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An observational study of sleep characteristics in elite endurance athletes during an altitude training camp at 1800 m

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ABSTRACT

Objectives: To observe changes in sleep from baseline and during an altitude training camp in elite endurance athletes.
Design: Prospective, observational.
Setting: Baseline monitoring at <500 m for 2 weeks and altitude monitoring at 1800 m for 17-22 days.
Participants: Thirty-three senior national-team endurance athletes (mean age 25.8 ± S.D. 2.8 years, 16 women).
Measurements: Daily measurements of sleep (using a microwave Doppler radar at baseline and altitude), oxygen saturation (SpO_2), training load and subjective recovery (at altitude).
Results: At altitude vs. baseline, sleep duration ($P = .036$) and light sleep ($P < .001$) decreased, while deep sleep ($P < .001$) and respiration rate ($P = .020$) increased. During the first altitude week vs. baseline, deep sleep increased ($P = .001$). During the first vs. the second and third altitude weeks, time in bed ($P = .005$), sleep duration ($P = .001$), and light sleep ($P < .001$) decreased. Generally, increased SpO_2 was associated with increased deep sleep while increased training load was associated with increased respiration rate.
Conclusion: This is the first study to document changes in sleep from near-sea-level baseline and during a training camp at 1800 m in elite endurance athletes. Ascending to altitude reduced total sleep time and light sleep, while deep sleep and respiration rate increased. SpO_2 and training load at altitude were associated with these responses. This research informs our understanding of the changes in sleep occurring in elite endurance athletes attending training camps at competition altitudes.

Introduction

Altitude training is a common strategy employed by elite endurance athletes to induce physiological adaptations, with a potential to improve subsequent performance at altitude and/or sea level.[1-3] Thus, elite endurance athletes commonly integrate training camps at low-to-moderate altitudes (eg, ~ 1400-2500 m) lasting 2–4 weeks into their annual training periodization.[2] The most extensively studied adaptive response linked to altitude training is the erythropoietin-driven increases in red blood cell volume and total hemoglobin mass.[2] Although there are conflicting views,[3,4] these hematological changes are considered to represent the main mechanism for improved endurance performance at sea level following periods of altitude training.[2]

Optimization of altitude adaptations depends on various factors, such as the hypoxic dose, training load and recovery, oxygen saturation (SpO_2), iron and energy availability and illness status.[2,5,6] The combined stressors of training and hypoxia at altitude place increased demands on recovery, posing a larger risk for illness, maladaptation, overreaching and/or overtraining.[2,5,7] Sleep is essential for physiological processes that facilitate recovery from training and is crucial for long-term performance development.[8] However, when acutely exposed to altitude athletes often report sleep disruptions, such as reduced sleep duration and sleep efficiency (for a review, see Roberts et al[9]). Living at altitude during a training camp may, therefore, have a detrimental effect on athletes' sleep, recovery and subsequent adaptations.

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Most existing studies on the altitude-related changes in sleep have investigated nonathletes exposed to high altitudes (eg, ~ 4000-5000 m).[10−13] Previous findings in athletes have shown that immediately upon ascent from near sea level (430 m) to high altitude (3600 m), a group of soccer players exhibited reduced rapid eye movement (REM) sleep, which was measured using polysomnography (PSG).[14] In the same group of athletes, actigraphic recordings showed acute effects of altitude leading to reduced sleep duration and sleep quality.[15] A comparison group of altitude-native peers did not experience such sleep changes, which suggests chronic adaptations or underlying differences in sleep patterns in athletes residing permanently at moderate-to-high altitudes.[15] Reductions in deep and REM sleep have also been reported in recreational endurance athletes acutely exposed to a simulated altitude of 2000 m.[16]

Sleep changes during longer sojourns at altitude have been examined using both terrestrial[14,15,17] and simulated[18] altitude. In the aforementioned group of soccer players ascending from near sea level to 3600 m, the reduction in REM sleep returned to sea-level values following 14 days of exposure.[14] In a study of recreational cyclists who slept at a simulated altitude of 2650 m for 15 nights, REM sleep increased on nights 8 and 15 compared to the first night.[18] Another study conducted on soccer players showed that acute reductions in total sleep time and subjectively-measured sleep quality following transmeridian travel and ascent to 1600 m stabilized after 4-6 days, and that a further ascent to 2150 m did not negatively influence sleep.[17] While these findings demonstrate changes in sleep at moderate-to-high altitudes, no previous studies have examined potential changes in sleep and sleep-stage distributions during acclimatization at low-to-moderate altitude (~ 1400-2500 m) in elite endurance athletes.

Existing studies have utilized PSG (considered the gold standard in sleep measurement),[14] actigraphy,[15] and sleep diaries.[17] While PSG can reliably detect the different sleep stages, it is costly and typically limited to short time periods (see eg, Sargent et al[14]). Actigraphy and sleep diaries are easy to use and cheaper than PSG, and have represented the primary choice for sleep monitoring in athletes' natural surroundings. However, actigraphy has limited specificity[19] and cannot differentiate between sleep stages, while sleep diaries may be subject to biases linked to recall,[20] common method[21] and social desirability.[22] Recently, the development of a reliable, noninvasive tool for sleep monitoring using the microwave Doppler radar (DR) technology has evidenced good estimation ability of sleep stage classification when compared to PSG[23]. The technology has already been applied in long-term monitoring of sleep in athletes.[24]

To extend our current understanding of elite endurance athletes' sleep characteristics at altitude, the present study aimed to observe sleep during an ~ 3-week altitude training camp with elite endurance athletes residing at 1800 m, and the time course of sleep changes in relation to near-sea-level baseline measures, using a novel, noninvasive microwave DR sleep monitor. It was hypothesized that exposure to altitude would lead to variations in athletes' sleep. Specifically, it was hypothesized that acute exposure to altitude would result in shorter total sleep time and changes in sleep-stage distributions (ie, reduced deep and REM sleep). It was further hypothesized that the acute effects of altitude on sleep would be diminished in the second and third weeks at altitude.

Materials and methods

Participants

Thirty-seven senior national-team endurance athletes, of which 25 were cross-country (XC) skiers and 12 were biathletes, volunteered to participate in the study. Of these athletes, 4 dropped out due to illness and early departure from the altitude training camp. Thus, 33 athletes completed the study (22 XC skiers and 11 biathletes; 16 women and 17 men). The mean ± S.D. characteristics of the final sample were: age 25.8 ± 2.8 years, body mass 71.3 ± 10.3 kg, height 171.9 ± 21.1 cm. All athletes were lifelong residents at near sea level (ie, 0−500 m), and none suffered from sleep disorders at the time of, or prior to the study. All athletes were fully informed about the nature of the study before providing written consent to participate. The study was conducted in accordance with the Declaration of Helsinki (1964) and its later revisions, and approved by the regional ethical review board in Umeå, Sweden (reference: 2018-46-31M).

Procedures

A prospective, observational design was employed to monitor sleep before and during an ~ 3-week-long altitude training camp. Prior to the data collection period, athletes received face-to-face and written instructions on how to place and operate the sleep monitoring equipment. Sleep was initially monitored at baseline (ie, <500 m) for 2 weeks, then at a terrestrial altitude of 1800 m (defined as low altitude by Bärtch and Saltin[25]) during the teams' preseason training camps in Font Romeu, France. This altitude was chosen based on its relevance to the Beijing 2022 Olympic Winter Games, where most endurance events are due to be held at an altitude of ~ 1650-1700 m. During the training camps, the XC skiers and biathletes spent 17 and 22 nights at altitude, respectively. Athletes slept and trained at altitude, employing the so-called "live high-train high" method.[2,26] No sleep education was provided before or during the sleep-monitoring period and athletes were free to consume caffeine and other nutritional supplements under the guidance of their coaches and support staff. Athletes were offered technical support throughout the data collection period to address and solve issues related to the sleep monitoring. In addition to daily monitoring of sleep, athletes' resting peripheral, training load and subjective recovery were measured daily during the altitude training camp.

Measurements

Sleep

Sleep was monitored using fully unobtrusive microwave DR (Somnofy version 0.7, VitalThings AS, Norway), which utilizes impulse radio ultra-wideband (IR-UWB) pulse radar, Doppler effect and fast Fourier transformation to measure the movement and respiration rate of a sleeping individual.[23] The raw data for movement and respiration, processed by a machine-learning algorithm, are used to calculate time in bed, sleep onset latency, total sleep time, light sleep, deep/slow wave sleep, REM sleep, sleep efficiency and respiration rate during non-REM sleep (see Table 1 for descriptions and abbreviations of the sleep variables). Recently, a full validation against PSG showed that the accuracy of the Somnofy sleep monitor was 0.97 for sleep, 0.72 for wake, 0.75 for light sleep, 0.74 for deep sleep and 0.78 for REM sleep. The overall Cohen's kappa for the Somnofy monitor was 0.63, indicating substantial agreement with PSG. Thus, the sleep monitor represents an adequate alternative to PSG for quantifying and classifying sleep, wake, and sleep-stage measurements in healthy adults,[23] suggesting superiority to other portable and unobtrusive tools for sleep assessment.[27]

Oxygen saturation

Resting peripheral SpO_2 was monitored daily during the altitude training camp before breakfast in a fasted state, between ~ 6:30 and 9:30 AM, in a field laboratory. Athletes wore a finger-clip pulse oximeter (Onyx Vantage, Nonin Medical B.V., Netherlands) on the index finger while seated for 60 seconds. Four individual measurements of SpO_2 were taken at 5-second intervals between 45-60 seconds. The 4 measurements were averaged and reported as the daily value.

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Table 1
Descriptions of the assessed sleep variables

Sleep variable	Abbreviation	Units	Characteristics of sleep variable
Time in bed	TIB	h	Time spent in bed, including time awake
Sleep onset latency	SOL	h	Time from when the athlete intends to sleep to sleep onset
Total sleep time	TST	h	Total sleep time obtained from sleep onset to sleep offset
Light sleep	LS	h	Total time in light sleep (stage N1 and N2)
Deep/slow wave sleep	SWS	h	Total time in deep sleep (stage N3)
Rapid eye movement sleep	REM	h	Total time in REM sleep
Sleep efficiency	SE	%	The percentage of total time in bed spent asleep
Respiration rate during non-REM sleep	NREM RPM	n	The number of respiratory ventilations per minute during non-REM sleep

Training load

Training load (arbitrary units, AU) was quantified by multiplying total training duration by the session rating of perceived exertion (sRPE). Total training duration, measured in minutes, was retrieved from athletes' training diaries and verified against athletes' heart rate data. sRPE was rated on a modified Borg category-ratio scale, utilizing a category scale with ratio properties ranging from 0 (“No exertion at all”) to 10 (“Maximal”).[28,29] The sRPE has been shown to be a valid marker of exercise intensity.[30] Although measurements of training load were available at altitude only, athletes and coaches provided verbal confirmation that training loads did not differ considerably between the baseline and altitude training camp measurement periods.

Subjective recovery

Subjective recovery was assessed immediately before the SpO_2 measurements were made, using the Overall Recovery item from the Short Recovery and Stress Scale (SRSS).[31] The Overall Recovery item was described as “Recovered, rested, muscle relaxation, physically relaxed”. Athletes rated their current subjective perception of recovery at altitude, in relation to their best subjective recovery state, on a 7-point Likert scale, ranging from 0 (“Does not apply at all) to 6 (“Fully applies”). Thus, higher scores indicated better subjective recovery. The SRSS has been shown to be both valid and reliable for the monitoring of athletes' recovery-stress states.[32]

Data collection compliance

The total number of nights in the study was 1078. However, some sleep data were lost due to technical issues with connecting the sleep monitor to the Wi-Fi, or a lack of access to electrical power. Data points were further removed when identified as extreme outliers (defined as data points >3 box lengths from either hinge of the boxplot). In total, 125 nights of baseline sleep data and 173 nights of altitude sleep data were removed. Thus, 780 sleep data points were analyzed, reflecting 72.4% compliance. For SpO_2 and subjective recovery, 3.3% of the data collected at altitude was lost due to illness. 6.2% of training load data at altitude was lost due to illness and mistakes with reporting training durations.

Statistical analysis

The collected data created a clustered data structure, in which repeated measurements of objective sleep data were clustered within the individual athletes. By virtue of the clustered data structures, there are dependencies of the repeated measurements within individuals. If this dependence is not taken into consideration in the statistical approach, an issue with excessive type I errors and biased parameter estimates might occur. Therefore, multi-level modeling in Mplus, version 8.3,[33] was utilized to carry out the statistical analyses by clustering the repeated measurements (level-1) within the athletes (level-2).

Random intercept models were used to investigate whether sleep varied between baseline (near sea level) and the training camp (at altitude), taking into consideration the influence of SpO_2, training load and subjective recovery as covariates. The duration of the altitude training camp was divided into the first week, and the second and third weeks. The second and third weeks were merged because not all athletes spent the full third week at altitude. Random intercept models assume that the only variation between individuals is at their intercept and that the effects of the predictor variables are the same for each individual (fixed slope). Three sets of random intercept models were tested: (1) effects of altitude (predictor, 0 = baseline, 1 = pooled days at altitude), SpO_2 (predictor, continuous variable), training load (predictor, continuous variable) and subjective recovery (predictor, continuous variable) on sleep (outcome), (2) effects of the first week at altitude (predictor, 0 = baseline, 1 = first week at altitude), SpO_2, training load and subjective recovery on sleep (outcome), and (3) effects of the second and third weeks at altitude (predictor, 0 = first week at altitude, 1 = second and third weeks at altitude), SpO_2, training load and subjective recovery on sleep (outcome).

Associations on the within level refer to the effects of the day-to-day variation within each athlete, and with the between-level effects (ie, the average differences between athletes) removed. These results are presented by reporting the estimated effect ± standard error (S.E.) and associated *P*value. On the between level, the results show the estimated variances of the predictor variables across athletes (ie, interindividual variances). For each random intercept model, the intraclass correlation (ICC) was calculated, representing the extent to which the dependent values of occasions of measurement in the same participant resemble each other compared to those from different athletes. For all random intercept models, R^2 values stating the explained variance on the within level were reported. The alpha level was set at $P< .05$ for all models. IBM SPSS (version 25.0) was used to conduct demographic and descriptive statistical analyses, which are presented as mean ± standard deviation (S.D.).

Results

Descriptive statistics for the sleep variables at baseline and the sleep variables, SpO_2, training load and subjective recovery at altitude during the first week, the second and third weeks combined and overall are shown in Table 2. Mean sleep data (with 95% confidence intervals) are visually presented in Fig. 1.

Changes in sleep from baseline to altitude

Random intercept models showed that for altitude overall, TST decreased by 9.0 ± 4.2 minutes ($P= .036$), LS decreased by 12.0 ± 3.0 minutes ($P< .001$), SWS increased by 7.8 ± 1.8 minutes ($P \leq .001$) and NREM RPM increased by 0.22 ± 0.09 respirations per min ($P= .020$) compared to baseline (Fig. 2). Additionally, each unit increase in SpO_2 was associated with an increase in SWS by 1.8 ± 0.6 minutes ($P= .017$), and each unit increase in training load was associated with an increase in NREM RPM by 0.02 ± 0.01 respirations per minutes ($P= .006$). The explained within-athlete variances (R^2) of these effects on sleep were low, ranging from 0.9% to 7.5%. Between-athlete variances

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Table 2
Descriptive statistics for objectively-measured sleep variables, resting peripheral oxygen saturation, training load, and subjective recovery in 33 elite endurance athletes at near sea level (baseline) and at altitude (overall, week 1, and weeks 2 and 3 combined)

	Near sea level		Altitude					
	Baseline		Overall		Week 1		Weeks 2/3	
Variable	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Time in bed (h)	9.15	0.93	9.08	0.82	9.18	0.70	9.01	0.90
Sleep onset latency (h)	0.51	0.34	0.44	0.36	0.43	0.37	0.44	0.34
Total sleep time (h)	7.44	0.99	7.35	0.87	7.44	0.79	7.29	0.93
Light sleep (h)	4.60	0.72	4.43	0.68	4.52	0.59	4.35	0.73
Deep/slow wave sleep (h)	1.06	0.40	1.20	0.37	1.20	0.37	1.19	0.36
REM sleep (h)	1.78	0.56	1.73	0.53	1.72	0.52	1.74	0.54
Sleep efficiency (%)	80.93	8.01	80.49	6.70	80.56	6.78	80.43	6.65
NREM RPM (N)	15.26	2.06	15.49	2.08	15.43	2.08	15.54	2.09
Resting peripheral oxygen saturation (%)	-	-	96.6	.1	96.8	.1	96.5	.1
Training load (au)	-	-	772.79	19.05	638.71	25.95	843.28	24.87
Subjective recovery (au)	-	-	3.54	.03	3.79	.06	3.41	.04

au, arbitrary values; NREM RPM, non-REM respirations per minute; REM, rapid eye movement sleep; S.D., standard deviation.

were significant for all sleep variables (TIB: P= .027; SOL: P= .002; TST, LS, SWS, REM, SE, NREM RPM: $P< .001$). ICC values showed that 14% to 32% of the total variance in TIB, SOL, TST, LS, SWS, REM and SE was due to differences between athletes, while 91% of the variance in NREM RPM was due to differences between athletes. Full results for the ICC values and between-athlete variances in sleep, comparing altitude to baseline, are presented in Table 3A.

Random intercept models investigating the effects of the 1st week at altitude on sleep showed that SWS increased by 7.8 ± 2.4 minutes (P= .001) compared to baseline (Fig. 2). Additionally, each unit increase in SpO_2 was associated with a decrease in LS by 4.8 ± 2.4 minutes (P= .023) and with an increase in SWS by 1.8 ± 1.2 minutes (P= .049). Furthermore, each unit increase in training load was associated with an increase in NREM RPM by 0.04 ± 0.01 respirations per minutes ($P< .001$). The explained within-athlete variances (R^2) of these effects on sleep ranged from 0.3% to 8.7%. Between-athlete variances were significant for all sleep variables (SOL, TST, LS: -P= .001; SWS, NREM RPM: $P< .001$; REM, SE: P= .002), except for TIB (P= .061). ICC values showed that 12%-32% of the total variance in TIB, SOL, TST, LS, SWS, REM and SE was due to differences between athletes, while 91% of the variance in NREM RPM was due to differences between athletes. Full results for the ICC values and between-athlete variances in sleep, comparing the first week of altitude to baseline, are presented in Table 3B.

Changes in sleep from the first week to the second and third weeks at altitude

Random intercept models investigating the effects of the second and third weeks at altitude on sleep showed that TIB decreased by 14.4 ± 5.4 minutes (P= .005), TST decreased by 13.8 ± 4.2 minutes (p= .001), and LS decreased by 13.8 ± 3.6 minutes ($P< .001$) compared to the first week at altitude (Fig. 2). Additionally, each unit increase in SpO_2 was associated with a decrease in TIB by 3.0 ± 1.8 minutes (P= .047) and each unit increase in training load was associated with an increase in NREM RPM by 0.02 ± 0.01 respirations per min ($P< .011$). The explained within-athlete variances (R^2) of these effects on sleep ranged from $<$ 0.1% to 4.7%. Between-athlete variances were significant for all sleep variables (TIB: P= .017; SOL, REM, SE, NREM RPM: $P< .001$; LS, SWS: P= .001; TST: P= .009). ICC values showed that 14%-40% of the total variance in TIB, SOL, TST, LS, SWS, REM, and SE was due to differences between athletes, while 94% of the variance in NREM RPM was due to differences between athletes. Full results for the ICC values and between-athlete in sleep, comparing the effect of the second and third weeks of altitude to the first week, are presented in Table 3C.

Discussion

This is to our knowledge the first study to observe sleep changes in elite endurance athletes from near-sea-level baseline and during an entire ~ 3-week altitude training camp at 1800 m. Sleep was monitored using a novel, noninvasive microwave DR sleep monitor and the main findings were that: (1) TST and LS decreased at altitude compared to near-sea-level baseline measures and these changes occurred between the first and second/third weeks at altitude; (2) SWS and NREM RPM increased at altitude compared to near-sea-level baseline measures and these changes were already present in the first week at altitude; (3) Increased training load was associated with increased NREM RPM throughout the entire duration of the altitude training camp. At altitude, increased SpO_2 was associated with increased SWS, decreased LS and with decreased TIB.

Some accounts of changes in athletes' sleep patterns from near sea level to altitude have been reported in the scientific literature,[14-18,34,35] but these studies have been conducted at altitudes of 2000-3600 m. In the present study an altitude of 1800 m was used for its relevance to the Beijing 2022 Olympic Winter Games. When pooling all nights during the ~ 3-week altitude training camp, TST and LS were reduced compared to the near-sea-level ($<$500 m) baseline measures for these elite endurance athletes, while SWS and NREM RPM were increased. Previous research at terrestrial altitude has ascribed the observed reductions in TST to sleep disturbances associated with ascending to altitude.[15,17] However, the mechanisms at play are unclear, as neither SpO_2, training load nor subjective recovery explained the reductions in TST and LS in the present study. Following ascent to altitude as compared to baseline, SpO_2 typically decreases.[14] Acclimatization to altitude is in turn associated with restored levels of SpO_2 [13]. In the present study, increases in SpO_2 were associated with an increase in SWS. Since TST was reduced, the increase in SWS may have further led to a concurrent, compensatory decrease in LS. It is worth noting that although the day-to-day variations in sleep only ranged from 7.8 to 12.0 minutes, accumulated effects of these variations over the entire ~ 3-week period could have a substantial influence on subjective recovery, adaptations and performance optimization in elite athletes. This is consistent with the suggestion of Lastella et al,[36] who have previously hypothesized that the cumulative effect of sleep loss over multiple days may negatively influence athletic performance.

A major effect of acute hypoxia relates to an increase in ventilation (ie, respiration rate) and sympathetic activity.[37] Increases in respiration rate may be caused by the arterial desaturation that occurs at altitude, leading to hypoxic ventilatory response.[37] Importantly, changes in ventilation due to hypoxia disrupt breathing during sleep, inducing respiratory events and periodic breathing.[34,38,39] Consistent with these effects, NREM RPM increased throughout the duration of the altitude training camp in

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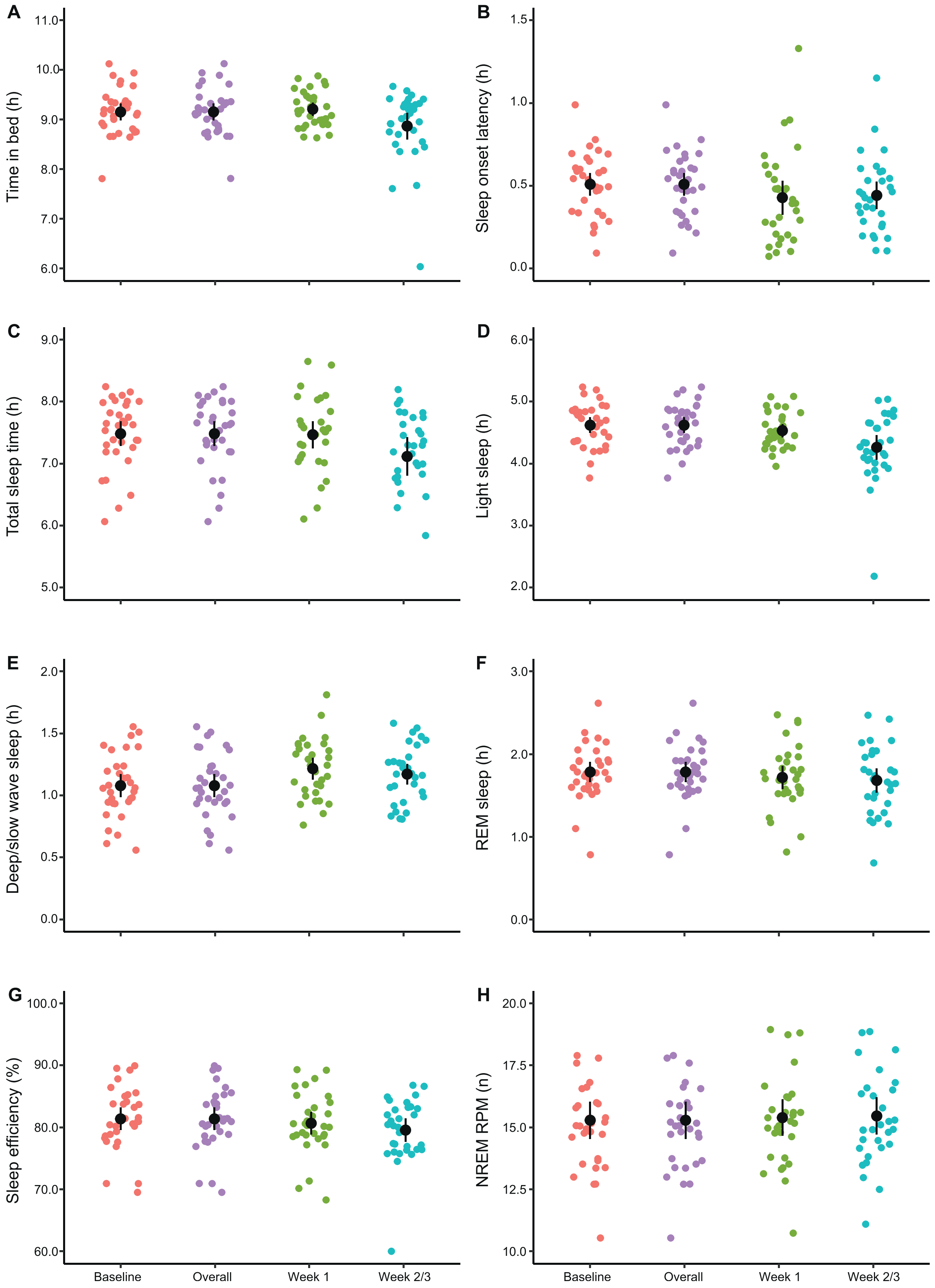

Fig. 1. Visualization of the descriptive data for the measured sleep variables. Data is based on sleep monitoring in 33 elite endurance athletes at near-sea-level baseline (pink dots) and at altitude overall (purple dots), week 1 (green dots) and weeks 2 and 3 combined (blue dots). Each data point represents each athletes' mean score in the respective sleep variables. The filled black dot represents the mean for the whole group, with upper and lower 95% confidence intervals represented by the bar intersecting the mean. (Color version of figure is available online.)

the present study. Increases in training load over the ~ 3 weeks, possibly attributable to increased perceived exertion associated with training at altitude, were related to this increase. Thus, the increased NREM RPM observed during the altitude training camp may be attributed to the process of acclimatization to increasing altitude, and to increasing training loads. However, further studies are required to examine the underlying

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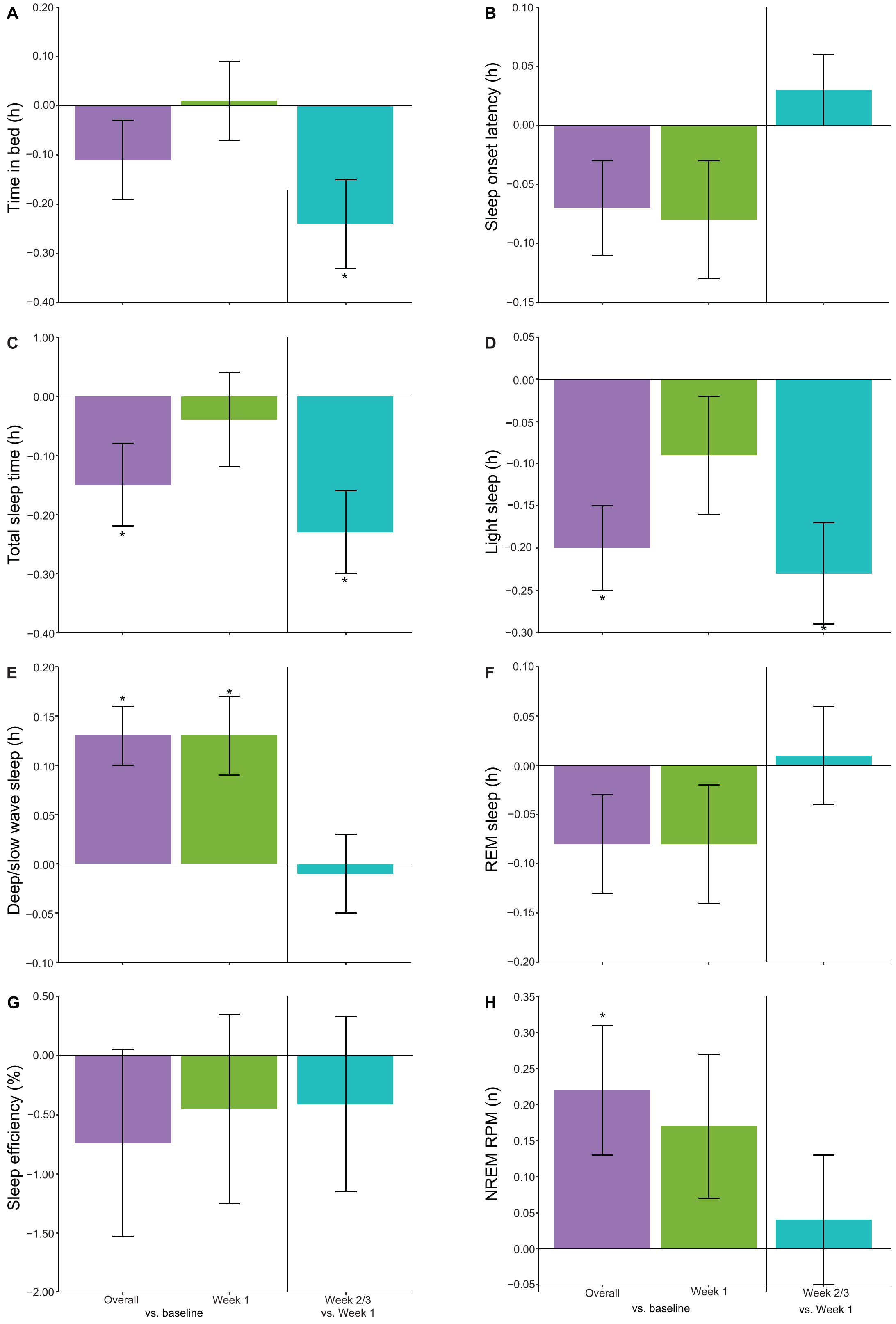

Fig. 2. Visualization of the within-athlete variations in the measured sleep variables across the different altitude periods. Data is based on sleep monitoring in 33 elite endurance athletes at near-sea-level baseline vs. altitude overall (purple bars) and vs. week 1 (green bars), and at week 1 vs. weeks 2 and 3 combined (blue bars). The error bars represent the S.E. * represents a significant change, $P < .05$. (Color version of figure is available online.)

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Table 3
The ICC values and between-athlete variance in sleep across the analyzed time periods, controlling for the effect of resting peripheral oxygen saturation, training load and subjective recovery, based on data from 33 elite endurance athletes

	(A) Effect of altitude (IV, 0 = baseline, 1 = altitude), SpO$_2$, training load and subjective recovery (IVs) on sleep (DV)				(B) Effect of the first week at altitude (IV, 0 = baseline, 1 = first week at altitude), SpO$_2$, training load and subjective recovery (IVs) on sleep (DV)				(C) Effect of the second and third weeks at altitude (IV, 0 = first week at altitude, 1 = second and third weeks at altitude), SpO$_2$, training load and subjective recovery (IVs) on sleep (DV)			
DV	ICC	Est.	S.E.	Sig.	ICC	Est.	S.E.	Sig.	ICC	Est.	S.E.	Sig.
Time in bed (h)	0.16	0.12	0.06	*0.027*	0.12	0.09	0.05	0.061	0.17	0.12	0.05	*0.017*
Sleep onset latency (h)	0.22	0.03	0.01	*0.002*	0.22	0.03	0.01	*0.001*	0.37	0.05	0.01	*<0.001*
Total sleep time (h)	0.27	0.23	0.06	*<0.001*	0.28	0.23	0.07	*0.001*	0.40	0.33	0.13	*0.009*
Light sleep (h)	0.14	0.07	0.02	*<0.001*	0.13	0.06	0.02	*0.001*	0.14	0.07	0.02	*0.001*
Deep/slow wave sleep (h)	0.22	0.03	0.01	*<0.001*	0.26	0.04	0.01	*<0.001*	0.20	0.03	0.01	*0.001*
REM sleep (h)	0.32	0.09	0.03	*<0.001*	0.32	0.09	0.03	*0.002*	0.36	0.10	0.03	*<0.001*
Sleep efficiency (%)	0.26	13.36	3.16	*<0.001*	0.32	17.82	5.72	*0.002*	0.31	14.04	3.46	*<0.001*
NREM RPM (N)	0.91	3.75	1.02	*<0.001*	0.91	3.82	1.06	*<0.001*	0.94	3.71	1.00	*<0.001*

DV, dependent variable; Est., estimate; ICC, intraclass correlation; IV, independent variable; NREM RPM, non-REM respirations per minute; REM, rapid eye movement sleep; S.E., standard error; Sig., significance; SpO$_2$, resting peripheral oxygen saturation.
Regressions were clustered on participant. Values are unstandardized. Significant results are italicized.

mechanisms of increased respiration rate and the potential incidence of periodic breathing in elite endurance athletes at low-to-moderate terrestrial altitudes.

When comparing sleep in the first week at altitude with near-sea-level measures, only increases in SWS and NREM RPM were observed. In addition, the increase in SWS was associated with an increase in SpO$_2$, which was also related to a decrease in LS. This is contrary to previous findings, which have typically reported reduced TST and REM sleep during the first nights following ascent from near sea level to altitude.[14,15,17] These conflicting findings might be explained by the fact that all nights for the first week at altitude were pooled, thereby limiting the possibility to detect potential changes in sleep during the initial nights at altitude. Alternatively, the differences in findings could be caused by the altitude used in the present study (ie, 1800 m), which was considerably lower than in previous studies (ie, 3600 m).[14,15] This would have posed a lower hypoxic stress in the present study and possibly less-pronounced changes in sleep compared to near-sea-level baseline measures as a result. This is further supported by studies performed under more extreme conditions (ie, 4559 m), where both SWS and REM sleep were substantially reduced or eliminated entirely following ascent to altitude.[12,13] Although increases in SWS from near sea level remained stable during the $\sim$ 3-week period, TIB, TST and LS were reduced in the combined second and third weeks of the training camp. While increases in SpO$_2$ were related to the reduction in TIB, it is unclear whether any other factors and their associated explanatory mechanisms contributed to these delayed changes throughout the sojourn at altitude. These findings require further examination using appropriate experimental designs. For instance, reduced levels of psychological stress as athletes became more familiar with their new routines and activities, or strategic use of napping during the daytime, should be taken into consideration in future research.

Limitations

A limitation of our design was the lack of detailed information on daytime napping routines at near sea level vs. altitude, which might have influenced the sleep measures reported in the present study. However, verbal communication with all athletes and their respective coaches revealed that daytime napping routines did not differ considerably between the baseline and altitude training camp measurement periods. In addition, it would have been beneficial to measure subjective sleep (using sleep diaries) and psychological stress in the present study. The absence of relevant variables in the tested statistical models may explain the low R^2 values of the reported results. Moreover, the low number of participants may have influenced the power to detect significant associations in the investigated multilevel statistical analyses. The use of novel technology for the measurement of sleep represents another relevant limitation. The Somnofy sleep monitor allowed us to monitor the sleep of 33 elite athletes over 2 weeks at baseline and for up to 3 weeks at altitude. The device shows limitations in terms of accuracy of sleep stage classification (0.75 for LS, 0.74 for SWS, and 0.78 for REM),[23] and its validity of estimations on a night-to-night basis has so far not been established.

In conclusion, the present study demonstrated that TST and LS decreased, while SWS and NREM RPM increased, compared to near-sea-level baseline measurements in elite endurance athletes during an $\sim$ 3-week altitude training camp at 1800 m. Increases in SpO$_2$ and training load were implicated in the observed variations in SWS and NREM RPM, respectively. Further experimental studies are needed to elucidate the role of sleep changes during altitude training camps in elite endurance athletes.

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Declaration of conflict of interest

The authors have declared no conflicts of interest.

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