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The impact of altitude on the sleep of young elite soccer players (ISA3600)

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ABSTRACT

Background Altitude training is used by elite athletes to improve sports performance, but it may also disrupt sleep. The aim of this study was to examine the effects of 2 weeks at high altitude on the sleep of young elite athletes.

Methods Participants (n=10) were members of the Australian under-17 soccer team on an 18-day (19-night) training camp in Bolivia, with six nights at near sea level in Santa Cruz (430 m) and 13 nights at high altitude in La Paz (3600 m). Sleep was monitored using polysomnography during a baseline night at 430 m and three nights at 3600 m (immediately after ascent, 1 week after ascent and 2 weeks after ascent). Data were analysed using effect size statistics.

Results All results are reported as comparisons with baseline. Rapid eye movement (REM) sleep was likely lower immediately upon ascent to altitude, possibly lower after 1 week and similar after 2 weeks. On all three nights at altitude, hypopneas and desaturations were almost certainly higher; oxygen saturation was almost certainly lower; and central apnoeas, respiratory arousals and periodic breathing were very likely higher. The effects on REM sleep were common to all but one participant, but the effects on breathing were specific to only half the participants.

Conclusions The immediate effects of terrestrial altitude of 3600 m are to reduce the amount of REM sleep obtained by young elite athletes, and to cause 50% of them to have impaired breathing during sleep. REM sleep returns to normal after 2 weeks at altitude, but impaired breathing does not improve.

INTRODUCTION

Altitude training is used by elite athletes from individual and team sports in an attempt to improve sports performance. The rationale for this type of training is that the hypoxia of altitude can stimulate the production of red blood cells, which in turn can increase maximal oxygen consumption on return to sea level.[1] Although altitude training can be beneficial, it also has risks and costs. In particular, altitude training camps are expensive,[2] training cannot be conducted at the same intensity at altitude as at sea level[1 3] and the athletes are susceptible to illness at the start of camp due to the acute effects of altitude[4] and, later in the camp, due to suppressed immune function.[5] Furthermore, there is some indirect evidence to indicate that altitude training may interfere with athletes' sleep.

Sleep is an essential component of daytime functioning,[6] so any intervention that potentially disrupts athletes' sleep should be closely examined. Human sleep is comprised of rapid eye movement (REM) sleep and non-rapid eye movement (NREM) sleep. NREM sleep is further divided into three stages representing a continuum from 'light' sleep to 'deep' sleep.[7] REM sleep and deep sleep are the most important stages because the former aids mental recovery, learning and memory consolidation,[8] and the latter aids physical recovery and growth.[9 10] The normal composition of sleep stages within a sleep period, that is, 'sleep architecture', is 61% light, 16% deep, and 23% REM for adults (aged 30 years) and 57% light, 22% deep, and 21% REM for adolescents (aged 16 years).[11]

The effects of high altitude, either terrestrial or simulated, on elite athletes' sleep are currently unknown. Nevertheless, comparisons of untrained adults' sleep at sea level and at high terrestrial altitude (ie, 4559–5050 m) indicate that sleep periods at altitude have more arousals,[12–16] more wakefulness[13] and less deep sleep.[12–15] Similarly, compared with living at sea level, when recreational athletes live at low and moderate simulated altitudes (ie, 2000–2650 m), their sleep periods contain more arousals,[17] more abnormal breathing,[18–20] and less deep sleep.[18] These athlete studies provide a good indication of the potential effects, but the degree to which their results can be generalised to elite athletes in high terrestrial altitude training camps over several days is limited because the participants were recreational athletes, the altitude was simulated and at a low-moderate level and only acute effects were assessed.

The aim of this study was to examine the effects of living at high altitude for several days on the sleep of young elite athletes who usually live at sea level. This was achieved by assessing the sleep of soccer players in an underage national team during an 18-day training camp in Bolivia. Sleep periods were monitored once at near sea level, then three times over a 2-week period at high altitude.

METHODS

Participants

Ten male soccer players (age 15.6±0.5 years, mass 71.0±5.8 kg; mean±SD), who were all sea-level natives and members of the Australian under-17 Soccer Team (the 'Joeys'), volunteered to participate in the study. Originally, 12 players were recruited to participate, but data sets were

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incomplete for two of them due to illness and/or equipment failure, so these were excluded from all analyses. The study was approved by ethics committees at the Australian Institute of Sport and the Universidad Mayor de San Andrés. Written informed consent was obtained from participants and their guardians.

Protocol

In August 2012, the Joeys participated in a training camp in preparation for the 2012 Asian Soccer Confederation under-16 Championship. The team travelled from Sydney, Australia, then lived and trained in Bolivia for 18 days (and 19 nights)—the first six nights at near sea level in Santa Cruz (430 m), and the next 13 nights at high altitude in La Paz (3600 m). A general description of the study methods, and data related to blood gases, wellness, running performance, activity during matches and sleep/wake behaviours, are reported in our companion papers in this issue.[2 21–25]

Each participant was observed during a baseline sleep at near sea level and three sleeps at high altitude (figure 1). Only six sets of data acquisition equipment were available, so for each time point, data collection occurred on two successive nights, each with six participants. Ultimately then, data were collected 1–2 nights prior to ascent to altitude, immediately after ascent (nights 1–2 at altitude), 1 week after ascent (nights 6–7 at altitude) and 2 weeks after ascent (nights 12–13 at altitude). All team members were accommodated in two-person hotel rooms, so participants were paired together. Participants were required to attend a brief prebreakfast testing session by 09:30 h each day, but other than that they were free to chose their own bedtimes and get-up times.

Sleep was recorded using portable polysomnography equipment (Compumedics; Victoria, Australia) with a standard montage of electrodes. The montage included two EEGs (C4-M1, C3-M2), two electro-oculograms (left outer canthus, right outer canthus) and a submental electromyogram. Respiratory effort was measured with a piezoelectric band (Compumedics; Victoria, Australia) and oxygen saturation was measured with a pulse oximeter (Nonin Medical; Minnesota, USA).

In the 30 min prior to bedtime, each participant had polysomnography electrodes applied to their face and scalp, a respiratory band placed around their chest and a pulse oximeter attached to their index finger. Each pair of participants was given an electronic pager that could be used to call the researchers during the night if required. Once all equipment was in position and the participants were comfortable, the lights were turned off. The signals from each device were transmitted wirelessly to a laptop computer located in a separate room to be monitored overnight by the researchers. When participants wanted to get up in the morning, they paged the researchers and the monitoring equipment was removed.

Measures

All sleep records were blinded and then analysed in 30 s epochs by a trained technician. Sleep stages, respiratory events, arousals and desaturations were manually scored in accordance with established criteria.[26] For each sleep period, the following dependent variables were calculated: time in bed (h), total sleep time (h), sleep onset latency (min), light sleep (ie, time spent in stage N1 or stage N2 sleep; min), deep sleep (ie, time spent in stage N3 sleep; min), REM sleep (time spent in stage R sleep; min), wake after sleep onset (min), sleep efficiency (ie, total sleep time/time in bed×100; %), central apnoeas (count), hypopneas (count), respiratory disturbance index (ie, total number of central apnoeas and hypopneas/total sleep time; per hour), total arousals (count), spontaneous arousals (count), respiratory arousals (count), mean oxygen saturation (%), minimum oxygen saturation (%), desaturations (count) and periodic breathing (ie, time spent in periodic breathing/total sleep time×100; %).

Statistical analyses

Sleep architecture was compared between the baseline sleep and the three sleeps at altitude using separate χ^2 tests. For all other comparisons between the baseline sleep and the three sleeps at altitude, variables were log transformed to reduce bias due to non-uniformity of error. The magnitude of the observed effects was assessed using standardised differences (effect size statistics) with a customised Excel-based spreadsheet.[27] For each variable, three separate probabilities were calculated using Student's t test to estimate the chances that the mean value at altitude was greater, similar and lower than the mean value at baseline. For these analyses, the hypothesised difference, that is, the smallest worthwhile difference, was calculated as 0.5×the between-subject SD. A relatively conservative value was used for the smallest worthwhile difference (ie, 0.5 rather than the standard 0.2[27]) because the relationships between changes in sleep and changes in sports performance are not yet well quantified. The resultant probabilities were used to mechanically interpret the likelihood that an observed effect was a true effect using the following standards: <1%, almost certainly not; 1–5%, very unlikely; 5–25%, unlikely; 25–75%, possibly; 75–95%, likely; 95–99%, very likely; and >99%, almost certainly. If the probability that a mean value at altitude was greater than its corresponding mean value at baseline and the probability that it was lower were both >5%, then the effect was interpreted as unclear.

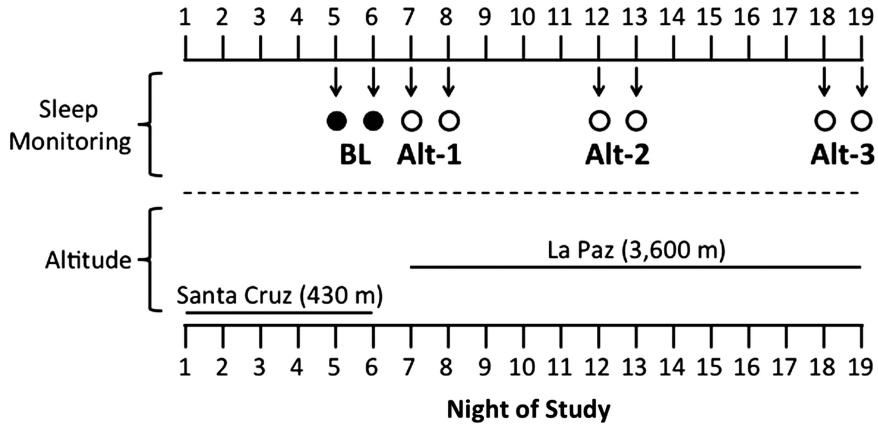

Figure 1 Sleep study protocol. The bottom section of the figure indicates when participants were at near sea level and when they were at high altitude. The top section of the figure indicates when participants had their sleep monitored. Only six sets of data acquisition equipment were available, so for each time point, data collection occurred on two successive nights, each with six participants.

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Table 1 Sleep architecture for adolescents

	Light sleep % (stages N1 & N2)	Deep sleep % (stage N3)	REM sleep % (stage R)
Normative	57.0	22.0	21.0
Baseline	50.5	27.1	22.4
Altitude-1	54.5	25.7	19.8
Altitude-2	55.5	24.7	19.8
Altitude-3	51.0	26.1	22.9

Sleep stages as a percentage of total sleep time. Data are presented for normal adolescent sleep (normative), and for the sleep of this study's participants during one night at near sea level (baseline) and three nights at high altitude (altitude-1, altitude-2 and altitude-3).

RESULTS

Sleep

The sleep architecture for the baseline sleep in this study was similar to the sleep architecture based on normative data for adolescents[11] ($\chi^2(2)1.0$, p=0.61; table 1). Furthermore, the sleep architecture at baseline was preserved for all three sleeps monitored at high altitude (table 1), which occurred immediately after ascent ($\chi^2(2)0.3$, p=0.84), 1 week after ascent ($\chi^2(2)0.5$, p=0.76) and 2 weeks after ascent ($\chi^2(2)0.0$, p=0.99).

On the first nights at altitude, REM sleep was likely lower (figure 2D); sleep efficiency (figure 2A), total sleep time (figure 2B), and deep sleep (figure 2C) were possibly lower; and wake after sleep onset was possibly higher than on the baseline night (table 2). These differences are indicators of poorer sleep at altitude than at near sea level. For all other sleep-related variables, there were no differences between sleep on the first nights at altitude and sleep on the baseline night (table 2).

After a week at altitude, deep sleep (figure 2C) and REM sleep (figure 2D) were possibly lower; and stage N2 sleep was possibly higher than on the baseline night (table 2). These differences are indicators of poorer sleep at altitude than at near sea level. For all other sleep-related variables, there were no differences between sleep after a week at altitude and sleep on the baseline night (table 2).

After 2 weeks at altitude, time in bed and deep sleep (figure 2C) were possibly lower than on the baseline night (table 2). These differences are indicators of poorer sleep at altitude than at near sea level. In contrast, sleep onset latency, stage N1 sleep and wake after sleep onset were possibly lower; and sleep efficiency

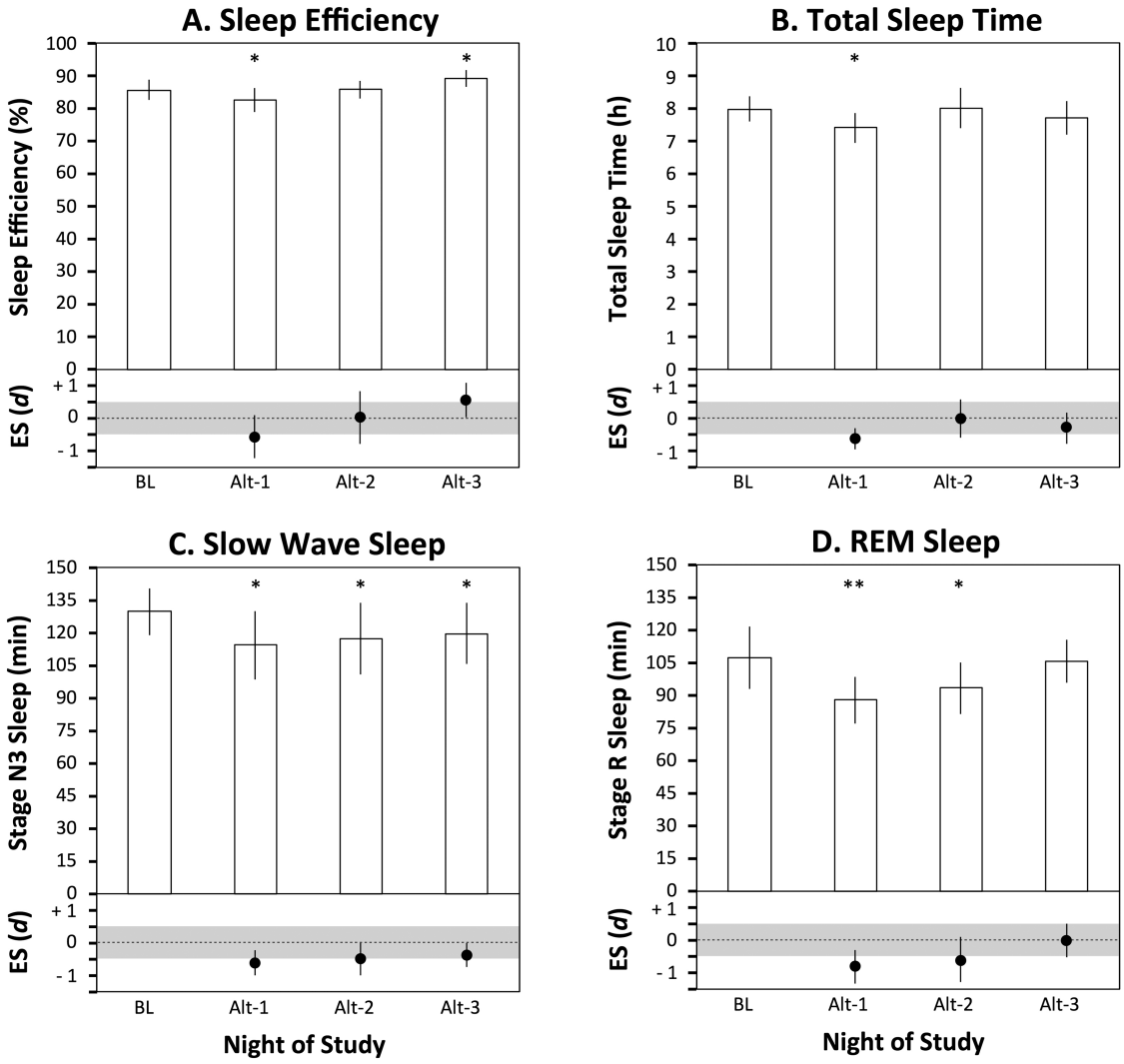

Figure 2 Characteristics of sleep for a baseline night at near sea level (BL) and three nights at high altitude (Alt-1, Alt-2 and Alt-3). In the top section of each panel, data for each night are presented as mean (±90% CIs). Asterisks indicate the likelihood that an observed effect, that is, difference from baseline, was a true effect: *possibly; **likely; ***very likely; ****almost certainly. In the bottom section of each panel, data are presented as the within-group standardised changes (±90% CIs) from baseline, and the shaded area represents trivial changes.

Table 2 Sleep, breathing, arousals and oxygen saturation for one night at near sea level and three nights at high altitude

	Baseline	Altitude-1 (immediately after ascent to altitude)			Altitude-2 (1 week after ascent to altitude)			Altitude-3 (2 weeks after ascent to altitude)		
Variable	Mean (SD)	Mean (SD)	Difference: effect sizewith 90% CL	Chances that value is greater/similar/lower than baseline value (%)	Mean (SD)	Difference: effect sizewith 90% CL	Chances that value is greater/similar/lower than baseline value (%)	Mean (SD)	Difference: effect sizewith 90% CL	Chances that value is greater/similar/lower than baseline value (%)
Time in bed (h)	9.3 (0.9)	9.0 (0.7)	−0.29 −0.48;−0.10	0/96/4	9.4 (1.4)	−0.02 −0.63; 0.58	7/84/9	8.7 (1.1)	−0.60 −1.11;−0.08	0/37/63
Total sleep Time (h)	8.0 (0.7)	7.4 (0.8)	−0.62 −0.95;−0.30	0/25/74	8.0 (1.1)	−0.01 −0.59; 0.58	7/85/8	7.7 (0.9)	−0.31 −0.78; 0.16	1/75/24
Sleep onset latency (min)	18 (8)	14 (7)	−0.22 −0.56; 0.11	0/92/8	23 (19)	0.12 −0.39; 0.63	10/87/3	12 (8)	−0.58 −0.94;−0.22	0/35/65
Stage N1 (min)	26 (13)	26 (13)	0.12 −0.31; 0.55	7/92/1	25 (10)	−0.01 −0.61; 0.59	8/84/8	20 (7)	−0.39 −0.88; 0.10	0/65/35
Stage N2 (min)	216 (28)	215 (37)	−0.04 −0.27; 0.19	0/100/0	245 (73)	0.42 −0.29; 1.13	42/56/2	216 (41)	−0.04 −0.56; 0.47	4/89/7
Stage N3 (min)	130 (19)	114 (27)	−0.63 −1.02;−0.24	0/28/72	117 (29)	−0.50 −1.00; 0.00	0/50/50	120 (24)	−0.39 −0.79;−0.01	0/71/29
Stage R (min)	107 (25)	88 (19)	−0.82 −1.33;−0.32	0/14/86	93 (21)	−0.59 −1.28; 0.10	1/40/59	106 (17)	−0.01 −0.52; 0.50	5/89/6
Wake after sleep onset (min)	64 (39)	81 (34)	0.58 −0.05; 1.21	59/40/1	59 (32)	−0.04 −0.77; 0.69	10/76/14	47 (31)	−0.51 −1.17; 0.16	1/48/51
Sleep efficiency (%)	86 (5)	82 (6)	−0.56 −1.21; 0.09	1/42/57	86 (5)	0.03 −0.78; 0.83	15/71/13	89 (4)	0.57 0.04; 1.09	59/41/0
Respiratory disturbance Index (h^{-1})	1 (1)	30 (39)	1.19 0.67; 1.71	98/2/0	45 (57)	1.34 0.78; 1.90	100/0/0	34 (41)	1.26 0.73; 1.79	99/1/0
Central apnoeas (count)	8 (7)	142 (216)	0.70 0.25; 1.16	78/21/0	285 (451)	0.78 0.19; 1.37	80/20/0	177 (231)	0.86 0.36; 1.34	89/11/0
Hypopneas (count)	2 (2)	92 (101)	1.81 1.26; 2.37	100/0/0	110 (114)	1.95 1.40; 2.50	100/0/0	98 (109)	1.92 1.45; 2.39	100/0/0
Periodic breathing (%)	0 (0)	13 (18)	0.91 0.38; 1.44	90/9/0	24 (32)	0.99 0.33; 1.65	90/10/0	21 (28)	0.95 0.31; 1.58	88/11/0
Total arousals (count)	88 (27)	143 (68)	0.58 0.27; 0.90	68/32/0	293 (303)	1.07 0.27; 1.86	89/11/0	144 (92)	0.53 0.11; 0.94	55/45/0
Spontaneous arousals (count)	86 (28)	83 (44)	−0.20 −0.64; 0.24	1/87/12	75 (76)	−0.59 −1.17;−0.01	0/39/61	72 (44)	−0.47 −1.05; 0.12	1/53/46
Respiratory arousals (count)	2 (3)	60 (84)	0.89 0.47; 1.31	94/6/0	155 (290)	0.97 0.40;1.53	92/8/0	72 (108)	0.90 0.38; 1.42	90/10/0
Mean SpO_2 (%)	97 (1)	82 (3)	−5.84 −6.52;−5.16	0/0/100	86 (3)	−4.06 −4.47;−3.42	0/0/100	88 (1)	−3.41 −3.64;−3.18	0/0/100
Minimum SpO_2 (%)	93 (2)	74 (4)	−3.73 −4.23;−3.23	0/0/100	77 (6)	−3.13 −3.98;−2.28	0/0/100	80 (3)	−2.37 −2.71;−2.03	0/0/100
Desaturations >3% (count)	3 (3)	214 (283)	1.55 1.07; 2.03	100/0/0	329 (407)	1.79 1.28; 2.29	100/0/0	256 (305)	1.75 1.30; 2.20	100/0/0

Variables related to sleep, breathing, arousals and oxygen saturation for one night at near sea level (baseline) and three nights at high altitude (altitude-1, altitude-2 and altitude-3). For each night, data are presented as means (±SD). For each night at altitude, data are also presented as standardised differences from baseline (ie, effect size with 90% CL), and the chances that an observed value is greater/similar/lower than the corresponding baseline value are given. Baseline occurred 1–2 nights before ascent to high altitude, altitude-1 occurred 1–2 nights after ascent, altitude-2 occurred 6–7 nights after ascent and altitude-3 occurred 12–13 nights after ascent.

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(figure 2A) was possibly higher after 2 weeks at altitude than on the baseline night (table 2). These differences are indicators of better sleep at altitude than at near sea level. For all other sleep-related variables, there were no differences between sleep after 2 weeks at altitude and sleep on the baseline night (table 2).

Respiratory events and periodic breathing

At baseline, none of the participants exhibited any signs of disordered breathing during sleep and there was almost no respiratory disturbance. However, ascending to altitude had a profound negative effect on breathing during sleep. Compared with baseline, the respiratory disturbance index (figure 3A) was very likely higher on the first nights at altitude and almost certainly higher after 1 and 2 weeks at altitude (table 2). On all three nights monitored at altitude, hypopneas were almost certainly higher; and central apnoeas and periodic breathing (figure 3B) were likely higher than on the baseline night (table 2). There were marked individual differences in susceptibility to periodic breathing. Half of the participants exhibited periodic breathing on all three nights monitored at altitude, while the other half did not exhibit periodic breathing on any of these nights (see figure 4 for an example). As a result of this finding, the athletes' data were split into two groups, and the sleep architecture of these groups was compared for all four sleeps at near sea level and altitude using χ^2 tests. There was no difference in sleep architecture between the two groups at near sea level ($\chi^2(2)0.1$, $p=0.95$), on the first nights at altitude ($\chi^2(2)0.1$, $p=0.95$), after 1 week at altitude ($\chi^2(2)0.1$, $p=0.96$) or after 2 weeks at altitude ($\chi^2(2)0.0$, $p=0.98$).

Arousals

Compared with baseline, total arousals were possibly higher on the first night at altitude, likely higher after 1 week at altitude and possibly higher after 2 weeks at altitude (table 2). These differences were not due to changes in spontaneous arousals, which were possibly lower after 1 and 2 weeks at altitude compared with baseline, but rather due to the likely increases in respiratory arousals (figure 3C) for all sleeps at altitude compared with baseline (table 2).

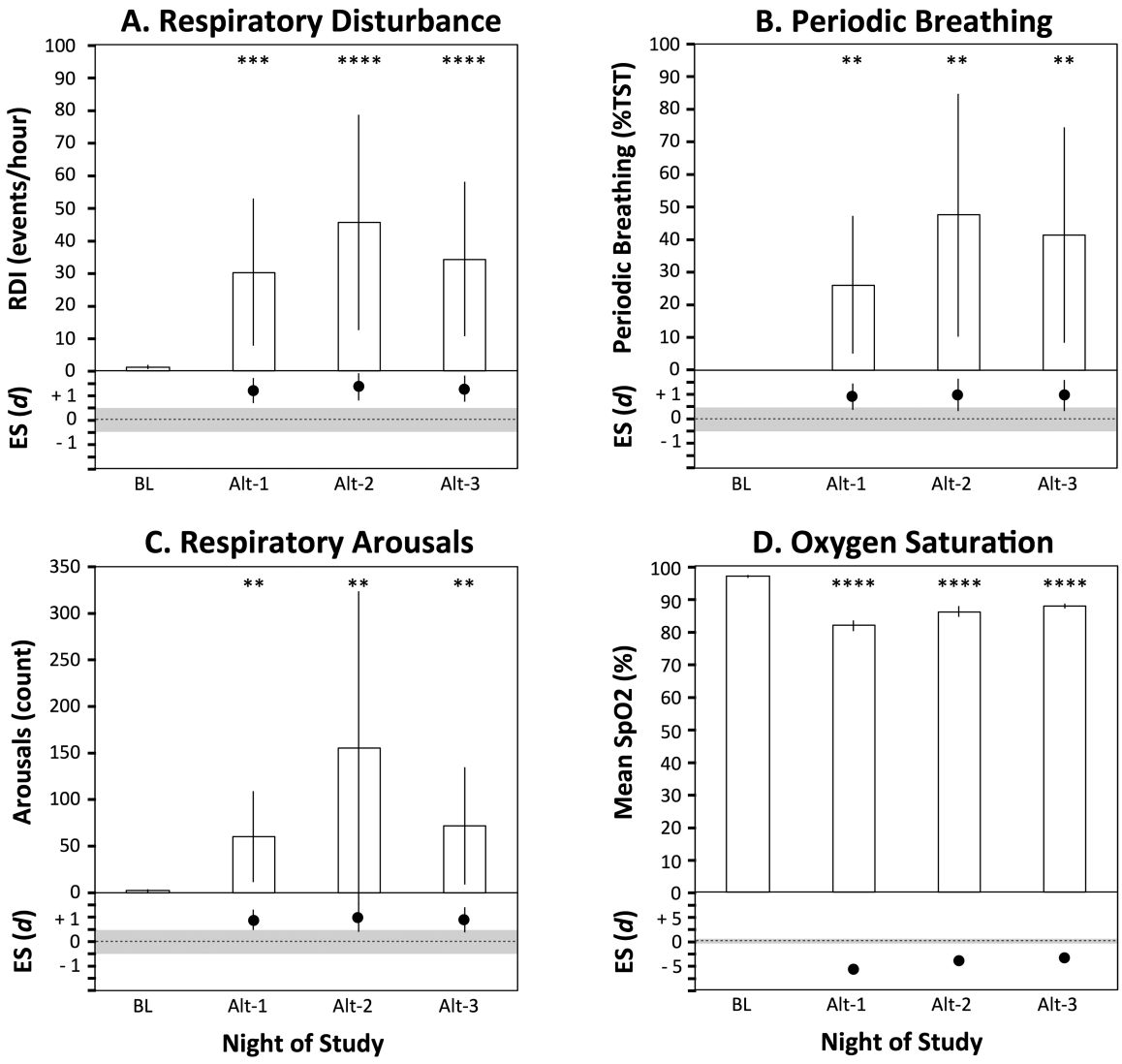

Figure 3 Characteristics of breathing, arousals, and oxygen saturation for a baseline night at near sea level (BL) and three nights at high altitude (Alt-1, Alt-2 and Alt-3). In the top section of each panel, data for each night are presented as mean (±90% CIs). Asterisks indicate the likelihood that an observed effect, that is, difference from baseline, was a true effect: *possibly; **likely; ***very likely; ****almost certainly. In the bottom section of each panel, data are presented as the within-group standardised changes (±90% CIs) from baseline, and the shaded area represents trivial changes.

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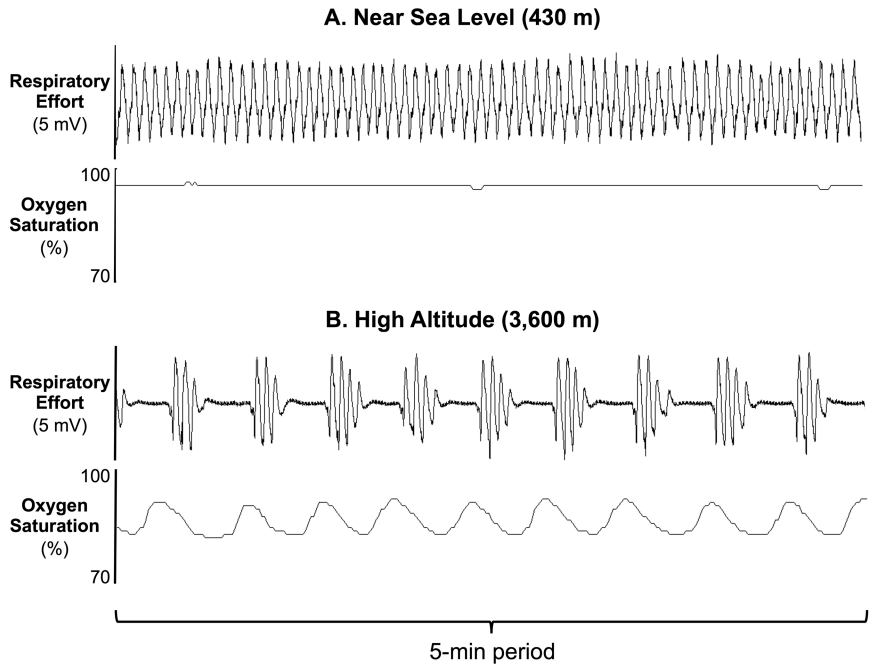

Figure 4 Examples, from one participant, of the respiratory effort and oxygen saturation associated with normal breathing during sleep at near sea level (A) and severe disordered breathing during sleep at high altitude (B).

Oxygen saturation

Compared with the baseline sleep, mean oxygen saturation (figure 3D) and minimum oxygen saturation were almost certainly lower; and desaturations were almost certainly higher for all sleeps at altitude (table 2).

DISCUSSION

Sleep

Exposure to high altitude caused some, but not major, disruption to the athletes' sleep. The main aspect of sleep that was affected was REM sleep, which was lower on the first nights at altitude (88 min) than at near sea level (107 min), improved after 1 week (93 min) and returned to its baseline level after 2 weeks (106 min; figure 2). This type of disruption in the early days of a camp could be harmful, particularly with young athletes, because losing REM sleep can impair learning and memory consolidation.[8] These results differ markedly from those of previous studies, which have found that exposure to high altitude substantially reduces, or even eliminates, deep sleep and/or REM sleep immediately after ascent.[12–15] The true explanation(s) for why altitude exposure was less disruptive to sleep in this study compared with previous studies cannot be known for certain, but there are some important differences between the studies that may be relevant. First, the previous studies were conducted under more extreme conditions than this study, as they occurred at higher altitudes (ie, ~5000 m), and in some cases participants had to sleep in tents or huts in subzero ambient temperatures. Second, the participants in the previous studies were considerably older (ie, mid-30 s) than in this study, and they were not elite athletes.

Disordered breathing

None of the athletes exhibited any signs of disordered breathing during sleep at near sea level, but immediately upon ascent to altitude, there was a marked increase in the number of central apnoeas and hypopneas, episodes of periodic breathing emerged and oxygen saturation was greatly reduced (figure 3). These indicators of disordered breathing during sleep had not improved after 2 weeks at altitude. The acute effects of terrestrial altitude on disordered breathing during sleep have been reported previously, albeit in older participants,[14 16] but this is the first study to show that these effects persist for at least 2 weeks. The 10 athletes in this study were split into two equal groups based on the degree to which their breathing during sleep was impaired: five of them were severely affected, and the other five were unaffected, on all three nights of testing at altitude. This is consistent with previous studies of sleep at altitudes of 3800 and 3900 m, which found severe disordered breathing in three of six participants[28] and 12 of 18 participants,[14] respectively. The results of this study, and one previous study,[14] indicate that the presence/absence of periodic breathing does not affect sleep architecture. The occurrence of disordered breathing during sleep at altitude may be due to hypoxia-induced instability in ventilatory control,[29 30] so individual differences in susceptibility to this instability[31 32] provides a possible explanation for the bifurcation of the sample in this study and others.

Arousals and fragmentation

The athletes had very few arousals associated with respiratory events during sleep at sea level (2), but this dramatically increased immediately on ascent to altitude (60), increased again after 1 week at altitude (155), and was still elevated after 2 weeks at altitude (72). It was beyond the scope of this study to determine whether the fragmentation of sleep caused by arousals was of any functional significance, but even in the presence of otherwise normal sleep architecture, frequent arousals can be harmful. For example, in experimental studies in which an auditory tone is used to induce arousals and fragment sleep without reducing the amount of deep sleep or REM sleep, the levels of cognitive impairment, mood disturbance and daytime sleepiness are similar to those observed following a night of total sleep deprivation.[33 34] It is unlikely that participants in this study would have experienced this degree of impairment, given that their arousals during sleep at altitude were less frequent (ie, every 2–3 min) than arousals during sleep in these experimental studies (ie, every 30–60 s), but there may have been some impairment nonetheless.

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Limitations

This study had two main limitations, neither of which was particularly serious. First, the baseline sleeps at near sea level occurred 4–5 nights after travelling from Australia to Bolivia (a time zone change of 10 h to the east), so it is possible that the athletes' body clocks had not fully adjusted before these assessments occurred. However, the athletes followed a schedule of sunlight exposure/avoidance to facilitate adjustment to the new time zone, based on the principles of circadian physiology,[35] and ultimately their baseline sleep was similar to, if not better than, normal sleep for adolescents. Second, to minimise the athletes' potential discomfort, the montage of sleep monitoring equipment that was employed did not include a sensor to measure airflow. The absence of an airflow sensor meant that central apnoeas/hypopneas were identified based on respiratory effort alone, rather than respiratory effort and airflow.[26] This is a reasonable approach, given that it is unlikely that airflow could be present in the absence of respiratory effort. Furthermore, it was not possible to identify obstructive apnoeas/hypopneas, but these were unlikely to have occurred given that none of the athletes had a history of obstructive sleep apnoea and altitude exposure does not typically cause obstructive events.[16]

CONCLUSIONS

This is the first study to examine the effects of high altitude on the sleep of young elite athletes. The immediate effects of altitude are to cause a general reduction in the amount of REM sleep obtained, and to severely disrupt breathing during sleep for half the athletes. There is some recovery in REM sleep after 1 week, and after 2 weeks it has returned to normal. In contrast, disordered breathing during sleep does not improve during 2 weeks of exposure to altitude. Future studies should examine whether or not breathing abnormalities during sleep at high altitude, and the accompanying sleep fragmentation, affect the efficacy of altitude training for young elite athletes.

What are the new findings?

- High altitude reduces rapid eye movement (REM) sleep in elite young athletes immediately on ascent. REM sleep improves after 1 week at altitude, and returns to normal after 2 weeks.
- High altitude causes severe disordered breathing in 50% of elite young athletes immediately on ascent. Disordered breathing does not improve after 2 weeks at altitude.

How might it impact on clinical practice in the near future?

Elite young athletes participating in live high, train low camps with simulated altitude should wear respiratory bands and/or pulse oximeters during their first few sleeps. If an athlete has disordered breathing and their daytime well-being and/or performance is also poor, then they may get more benefit from the camp if they sleep at sea level.

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