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Cardiovascular and Metabolic Responses to Altitude Exposure in Expert Hikers: Implications for Functional Capacity and Rehabilitation in Hypoxic Environments

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Abstract: Transitioning from sea level to low or moderate altitude can influence the cardiovascular and metabolic adaptations of hikers. This study aims to investigate the effect of different altitudes (sea level, low altitude, and moderate altitude) on cardiovascular and metabolic variables in expert mountain-hikers; (2) Methods: The study included 11 male volunteers who underwent measurements at three different locations: Sea level (less than 10m), low altitude (1500m), and moderate altitude (2500m) in Oman, covering 10km each time. Physiological variables such as peripheral oxygen saturation, blood lactate accumulation, blood glucose levels, and heart rate were measured during and after each hike. In addition, step count, step frequency, and hiking duration were also recorded; (3) Results: Findings showed that altitude significantly affected several cardiovascular and metabolic variables. Heart rate and blood lactate accumulation increased with altitude ($p<0.001$), while blood oxygen saturation, blood glucose levels, step length, and hiking speed decreased significantly at higher altitudes ($p<0.001$). Hiking time and step count were longer at higher altitudes ($p<0.001$). The differences in these variables were mostly pronounced at moderate altitude; (4) Conclusions: Our findings suggest that exposure to higher altitudes requires significant physiological adaptations, making it more difficult to maintain physical efficiency and optimal performance due to oxygen deficiency.

Keywords: Hikers; altitude; heart rate; blood lactate; blood glucose; oxygen saturation

1. Introduction

Altitude exposure presents a unique physiological challenge, particularly for individuals engaged in sustained physical activity. With increasing altitude, both atmospheric pressure and the partial pressure of oxygen decrease, affecting cardiovascular, respiratory, and metabolic functions [1]. This reduced oxygen availability impacts cardiovascular and respiratory function by inducing pulmonary vasoconstriction, altering cardiac control, and increasing respiratory rates [2-4]. A common response is elevated heart rate, which becomes more pronounced with greater altitude [5]. During physical exertion, cardiac output normally rises to supply oxygen and nutrients to working muscles, but at altitude this process is compromised, increasing the risk of overtraining and injury due to oxygen deficiency [6]. Furthermore, oxygen saturation declines progressively with altitude due to reduced atmospheric pressure and oxygen tension [7]. Hypoxic stress also accelerates lactate accumulation through anaerobic glycolysis, as glycogen breakdown is enhanced when

oxygen availability is limited [8]. Consequently, altitude exposure is consistently associated with higher respiratory rates, elevated heart rate, and increased blood lactate concentrations-indicators of heightened cardiovascular strain and potential health risks [7].

While the effects of high-altitude exposure (above 2,500 meters) have been extensively studied, the acute responses at low and moderate altitudes, ranging from 1,000 to 2,500 m, remain less understood, particularly among trained populations such as expert hikers [9]. Evidence suggests that even moderate altitudes can induce measurable changes in cardiovascular function, including increases in blood pressure and heart rate, likely mediated by enhanced sympathetic nervous system activity [10]. Furthermore, a short-term stay at moderate altitude (around 2,500 meters) has been associated with elevated blood pressure and heart rate, as well as improvements in lipid profiles and reductions in systemic inflammation, suggesting beneficial metabolic adaptations [10]. Understanding these physiological responses is crucial, as even moderate altitudes can affect oxygen delivery, autonomic regulation, and exercise performance, which in turn influence health and endurance during prolonged physical activity [11, 12]. However, systematic investigations of acute responses across different elevation levels are still limited, and standardized outcome measures are needed [2, 7].

Expert hikers, who regularly engage in sustained physical activity at varying altitudes, represent an ideal population for examining these responses. Understanding how cardiovascular and metabolic variables (such as heart rate, blood lactate, blood glucose, and oxygen saturation) respond during acute exposure to low and moderate altitudes is critical for optimizing performance, safety, and health outcomes during mountain activities. Therefore, this study aimed to investigate the acute effects of low and moderate altitude exposure on these parameters in male expert hikers, providing new insights into the physiological demands of mountain environments and identifying the most appropriate exercise elevations to minimize adverse effects. We hypothesized that different altitudes (i.e., SL, LA, and MA) would significantly affect cardiovascular and metabolic variables in mountain hiking practitioners. The second hypothesis was that the most pronounced physiological changes would be observed at MA (i.e., 2500 meters).

2. Materials and Methods

2.1. Participants

The sample size of 11 participants was established based on an a priori power analysis conducted using G*Power software (Version 3.1, University of Dusseldorf, Germany [13]). This analysis set the power at 0.80, with an alpha level of 0.05, anticipating a moderate effect size (f = 0.30, d = 0.60 and critical F = 3.492). Consequently, eleven volunteer expert male hikers (age: 27.28 ± 3.5 years; height: 1.67 ± 0.05 m; body mass: 57.55 ± 5.02 kg; BMI: 20.81 ± 1.3; experience: 5 ± 2 years) consented to participate in the study. The study sample showed no significant variation and was homogeneous, ensuring more objective and accurate measurement of the variables.

Each participant was thoroughly briefed about the study's procedures, methods, potential benefits, and risks beforehand and signed a consent form to confirm their participation. The experimental protocol was implemented between 20 May and 11 June 2024, in alignment with the ethical standards of the Declaration of Helsinki for human experimentation [14] and received approval from the Ethical Committee of Sultan Qaboos University (OM220050).

2.2. Experimental design and procedures

This study comprised three separate randomized assessments [15], each conducted on different days with minimum 72 hours between assessments. These assessments were consistently conducted on hiking trails at three different altitude levels: sea level (<10 m), low altitude (1500 m), and moderate altitude (2500 m), during

the same time window (17:00–19:00) to control for diurnal variation. Each session consisted of a 10,000 m hiking trial. Participants were required to complete the specified distance using self-effort that simulated walking on mountain trails at all different altitudes. Walking speed was individual and not predetermined for the participants. Environmental conditions were recorded at each altitude level as follows:

a) **Sea level** (SL) [i.e., less than 10m (GPS coordinate: latitude 23.697938250311072°, longitude 58.149409328842786°)] 10,000m hiking trail at Seeb Beach Muscat Oman (Figure 1a). Mean ambient temperature was 33°C, average wind speed was 1.6 ± 0.8 km/h, relative humidity was 41%, and slope angle was 0°.

b) **Low altitude** (LA) [i.e., 1500m (GPS coordinate: latitude 23.079706319322625°, longitude 57.70143247183392°)] 10,000m hiking trail at Ad Dakhiliyah, Nizwa, Oman (Figure 1b). Mean ambient temperature was 28°C, average wind speed was 2.0 ± 0.4 km/h, relative humidity was 23%, and slope angle was 0.04°.

c) **Moderate altitude** (MA) [i.e., 2500m (GPS coordinate: latitude 23.11852743006907°, longitude 57.63673592017937°)] 10,000m hiking trail at Al Jabal Al Akhdar, Nizwa, Oman (Figure 1c). Mean ambient temperature was 21°C, average wind speed was 1.6 ± 0.6 km/h, relative humidity was 11%, and slope angle was 0.115°.

Figure 1. Hiking path of sea level (a), low altitude (b), and medium altitude (c).

All trails were on a quasi-flat path (between 0° and 0.115°) to avoid the impact of trail difficulty on the measured variables. Furthermore, the study sample arrived at the designated altitude by car 24 hours before the experiment to acclimatize and minimize the effects associated with reaching altitude.

In each assessment, cardiovascular and metabolic variables were studied as follows:

- Peripheral oxygen saturation (SpO_2), using a fingertip pulse oximeter (RoHS, China [16]) was measured at the beginning and end of the hike.

- Blood lactate (BLa), using a lactate Pro 2 analyser (Arkray, Japan [17]) was collected at the beginning and end of the hike, 5 minutes after completion.

- Blood glucose (BGl), using a Accu-Chek instant glucose meter (Roche, United State [18]), was collected at the beginning and end of the hike.

- Heart rate (HR), using a Garmin Forerunner 945 (Garmin, United States [19]) was registered at rest (HRrest), at peak during the hike (HRpeak), and recovery (HRrecov) at 1, 3 and 5 minutes after the hike.

In addition, tracking distance, hiking time, hiking velocity, and step numbers (Step No.), were measured using the Garmin Connect Android application (Garmin, United States [19]), via Garmin Forerunner 945 smartwatch, throughout the hike. Hikers were only; no carbohydrate or food supplements were permitted during the hike.

Moreover, the external load was the same for all participants across all stages of the three experiments, as they walked without any external load that could affect the measured variables. The timing and type of food intake were also controlled during the 48 hours preceding the experiments. In addition, participants received sufficient sleep and avoided intense physical exertion during the two days before the experiments.

2.3. Statistical analysis

For statistical analysis, the SPSS 20 package (SPSS, Chicago, IL, USA) program was used. Descriptive statistics (i.e., means ± SD) were calculated for all variables. The effect size was conducted using G*Power software Version 3.1 [13]. The following scale was used for the interpretation of *d*: < 0.2, trivial; 0.2 – 0.6, small; 0.6 – 1.2, moderate; 1.2 – 2.0, large; and > 2.0, very large [20]. The normality of distribution estimated by the Kolmogorov-Smirnov test was acceptable for all variables ($p > 0.05$). Consequently, a one-way repeated-measures ANOVA (i.e., SL, LA, and MA) was used to compare benchmarks across different conditions. Bonferroni test was applied for pairwise comparisons. Additionally, effect sizes (*d*) were determined from ANOVA output by converting partial eta-squared to Cohen's d. An a priori significance level of $p \leq 0.05$ was used as the criterion for significance.

3. Results

The repeated measures ANOVA showed a significant difference ($p < 0.001$; d = 2.069 to 6.067) between altitude levels (i.e., SL, LA, and MA) in all variables except heart rate at rest and step frequencies which are not significant (table 1).

Table 1. One-way repeated measure ANOVA.

Variables	df	Mean Square	F	Sig.	Effect Size	Power
HR-rest	2	44.000	1.515	0.244	0.749	0.284
HR-peak	2	4452.212	54.133	0.000**	4.652	1.000
HR-recov1	2	1110.636	50.477	0.000**	4.499	1.000
HR-recov3	2	363.848	36.573	0.000**	3.821	1.000
HR-recov5	2	247.758	39.176	0.000**	3.962	1.000
ΔRecov3	2	318.058	10.684	0.001**	2.069	0.976
ΔRecov5	2	275.584	14.100	0.000**	2.374	0.995
SpO_2-before	2	27.848	38.941	0.000**	3.951	1.000
SpO_2-after	2	102.091	43.924	0.000**	4.197	1.000
BLa-before	2	2.780	21.502	0.000**	2.935	1.000
BLa-after	2	85.424	45.666	0.000**	4.268	1.000
BGl-before	2	0.593	15.594	0.000**	2.496	0.998
BGl-after	2	1.313	56.925	0.000**	4.779	1.000
Step Number	2	11232000.848	76.765	0.000**	5.548	1.000
Step Length	2	0.020	92.520	0.000**	6.067	1.000
Step Frequency	2	0.000	1.360	0.279	0.738	0.258
Hiking Time	2	296.455	27.062	0.000**	3.288	1.000
Hiking Velocity	2	0.961	23.550	0.000**	3.069	1.000

(HR-rest) Heart rate at rest; (HR-peak) Heart rate peak in the hike; (HR-recov1) Heart rate at 1 min recovery; (HR-recov3) Heart rate at 3 min recovery; (HR-recov5) Heart rate at 5 min recovery; (ΔRecov3) Percentage of recovery at 3 min; (ΔRecov5) Percentage of recovery at 5 min;(SpO_2-befor) Peripheral oxygen saturation befor hiking; (SpO_2-after) Peripheral oxygen saturation after hiking; (BLa-befor) Blood lactate concentration before hiking; (BLa-after) Blood lactate concentration after hiking; (BGl-befor) Blood glucose concentration before hiking; (BGl-after) Blood glucose concentration after hiking; (*) Significant at $p < 0.05$; (**) Significant at $p < 0.01$.

Pairwise comparison showed an increase in heart rate (i.e., HR-peak and HR-recovery), blood lactate, hiking time, and step number when increasing altitude level (figure 2a, 2b, 2c, and 2d). In contrast, blood glucose, SpO_2, recovery percentage at 3 and 5 minutes, step length, and hiking speed decreased significantly when increasing altitude level (table 2, figure 3a, 3b, 3c, 3d).

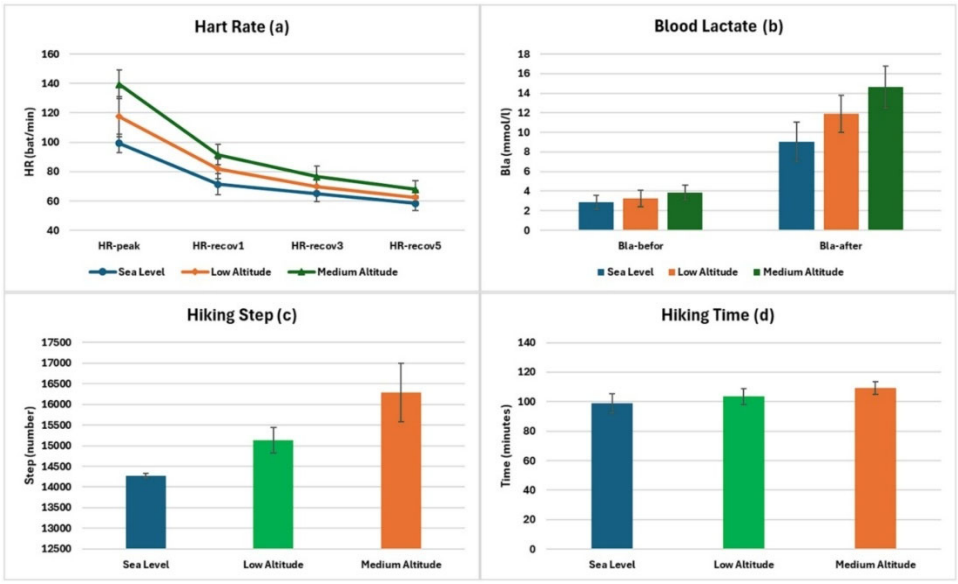

Figure 2. Variables increased with increasing altitude level; (a) heart rate peak and recovery; (b) blood lactate; (c) hiking steps; (d) hiking time.

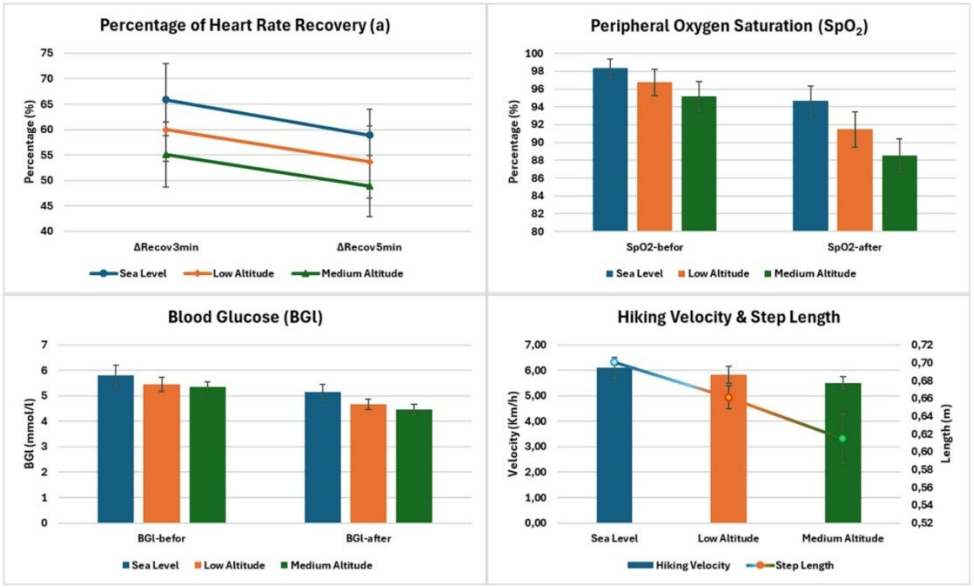

Figure 3. Variables decreased with increasing altitude level; (a) percentage of heart rate recovery; (b) peripheral oxygen saturation; (c) blood glucose; (d) velocity and step length.

Table 2. Pairwise comparison between altitude levels.

Measure		Mean Diff.	SE Diff.	Sig.	Effect Size
HR-peak	SL vs. LA	-18.273	4.571	0.003**	3.997
	SL vs. MA	-40.182	2.990	0.000**	8.052
	LA vs. MA	-21.909	3.876	0.000**	5.652
HR-recov1	SL vs. LA	-10.455	2.217	0.001**	4.715
	SL vs. MA	-20.091	1.937	0.000**	6.840
	LA vs. MA	-9.636	1.825	0.000**	5.281
HR-recov3	SL vs. LA	-4.818	1.102	0.001**	4.372
	SL vs. MA	-11.455	1.681	0.000**	6.814
	LA vs. MA	-6.636	1.178	0.000**	5.633
HR-recov5	SL vs. LA	-4.000	0.944	0.002**	4.237
	SL vs. MA	-9.455	1.209	0.000**	6.820
	LA vs. MA	-5.455	1.048	0.000**	5.205
ΔRecov3	SL vs. LA	5.866	2.830	0.065	0.897
	SL vs. MA	10.739	2.382	0.001**	4.612
	LA vs. MA	4.873	1.599	0.012*	3.047
ΔRecov5	SL vs. LA	5.281	2.331	0.047*	1.594
	SL vs. MA	10.005	1.543	0.000**	6.484
	LA vs. MA	4.724	1.688	0.019*	1.757
SpO_2-befor	SL vs. LA	1.636	0.244	0.000**	6.704
	SL vs. MA	3.182	0.423	0.000**	6.084
	LA vs. MA	1.545	0.390	0.003**	3.961
SpO_2-after	SL vs. LA	3.182	0.464	0.000**	6.587
	SL vs. MA	6.091	0.732	0.000**	8.321
	LA vs. MA	2.909	0.719	0.002**	4.045
BLa-befor	SL vs. LA	-0.409	0.166	0.033*	1.537
	SL vs. MA	-1.000	0.148	0.000**	6.567
	LA vs. MA	-0.591	0.146	0.002**	4.047
BLa-after	SL vs. LA	-2.864	0.565	0.000**	5.069
	SL vs. MA	-5.573	0.610	0.000**	6.136
	LA vs. MA	-2.709	0.573	0.001**	4.727
BGl-befor	SL vs. LA	0.336	0.090	0.004**	3.733
	SL vs. MA	0.445	0.100	0.001**	4.449
	LA vs. MA	0.109	0.051	0.059	0.938
BGl-after	SL vs. LA	0.473	0.074	0.000**	6.391
	SL vs. MA	0.673	0.078	0.000**	6.628
	LA vs. MA	0.200	0.033	0.000**	6.060
Step Number	SL vs. LA	-855.818	83.520	0.000**	6.113
	SL vs. MA	-2013.455	205.838	0.000**	6.781
	LA vs. MA	-1157.636	174.540	0.000**	6.632
Step Lenght	SL vs. LA	0.039	0.004	0.000**	6.975
	SL vs. MA	0.086	0.008	0.000**	6.107
	LA vs. MA	0.046	0.007	0.000**	6.571
Hiking Time	SL vs. LA	-4.636	1.260	0.004**	3.679
	SL vs. MA	-10.364	1.754	0.000**	5.908
	LA vs. MA	-5.727	1.145	0.001**	5.001
Hiking Velocity	SL vs. LA	0.282	0.079	0.005**	3.567
	SL vs. MA	0.591	0.109	0.000**	5.422
	LA vs. MA	0.309	0.064	0.001**	4.828

* (SL) Sea level; (LA) Low altitude; (MA) Medium altitude; (HR-rest) Heart rate at rest; (HR-peak) Heart rate peak in the hike; (HR-recov1) Heart rate at 1 min recovery; (HR-recov3) Heart rate at 3 min recovery; (HR-recov5) Heart rate at 5 min recovery; (ΔRecov3) Percentage of recovery at 3 min; (ΔRecov5) Percentage of recovery at 5 min;(SpO_2-befor) Peripheral oxygen saturation before hiking; (SpO_2-after) Peripheral oxygen saturation after hiking; (BLa-befor) Blood lactate concentration before hiking; (BLa-after) Blood lactate concentration after hiking; (BGl-before) Blood glucose concentration before hiking; (BGl-after) Blood glucose concentration after hiking; (*) Significant at $p < 0.05$; (**) Significant at $p < 0.01$.

4. Discussion

The aim of this study was to investigate the acute effects of varying altitude levels (i.e., SL, LA, and MA) on cardiovascular and metabolic indicators in hikers. Our results demonstrated a direct correlation between altitude and parameters such as HR, BLa, step number, and walking duration. Conversely, an inverse relationship was observed with SpO_2, BGl, and step length. Numerous studies support the idea that exposure to low and moderate altitudes significantly influences cardiovascular and metabolic variables, particularly in experienced mountain climbers and physically active individuals [3, 21]. At these altitudes, physiological adaptations occur in response to reduced atmospheric pressure, which decreases oxygen availability in inspired air, thereby affecting oxygen delivery and utilization [22]. The magnitude and nature of these effects can vary considerably depending on the specific altitude level and duration of exposure [22].

Our results revealed statistically significant differences in heart rate (HR) variables ($p < 0.01$) during exercise and across all three recovery periods among the three altitude conditions (i.e., SL, LA, and MA). Resting HR remained unchanged, confirming equivalent baseline physiological states at trial onset. Strikingly, altitude modulated HR responses during exercise and recovery (HR-peak, HR-recov1, HR-recov3, HR-recov5), with MA eliciting the most pronounced elevations. HR peaked at MA (139.45 ± 9.61 bpm), indicating substantially higher cardiovascular strain versus LA and SL (117.51 ± 13.59 bpm and 99.27 ± 6.31 bpm respectively). This pattern underscores MA's greater metabolic challenge, driven by hypobaric hypoxia that impairs oxygen diffusion, heightens sympathetic outflow, and accelerates HR to optimize cardiac output and tissue oxygenation amid escalating energetic demands [23]. In addition, HR recovery at 1, 3, and 5 min post-exercise showed the slowest rates at MA, where the highest mean values were recorded compared with the other two altitude conditions (i.e., 91.55 ± 7.07 bpm, 81.91 ± 6.77 bpm, and 71.45 ± 7.09 bpm at 1 min; 76.55 ± 7.39 bpm, 69.91 ± 4.91 bpm, and 65.09 ± 5.45 bpm at 3 min; 67.82 ± 5.93 bpm, 62.36 ± 5.18 bpm, and 58.36 ± 4.98 bpm at 5 min, respectively MA, LA, and SL). This delayed recovery may reflect reduced oxygen availability at moderate altitude, which elevates HR to sustain oxygen delivery to working muscles during and after exercise. These findings align with Netzer et al. [23], who reported HR increases with altitude as a compensatory response to hypoxia. Such adaptations mirror established literature on hypoxia-triggered autonomic shifts and HR escalation at altitude [24, 25].

Additionally, SpO_2 revealed a significant decrease in post-exercise at all altitudes (i.e., SL, LA, and MA) ($p < 0.01$), with MA showing the lowest mean SpO_2 (i.e., 88.55%). Consequently, our study showed that as altitude increases, SpO_2 decreases, accompanied by a noticeable increase in HR to compensate for the oxygen required by tissues and muscles to continue physical activity (table 1; figures 1a and 2b). Many studies have confirmed this and established that the decline in SpO_2 is inversely correlated with HR increases, consistent with compensatory cardiovascular responses to tissue hypoxia [26-29]. Additionally, Zupet et al. [30] and Aebi et al. [31] confirmed that reduced atmospheric pressure at high altitudes leads to oxygen deficiency that influences HR variability.

Additionally, BLa and BGl both showed significant changes ($p < 0.01$) from pre- to post-exercise (table 1; figure 1b). Furthermore, comparisons among the altitude levels (i.e., SL, LA, and MA) revealed significant differences ($p < 0.01$) in post-exercise Bla (14.618 ± 2.17 mmol/L) and BGl (4.46 ± 0.20 mmol/L), which peaked at MA, indicating greater metabolic stress. Therefore, at higher altitudes, the reduced oxygen availability limits reliance on aerobic metabolism, increasing the dependence on glucose utilization for energy production during exercise. This metabolic adaptation poses greater challenges in maintaining energy homeostasis, which is crucial for optimal performance under hypoxic conditions. Recent research supports these find-

ings, showing that hypoxia during exercise stimulates increased carbohydrate oxidation and influences blood glucose regulation, highlighting the heightened risk of exercise-induced hypoglycemia at high altitudes [32].

The results demonstrate that step number, step length, velocity, and hiking time significantly differed across SL, LA, and MA trials ($p < 0.01$), while step frequency remained unchanged. Post-hoc analysis confirmed that MA had the most pronounced effects, characterized by the highest step number (16,288.73 ± 707.73), shortest step length (0.615 ± 0.03 m), longest completion time (109.09 ± 4.41 min), and lowest step velocity (5.51 ± 0.24 m/s). In fact, increasing altitude was associated with shorter step lengths and longer walking duration, likely due to increased fatigue from oxygen deprivation. Our findings are consistent with prior studies demonstrating that altitude-induced hypoxia impairs locomotor efficiency and endurance through physiological adaptations to decreased oxygen pressure [11, 12, 33]. Moreover, altitude-related reductions in oxygen trigger oxidative stress, potentially compromising cellular function and overall performance during high-intensity exercise [34].

Beyond exercise and altitude physiology, this study offers important implications for rehabilitation medicine. The findings illuminate cardiopulmonary reserve under hypoxic conditions, showing how tissue oxygenation capacity is compromised at moderate altitude. Oxygen delivery during exercise is directly affected by reduced atmospheric pressure, impacting rehabilitation program planning for patients with limited cardiopulmonary reserves. Exercise tolerance monitoring (HR, SpO_2, walking parameters) provides clinical indicators for assessing functional capacity. Physiological assessment during rehabilitation could integrate these parameters (HR, SpO_2, BLa, step length) to monitor adaptation to progressive exercise loads. Finally, results inform cardiopulmonary rehabilitation under hypoxic conditions: moderate altitude may serve as a controlled stimulus to improve endurance but requires cautious application and rigorous monitoring of oxygen delivery and exercise tolerance in patients with reduced cardiopulmonary reserve.

This study has several limitations that should be considered when interpreting the findings. First, exercise intensity was self-selected by the participants, which may have resulted in differences in pacing and limited strict control of workload. In addition, environmental conditions such as temperature and humidity may have varied slightly between the different altitude locations, despite efforts to standardize the testing procedures. Another limitation is that continuous monitoring of oxygen consumption was not conducted, which restricted the precise evaluation of exercise intensity and energy expenditure. Finally, the inclusion of only male participants may limit the extent to which these results can be generalized to other populations.

5. Conclusions

This study demonstrates that altitude significantly influences cardiovascular and metabolic responses in expert hikers. Exposure to moderate altitudes (1500m and 2500m) elicited distinct physiological adaptations: heart rate (HR) and blood lactate (BLa) accumulation increased progressively, while SpO_2, blood glucose (BGl), step length, and walking speed decreased. The most pronounced effects occurred at 2500m, characterized by elevated HR (139.45 ± 9.61 bpm), reduced SpO_2 (88.55%), and extended hiking duration.

These findings have practical implications for mountain activities and cardiopulmonary rehabilitation. Hikers should assess physical capacity before ascending to higher altitudes and implement gradual adaptation strategies to mitigate oxygen deprivation risks. Clinically, HR, SpO_2, and walking parameters serve as valuable indicators for monitoring exercise tolerance and functional capacity under hypoxic conditions.

Future research should investigate continuous oxygen consumption monitoring during altitude exposure, examine sex-specific responses (given our male-only sample), and explore the therapeutic potential of controlled hypoxic training for cardiopulmonary rehabilitation programs.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Conflicts of Interest: The authors declare no conflict of interest.

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