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Effect of Cluster versus Traditional Set Resistance Exercises on Sleep Architecture and Oxygen Saturation

Küme Set ve Geleneksel Set Direnç Egzersizlerinin Uyku Mimarisi ve Oksijen Satürasyonu Üzerine Etkisi

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Abstract

Objective: The aim of this study was to compare the effect of cluster set (CLS) and traditional set (TS) resistance exercises on sleep architecture and nocturnal oxygen saturation (SpO_2) in healthy recreationally active males.

Materials and Methods: Fourteen males (22.41 ± 2.38 years; 177.79 ± 6.55 cm; 77.15 ± 12.65 kg) completed CLS or TS resistance training in a randomized crossover design (72 ± 24-hour washout). Exercises were performed at 85% intensity. TS was applied with 4 x 6 repetitions and 3-minute rest between sets and exercises; CLS was performed as 4 x (3 x 2) repetitions with 30–second intraset and 3-minute rest interset and exercise. Blood lactate was assessed pre- and post-exercise, anxiety was evaluated pre-sleep, and polysomnography was conducted post-exercise (11:00 p.m.–7:00 a.m.).

Results: Repeated-measures ANOVA and paired samples t-tests indicated that sleep architecture parameters and SpO_2 after TS or CLS resistance exercise did not differ significantly from the control sleep session ($p > 0.05$).

Conclusion: In this study, neither set configuration significantly affected sleep architecture parameters or nocturnal SpO_2 compared to control sleep.

Keywords: Polysomnography, REM sleep, NREM sleep, strength training, set structure

Öz

Amaç: Bu çalışmanın amacı, sağlıklı rekreasyonel aktif erkeklerde küme set (CLS) ve geleneksel set (TS) direnç egzersizlerinin; uyku mimarisi ve uyku sırasındaki oksijen satürasyonu (SpO_2) üzerine olan etkilerini karşılaştırmaktır.

Gereç ve Yöntem: On dört erkek (22,41 ± 2,38 yaş; 177,79 ± 6,55 cm; 77,15 ± 12,65 kg) rastgeleleştirilmiş çapraz geçişli bir tasarımda (72 ± 24 saat aralıkla) CLS veya TS direnç antrenmanlarını tamamladı. Egzersizler %85 şiddette uygulandı. TS 4 x 6 tekrar, set arası ve hareketler arası 3 dakika dinlenme ile; CLS 4 x (3 x 2) tekrar, set içi 30 saniye, setler ve hareketler arası 3 dakika dinlenme ile gerçekleştirildi. Kan laktadı egzersiz öncesi ve sonrasında değerlendirildi. Uyku öncesi anksiyete değerlendirildi; polisomnografi egzersiz sonrasında (23:00–07:00) uygulandı.

Bulgular: Tekrarlı ölçümlerde ANOVA ve eşleştirilmiş örneklemler t-testleri, TS veya CLS direnç egzersizleri sonrasında uyku mimarisi parametreleri ve SpO_2'nin kontrol uykusuna kıyasla anlamlı farklılık göstermediğini ortaya koydu ($p > 0,05$).

Sonuç: Çalışmanın sonuçlarına göre, hiçbir set konfigürasyonu uyku mimarisi parametrelerini veya SpO_2'yi kontrol uykusu değerlendirmesinden farklı etkilememiştir.

Anahtar Kelimeler: Polisomnografi, REM uykusu, NREM uykusu, kuvvet antrenmanı, set yapısı

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Introduction

Despite evidence showing the effects of training and competition on athletes' sleep (1), the magnitude and underlying mechanisms of these effects have not yet been fully elucidated (2). Although the neural mechanisms involved in sleep (3) and central or peripheral nervous system fatigue (4) may appear different, exercise protocols are known to impact sleep (5). Aerobic exercise is recognized as a non-pharmacological method to increase both the amount and quality of sleep (6). In addition to aerobic exercise, reports indicate that progressive resistance exercises can also improve sleep quality (7). However, resistance exercises have numerous training variables (exercise type, set configuration, load lifted, training volume, rest between sets, and repetition speed), with manipulation of these variables producing different results. Therefore, it is challenging yet important to determine the effects of resistance exercises on human health.

Kovacevic et al. (7) reported that chronic resistance exercise improves various aspects of sleep, particularly sleep quality. Previous studies examining the effects of acute and chronic resistance exercise on sleep architecture and quality have typically utilized traditional set (TS) protocols (8,9). The TS method causes a temporary decrease in muscle power and movement velocity due to phosphocreatine depletion and increased blood lactate [$(La^-)_b$] concentrations (10). High ammonia concentrations and decreased neural activation can also lead to muscle fatigue (11,12). Therefore, it has been suggested that TS configurations may not be the most appropriate choice in clinical applications (13).

In contrast to these disadvantages of the TS method of resistance training, cluster set (CLS) configurations have been associated with lower [$(La^-)_b$] concentrations and increased repetition velocity compared to TS in both acute and long-term training (14,15). Given that sleep and exercise interact through complex physiological and psychological pathways (5), the effects of fatigue induced by TS and CLS exercise on sleep architecture and oxygen saturation (SpO_2) during sleep are still not fully understood. Therefore, this study aimed to examine the acute effects of TS and CLS resistance training on sleep architecture and nocturnal SpO_2. We hypothesized that CLS would lead to improved sleep architecture and nocturnal SpO_2 compared to TS.

Materials and Methods

Experimental Design

This study employed a randomized, controlled, crossover experimental design with repeated - measures. Participants underwent three conditions-control (no exercise), TS exercise, and CLS exercise-each separated by a 72 ± 24-hour washout period. Condition order was randomized using a computer program to mitigate the "first-night effect" associated with polysomnography (PSG) and potential carry-over effects between interventions.

During the initial screening session, participants' anthropometric data, sleep quality, anxiety level, and chronotype (natural circadian rhythm) were assessed. In the second session, a 10-repetition maximum (10-RM) test was conducted for the six resistance exercises. In subsequent sessions, participants followed one of three assigned conditions. In the control condition, resting [$(La^-)_b$] concentration was measured, followed by overnight PSG evaluation. In the TS and CLS conditions, [$(La^-)_b$] concentration was measured pre- and post-exercise, and the resistance training protocol was followed by overnight PSG evaluation.

Participants

A G*Power F-test analysis was performed to determine the number of volunteers to enroll in the study. With effect size (f) = 0.36, type I error rate (α) = 0.05, and power (1-β) = 0.80 for a single-group, repeated-measures design with 3 repetitions, the required sample size was 14. Accordingly, 14 recreationally active men (age; 22.41 ± 2.38 years; height; 177.79 ± 6.55 cm; weight; 77.15 ± 12.65 kg) were recruited to participate in this study. The participants were involved in sports such as swimming, football, basketball, tennis, athletics, and fitness.

Inclusion criteria were as follows: (a) male sex, aged 18 to 30 years; (b) recreational sports participation at least 3 days per week for at least 3 months; (c) no physical or psychological illness; (d) no tobacco, alcohol, medication, or caffeine use; (e) no diagnosed sleep disorder; and (f) intermediate chronotype.

Ethics committee approval for the study was obtained from the Medical Research Ethics Committee of the Ege University Faculty of Medicine (decision number: 21-3.1T/60, date: 18.03.2021). All participants were informed about the purpose of the study and signed a voluntary consent form prior to participation, in accordance with the Declaration of Helsinki.

Data Collection Tools

Anthropometric Measurements

Height was measured barefoot using a Seca stadiometer. Body weight, body mass index, and body fat percentage were assessed using a Tanita BC-418 body composition analyzer, at the same time of day to ensure consistency.

Chronotype Assessment

Chronotype was assessed using a culturally adapted and validated version of the Horne-Ostberg questionnaire (16). Only participants with intermediate types were included in the study.

Sleep Quality Assessment

A culturally adapted and validated version of the Pittsburgh Sleep Quality Index was used to evaluate the participants' sleep quality prior to the training sessions (17).

Anxiety Assessment

A validated, culturally adapted version of the State-Trait Anxiety Inventory (Form X) was administered before each PSG evaluation to assess anxiety levels (18).

Calorie Determination

Daily caloric requirements were estimated using the Harris-Benedict equation (basal metabolic rate x 1.55 activity factor) (19). The timing and content of pre-sleep meals were standardized across all conditions.

Ten-Repetition Maximal Strength

Individual training loads were determined using a 10-RM protocol for each prescribed exercise (20).

Blood Lactate Measurement

Resting [$(La^-)_b$] was measured after 10 minutes of passive rest upon arriving at the laboratory using a YSI 1500 Lactate Analyzer (Yellow Springs Instruments, Ohio, USA). For the TS and CLS conditions, a second measurement was performed 5 minutes post-exercise.

Polysomnographic Sleep Analysis

PSG recordings were carried out in the Sleep Disorders Monitoring Center in the Neurology Department of University of Health Sciences Türkiye, İzmir Bozyaka Training and Research Hospital. The sleep room was a controlled environment with a noise level of 27 dB (decibels), temperature of 21 °C, and 0 lx (lux) illumination. Recordings commenced at 11:00 p.m. and concluded at 7:00 a.m.

Sleep data were acquired and analyzed using an EMBLA N7000 PSG system and RemLogic software (version 3.4.1). Neuro-ocular recordings were obtained using three-channel (F4-M1, C4-M1, O2-M1) electroencephalography in the frontal, central, and occipital regions of the head and two-channel electrooculography near the right and left eyes. Myographic data were obtained by surface electromyography of the chin and bilateral tibialis anterior muscles. Respiratory and position data were obtained via cannula and thermistor for oronasal airflow to the nose; respiratory belts in the relevant areas for thoracic and abdominal respiratory movements; a body position control belt; finger pulse oximeter for SpO_2; and respiratory sound and synchronous video recording.

Sleep datasets were staged in 30-second epochs and scored according to the 2016 American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events (21).

Strength Training Program

The resistance training protocol (85% intensity) included six exercises: Smith machine front squat, bench press, lat pull-down, cable face pull, barbell preacher curl, and triceps pushdown. The TS condition comprised 4 x 6 reps with 3-min rest interset and exercise. The CLS configuration involved 4 sets of 3 x 2 reps with 30-s intraset and 3-min rest interset and exercise.

Participant Schedule

Participants continued their routine daily activities (without exercise) and maintained a regular sleep schedule (11:00 p.m. to 7:00 a.m.) on non-testing days and during washout periods (Figure 1A). On sleep measurement days, participants rose at 7:00 a.m. and reported to the laboratory in a rested state. After baseline [$(La^-)_b$] measurements, participants trained from 4:00 p.m. to 6:00 p.m., and the final meal was consumed between 6:00 p.m. and 7:00 pm. Between 7:00 p.m. and 11:00 p.m., the participants rested, traveled to the clinic, and underwent preparations for PSG evaluation, which was conducted between 11:00 p.m. and 7:00 a.m. (Figure 1B).

Statistical Analysis

Results were evaluated using SPSS 25.0 (IBM Corp., Armonk, NY). According to the Shapiro-Wilk test, 15 parameters were found to be normally distributed (TRT, TST, N1, N2, N3, R, RL, SE, mean SpO2 during non-rapid eye movement (NREM), rapid-eye movement (REM), wakefulness, state anxiety, pre-exercise [$(La^-)_b$], post-exercise [$(La^-)_b$] and $\Delta[(La^-)_b]$) and 9 parameters were non-normally distributed (SOL, N1L, N2L, N3L, W, WASO, AHI, PLMS, and trait anxiety).

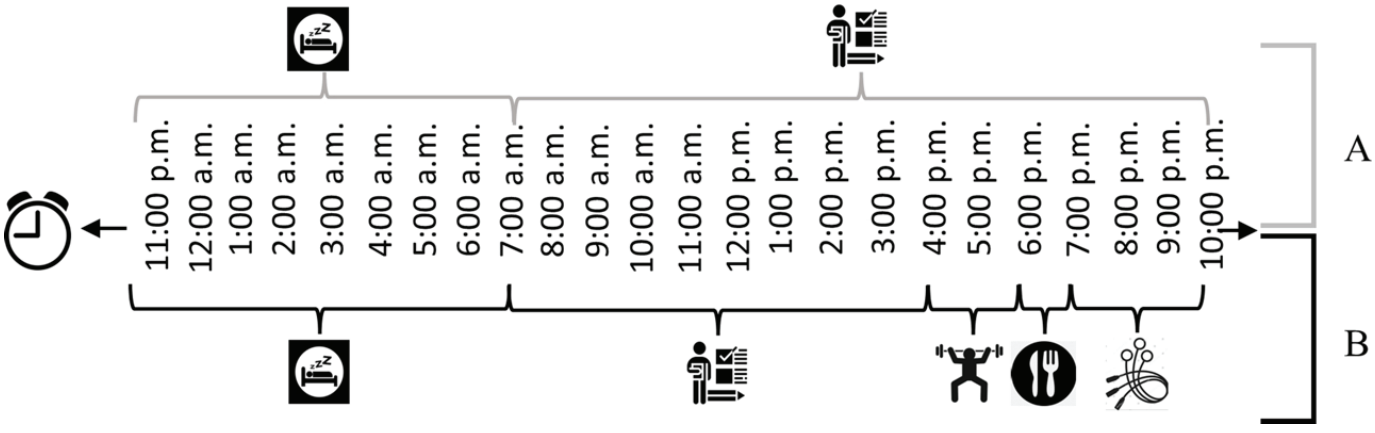

Figure 1. A) Participants' daily schedule before and between sleep measurement. B) Daily schedule on the day of sleep measurement.

Parametric data were analyzed using repeated-measures ANOVA. Partial eta-squared (η_p^2) was calculated as the effect size and was categorized as small (0.01–0.06), medium (0.06–0.14), and large (>0.14) (22). Pairwise comparisons were performed with Bonferroni correction, and Cohen's d was determined as the effect size, categorized as small (0.2–0.5), medium (0.5–0.8), and large (>0.8) (22). Paired samples t-test and Cohen's d effect size were used for pairwise comparisons of post-exercise $[(La^-)_b]$ and $\Delta[(La^-)_b]$.

Repeated measures analysis of non-parametric data was performed using the Friedman test with Kendall's W effect sizes, categorized as small (0.1–0.3), medium (0.3–0.6), large (0.6–1.0), and very large (>1.0). For pairwise comparisons of non-parametric data, the Conover test was used and effect sizes were presented with Cohen's d. Statistical significance was set at $p < 0.05$.

Results

Descriptive statistics of the participants are given in Table 1.

Based on the repeated-measures ANOVA test and Friedman test, a significant difference was observed only in REM latency (RL) ($F_{2,26}=3.812$, $p=0.035$, $\eta_p^2=0.227$). No statistically significant difference was found in repeated measures for any other parameter ($p>0.05$) (Table 2). Paired samples t-test revealed a significant difference in post-exercise $[(La^-)_b]$ ($t_{13}=5.766$, $p\leq0.001$) and $\Delta[(La^-)_b]$ ($t_{13}=5.375$, $p\leq0.001$) (Table 2). No other significant differences were identified in pairwise comparisons between interventions ($p > 0.05$) (Table 2).

Discussion

This study aimed to examine the effects of strength training with CLS and TS configurations on sleep architecture and changes in SpO_2 during sleep. Contrary to our hypothesis that the CLS resistance training protocol would improve sleep architecture and nocturnal SpO_2 values compared to the TS protocol, our findings did not support this prediction. The main results indicated that the CLS and TS interventions yielded no statistically significant differences in sleep architecture parameters or SpO_2 during sleep ($p>0.05$).

Table 1. Descriptive statistics of the participants (n = 14).

	$\bar{X}\pm$ SD
Age (years)	22.41 ± 2.38
Height (cm)	177.79 ± 6.55
Weight (kg)	77.15 ± 12.65
Body mass index (kg/m²)	24.36 ± 3.21
Body fat ratio (%)	12.27 ± 4.85
Pittsburgh Sleep Quality Inventory (PSQI) score	3.07 ± 1.38
Horne–Östberg chronotype score	51.29 ± 6.01
Daily calorie requirement (kcal.)	2886.54 ± 310.11

$\bar{X}$ ± SD: Mean ± standard deviation

The absence of a statistical difference in any of the study parameters may be explained by the ceiling and floor effect hypothesis. This theory suggests that better baseline sleep equates to less potential for improvement in sleep parameters (23). As our participants were recreationally active and reported no sleep problems, there may not have been a measurable change or improvement in their sleep architecture (24,25). Acute physical exercise can increase REM latency, reduce REM duration, and slightly increase slow-wave sleep (25). It has also been reported that the positive effects of acute exercise on sleep are observable after exercise sessions lasting at least one hour (24). Nevertheless, acute physical exercise is reported to have a small to moderate impact on sleep (26). This suggests that although the exercise protocol in our study lasted longer than one hour, the time spent under load in the single sessions was not sufficient to alter sleep parameters in these participants.

Although numerous studies have examined the relationship between exercise and sleep, comparing their results is challenging because of differences in sample groups (e.g., age, clinical diagnoses), sleep evaluation methods, specific parameters assessed, and interventions (8,27). In the literature, resistance training studies employing TS configurations with comparable research designs and study populations have consistently reported no statistically significant differences in any parameters related to sleep architecture ($p > 0.05$) (8,28,29).

Ramos-Campo et al. (8) evaluated 15 resistance-trained men (age; 23.4 ± 2.4) performing 4 sets of 10 repetitions or 5 sets of 8 repetitions at the same intensity (75% of 1-RM). Using actigraphy to assess sleep quality, they reported no statistically significant difference in sleep onset latency (SOL), sleep efficiency (SE), or time in bed between the intervention groups ($p > 0.05$). Although their study is the most similar to ours in terms of participant characteristics and research design, the use of actigraphy rather than PSG is a major methodological distinction (8). Nevertheless, some of their findings align with ours, particularly regarding the lack of significant impact on SOL and SE following TS exercise.

In a study by de Faria et al. (28) 27 sedentary men with no sleep problems or diagnosed disease performed TS resistance exercise in 3 sets of 15 repetitions at 50% of 1-RM at one of three times of day: morning, afternoon, or evening. The training lasted 40–45 minutes with 90-second intraset rest, and sleep was assessed by PSG on the same night after a single exercise session. The authors reported no significant difference in any sleep parameter (SOL, RL, TST, SE, N1, N2, N3, N4, SWS, R, and W) compared to the baseline measurements ($p > 0.05$). Although this study is also similar to ours in that the sample group comprised healthy young men and sleep was evaluated by PSG, the lower intensity exercise and inclusion of sedentary participants are major differences. However, the results reported by de Faria et al. (28) are similar to the findings we obtained after TS.

Table 2. Comparisons of sleep architecture and oxygen saturation parameters.

	Control $\bar{X} \pm SD$	TS $\bar{X} \pm SD$	CLS $\bar{X} \pm SD$	ANOVA p	ηp^2	Friedman p	Kendall's W	Control vs. TS			Control vs. CLS			TS vs. CLS		
								Δ	p	ES	Δ	p	ES	Δ	p	ES
TRT (min)	434.47 ± 37.93	451.87 ± 36.72	425.40 ± 49.92	0.069	0.186			17.4	0.45	0.46	9.07	0.826	0.20	26.47	0.195	0.60
TST (min)	368.91 ± 63.87	394.84 ± 58.16	378.21 ± 58.93	0.211	0.113			25.92	0.44	0.42	9.30	0.988	0.15	16.62	0.965	0.28
N1 (min)	31.77 ± 14.32	28.71 ± 13.74	27.57 ± 18.99	0.670	0.030			3.057	1.0	0.21	4.20	1.0	0.24	1.14	1.0	0.24
N2 (min)	249.94 ± 56.26	268.80 ± 50.33	257.19 ± 58.89	0.352	0.077			18.86	0.527	0.35	7.25	1.0	0.12	11.61	1.0	0.21
N3 (min)	38.78 ± 25.45	39.0 ± 23.51	38.56 ± 28.48	0.997	< 0.001			0.214	1.0	0.008	0.221	1.0	0.008	0.221	1.0	0.016
R (min)	48.41 ± 21.99	58.32 ± 24.23	52.97 ± 18.49	0.375	0.151			9.907	0.684	0.42	4.564	0.727	0.22	5.343	1.0	0.24
RL (min)	172.92 ± 90.86	126.10 ± 64.42	109.35 ± 46.43	0.035*	0.227			46.82	0.229	0.62	63.57	0.079	0.88	16.75	1.0	0.88
SE (%)	84.82 ± 12.09	87.27 ± 9.48	88.39 ± 8.09	0.325	0.083			2.457	1.0	0.22	3.571	0.194	0.34	1.114	1.0	0.12
SpO_2 during wakefulness (%)	95.62 ± 1.00	95.55 ± 1.18	95.55 ± 0.87	0.905	0.008			0.071	1.0	0.05	0.071	1.0	0.07	<0.001	1.0	0.0
SpO_2 during NREM (%)	94.63 ± 1.20	94.72 ± 1.35	94.75 ± 1.12	0.839	0.013			0.086	1.0	0.07	0.121	1.0	0.10	0.036	1.0	0.02
SpO_2 during REM (%)	95.10 ± 1.11	95.27 ± 1.30	95.42 ± 1.15	0.334	0.081			0.164	1.0	0.14	0.314	0.334	0.28	0.150	1.0	0.12
State anxiety	46.57 ± 3.18	45.50 ± 4.25	46.36 ± 3.67	0.627	0.035			1.071	1.0	0.018	0.214	1.0	0.06	0.857	1.0	0.21
Pre-exercise $[(La^-)_b]$ $(mmol \cdot L^{-1})$	1.47 ± 0.38	1.51 ± 0.32	1.46 ± 0.46	0.887	0.009			0.048	1.0	0.11	0.006	1.0	0.02	0.054	1.0	0.12
Post-exercise $[(La^-)_b]$ $(mmol \cdot L^{-1})$	—	7.14 ± 1.77	4.57 ± 1.00	—	—			—	—	—	—	—	—	2.56	<0.001*	1.78
$\Delta[(La^-)_b]$ $(mmol \cdot L^{-1})$	—	5.62 ± 1.72	3.11 ± 0.90	—	—			—	—	—	—	—	—	2.51	<0.001*	1.82
SOL (min)	10.56 ± 8.03	10.10 ± 11.80	11.63 ± 12.30			0.751	0.020	0.464	1.0	0.043	1.071	1.0	0.09	1.536	1.0	0.14
N1L (min)	10.56 ± 8.03	10.10 ± 11.80	11.63 ± 12.30			0.751	0.020	0.464	1.0	0.043	1.071	1.0	0.09	1.536	1.0	0.14
N2L (min)	16.49 ± 12.14	12.52 ± 11.85	14.58 ± 12.62			0.607	0.036	3.964	1.0	0.325	1.907	1.0	0.15	2.057	1.0	0.16
N3L (min)	45.10 ± 34.38	54.17 ± 47.43	58.33 ± 51.73			0.849	0.012	9.071	1.0	0.201	13.23	1.0	0.29	4.164	1.0	0.09
W (min)	65.55 ± 52.32	57.01 ± 42.83	49.07 ± 36.38			0.109	0.158	8.543	1.0	0.193	16.47	0.1	0.37	10.89	0.5	0.17
WASO (min)	54.97 ± 49.47	46.92 ± 33.38	37.49 ± 37.42			0.257	0.097	8.057	1.0	0.198	17.48	0.428	0.43	9.429	0.5	0.23
AHI	1.95 ± 2.35	2.30 ± 2.77	2.38 ± 2.29			0.982	0.001	0.350	1.0	0.143	0.436	1.0	0.178	0.086	1.0	0.03
PLMS/h	0.357 ± 1.19	0.036 ± 0.13	0.0001 ± 0.0001			0.368	0.071	0.321	1.0	0.461	0.357	0.237	0.512	0.036	1.0	0.05
Trait anxiety	42.50 ± 4.70	42.21 ± 4.13	41.21 ± 3.16			0.819	0.014	0.286	1.0	0.071	1.286	1.0	0.371	1.00	1.0	0.24

*p < 0.05: $\bar{X} \pm SD$: Mean ± SD, η_p^2: Partial eta-squared, Kendall's W: Friedman test effect size, Δ: Difference of means, ES: Cohen's d effect size, TS: Traditional set, CLS: Cluster set, TRT: Total record time, SOL: Sleep onset latency, SE: Sleep efficiency, N1: NREM stage 1, N2: NREM stage 2, N3: NREM stage 3, R: REM stage, W: Wakefulness during sleep period, N1L: NREM stage 1 latency, N2L: NREM stage 2 latency, N3L: NREM stage 3 latency, RL: REM stage latency, TST: Total sleep time, SpO2: Oxygen saturation, WASO: Wake after sleep onset, AHI: Apnea-hypopnea index, PLMS: Periodic limb movements in sleep, $[(La\text{-})_b]$: Blood lactate.

Another important parameter for sleep architecture and quality is the Apnea-Hypopnea Index (AHI), which is the average number of apneas and hypopneas occurring per hour of sleep (30). According to the AASM, obstructive sleep apnea (OSA) is classified according to AHI as mild (5–15), moderate (15–30), and severe (>30). The prevalence of OSA in the general population varies between 9% and 38%, with rates higher in men and increasing with age (31). The participants in our study had a normal AHI (<5) in the control sleep session. Although an increase was observed after the TS and CLS interventions, it was not statistically significant. Rossi et al. (29) reported that acute resistance training with 3 sets of 15 repetitions at 50% of 1-RM with 90-s interset rest did not result in significant changes in AHI or SpO_2 parameters. Similarly, we observed no statistically significant difference in mean SpO_2 during NREM or REM sleep. This is likely related to the low AHI values in our participants.

Study Limitations

This study has several limitations that should be considered. First, the sample consisted of recreationally active men without sleep problems. Therefore, the findings of this study cannot be generalized to clinical populations with sleep disorders, sedentary individuals, elite athletes, or female athletes. Second, as this study examined only acute effects, the findings may not reflect the chronic effects of different resistance training set configurations. Finally, the time under tension (TUT) employed in our protocol may not have provided a sufficient stimulus to elicit measurable changes. Future research should include participants with clinically diagnosed sleep disorders, recruit across different performance levels and both sexes, and employ protocols with longer TUT to determine whether different outcomes emerge. In addition, to ensure sample homogeneity, future studies may consider incorporating biochemical and neuronal homogeneity criteria related to sleep.

Conclusion

A single session of resistance training using either a TS or CLS configuration did not significantly alter sleep architecture or nocturnal SpO_2 in healthy, recreationally active young men without sleep disorders.

Ethics

Ethics Committee Approval: Ethics committee approval for the study was obtained from the Medical Research Ethics Committee of the Ege University Faculty of Medicine (decision number: 21-3.1T/60, date: 18.03.2021).

Informed Consent: All participants were informed about the purpose of the study and signed a voluntary consent form prior to participation, in accordance with the Declaration of Helsinki.

Footnotes

Authorship Contributions

Concept: O.D., C.K., Design: O.D., P.O., B.Ö., Data Collection or Processing: O.D., P.O., Analysis or Interpretation: O.D., C.K., P.O., B.Ö., Literature Search: O.D., Writing: O.D.

Conflicts of Interest: No conflict of interest was declared by the authors.

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Data Availability Statement: The data that support the findings of this study are available from the corresponding author from the upon reasonable request.

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