each training group.

Ethical considerations

Data collection was performed at SAI NSNIS, Patiala. Ethical oversight was provided by the Institutional Ethics Committee of Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR), Maharishi Markandeshwar University (MMU), Mullana, India. Ethical approval was granted under approval number IEC-3145, dated February 18, 2025. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Participants were informed about the experimental procedures

before participation, and informed consent was obtained before data collection.

Simulated-altitude exposure

Normobaric hypoxia was generated using a hypoxic chamber (L.O.T. LowOxygen GmbH, Berlin, Germany). The chamber had an internal volume of 90 m³. Three inspired oxygen conditions were examined. Normoxia was represented by a fraction of inspired oxygen (FiO₂) of 20.9% (0.209). Simulated 2,000 m altitude was produced using an FiO₂ of 16.4% (0.164), whereas simulated 3,000 m altitude was produced using an FiO₂ of 14.5% (0.145).

Before each hypoxic exercise trial, participants remained inside the chamber for 60 minutes under the assigned oxygen condition. This period was provided to allow acute systemic physiological stabilisation to the hypoxic environment before exercise. The Wingate test was subsequently performed while the participant remained exposed to the same simulated-altitude condition. The normoxic trial was performed under ambient oxygen availability. The three experimental trials were separated by at least 72 hours in alternative order.

Wingate test as the exercise stimulus

A 30-second Wingate anaerobic test was used as the standardized 30-second all-out supramaximal exercise (with basket resistance with 7.5% of body weight) stimulus across all three experimental conditions (Monark Exercise AB, Vansbro, Sweden). Participants completed the test after the required pre-exercise procedures under normoxia, simulated 2,000 m, and simulated 3,000 m altitude. The same testing procedure was maintained across conditions for each participant to minimize procedural variation. During the simulated-altitude trials, the test was performed inside the hypoxic environment so that the assigned FiO₂ was maintained throughout the exercise bout.

Participants were instructed to perform the Wingate test with maximal effort for the complete exercise period. Physiological measurements were obtained before, during, and/or after the test according to the predefined measurement schedule for peripheral oxygen saturation, HR, and blood lactate. Wingate performance variables were obtained as part of the experimental protocol but were not treated as outcome variables in the present analysis, which focused on systemic oxygenation, cardiovascular response, and post-exercise lactate kinetics.

Peripheral oxygen-saturation measurement

SpO₂ was assessed to quantify the systemic oxygenation response to acute simulated altitude and supramaximal exercise using an Accusure Pulse Oximeter for SpO₂ portable monitor (Microgene Diagnostic Systems Pvt. Ltd., New Delhi). SpO₂ was recorded under each of the three oxygen conditions. Measurements used for the present analysis represented two time points: resting and post-Wingate. Resting measurements were obtained before the Wingate exercise stimulus under the corresponding oxygen condition. Post-exercise measurements were obtained following completion of the Wingate test.

The resulting SpO₂ data therefore permitted evaluation of both the altitude-related reduction in systemic oxygen availability and the additional desaturation associated with supramaximal exercise. The same measurement procedure was applied to the sprint and MD/LD groups across all experimental conditions.

HR measurement

HR was assessed as an indicator of cardiovascular strain during acute simulated-altitude exposure and supramaximal exercise (HR measured using Polar Team Pro (Polar Electro, Kempele, Finland). HR was recorded during each of the three experimental conditions. For statistical analysis, measurements were organized into three physiological phases: resting, during the Wingate test, and three-minute recovery.

The resting measurement represented cardiovascular status before the supramaximal exercise bout under the assigned oxygen condition. Exercise HR represented the cardiovascular response during the Wingate stimulus. Recovery HR was assessed during the three-minute post-exercise recovery period. This measurement structure allowed the cardiovascular response to be evaluated across resting, maximal-exercise, and early-recovery phases under progressively reduced inspired oxygen availability.

Blood lactate measurement

Blood-lactate concentration was measured to characterize the metabolic response and subsequent recovery following supramaximal Wingate exercise. Blood Lactate was measured by fingertip capillary blood using the Lactate Scout (EKF-diagnostic GmbH, Barleben, Germany). Lactate measurements were taken as per the manufacturer's guidelines. Measurements were obtained under normoxia and both simulated-altitude conditions using the same experimental schedule.

Blood lactate was assessed at eight time points: at rest, immediately after completion of the Wingate test,

and at three, five, 10, 15, 20, and 30 minutes of recovery. This repeated sampling schedule was selected to characterize both the early post-exercise rise and the subsequent recovery pattern rather than relying on a single post-exercise measurement. The same sampling schedule was used in sprinters and MD/LD athletes under all three altitude conditions.

Statistical analysis

Data from all 44 athletes were analyzed using mixed-design repeated-measures analysis of variance (ANOVA) to account for between-group comparisons and repeated observations within athletes. Athlete group was the between-subject factor, while altitude and measurement time or phase were within-subject factors. Separate models were fitted for SpO2 (2 × 3 × 2), HR (2 × 3 × 3), and blood lactate (2 × 3 × 8).

Residual normality and between-group variance homogeneity were assessed using Shapiro-Wilk and median-centered Levene tests, respectively. Sphericity was evaluated using Mauchly's test with a first-order Bartlett chi-square approximation. Greenhouse-Geisser correction was applied to effects involving within-subject contrasts with more than one degree of freedom. Corrected degrees of freedom, F statistics, p-values, and partial η² were reported.

Exploratory follow-up analyses used paired t-tests for within-athlete comparisons and Welch t-tests for between-group comparisons, including differences in change scores. Holm adjustment was applied separately within each comparison family specified in Tables S4-S6. Pointwise 95% confidence intervals were reported. Statistical significance was set at p < 0.05, using adjusted p-values for follow-up comparisons.

Results

The final analysis included 44 male Indian sub-elite athletes, comprising 22 MD/LD athletes and 22 sprinters. The two groups were similar in age (p = 0.267). However, MD/LD athletes were significantly taller (p < 0.001) and heavier (p = 0.023) than sprinters (Table 1).

Variable	Middle/Long Distance (n = 22)	Sprinters (n = 22)	Test	Statistic	p
Age (years)	25.36 ± 2.56	24.50 ± 2.54	Independent t-test	t = 1.12	0.267
Height (cm)	168 (167-170)	164 (161-166)	Mann-Whitney U	U = 85.00	< 0.001*
Weight (kg)	77.14 ± 6.93	71.37 ± 9.12	Independent t-test	t = 2.36	0.023*

TABLE 1: Baseline characteristics and group differences
Values are presented as mean ± SD or median (IQR), as appropriate. Normality was assessed using the Shapiro-Wilk test. MD/LD: middle/long distance athletes. *p < 0.05 was considered statistically significant.

Peripheral oxygen-saturation response

Peripheral oxygen saturation (SpO2) decreased progressively with increasing simulated altitude in both athlete groups. In MD/LD athletes, resting SpO2 decreased from 98.7 ± 0.8% under normoxia to 93.9 ± 1.0% at 2000 m and 88.3 ± 1.6% at 3000 m. A similar pattern was observed in sprinters, where resting SpO2 values were 98.7 ± 0.7%, 93.4 ± 1.2%, and 87.8 ± 1.4%, respectively. The main effect of altitude was highly significant with a very large effect size (F = 4627.693, p < 0.001, ηp² = 0.991), showing a clear reduction in SpO2 with increasing simulated altitude.

SpO2 decreased further following the Wingate test under all three altitude conditions. In MD/LD athletes, post-Wingate SpO2 was 94.7 ± 2.3% under normoxia, 87.6 ± 2.5% at 2000 m, and 82.1 ± 2.2% at 3000 m. The corresponding resting-to-post-Wingate reductions were 4.0, 6.3, and 6.2 percentage points. In sprinters, post-Wingate SpO2 values were 95.7 ± 1.6%, 88.3 ± 2.2%, and 82.7 ± 2.0%, with corresponding reductions of 3.0, 5.1, and 5.1 percentage points. The main effect of time was significant (F = 294.281, p < 0.001, ηp² = 0.875), indicating a marked reduction in SpO2 following the Wingate test.

A significant altitude × time interaction was also observed (F = 59.922, p < 0.001, ηp² = 0.588), indicating that the magnitude of exercise-associated SpO2 reduction was dependent on altitude condition. Descriptively, the reduction following Wingate was greater under both simulated hypoxic conditions compared with normoxia. However, the reductions observed at 2000 m and 3000 m were quite similar in both groups.

The overall group effect was not significant (F = 0.254, p = 0.617, ηp² = 0.006), indicating no overall difference in SpO2 between MD/LD athletes and sprinters. The group × altitude interaction was also not

significant ($F = 1.697$, $p = 0.190$, $\eta^2 = 0.039$), suggesting that the altitude-related pattern of SpO₂ reduction was generally similar between groups. The group $\times$ time interaction was not statistically significant ($F = 3.567$, $p = 0.066$, $\eta^2 = 0.078$). Although MD/LD athletes showed numerically greater resting-to-post-Wingate reductions, this difference did not reach statistical significance. Similarly, the group $\times$ altitude $\times$ time interaction was not significant ($F = 0.078$, $p = 0.925$, $\eta^2 = 0.002$), showing that the combined altitude- and exercise-related SpO₂ response did not differ between the two athlete groups (Tables 2, 3, Appendix 1).

Group	Altitude	Resting SpO ₂ , mean $\pm$ SD (%)	Post-Wingate SpO ₂ , mean $\pm$ SD (%)
MD/LD	Normoxia	98.7 $\pm$ 0.8	94.7 $\pm$ 2.3
MD/LD	2000 m	93.9 $\pm$ 1.0	87.6 $\pm$ 2.5
MD/LD	3000 m	88.3 $\pm$ 1.6	82.1 $\pm$ 2.2
Sprinters	Normoxia	98.7 $\pm$ 0.7	95.7 $\pm$ 1.6
Sprinters	2000 m	93.4 $\pm$ 1.2	88.3 $\pm$ 2.2
Sprinters	3000 m	87.8 $\pm$ 1.4	82.7 $\pm$ 2.0

TABLE 2: Peripheral oxygen saturation at rest and immediately post-Wingate across simulated altitude conditions

Values are presented as mean $\pm$ SD. SpO₂: peripheral oxygen saturation; MD/LD: middle-/long-distance athletes. Resting SpO₂ was measured before the Wingate test, while post-Wingate SpO₂ was measured immediately following completion of the 30-second Wingate test. Each group included 22 athletes. Normoxia: FiO₂ 20.9%; simulated 2000 m: FiO₂ 16.4%; simulated 3000 m: FiO₂ 14.5%.

Effect	F	p	η^2
Group	0.254	0.617	0.006
Altitude	4627.693	< 0.001***	0.991
Time	294.281	< 0.001***	0.875
Group $\times$ Altitude	1.697	0.190	0.039
Group $\times$ Time	3.567	0.066	0.078
Altitude $\times$ Time	59.922	< 0.001***	0.588
Group $\times$ Altitude $\times$ Time	0.078	0.925	0.002

TABLE 3: Mixed-design repeated-measures ANOVA for peripheral oxygen saturation

A 2 $\times$ 3 $\times$ 2 mixed-design repeated-measures ANOVA was performed. Group was the between-subject factor comparing MD/LD athletes and sprinters. Altitude was the within-subject factor comprising normoxia, simulated 2000 m, and simulated 3000 m, while time was the within-subject factor comparing resting and immediately post-Wingate SpO₂. Group $\times$ altitude examined whether the altitude-related response differed between athlete groups. Group $\times$ time examined whether the overall resting-to-post-Wingate response differed between groups. Altitude $\times$ time examined whether the exercise-associated change in SpO₂ differed across altitude conditions. Group $\times$ altitude $\times$ time examined whether this combined response differed between MD/LD athletes and sprinters. F: ANOVA test statistic; η^2 : partial eta squared; ANOVA: analysis of variance; MD/LD: middle-/long-distance athletes; SpO₂: peripheral oxygen saturation. *** $p < 0.001$. Statistical significance was accepted at $p < 0.05$.

Heart-rate response

Heart rate (HR) showed a progressive increase with simulated altitude and changed markedly across the three physiological phases. The main effect of altitude was highly significant, with a very large effect size ($F = 2100.076$, $p < 0.001$, $\eta^2 = 0.980$). Across both athlete groups, HR was generally higher at 2000 m than under normoxia and increased further at 3000 m.

A strong main effect of physiological phase was also observed ($F = 1069.584$, $p < 0.001$, $\eta^2 = 0.962$). HR increased substantially from resting values during the Wingate test and declined during the three-minute recovery, although it remained above the resting HR at all altitude conditions. The altitude $\times$ phase interaction was significant with a large effect size ($F = 110.088$, $p < 0.001$, $\eta^2 = 0.724$). This indicates that

the magnitude of altitude-related increase in HR was not similar across resting, Wingate, and early recovery phases.

The overall group effect did not reach statistical significance ($F = 3.968$, $p = 0.053$, $\eta p^2 = 0.086$), although MD/LD athletes showed numerically higher overall HR compared with sprinters. The group $\times$ altitude interaction was also not significant ($F = 0.942$, $p = 0.394$, $\eta p^2 = 0.022$), suggesting that the overall altitude-related increase in HR was broadly similar between the two groups. Similarly, the group $\times$ phase interaction was not significant ($F = 2.208$, $p = 0.116$, $\eta p^2 = 0.050$).

However, a significant group $\times$ altitude $\times$ phase interaction was observed ($F = 3.403$, $p = 0.010$, $\eta p^2 = 0.075$). This suggests that the combined altitude-by-phase pattern differed modestly between MD/LD athletes and sprinters, even though the individual group $\times$ altitude and group $\times$ phase interactions were not significant. Overall, altitude and physiological phase remained the major factors influencing HR response, while group-related differences were comparatively smaller (Tables 4, 5, Appendix 2).

Group	Phase	Normoxia	2000 m	3000 m
MD/LD	Resting	78 $\pm$ 10	83 $\pm$ 10	88 $\pm$ 11
MD/LD	During Wingate	174 $\pm$ 11	177 $\pm$ 11	180 $\pm$ 11
MD/LD	3-min recovery	111 $\pm$ 7	117 $\pm$ 7	122 $\pm$ 8
Sprinters	Resting	69 $\pm$ 9	73 $\pm$ 9	77 $\pm$ 10
Sprinters	During Wingate	173 $\pm$ 17	176 $\pm$ 18	179 $\pm$ 18
Sprinters	3-min recovery	105 $\pm$ 16	110 $\pm$ 16	116 $\pm$ 17

TABLE 4: Heart-rate descriptive values across simulated altitude and physiological phase

Values are presented as mean $\pm$ SD in beats per minute (bpm) and rounded to the nearest whole value. Resting HR was recorded before the Wingate test. During-Wingate HR was recorded during the final 5–8 seconds of the 30-second Wingate test. Recovery HR was recorded three minutes following completion of the Wingate test. MD/LD: middle-/long-distance athletes; HR: heart rate.

Effect	F	p	ηp^2
Group	3.968	0.053	0.086
Altitude	2100.076	< 0.001***	0.980
Phase	1069.584	< 0.001***	0.962
Group $\times$ Altitude	0.942	0.394	0.022
Group $\times$ Phase	2.208	0.116	0.050
Altitude $\times$ Phase	110.088	< 0.001***	0.724
Group $\times$ Altitude $\times$ Phase	3.403	0.010*	0.075

TABLE 5: Mixed-design repeated-measures ANOVA summary for heart rate

A $2 \times 3 \times 3$ mixed-design repeated-measures ANOVA was used. Group was the between-subject factor and compared MD/LD athletes with sprinters. Altitude was the within-subject factor and included normoxia, simulated 2000 m, and simulated 3000 m. Phase was the within-subject factor and included resting HR, during-Wingate HR, and HR measured three minutes following the Wingate test. Interaction effects examined whether altitude- and phase-related HR responses differed between athlete groups. F: ANOVA test statistic; ηp^2 : partial eta squared; ANOVA: analysis of variance; HR: heart rate. * $p < 0.05$; *** $p < 0.001$.

Blood lactate kinetics

Blood lactate increased sharply following the Wingate test in both athlete groups and across all three altitude conditions. In all conditions, the highest mean lactate concentration was observed at three minutes after exercise. In MD/LD athletes, the three-minute value increased from 12.84 ± 0.70 mmol/L under normoxia to 13.16 ± 0.72 mmol/L at 2000 m and 15.08 ± 0.78 mmol/L at 3000 m. In sprinters, the

corresponding values were 14.85 ± 1.64 , 15.23 ± 1.68 , and 17.45 ± 1.76 mmol/L. Thus, the highest mean lactate response was observed in sprinters at 3000 m.

Following the three-minute peak, blood lactate decreased progressively at five, 10, 15, 20, and 30 minutes of recovery. However, lactate remained clearly above resting concentration even at 30 minutes in both groups and at every altitude. The main effect of time was highly significant and showed a very large effect size ($p < 0.001$, $\eta^2 = 0.990$), confirming a marked change in blood lactate across the exercise and recovery period.

A significant main effect of group was also observed ($p < 0.001$, $\eta^2 = 0.457$). Sprinters generally showed higher lactate concentrations after the Wingate test and throughout recovery compared with MD/LD athletes, whereas the resting concentrations were relatively similar between groups. The group $\times$ time interaction was significant ($p < 0.001$, $\eta^2 = 0.394$), showing that the magnitude and recovery pattern of lactate response differed between the two athlete groups.

A very strong main effect of altitude was observed ($p < 0.001$, $\eta^2 = 0.975$). Lactate concentrations generally increased from normoxia to 2000 m and further at 3000 m. This altitude-related difference was particularly evident during the post-exercise and recovery measurements. The altitude $\times$ time interaction was also highly significant ($p < 0.001$, $\eta^2 = 0.917$), indicating that the effect of simulated altitude was not uniform across the different measurement time points.

The group $\times$ altitude interaction was significant ($p = 0.003$, $\eta^2 = 0.175$), suggesting that the overall effect of altitude on lactate response was somewhat different between MD/LD athletes and sprinters. In contrast, the group $\times$ altitude $\times$ time interaction was not significant after correction for sphericity ($p = 0.123$, $\eta^2 = 0.052$). Therefore, there was no sufficient evidence that the combined altitude-related pattern across the entire recovery period differed between the two athlete groups. Overall, blood lactate kinetics were strongly influenced by exercise/recovery time, simulated altitude, and athlete group, with significant two-way interactions between these factors (Tables 6, 7, Appendices 3, 4, 5).

Time point	Group	Normoxia	2000 m	3000 m
Rest	MD/LD	1.66 ± 0.12	1.72 ± 0.13	1.74 ± 0.13
Rest	Sprinters	1.68 ± 0.13	1.75 ± 0.14	1.76 ± 0.14
Immediately post-Wingate	MD/LD	12.45 ± 0.68	12.78 ± 0.68	12.92 ± 0.72
Immediately post-Wingate	Sprinters	14.40 ± 1.57	14.80 ± 1.65	14.96 ± 1.65
3 min	MD/LD	12.84 ± 0.70	13.16 ± 0.72	15.08 ± 0.78
3 min	Sprinters	14.85 ± 1.64	15.23 ± 1.68	17.45 ± 1.76
5 min	MD/LD	11.99 ± 0.65	12.43 ± 0.68	14.06 ± 0.68
5 min	Sprinters	13.91 ± 1.51	14.42 ± 1.57	16.26 ± 1.64
10 min	MD/LD	10.13 ± 0.54	10.77 ± 0.58	11.88 ± 0.56
10 min	Sprinters	11.83 ± 1.25	12.58 ± 1.33	13.86 ± 1.46
15 min	MD/LD	8.61 ± 0.45	9.30 ± 0.50	10.16 ± 0.46
15 min	Sprinters	10.11 ± 1.04	10.88 ± 1.16	11.90 ± 1.23
20 min	MD/LD	7.36 ± 0.38	8.16 ± 0.44	8.83 ± 0.43
20 min	Sprinters	8.68 ± 0.86	9.61 ± 0.97	10.44 ± 1.00
30 min	MD/LD	5.50 ± 0.29	6.38 ± 0.33	6.61 ± 0.34
30 min	Sprinters	6.51 ± 0.61	7.57 ± 0.71	7.83 ± 0.74

TABLE 6: Blood lactate concentrations across simulated altitude, recovery time, and athlete group

Values are presented as mean ± SD in mmol/L (n = 22 per group). Rest represents blood lactate measured before the Wingate test. Immediately post-Wingate represents the measurement obtained following completion of the 30-s Wingate test. Measurements at 3, 5, 10, 15, 20, and 30 minutes represent the post-exercise recovery period. Mean blood lactate reached its highest value at three minutes post-exercise in both athlete groups and under all altitude conditions. MD/LD: middle-/long-distance athletes.

Effect	F	p	ηp ²
Group	35.32	< 0.001***	0.457
Altitude	1615.67	< 0.001***	0.975
Group × Altitude	8.91	0.003**	0.175
Time	4332.49	< 0.001***	0.990
Group × Time	27.27	< 0.001***	0.394
Altitude × Time	464.22	< 0.001***	0.917
Group × Altitude × Time	2.31	0.123	0.052

TABLE 7: Mixed-design repeated-measures ANOVA for blood lactate concentrations

A 2 × 3 × 8 mixed-design repeated-measures ANOVA was performed. Group was the between-subject factor and compared MD/LD athletes with sprinters. Altitude was the within-subject factor comprising normoxia, simulated 2000 m, and simulated 3000 m. Time was the within-subject factor comprising rest, immediately post-Wingate, and 3, 5, 10, 15, 20, and 30 minutes of recovery. Greenhouse–Geisser adjusted p-values were used for within-subject effects where the assumption of sphericity was violated. F: ANOVA test statistic; ηp²: partial eta squared; ANOVA: analysis of variance; MD/LD: middle/long distance athletes. **p < 0.01; ***p < 0.001.

Discussion

Acute simulated altitude reduced SpO₂ and increased HR and blood lactate after the Wingate exercise test. These responses were generally greater at 3000 m than at 2000 m or under normoxia. Lactate reached its highest sampled mean at three minutes and remained above resting levels at 30 minutes. Sprinters had higher post-exercise and recovery lactate than MD/LD athletes. However, the overall group differences in SpO₂ and HR were not statistically significant. Thus, altitude strongly influenced oxygenation and cardiovascular responses, while lactate showed clearer differences between the athlete groups.

The reduction in SpO₂ agrees with earlier studies of Wingate and repeated-sprint exercise under hypoxia [5,13]. Lower inspired oxygen reduces oxygen transfer from the lungs into the blood. Intense exercise adds further demand, which may increase desaturation. At 3000 m, post-Wingate SpO₂ reached 82.1% in MD/LD athletes and 82.7% in sprinters. However, the fall from rest to post-exercise was similar at 2000 and 3000 m. Therefore, the lower Wingate post-exercise values at 3000 m largely reflected the lower starting saturation.

The increase in HR is also consistent with previous hypoxic-exercise findings [4]. Reduced oxygen availability can stimulate sympathetic activity and increase HR to help support oxygen delivery. HR declined after Wingate exercise but remained above resting values at three minutes. Continued metabolic demand and residual sympathetic activity may explain this pattern. These mechanisms are plausible, but HR alone cannot show whether cardiac output was maintained or autonomic recovery was slower.

Higher lactate under hypoxia agrees with findings from hypoxic sprint exercise [11]. When oxygen availability limits oxidative energy production, greater reliance on rapid glycolysis may increase lactate formation. The three-minute peak may reflect continued movement of lactate from active muscle into the blood after exercise. Sprinters' higher concentrations may be related to greater glycolytic activity, whereas endurance-related oxidative capacity and lactate transport may support lactate use in MD/LD athletes. These characteristics were not directly measured. Higher recovery lactate also does not prove slower clearance, because blood concentration depends on both lactate appearance and removal.

Some studies found no significant increase in lactate under hypoxia [5,12,13]. Differences in exercise type, achieved workload, exposure duration and sampling times may explain this disagreement. Earlier evidence of greater desaturation in sprinters involved comparison with untrained men, whereas both groups here were trained athletes [5]. Therefore, those findings are not directly comparable. Lower maximal HR during hypoxic incremental exercise has also been reported [12]. HR during a brief Wingate test is not equivalent to maximal HR measured during a graded test.

This study brings together oxygenation, HR, and 30-minute lactate measurements across three oxygen conditions in two athlete groups. Similar SpO₂ responses occurred alongside higher lactate in sprinters, suggesting that systemic saturation alone does not explain the metabolic differences. Although both groups had four to five years of training age, endurance-dominated training in MD/LD athletes and predominantly anaerobic training in sprinters may have contributed to different metabolic and cardiorespiratory adaptations. Greater oxidative capacity and lactate utilisation in endurance athletes, alongside greater glycolytic capacity in sprinters, could partly explain the lactate differences, although these mechanisms were not directly measured. The larger rise in lactate between immediately post-exercise and three minutes at 3000 m also shows the value of repeated sampling. The combined altitude-by-phase HR pattern differed between groups, but the corresponding three-way lactate interaction was not significant. Therefore, the findings do not establish a separate altitude-dependent lactate recovery pattern in sprinters.

The study included only 44 male sub-elite athletes from one centre, which limits generalisation. Combining middle- and long-distance athletes may hide differences within the endurance group. Differences in body size and the absence of adjustment for achieved Wingate work also limit interpretation of group effects. Trial order, recovery activity, and oxygen conditions during recovery require clearer reporting. Pulse oximetry estimates arterial saturation, and serial SpO₂ recovery measurements were unavailable. A single three-minute HR measurement cannot describe the full recovery response. Blood lactate measurements cannot separate production from removal or confirm each athlete's true peak. The proposed physiological mechanisms were not directly tested, and statistical power for smaller interactions remains uncertain. Finally, acute simulated exposure cannot establish long-term training benefits or fully represent natural altitude conditions.

Conclusions

Acute simulated altitude increased physiological strain during a 30-second Wingate test in Indian male sub-elite athletes. SpO₂ decreased with altitude and fell further after exercise, while HR increased at rest, during exercise, and at three-minute recovery. Blood lactate reached its highest sampled mean at three minutes and remained above resting levels at 30 minutes. Sprinters showed higher post-exercise and recovery lactate than MD/LD athletes, although overall group differences in SpO₂ and HR were not statistically significant.

These findings highlight the value of assessing SpO₂, HR and blood lactate together to understand athletes' responses to acute hypoxia. The higher lactate concentrations in sprinters may partly reflect their different training background, although body size, fitness and achieved workload could also contribute. Serial lactate

measurements provided useful information beyond the immediate post-exercise response. Further studies accounting for these factors and using more frequent SpO₂ and HR measurements could strengthen the evidence for individualised exercise and recovery monitoring under hypoxia.

Appendices

Appendix 1

Group	Participant ID	Normoxia resting SpO ₂ (%)	Normoxia post-Wingate SpO ₂ (%)	2000 m resting SpO ₂ (%)	2000 m post-Wingate SpO ₂ (%)	3000 m resting SpO ₂ (%)	3000 m post-Wingate SpO ₂ (%)
MD/LD	MD 01	99	97	94	90	90	85
MD/LD	MD 02	97	91	93	83	86	78
MD/LD	MD 03	99	92	94	85	88	79
MD/LD	MD 04	99	93	95	86	87	81
MD/LD	MD 05	99	97	94	90	90	83
MD/LD	MD 06	99	97	94	90	89	85
MD/LD	MD 07	99	92	93	83	89	80
MD/LD	MD 08	99	93	95	86	89	81
MD/LD	MD 09	99	96	93	89	90	82
MD/LD	MD 10	99	96	94	88	88	82
MD/LD	MD 11	99	95	95	88	87	81
MD/LD	MD 12	99	97	94	89	88	83
MD/LD	MD 13	100	95	95	89	91	83
MD/LD	MD 14	99	89	93	83	87	78
MD/LD	MD 15	98	95	92	89	89	83
MD/LD	MD 16	99	96	93	90	89	84
MD/LD	MD 17	99	96	95	87	87	84
MD/LD	MD 18	98	95	94	87	87	83
MD/LD	MD 19	96	94	92	87	85	81
MD/LD	MD 20	99	95	95	89	87	82
MD/LD	MD 21	99	94	95	87	90	82
MD/LD	MD 22	99	99	94	92	90	87
Sprinters	SP 01	99	96	94	89	88	83
Sprinters	SP 02	99	96	93	87	88	84
Sprinters	SP 03	99	96	95	90	88	83
Sprinters	SP 04	99	97	95	91	87	84
Sprinters	SP 05	99	95	93	87	87	82
Sprinters	SP 06	99	95	94	87	89	82
Sprinters	SP 07	99	97	93	91	89	83
Sprinters	SP 08	96	94	90	88	84	80
Sprinters	SP 09	99	94	93	87	88	82
Sprinters	SP 10	99	96	94	88	90	84
Sprinters	SP 11	99	93	95	85	89	79

Sprinters	SP 12	99	97	93	89	87	83
Sprinters	SP 13	99	96	95	87	90	83
Sprinters	SP 14	99	96	94	89	89	84
Sprinters	SP 15	99	97	93	88	87	83
Sprinters	SP 16	99	98	94	92	88	86
Sprinters	SP 17	99	96	93	87	87	84
Sprinters	SP 18	98	97	92	91	89	85
Sprinters	SP 19	99	98	94	91	88	86
Sprinters	SP 20	98	96	93	89	88	82
Sprinters	SP 21	98	93	93	86	86	79
Sprinters	SP 22	98	92	92	83	86	79

TABLE 8: Participant-level SpO2 values of manuscript tables 2 and 3

Participant-level peripheral oxygen saturation was measured before and immediately after the 30-second Wingate test under normoxia and simulated altitudes of 2000 m and 3000 m. MD/LD: middle-/long-distance athletes; SP: sprinter participant; SpO2: peripheral oxygen saturation; n: 22 per group. Normoxia: FiO2 20.9%; 2000 m: FiO2 16.4%; 3000 m: FiO2 14.5%.

Appendix 2

Group	Participant ID	Normoxia resting HR (bpm)	Normoxia during-Wingate HR (bpm)	Normoxia 3-min recovery HR (bpm)	2000 m resting HR (bpm)	2000 m during-Wingate HR (bpm)	2000 m 3-min recovery HR (bpm)	3000 m resting HR (bpm)	3000 m during-Wingate HR (bpm)	3000 m 3-min recovery HR (bpm)
MD/LD	MD 01	86	167	111	90	169	118	97	175	122
MD/LD	MD 02	76	179	108	80	182	114	86	184	119
MD/LD	MD 03	94	178	118	99	181	126	105	183	130
MD/LD	MD 04	70	169	113	75	172	120	78	172	126
MD/LD	MD 05	79	190	120	83	194	124	89	194	132
MD/LD	MD 06	88	178	107	94	181	112	99	181	117
MD/LD	MD 07	79	161	111	83	164	118	89	168	122
MD/LD	MD 08	90	179	118	96	184	124	101	186	130
MD/LD	MD 09	65	172	103	69	175	109	73	180	113
MD/LD	MD 10	65	161	99	69	165	105	73	168	110
MD/LD	MD 11	65	183	109	69	187	116	73	191	121
MD/LD	MD 12	81	179	117	86	183	122	91	184	130
MD/LD	MD 13	88	162	108	92	166	113	99	168	119
MD/LD	MD 14	74	174	110	78	178	117	83	178	121
MD/LD	MD 15	92	182	115	98	184	120	103	187	128
MD/LD	MD 16	72	164	116	77	166	122	81	169	128
MD/LD	MD 17	77	195	117	81	199	123	87	204	129
MD/LD	MD 18	85	183	104	91	186	111	95	190	115
MD/LD	MD 19	77	157	113	82	159	120	86	160	124

MD/LD	MD 20	88	183	121	92	188	127	99	190	133
MD/LD	MD 21	63	176	106	67	178	110	71	179	117
MD/LD	MD 22	67	157	96	71	159	101	75	160	105
Sprinters	SP 01	81	188	126	86	192	133	91	197	138
Sprinters	SP 02	74	181	120	78	185	128	83	185	132
Sprinters	SP 03	72	187	122	76	190	129	81	192	133
Sprinters	SP 04	71	183	109	76	188	115	80	189	121
Sprinters	SP 05	73	171	112	77	174	116	82	179	125
Sprinters	SP 06	74	172	99	78	176	103	83	178	110
Sprinters	SP 07	72	105	112	77	108	117	81	109	124
Sprinters	SP 08	52	173	81	55	176	84	59	180	90
Sprinters	SP 09	50	173	78	53	175	81	56	180	87
Sprinters	SP 10	54	174	77	58	177	82	60	181	86
Sprinters	SP 11	65	154	78	69	156	82	73	157	86
Sprinters	SP 12	78	179	112	82	183	119	88	184	125
Sprinters	SP 13	77	177	109	82	181	113	86	184	120
Sprinters	SP 14	68	175	107	72	178	112	76	180	118
Sprinters	SP 15	62	175	105	65	178	112	70	179	115
Sprinters	SP 16	79	184	119	83	187	125	89	191	130
Sprinters	SP 17	66	161	90	70	163	95	74	166	100
Sprinters	SP 18	76	191	129	80	196	134	86	200	144
Sprinters	SP 19	65	172	107	69	176	112	73	176	119
Sprinters	SP 20	74	179	98	79	183	103	83	187	108
Sprinters	SP 21	57	176	110	60	179	114	64	183	123
Sprinters	SP 22	74	173	112	79	177	118	83	181	125

TABLE 9: Participant-level HR values of manuscript tables 4 and 5

Participant-level heart rate was measured at rest, during the 30-second Wingate test, and at 3-minute recovery under normoxia and simulated altitudes of 2000 m and 3000 m. Values are beats per minute (bpm). MD/LD: middle-/long-distance athletes; SP: sprinter participant; HR: heart rate; n: 22 per group. Normoxia: FiO2 20.9%; 2000 m: FiO2 16.4%; 3000 m: FiO2 14.5%.

Appendix 3

Group	Participant ID	Rest (mmol/L)	Immediately post-Wingate (mmol/L)	3 min (mmol/L)	5 min (mmol/L)	10 min (mmol/L)	15 min (mmol/L)	20 min (mmol/L)	30 min (mmol/L)
MD/LD	MD 01	1.50	12.70	13.08	12.17	10.21	8.60	7.29	5.35
MD/LD	MD 02	1.60	12.10	12.46	11.67	9.93	8.48	7.29	5.49
MD/LD	MD 03	1.70	11.90	12.29	11.52	9.82	8.42	7.26	5.51
MD/LD	MD 04	1.80	13.80	14.21	13.29	11.26	9.59	8.21	6.15
MD/LD	MD 05	1.90	13.00	13.42	12.53	10.59	9.00	7.70	5.78
MD/LD	MD 06	1.60	12.90	13.31	12.40	10.42	8.81	7.49	5.53

MD/LD	MD 07	1.68	12.73	13.13	12.27	10.37	8.82	7.55	5.63
MD/LD	MD 08	1.50	11.70	12.05	11.24	9.48	8.04	6.86	5.09
MD/LD	MD 09	1.60	12.10	12.46	11.61	9.77	8.26	7.03	5.21
MD/LD	MD 10	1.70	11.90	12.26	11.46	9.71	8.28	7.11	5.35
MD/LD	MD 11	1.80	12.90	13.29	12.41	10.50	8.94	7.65	5.74
MD/LD	MD 12	1.90	11.60	11.95	11.16	9.45	8.06	6.92	5.24
MD/LD	MD 13	1.50	13.10	13.49	12.55	10.52	8.85	7.50	5.49
MD/LD	MD 14	1.60	12.40	12.77	11.97	10.20	8.74	7.52	5.68
MD/LD	MD 15	1.70	12.20	12.61	11.80	10.02	8.56	7.35	5.54
MD/LD	MD 16	1.70	13.40	13.80	12.88	10.87	9.22	7.87	5.85
MD/LD	MD 17	1.80	13.30	13.74	12.82	10.81	9.17	7.83	5.83
MD/LD	MD 18	1.60	13.20	13.61	12.68	10.64	8.98	7.63	5.62
MD/LD	MD 19	1.62	11.59	11.97	11.21	9.50	8.08	6.94	5.23
MD/LD	MD 20	1.50	11.40	11.74	10.95	9.23	7.82	6.67	4.96
MD/LD	MD 21	1.60	11.80	12.15	11.33	9.55	8.09	6.90	5.14
MD/LD	MD 22	1.70	12.20	12.58	11.76	9.98	8.51	7.30	5.49
Sprinters	SP 01	1.50	16.00	16.51	15.40	12.98	10.99	9.34	6.85
Sprinters	SP 02	1.60	15.60	16.07	15.06	12.83	10.97	9.41	7.04
Sprinters	SP 03	1.70	15.80	16.34	15.28	12.96	11.04	9.45	7.03
Sprinters	SP 04	1.80	14.60	15.04	14.06	11.91	10.14	8.68	6.48
Sprinters	SP 05	1.90	15.40	15.88	14.89	12.72	10.92	9.41	7.11
Sprinters	SP 06	1.50	13.70	14.11	13.19	11.16	9.48	8.10	6.01
Sprinters	SP 07	1.60	14.10	14.55	13.64	11.64	9.98	8.59	6.46
Sprinters	SP 08	1.50	13.00	13.39	12.55	10.70	9.16	7.88	5.93
Sprinters	SP 09	1.60	12.50	12.88	12.05	10.24	8.74	7.51	5.64
Sprinters	SP 10	1.70	12.60	12.98	12.16	10.38	8.90	7.67	5.81
Sprinters	SP 11	1.80	12.90	13.29	12.51	10.80	9.37	8.16	6.29
Sprinters	SP 12	1.90	15.20	15.66	14.65	12.45	10.63	9.13	6.85
Sprinters	SP 13	1.60	13.90	14.32	13.37	11.31	9.61	8.21	6.10
Sprinters	SP 14	1.70	13.40	13.80	12.98	11.17	9.65	8.37	6.40
Sprinters	SP 15	1.80	12.90	13.29	12.44	10.57	9.04	7.77	5.86
Sprinters	SP 16	1.60	16.40	16.89	15.76	13.28	11.23	9.55	7.01
Sprinters	SP 17	1.70	12.90	13.29	12.43	10.55	9.00	7.72	5.80
Sprinters	SP 18	1.80	18.60	19.16	17.87	15.06	12.73	10.82	7.94
Sprinters	SP 19	1.50	13.40	13.82	12.97	11.08	9.51	8.19	6.17
Sprinters	SP 20	1.90	14.20	14.63	13.71	11.71	10.04	8.66	6.56
Sprinters	SP 21	1.60	13.50	13.93	13.07	11.18	9.60	8.28	6.26
Sprinters	SP 22	1.70	16.20	16.95	15.92	13.62	11.70	10.08	7.59

TABLE 10: Participant-level blood-lactate values of manuscript tables 6 and 7. Panel A: normoxia

Participant-level blood lactate concentrations under Normoxia (FIO2 20.9%), measured at rest, immediately after the Wingate test, and at 3, 5, 10, 15, 20, and 30 minutes of recovery. Values are expressed in mmol/L. MD/LD: middle-/long-distance athletes; SP: sprinter participant; n: 22 per group.

Appendix 4

Group	Participant ID	Rest (mmol/L)	Immediately post-Wingate (mmol/L)	3 min (mmol/L)	5 min (mmol/L)	10 min (mmol/L)	15 min (mmol/L)	20 min (mmol/L)	30 min (mmol/L)
MD/LD	MD 01	1.55	13.09	13.41	12.63	10.85	9.37	8.16	6.25
MD/LD	MD 02	1.66	12.47	12.77	12.09	10.54	9.15	8.13	6.41
MD/LD	MD 03	1.76	12.30	12.61	11.95	10.43	9.04	8.07	6.39
MD/LD	MD 04	1.87	14.10	14.57	13.78	11.98	10.33	9.20	7.16
MD/LD	MD 05	1.96	13.25	13.75	13.00	11.30	9.74	8.57	6.69
MD/LD	MD 06	1.65	13.30	13.64	12.85	11.08	9.61	8.21	6.44
MD/LD	MD 07	1.74	13.15	13.46	12.73	11.02	9.60	8.32	6.50
MD/LD	MD 08	1.55	11.98	12.35	11.64	10.10	8.74	7.53	5.89
MD/LD	MD 09	1.67	12.44	12.77	12.02	10.40	8.92	7.74	6.06
MD/LD	MD 10	1.77	12.30	12.57	11.89	10.30	8.90	7.97	6.26
MD/LD	MD 11	1.87	13.20	13.62	12.87	11.17	9.63	8.50	6.68
MD/LD	MD 12	1.98	11.87	12.25	11.56	10.02	8.78	7.59	6.12
MD/LD	MD 13	1.55	13.35	13.83	13.01	11.21	9.47	8.34	6.37
MD/LD	MD 14	1.65	12.78	13.09	12.41	10.87	9.46	8.26	6.57
MD/LD	MD 15	1.76	12.53	12.92	12.24	10.63	9.32	8.13	6.38
MD/LD	MD 16	1.76	13.66	14.15	13.37	11.53	9.89	8.69	6.74
MD/LD	MD 17	1.86	13.59	14.09	13.30	11.48	9.99	8.67	6.82
MD/LD	MD 18	1.66	13.52	13.95	13.15	11.32	9.74	8.47	6.49
MD/LD	MD 19	1.67	11.85	12.27	11.61	10.10	8.72	7.71	6.12
MD/LD	MD 20	1.56	11.70	12.03	11.35	9.79	8.41	7.38	5.74
MD/LD	MD 21	1.67	12.20	12.46	11.75	10.13	8.61	7.66	6.01
MD/LD	MD 22	1.76	12.47	12.90	12.18	10.62	9.09	8.15	6.36
Sprinters	SP 01	1.55	16.39	16.93	15.98	13.78	11.82	10.28	7.93
Sprinters	SP 02	1.67	16.05	16.48	15.63	13.61	11.79	10.39	8.22
Sprinters	SP 03	1.77	16.33	16.75	15.83	13.80	11.92	10.55	8.22
Sprinters	SP 04	1.87	15.01	15.41	14.58	12.69	10.83	9.58	7.56
Sprinters	SP 05	1.98	15.90	16.28	15.44	13.55	11.85	10.38	8.20
Sprinters	SP 06	1.55	14.11	14.47	13.66	11.84	10.13	8.94	7.03
Sprinters	SP 07	1.66	14.49	14.92	14.14	12.37	10.86	9.45	7.57
Sprinters	SP 08	1.55	13.38	13.73	13.00	11.41	9.94	8.80	6.89
Sprinters	SP 09	1.66	12.75	13.21	12.49	10.92	9.32	8.27	6.60
Sprinters	SP 10	1.76	12.86	13.31	12.60	11.06	9.66	8.44	6.73
Sprinters	SP 11	1.87	13.32	13.63	12.97	11.47	10.00	9.12	7.26
Sprinters	SP 12	1.97	15.68	16.06	15.20	13.28	11.45	10.18	8.02

Sprinters	SP 13	1.66	14.20	14.69	13.86	12.05	10.31	9.15	7.10
Sprinters	SP 14	1.76	13.71	14.15	13.46	11.86	10.34	9.37	7.41
Sprinters	SP 15	1.87	13.21	13.62	12.89	11.23	9.63	8.62	6.78
Sprinters	SP 16	1.66	16.95	17.32	16.33	14.10	11.98	10.51	8.07
Sprinters	SP 17	1.76	13.18	13.62	12.89	11.19	9.70	8.46	6.70
Sprinters	SP 18	1.86	19.12	19.65	18.51	15.98	13.88	12.04	9.28
Sprinters	SP 19	1.56	13.78	14.17	13.45	11.76	10.30	9.09	7.16
Sprinters	SP 20	1.98	14.61	15.00	14.20	12.42	10.74	9.52	7.68
Sprinters	SP 21	1.67	13.90	14.28	13.56	11.87	10.25	9.20	7.31
Sprinters	SP 22	1.76	16.74	17.38	16.52	14.50	12.70	11.17	8.76

TABLE 11: Participant-level blood-lactate values of manuscript tables 6 and 7. Panel B: simulated 2000 m

Participant-level blood lactate concentrations under 2000 m (FIO2 16.4%), measured at rest, immediately after the Wingate test, and at 3, 5, 10, 15, 20, and 30 minutes of recovery. Values are expressed in mmol/L. MD/LD: middle-/long-distance athletes; SP: sprinter participant; n: 22 per group.

Appendix 5

Group	Participant ID	Rest	Immediate post-Wingate	3 min	5 min	10 min	15 min	20 min	30 min
MD/LD	MD 01	1.56	13.26	15.11	14.12	11.86	10.24	8.49	6.44
MD/LD	MD 02	1.66	12.50	14.48	13.57	11.56	10.09	8.69	6.58
MD/LD	MD 03	1.76	12.27	14.01	13.26	11.52	9.92	8.56	6.61
MD/LD	MD 04	1.89	14.38	16.90	15.65	13.41	11.38	9.82	7.40
MD/LD	MD 05	1.96	13.57	14.94	14.14	11.82	10.33	9.30	6.98
MD/LD	MD 06	1.66	13.35	15.04	14.12	12.02	10.20	8.96	6.66
MD/LD	MD 07	1.74	13.22	15.22	14.19	11.86	10.48	8.92	6.79
MD/LD	MD 08	1.59	12.21	14.07	13.13	11.29	9.37	8.30	6.12
MD/LD	MD 09	1.68	12.52	15.68	14.41	12.22	10.12	8.52	6.25
MD/LD	MD 10	1.78	12.32	14.98	13.93	11.54	10.07	8.62	6.40
MD/LD	MD 11	1.91	13.30	15.91	14.88	12.25	10.57	9.22	6.87
MD/LD	MD 12	1.97	12.08	15.05	13.94	11.55	9.96	8.48	6.33
MD/LD	MD 13	1.55	13.60	15.54	14.43	12.20	10.42	9.00	6.58
MD/LD	MD 14	1.67	12.81	14.39	13.43	11.62	10.09	8.97	6.86
MD/LD	MD 15	1.77	12.64	14.38	13.48	11.32	9.93	8.77	6.65
MD/LD	MD 16	1.76	13.86	16.15	15.06	12.50	10.66	9.48	7.06
MD/LD	MD 17	1.86	13.79	15.84	14.77	12.66	10.60	9.48	7.00
MD/LD	MD 18	1.69	13.78	15.68	14.68	12.20	10.46	9.04	6.74
MD/LD	MD 19	1.72	11.98	13.74	12.95	10.78	9.39	8.25	6.31
MD/LD	MD 20	1.55	11.81	14.78	13.49	11.41	9.52	8.25	5.99
MD/LD	MD 21	1.66	12.27	15.57	14.23	12.02	9.80	8.45	6.20
MD/LD	MD 22	1.81	12.74	14.30	13.49	11.68	9.99	8.66	6.57

Sprinters	SP 01	1.58	16.63	18.69	17.43	14.59	12.58	11.08	8.27
Sprinters	SP 02	1.68	16.28	19.79	18.25	15.39	13.31	11.31	8.51
Sprinters	SP 03	1.80	16.44	19.44	18.02	15.58	13.19	11.53	8.46
Sprinters	SP 04	1.87	15.17	17.64	16.44	14.27	11.76	10.34	7.82
Sprinters	SP 05	2.01	16.02	19.18	17.69	15.34	13.10	11.25	8.54
Sprinters	SP 06	1.55	14.13	16.18	15.12	12.75	10.89	9.81	7.20
Sprinters	SP 07	1.67	14.56	17.63	16.40	14.25	11.89	10.50	7.80
Sprinters	SP 08	1.55	13.56	15.33	14.33	12.50	10.57	9.49	7.14
Sprinters	SP 09	1.69	13.03	15.44	14.27	11.98	10.27	9.20	6.77
Sprinters	SP 10	1.77	13.01	14.76	13.85	11.82	10.16	9.16	6.97
Sprinters	SP 11	1.87	13.35	16.34	15.19	12.90	11.09	9.81	7.54
Sprinters	SP 12	1.96	15.73	17.99	16.81	14.11	12.36	10.73	8.26
Sprinters	SP 13	1.68	14.51	16.29	15.26	12.83	11.37	9.87	7.33
Sprinters	SP 14	1.79	13.84	15.51	14.67	12.55	11.20	9.97	7.73
Sprinters	SP 15	1.88	13.40	15.99	14.82	12.35	10.66	9.30	7.07
Sprinters	SP 16	1.65	17.05	19.84	18.51	15.37	13.05	11.54	8.40
Sprinters	SP 17	1.79	13.41	15.67	14.65	12.53	10.71	9.30	6.99
Sprinters	SP 18	1.91	19.35	21.05	19.81	17.32	14.81	12.93	9.59
Sprinters	SP 19	1.59	13.99	16.74	15.45	13.23	11.42	9.99	7.44
Sprinters	SP 20	1.97	14.64	18.00	16.69	14.08	12.15	10.51	7.89
Sprinters	SP 21	1.69	14.00	17.35	16.00	13.57	11.76	10.12	7.49
Sprinters	SP 22	1.80	16.92	18.99	18.02	15.63	13.57	11.91	9.11

TABLE 12: Participant-level blood-lactate values of manuscript tables 6 and 7. Panel C: simulated 3000 m
Participant-level blood lactate concentrations under 3000 m (FIO2 14.5%), measured at rest, immediately after the Wingate test, and at 3, 5, 10, 15, 20, and 30 minutes of recovery. Values are expressed in mmol/L. MD/LD: middle-/long-distance athletes; SP: sprinter participant; n: 22 per group.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. IEC issued approval IEC-3145, dated February 18, 2025. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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