

Short Communication

Dynamics of Sleep Architecture and Nocturnal Oxygen Saturation During Acclimatization at Intermediate Altitude: A Longitudinal Observational Study in Elite Athletes

Giorgio Varesco^{1,2,3,*}, Jeroen Van Cutsem^{4,5}, Nathalie Pattyn^{1,4}, Nicolas Bourrel³, François Bieuzen^{3,†}, Guido Simonelli^{1,2,6,†}

¹Center for Advanced Research in Sleep Medicine, Hôpital du Sacré-Coeur de Montréal, Santé Québec Nord-de-l'Île-de-Montréal Universitaire, Montréal, QC H4J 1C5, Canada

²Department of Medicine, Université de Montréal, Montréal, QC H3T 1J4, Canada

³National Institute of Sport of Québec, Montréal, QC H1V 3N7, Canada

⁴VIPER Research Unit, LIFE Department, Royal Military Academy, 1000 Brussels, Belgium

⁵Human Physiology and Sports Physiotherapy Research Group, Faculty of Physical Education and Physiotherapy, Vrije Universiteit Brussel, 1050 Brussels, Belgium

⁶Department of Neuroscience, Université de Montréal, Montréal, QC H3T 1J4, Canada

*Correspondence: giorgio.varesco@umontreal.ca (Giorgio Varesco)

†These authors contributed equally.

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Abstract

Background: Athletes typically undergo training camps living and sleeping at intermediate altitudes (1500–2500 m). Hypoxia reshapes sleep through central circuits regulating arousal, chemoreflex gain and sympathetic outflow. However, the relationship between peripheral oxygen saturation (SpO₂) and sleep architecture and how it co-evolves during acclimatization, is unclear. **Methods:** We tested whether SpO₂ differs across sleep stages at intermediate altitude and whether its recovery is stage specific. Seventeen mogul-ski athletes (seven women) travelled from Montréal (Canada) to Tignes (France) for a 2-week altitude training camp (AltitudeCamp; 2100 m) in June 2025. During the first week (6 nights) and the last night of AltitudeCamp, athletes were provided with a validated ring pulse oximeter and an electroencephalography (EEG) headband. We evaluated the changes in sleep stage distribution across successive nights, scored on 30-second epochs and synchronized with oximetry data. Model estimates are reported as mean ± Standard Error (SE), raw data as mean ± Standard Deviation (SD). **Results:** Average total sleep time was 429 ± 47 min (mean ± SD) and did not change during AltitudeCamp ($p = 0.939$). Across the first week, %N3 decreased logarithmically ($\beta = -2.35 \pm 0.72\%/ \ln(\text{night})$, $p = 0.002$) and %Rapid eye movement sleep (REM) increased logarithmically ($\beta = 3.13 \pm 0.88\%/ \ln(\text{night})$, $p < 0.001$), with no changes in %Wake, %N1, or %N2. SpO₂ was lower during AltitudeCamp ($95.2 \pm 0.3\%$) than measures obtained in normoxia ($98 \pm 0.4\%$; $p < 0.001$), and overnight heart rate (HR) was 7 ± 2 beats per minute (bpm) higher than the habitual 58 ± 3 bpm observed in normoxia ($p = 0.005$). Wake SpO₂ increased by 0.13 ± 0.01 percentage points per night ($p < 0.001$), with faster recovery in N1 (0.19 ± 0.02 percentage points/night; $p = 0.006$) and REM (0.23 ± 0.01 percentage points/night; $p < 0.001$), while N2 and N3 did not differ from Wake. HR declined by 0.79 bpm/night during Wake, similar to N3 ($p = 0.26$), but larger than the decline in N1 ($p = 0.024$) and REM ($p < 0.001$). In N2, HR did not evolve during the first week at altitude. **Conclusions:** At intermediate altitude, nocturnal SpO₂ and HR show sleep-stage-specific dynamics that mirror central ventilatory and autonomic adaptation to hypoxia. Our data suggest that acclimatization is not uniform across sleep stages, and that the first 2 nights after ascent may capture the most rapid adjustments despite similar total sleep duration. Sleep therefore offers a heterogeneous and ecologically valid index of central nervous system (CNS) acclimatization, which can be monitored longitudinally with wearable EEG–oximetry.

Keywords: hypoxia; wearable electronic devices; acclimatization; REM; athletes

1. Introduction

More than 100 million people transiently visit high altitude each year for recreation, sport, or work. Exposure above approximately 1500 m can impose a substantial hypoxic load on cardiorespiratory control. Hypoxemia engages a fine-tuned respiratory control network that begins with peripheral carotid-body afferents projecting via the glossopharyngeal nerve to the nucleus of the solitary tract,

and then recruits pontomedullary circuits that shape ventilatory and sympathetic output [1,2]. Central chemosensitive regions, including the retrotrapezoid nucleus, contribute to the hypercapnic ventilatory responses, while higher-order brain networks involved in interoception and affective processing help shape the conscious experience of dyspnea, effort, and fatigue during hypoxemia [2,3]. Accordingly, hypoxemia can increase fatigability, which in turn could in-



crease the risk of accidents and injuries at altitude, especially with insufficient rest [4]. Sleep is essential for recovery, but altitude perturbs sleep architecture and breathing stability in unacclimatized individuals, with the strongest and most consistent effects typically appearing during the first nights after ascent [5,6]. During sleep, ventilatory responses to hypoxemia and hypercapnia are reduced compared with wakefulness. Rapid eye movement sleep (REM) sleep is particularly unstable, and it is characterized by irregular patterns and periodic breathing [7]. In contrast, non-rapid eye movement (NREM) sleep is characterized by more stable autonomic control, greater parasympathetic predominance, and lower sympathetic outflow [8,9].

Sleep at altitude therefore offers a unique probe of central state-dependent regulation of chemoreflex and autonomic control. Nussbaumer-Ochsner et al. [10] reported progressive recovery of REM and slow-wave sleep across three nights at 4559 m, accompanied by a rise in peripheral oxygen saturation (SpO_2), and Ju et al. [11] showed that nocturnal oxygenation increased while periodic breathing persisted, suggesting dissociable time-courses for ventilatory drive and arousal-related instability. However, previous polysomnographic work at altitude has largely been limited to high altitudes (>2500 m) and to one or two nights [5,6]. This lack of studies on sleep architecture and acclimatization could result from the difficulty in implementing polysomnographic electroencephalography (EEG) for multiple nights, which requires specialized equipment and trained personnel. Recent work has focused on validating Portable EEG devices against polysomnography [12], enabling more cost-effective and ecologically valid longitudinal monitoring in unsupervised settings. Importantly, they facilitate simultaneous recording in multiple participants. It is not unusual that large groups of individuals move collectively to altitude to sojourn temporarily. One typical case is athletes undergoing altitude training camps taking place at intermediate altitude, either to practice winter sports or to seek physiological adaptation favoring endurance performance [13]. Focusing on intermediate altitude is also relevant for individuals visiting mountain regions for leisure or work, as infrastructure or permanent mountain settlements are scarce above 2500 m.

This study investigates whether acclimatization of nocturnal SpO_2 is sleep-stage dependent at intermediate altitude. We used recent multi-participant, dry-electrode validated EEG technology [12] coupled with a ring pulse oximeter in elite ski-mogul athletes during a 2-week altitude training camp at 2100 m. We hypothesized that (i) SpO_2 would differ across sleep stages, consistent with stage-dependent ventilatory response and autonomic outflow, and that (ii) SpO_2 recovery across successive nights would be stage-specific, reflecting differential plasticity of central neural circuits regulating breathing and sympathetic tone during sleep.

2. Materials and Methods

2.1 Participants and Design

Seventeen youth elite athletes of the National Freestyle Moguls team of Québec, Canada (7 women; 18 ± 2 years; 69 ± 10 kg; 175 ± 10 cm; 9 ± 2 years of competitive experience) participated in this observational study. Screening questionnaires excluded clinically meaningful sleep or vigilance disturbance (Pittsburgh Sleep Quality Index 5 ± 2 ; Insomnia Severity Index 6 ± 4 ; Epworth 7 ± 3 ; Caen chronotype V2 $58 \pm 16\%$; Brugmann 3 ± 1 mental item/ 3 ± 2 physical item). Exclusion criteria were epilepsy, cardiac disease, or any condition increasing risk at altitude. Athletes travelled from Montréal to Tignes (France) for a 2-week training camp (AltitudeCamp, June 2025). The first and last days were travel days. Thus, AltitudeCamp refers to the twelve nights spent sleeping at 2100 m (Tignes-Le-Lac). Athletes trained in the morning (8 AM–12 PM) on the Grande-Motte glacier (2800–3456 m). Measures presented in this short communication covered all nights of the first week of AltitudeCamp (i.e., nights 1 to 6), plus the last night (i.e., night 12). For comparison, data at baseline (i.e., normoxia) were also collected during a summer training block in Beauport, Québec (2 nights, altitude: 150 m). The study reporting follows Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) (see **Supplementary Material** for completed checklist).

2.2 Assessment Tools

SpO_2 , heart rate (HR) and activity counts were recorded at 1 Hz by a validated medical-grade ring oximeter (O_2Ring , Viatom Technology Co., Ltd., Shenzhen, China; FDA K242876; accuracy $\pm 2\%$ at 80–100% SpO_2 [14]). Sleep was recorded with the Muse-S headband (MS-02, InteraXon Inc., Toronto, Canada), a four-channel dry-electrode EEG (Fp1, Fp2, Tp9, Tp10, referenced to Fpz). Automated sleep staging (InteraXon v2 algorithm) was validated against level-1 polysomnography (Cohen's $\kappa = 0.76$; accuracy 88–96%; sensitivity 79–92%; specificity 90–99%) [12]. Per manufacturer recommendation, nights with a signal-quality index <0.6 AU or valid recording time $<70\%$ of the lights-off/lights-on interval were excluded. Both devices transmitted to an iPhone 8 (Apple Inc., Cupertino, CA, USA) provided to each participant, which generated the same absolute timestamps.

2.3 Data Processing and Statistical Analysis

Sleep-stage percentages were computed as the proportion of 30-s epochs scored in each stage over time-in-bed. Total sleep time and sleep efficiency were also computed. Oximetry was averaged into 30-s bins aligned to EEG epochs via absolute timestamps (illustrative synchronized recording is available in **Supplementary Material**). Night-in-study was indexed from night 1 to 6 of AltitudeCamp, and night 12, i.e., the nights recorded; time-of-night was expressed in hours from recording start. Nights were also sep-

arated in two-halves to inform overnight macrovariations. Analyses were performed in R (version 4.5.2, R Foundation for Statistical Computing, Vienna, Austria) with data modelled using linear mixed models. Models and fit details can be found in supplemental materials. First, condition (Baseline vs AltitudeCamp) effects on SpO₂, HR and sleep stages were estimated. Second, night-half effects were tested. Third, trajectories across AltitudeCamp nights were modelled by selecting between linear, quadratic, logarithmic and cubic functional forms via Akaike Information Criterion (AIC). Finally, stage×night interactions were fitted. The stage×night model was restricted to nights 1–6 for the primary inference on acclimatization and because of the large missing interval between night 6 and 12. The significance threshold was set at $\alpha = 0.05$. Model estimates are reported as mean $\pm$ standard error (SE); raw data are presented as mean $\pm$ standard deviation (SD).

3. Results

Of 153 expected nights, 118 were recorded (35 lost to technical failure) and 66 passed quality control (15 athletes with ≥ 1 valid night; 67,285 synchronized 30-s epochs; 55,691 in AltitudeCamp). Examples of technical failures or low data quality are low device battery, synchronization errors or headband displacing during the night. Residuals showed minor tail departures attributable to episodic desaturations (minimum SpO₂ = 87%), but evaluation of Wald and model-based confidence intervals confirmed inference robustness. Full analysis output is available at <https://doi.org/10.17605/OSF.IO/3PMYF>.

3.1 Sleep Architecture Trajectories Across AltitudeCamp

Total sleep time estimated by the model was 429 ± 10 min (mean $\pm$ SE) and did not change across AltitudeCamp nights ($\beta = -0.15 \pm 1.9$ min/night; $p = 0.939$). AIC-guided selection indicated that Wake, N1 and N2 were best fit by non-significant linear terms (all $p \geq 0.365$). N3 decreased logarithmically ($\beta = -2.35 \pm 0.72\%/ \ln(\text{night})$; $p = 0.002$) and REM increased logarithmically ($\beta = 3.13 \pm 0.88\%/ \ln(\text{night})$; $p < 0.001$), with the steepest change across nights 1 and 2 (Fig. 1A; Table 1). Night-half contrasts showed more N2 ($5.41 \pm 1.94\%$; $p = 0.006$) and REM ($5.49 \pm 1.64\%$; $p = 0.001$) in the second half and more N3 in the first half ($-12.1 \pm 1.75\%$; $p < 0.001$), with no differences for Wake or N1 (Fig. 1B). Trajectories for SpO₂ and HR averaged over the whole duration of each night are presented in the **Supplementary Material**.

3.2 Stage-Specific Signatures in SpO₂ and HR

Mean SpO₂ in AltitudeCamp was $95.2 \pm 0.3\%$, or 3.1 ± 0.3 percentage points below Baseline ($98 \pm 0.4\%$; $p < 0.001$), and nocturnal HR was 6.62 ± 2.27 bpm higher ($p = 0.005$); overnight activity counts did not differ across conditions ($p = 0.979$). In normoxia, SpO₂ did not vary with stage (all $p \geq 0.11$). In AltitudeCamp, a clear hierarchy

emerged relative to Wake ($95 \pm 0.3\%$): N1 = -0.32 ± 0.10 percentage points, REM = -0.44 ± 0.06 percentage points, N2 = -0.52 ± 0.05 percentage points, N3 = -0.67 ± 0.05 percentage points (all $p \leq 0.018$). HR in AltitudeCamp showed the greatest reduction from Wake in N2 (-6.82 ± 0.34 bpm; $p < 0.001$), with smaller stage differences in N1, N3 and REM (condition×stage interaction $p < 0.001$).

3.3 Stage-Specific Changes in SpO₂ and HR Across Nights at AltitudeCamp

Wake SpO₂ rose by 0.132 ± 0.010 percentage points per night ($p < 0.001$). REM-associated SpO₂ increased the fastest (0.225 percentage points per night; interaction $\beta = 0.093 \pm 0.012$; $p < 0.001$). N1 also increased (0.186 percentage points per night; $\beta = 0.054 \pm 0.020$; $p = 0.006$); N2 and N3 did not differ from Wake (all $p \geq 0.064$; Fig. 2A). HR in Wake decreased by 0.79 bpm/night. Compared to Wake, the decline in HR was similar in N3 (-0.71 bpm/night; night×stage interaction $p = 0.26$); and smaller in N1 (-0.51 bpm/night; interaction $p = 0.024$), and REM (-0.47 bpm/night; interaction $p < 0.001$). In N2, HR was approximately stable across nights (0.05 bpm/night), indicating a departure from the Wake slope (interaction: $p < 0.001$; Fig. 2B). Activity count was lowest in N3 and decreased across camp, with sleep-stage×night interactions (all $p \leq 0.008$) indicating progressive difference across stages.

4. Discussion

This was the first study investigating the evolution and relationship between sleep stages and SpO₂ at intermediate altitude. Another novel aspect is the simultaneous recording across multiple athletes. The main results show that (i) the distribution of the sleep stages changed during AltitudeCamp, with an estimated total ~6% decrease in N3 (slow wave sleep) across the 12 days and a ~8% increase in REM sleep, with the most rapid changes occurring during the first two nights; (ii) SpO₂ increased by ~1% from day 1 to day 12; and (iii) HR acclimatization varied accordingly to the sleep stage, remaining stable only in N2 across nights and declining in all other phases.

4.1 Blood Saturation Differs Across Sleep Stages During an Altitude Training Camp

At Baseline (150 m), SpO₂ was stage-independent because arterial oxygen tension sits on the flat portion of the oxyhemoglobin dissociation curve. In AltitudeCamp, SpO₂ declined progressively from wakefulness through REM and NREM, reaching the lowest values in N3. Because pulse oximetry is subject to measurement error, especially at lower saturation values, these results should be interpreted as group-level trends rather than as clinically meaningful changes for any single reading. This pattern is consistent with the known shift from wake-related cortical and behavioral ventilatory drive to the automatic brainstem respiratory network, including the NTS, ventral res-

Table 1. Sleep data across nights of baseline and the altitude training camp.

Condition	Night	n	TST (min)	Sleep efficiency (%)	Wake (%)	N1 (%)	N2 (%)	N3 (%)	REM (%)
Baseline	1	5	442 ± 35 [386; 473]	87 ± 5 [79; 92]	13 ± 4.8 [7.7; 20]	4.1 ± 1.5 [2.5; 6.5]	42.8 ± 4.1 [37.7; 48.2]	19.4 ± 2 [17.5; 21.9]	20.7 ± 4.4 [16.3; 27.1]
	2	6	477 ± 40 [417; 539]	89 ± 5 [83; 94]	11.3 ± 5.1 [5.9; 17.4]	2.8 ± 0.6 [2.1; 3.9]	44.3 ± 4.3 [39.1; 52.2]	17.6 ± 3.5 [14; 22.1]	24 ± 4.5 [20.1; 30.7]
Altitude camp	1	8	418 ± 45 [355; 482]	91 ± 7 [76; 96]	9.2 ± 6.7 [3.8; 23.6]	3 ± 1.6 [1.5; 6.2]	44.9 ± 8.8 [32.6; 55.6]	24.9 ± 4.2 [19.5; 30.1]	18 ± 5.7 [11.4; 26.8]
	2	13	443 ± 40 [358; 497]	89 ± 8 [73; 97]	9.5 ± 6.3 [3.3; 26.7]	3.6 ± 4.2 [1.5; 17.5]	45.9 ± 5.6 [34.8; 55.6]	20.6 ± 6.4 [11.4; 35.6]	20.5 ± 5.3 [9.6; 29.4]
	3	9	418 ± 58 [303; 482]	88 ± 9 [65; 95]	8.5 ± 3 [3.7; 12.5]	3.6 ± 1.4 [2; 5.7]	44.9 ± 4.8 [37.2; 53.6]	20.1 ± 4.2 [15.2; 28.8]	23 ± 2.5 [19.5; 28.2]
	4	7	423 ± 55 [310; 487]	82 ± 11 [63; 93]	10.8 ± 6 [5.2; 19.5]	3.6 ± 1.9 [2.3; 7.9]	42.7 ± 5.2 [35.8; 48.7]	20.6 ± 3.7 [14.7; 25.1]	22.2 ± 5.7 [12.5; 28.2]
	5	6	453 ± 34 [405; 489]	89 ± 6 [78; 97]	9.1 ± 3.6 [3.5; 13.3]	4.4 ± 2 [2.3; 7.7]	44.2 ± 3.6 [38.9; 49.8]	20 ± 4.1 [16; 27.5]	22.3 ± 5.6 [13.9; 28.8]
	6	7	407 ± 33 [352; 442]	88 ± 9 [72; 96]	11.4 ± 8.4 [4.5; 28.5]	3.5 ± 2.2 [1.7; 8.3]	44.4 ± 4.8 [37.3; 49.7]	18.1 ± 3.3 [13.3; 22.5]	22.6 ± 6.6 [12.5; 29.7]
	12	7	430 ± 57 [336; 492]	89 ± 8 [73; 97]	9.2 ± 4.3 [3; 13.8]	3.2 ± 1.6 [1.5; 6.4]	43.1 ± 7.6 [34.7; 56.2]	18.6 ± 4.3 [14.9; 26.1]	26 ± 5 [17.1; 31.2]

Notes: data are mean ± SD [range]. SD, standard deviation; TST, total sleep time; REM, rapid eye movement sleep. The cohort was not fixed across nights: due to missing data, not all athletes contributed observations to every night.

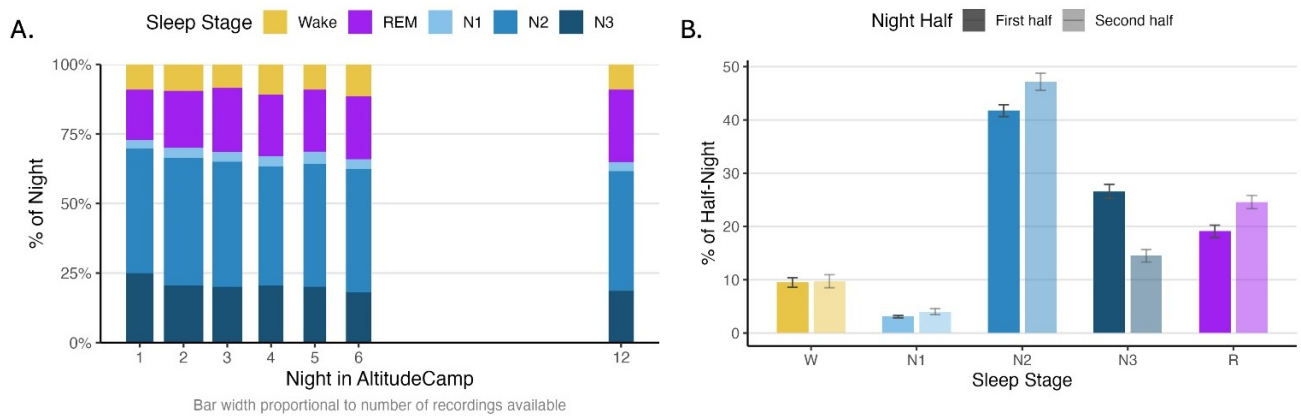


Fig. 1. Sleep architecture across the altitude training camp. (A) Stacked bar chart showing the mean percentage of the night spent in each sleep stage across successive camp nights. Bar width is proportional to the number of valid recordings contributing to each night; numerical summaries are provided in Table 1. (B) Grouped bar chart comparing the percentage of each sleep stage between the first (darker shades) and second (lighter shades) halves of the night, averaged across camp nights.

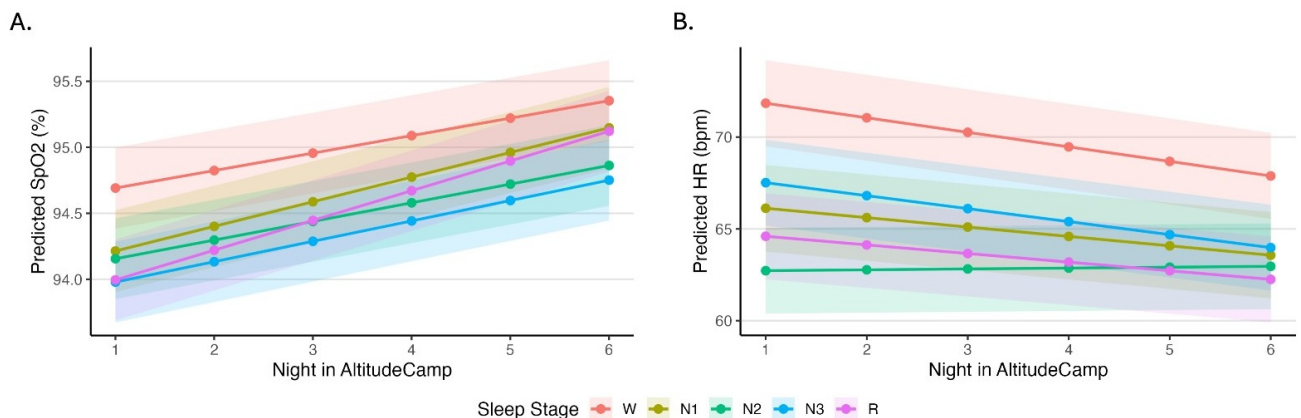


Fig. 2. Stage-specific changes in nocturnal SpO₂ (A) and heart rate (B) across the first week at 2100 m. Lines and shaded bands are linear mixed-effects model estimates with 95% confidence intervals for each sleep stage (Wake, N1, N2, N3, REM). REM-associated SpO₂ recovers faster than Wake ($\beta_{\text{interaction}} = +0.093$ percentage points per night; $p < 0.001$); In N2, HR was stable across nights, declining in the other phases. HR, heart rate.

piratory column/pre-Bötzinger complex, and pontine respiratory regions [15,16]. As sleep deepens, cortical facilitation of respiratory motor output declines [8,9], and hypoxic and hypercapnic ventilatory responsiveness becomes progressively blunted, reducing the ability to compensate for altitude-related hypoxemia [7,16]. At altitude, this suppression becomes relevant because we are now on the steep portion of the oxyhemoglobin dissociation curve, amplifying the desaturation effect of even modest reductions in ventilation [17]. N3 might therefore represent the most vulnerable stage, because sleep-related depression of hypoxic and hypercapnic ventilatory responses, together with a higher arousal threshold in N3, could limit recruitment of medullary chemoreflex circuits and corrective ventilatory responses. REM sleep showed relatively higher SpO₂ than deeper NREM, which may be explained by phasic pontine activity and micro-arousals intermittently increasing

ventilation (also causing a more variable ventilation in this stage), partially counteracting desaturation [11,16]. Overall, these findings suggest that altitude could unmask state-dependent differences in central respiratory and autonomic regulation.

4.2 Sleep Stages and Blood Saturation Changes Across Nights Spent in Altitude

Faster SpO₂ acclimatization during REM is compatible with progressive recovery from altitude-related disruption in pontine and brainstem respiratory-state control, and possible adaptations involving carotid-body sensitization combined with central integration within medullary chemoreflex circuits [16]. Because REM breathing depends more on phasic pontine-brainstem drive than on the more metabolically tonic control of NREM sleep, it is possible that acclimatization might have a larger impact during REM and could drive the increase in %REM with nights

spent at altitude. Slower N3 recovery is compatible with sleep-stage suppression of hypoxic and hypercapnic ventilatory responses [11], since slow wave sleep imposes a high arousal threshold and stronger cortical-brainstem gating of chemoreflex recruitment. Interestingly, there was a blunted HR acclimatization in N2. This may be explained by frequent micro-arousals (detected as passages from N2 to Wake) and K-complex–linked autonomic transient events, limiting full sympathetic withdrawal [8,9]. These aspects need to be analyzed in future studies. Overall, it is possible that the stage-specific dynamics of acclimatization could reflect central reorganization of respiratory and autonomic control rather than peripheral gas exchange mechanics alone.

4.3 Sleep Architecture Reorganization Is Compatible With Central Homeostatic Drive

The progressive decrease in N3 and increase in REM across nights is consistent with prior acclimatization studies at higher altitude [10] and with a two-process interpretation of sleep regulation [18]. Within this framework, the first nights would reflect elevated homeostatic sleep pressure from combined hypoxic and travel-related stress (to reach the altitude location) [19,20], followed by partial recovery as athletes acclimatized. In particular, %REM sleep may recover to baseline values as breathing and autonomic control become more stable across the camp. Because total sleep time did not change overtime, this likely reflects a shift in sleep architecture and central sleep-state regulation rather than a simple change in how long the athletes slept. Finally, the high fitness of athletes does not seem to be a protective factor against sleep impairment at altitude [21,22], making these results relevant for the broader non-athletic healthy population.

4.4 Implications and Limitations

Anchoring the findings in central nervous system (CNS) circuitry has immediate practical implications: during the first ~6 nights at intermediate altitude, motor consolidation and cognitively demanding learning are likely compromised, as both depend on %REM and on prefrontal/dACC network connectivity, which are both stressed under hypoxia [3,22]. Training loads and demanding sessions should therefore be scheduled later in the camp. Although athletes trained on the glacier and slept at a lower altitude, the short morning exposure and chairlift transport suggest only a limited influence on overnight sleep outcomes, although this effect cannot be completely ruled out. Key limitations include a low usable data rate and thus a risk of bias. Furthermore, caution is required when interpreting early-camp sleep changes, as the first days of altitude exposure coincided with transatlantic travel and likely circadian misalignment, both of which can independently alter sleep architecture. In addition, day-to-day variation in training load may have influenced sleep recovery, and the

observed trajectory across days could reflect the composite resolution of both acclimatization and recovery from travel-related fatigue. However, we argue that this combination of factors is common in individuals transiently visiting altitude areas, such visits typically involve several hours of travel. Thus, our study maximizes ecological validity by providing in-the-field data, while laboratory-based studies that simulate altitude have been shown to be useful for isolating the effects of altitude exposure alone. Future studies should examine how travel and training load interact with altitude acclimatization to shape sleep architecture, including markers such as micro-arousals and K-complexes that are directly implicated in motor memory consolidation. Extending this work across sport disciplines and travel duration would help determine whether the observed sleep trajectories reflect a general altitude response or are modulated by exercise, jet lag, or travel-related fatigue.

5. Conclusions

At intermediate altitude, nocturnal SpO₂ and HR show sleep-stage-specific trajectories that reflect central ventilatory and autonomic adaptation to hypoxia. Our data suggest that acclimatization is not uniform across sleep states, and that the first 2 nights after ascent may capture the most rapid adjustments despite similar total sleep duration. Sleep stages act as a heterogeneous probe of central acclimatization and argue for caution when scheduling demanding tasks during the first days at altitude.

Availability of Data and Materials

Analysis code, and detailed statistical outcomes are available on the Open Science Framework (<https://doi.org/10.17605/OSF.IO/3PMYF>).

Author Contributions

Conceptualization, Funding acquisition: GV, JVC, NP, NB, FB, GS; Methodology, Investigation, Data curation, Formal analysis, Visualization, Writing—original draft: GV; Writing—review & editing: JVC, NP, NB, FB, GS. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The project adheres to the Declaration of Helsinki (2013) and was approved by the Ethical Research Committee of the CIUSSS du Nord-de-l'Île-de-Montréal (CIUSSS-NIM, n. 2024-2718). All athletes provided written consent (co-signed by their legal tutor in case of a minor).

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Conflicts of Interest

The authors declare no conflicts of interest.

Declaration of AI and AI-Assisted Technologies in the Writing Process

DeepL was used for grammar checking. Claude Sonnet 4.6 was used to assist with ggplot2 figure code and to organize the OSF code repository. No AI tool generated scientific content or data.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at <https://doi.org/10.31083/JIN52881>.

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