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Comparative efficacy of exercise modalities on sleep architecture in adults with sleep disorders: a systematic review and network meta-analysis of randomized controlled trials

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ABSTRACT

Objective: This study aims to evaluate the efficacy of exercise interventions on sleep architecture in individuals with sleep disorders and to determine the optimal exercise modalities.
Methods: A comprehensive search of PubMed, Embase, EBSCO, Scopus, PsycINFO, and Web of Science was performed for randomized controlled trials (RCTs) published up to March 2025. Meta-analyses and Bayesian network meta-analyses (NMA) were performed using a random-effects model, with mean differences (MDs) as the effect size measure. Subgroup analyses were performed to explore potential moderating factors. The GRADE framework and Cochrane Risk of Bias 2.0 Tool were used to assess evidence certainty and risk of bias, respectively.
Results: Eighteen studies involving 1214 individuals were included. Exercise interventions improved sleep efficiency (SE) (MD = 2.85; 95 % CI = 0.85, 4.84), reduced wake after sleep onset (WASO) (MD = −10.16; 95 % CI = −15.68, −4.64) and extended slow wave sleep (SWS) (MD = 2.19; 95 % CI = 0.35, 4.03). Short-term interventions (8–12 weeks) with medium frequency (3 times/week) and duration (45–60 min/session) improved SE and WASO, while high frequency (≥4 times/week) were more effective in enhancing SWS, especially in individuals with obstructive sleep apnea (OSA). The NMA indicated that moderate-intensity aerobic exercise (MIAE) was most effective in improving SE and WASO.
Conclusion: Exercise interventions improve sleep architecture in adults with sleep disorders. Optimal exercise parameters in SE and WASO improved most with 8–12 week interventions of 3 weekly 45–60 min sessions. Interventions of four or more times per week were more effective at increasing SWS, especially in individuals with OSA. MIAE is recommended for clinical application to optimize sleep outcomes.

1. Introduction

Sleep is a fundamental physiological function in all living organisms, essential for maintaining homeostasis, facilitating physiological repair, and regulating psychological health [1]. Research indicates that approximately a third of a person's life goes to sleep [2], underscoring its critical role in both physical and mental well-being. However, with the rapid tempo of contemporary life and increasing work-life stress, sleep disorders have become increasingly prevalent, emerging as a significant global health problem that negatively influences people's quality of life and overall health [3]. The World Health Organization (WHO) reports that the global prevalence of sleep disorders has attained 27 % [4]. These disorders not only disrupt sleep architecture, such as reducing deep sleep and increasing wakefulness [5], but also affect physical and mental health through various physiological mechanisms. Studies suggest that sleep disorders can disturb circadian rhythms, which can leading to or exacerbating endocrine imbalances [6], neurological disorders [7], hypertension [8], cardiovascular diseases [9], and cognitive dysfunction [10–12]. Given the profound health implications of sleep disorders, there is an immediate necessity for effective, safe, and accessible therapies to alleviate these conditions.

Currently, clinical interventions to sleep disorders face two primary challenges. On one hand, pharmacological treatments may offer immediate benefits; nevertheless, continued use can result in drug dependency and tolerance, heightening the risk of adverse effects [2]. On the other hand, despite their demonstrated efficacy,

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non-pharmacological treatments like repetitive transcranial magnetic stimulation (rTMS) and cognitive behavioral therapy for insomnia (CBT-I) are challenging to scale because of a lack of qualified personnel and the high expense of equipment [13,14]. In this context, exercise interventions have increasingly been considered an effective supplement or even a viable alternative to pharmacological and psychological treatments, due to their low cost, ease of implementation, and broad applicability [15,16]. Exercise can be characterized as systematic, structured, and repeated physical activity intended for enhancing or preserving health [17]. Research indicates that exercise interventions may markedly improve sleep quality and sleep architecture among individuals with sleep disorders [18–20], including positively affect conditions such as obstructive sleep apnea (OSA) and insomnia [21–23]. Exercise improves body composition and cardiovascular function [24], and it regulates the autonomic nervous system by enhancing parasympathetic activity and suppressing excessive sympathetic activation [25]. Moreover, exercise optimizes sleep architecture through multiple mechanisms—such as improving circadian rhythms [26,27] and modulating thermoregulatory processes [28–31]—which lead to an increase in slow wave sleep (SWS) and ultimately enhance overall sleep quality [32,33].

Although meta-analyses have demonstrated the beneficial effects of exercise on sleep, several significant limitations remain in the existing literature. First, most reviews have primarily focused on subjective sleep quality [34–39], with relatively limited systematic analysis of objective sleep architecture. Subjective assessments are prone to biases related to recall and individual perception, which can undermine the external validity of the findings [40]. By contrast, sleep architecture—comprising critical factors including sleep efficiency (SE), wake after sleep onset (WASO), the percentages of rapid eye movement (REM) and non-rapid eye movement (NREM) sleep—is typically assessed using objective measures like polysomnography (PSG) or actigraphy. These objective parameters are essential for evaluating sleep quality and its restorative functions, providing insights into sleep depth, continuity, and stage distribution [25]. Second, existing reviews often conflate primary sleep disorders with comorbid conditions, such as depression or cancer [18, 41–43], or examine exercise as part of combined interventions (e.g., cognitive training or psychotherapy) [44]. This approach reduces the specificity of the research findings, making it difficult to evaluate the effect of exercise on sleep improvement independently, thereby preventing a clear determination of its therapeutic mechanisms. Finally, existing meta-analyses typically focus on a single type of exercise, while traditional analytical methods are often insufficient to assess the comparative effects of different exercise prescriptions on sleep architecture. Consequently, there remains a critical evidence gap regarding how key exercise parameters—such as exercise type, intervention length, session duration, and weekly training frequency—influence therapeutic outcomes for sleep disorders. These limitations hinder a comprehensive understanding of the optimal exercise parameters needed to achieve effective therapeutic outcomes in adults with sleep disorders.

In light of these limitations, the present study aims to rigorously define inclusion and exclusion criteria to eliminate confounding effects from comorbidities. Network meta-analysis (NMA) offers a unique advantage by enabling simultaneous comparison of multiple interventions and evaluating their relative efficacy in improving specific outcomes. By integrating traditional meta-analysis with NMA, this study will examine the effects of exercise interventions on sleep architecture in adults with sleep disorders and identify the most effective exercise modalities. Specifically, this study aims to answer three key questions: (1) Can exercise interventions improve objective sleep architecture in adults with sleep disorders? (2) Do exercise parameters (exercise type, intervention length, session duration, and weekly training frequency) and the types of sleep disorders moderate the effects of exercise on sleep architecture? (3) Which exercise modalities are most effective in improving sleep architecture? Ultimately, this study aims to provide precise exercise modalities tailored to individuals with different types of sleep disorders.

2. Methods

This investigation was carried out in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) and the PRISMA Network Meta-Analysis Extension Statement for Reporting of Systematic Reviews (PRISMA-NMA) guidelines [34] (Appendix14). A prospective registration of the study protocol in PROSPERO (CRD420251014941) was conducted.

2.1. Literature search

The databases of PubMed, EBSCO, Web of Science, Scopus, PsycINFO, and Embase were thoroughly screened by two independent authors (PW and AZ) from the inception of the study until March 22, 2025. The search strategy utilized a comprehensive set of controlled vocabulary and keywords related to exercise, sleep disorders, and RCTs, using Boolean operators ("AND" and "OR") to optimize term retrieval (Appendix 1).

2.2. Eligibility criteria

This meta-analysis adhered to defined inclusion and exclusion criteria:

(1) Participants: adults (≥18 years) diagnosed with sleep disorders (e.g., insomnia, OSA, restless legs syndrome (RLS) or poor sleep quality), based on clinical diagnostic criteria or self-reported assessments exceeding clinical thresholds (e.g., PSQI >5). Shift workers, pregnant woman, those with specific health conditions (e.g., depression, cancer), and individuals with professional athletic training will be excluded. (2) Intervention: regardless of intensity levels, any form of exercise program was included in the study, with the minimum intervention duration being as brief as a single session (acute intervention). Studies that involved pharmacological interventions (e.g., combined exercise and drug therapies) were excluded. (3) Comparison: control groups involving low intensity stretching, walking, non-exercise interventions (e.g., usual care, placebo), sleep hygiene education, or baseline lifestyle maintenance. (4) Outcomes: assessment of sleep architecture using indicators including SE, sleep onset latency (SOL), WASO, total sleep time (TST), and the distribution of sleep stages, including REM and NREM stages (N1, N2, and SWS). Studies that lacked objective sleep architecture data (e.g., PSG/actigraphy) or had insufficient data to calculate effect sizes were excluded. (5) Study design: RCTs with full-text availability in English were included, while duplicate publications, abstracts, protocols, and conference proceedings were excluded.

2.3. Data extraction

Study selection was performed by two independent authors (PW and AZ) who using EndNote 21 for screening titles and abstracts, subsequently conducting a review of the entire manuscript for prospective inclusion. Any discrepancies between the authors were settled by discussion or, if necessary, by involving another author (YC). Data extraction was carried out using standardized forms to collect the subsequent information (Appendix 3): (1) study characteristics (e.g., first author's last name, country, and publication year); (2) participant information (e.g., age, types of sleep disorders, and sample size for both intervention and control groups); (3) intervention protocol (e.g., exercise type, intervention length, session duration and weekly training frequency); and (4) outcome of interest (along with the measurement instruments used).

2.4. Data coding

Exercise interventions were classified based on exercise parameters:

Exercise Type: (1) Aerobic exercise, such as running or walking, that is usually intended to increase cardiovascular fitness, subdivided into low intensity (45–55 % maximal heart rate, 15–40 % heart rate reserve), moderate intensity (55–70 % maximal heart rate, 40–60 % heart rate reserve), and vigorous intensity (70–90 % maximal heart rate, 60–85 % heart rate reserve). (2) Resistance training which is composed of resistance bands, free weights, or weight equipment, and is designed to enhance muscle strength, endurance, and power. This type of resistance training is classified as moderate intensity (≤50 % one-repetition maximum). (3) Multicomponent exercise that is a combination of different exercise types. (4) Mind-Body exercise that includes activities such as Tai Chi, Qigong, or yoga [35,36].

Intervention length Categorized by the number of weeks of exercise, with acute (single session), short-term (8–12 weeks), medium-term (12–26 weeks), and long-term (≥26 weeks) lengths [35,37,38].

Weekly Training Frequency Categorized based on amount of training sessions per week: low (1–2 times per week), medium (3 times per week), or high (≥4 times per week) frequency [39].

Session Duration Defined by the overall length of each exercise session, including warm-up and cool-down: short (≤45 min/session), medium (45–60 min/session), and long (>60 min/session) [40].

2.5. Assessment of risk of bias

The Cochrane Risk of Bias 2.0 instrument was employed by two reviewers (PW and AZ) to independently evaluate the risk of bias [41]. The assessment encompassed five domains. The criterion for the domain of 'deviations from intended interventions' was altered by the infeasibility of blinding during exercise therapy [42]. The overall risk of bias for each domain was classified as follows: studies was judges to have a low risk of bias if all five domains were assessed as 'low risk'; a high risk of bias was designated if one or more domains were evaluated as 'high risk'; and studies that failed to meet the standards for either low or high risk were categorized as having 'some concerns' regarding bias.

2.6. Assessment of certainty of evidence

The degree of certainty in the evidence was assessed using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) approach, with direct evidence from RCTs having the highest degree of certainty. Risk of bias, indirectness, imprecision, and possible publishing bias all reduced certainty [43,44]. Inconsistency further degraded indirect comparisons. Using the shortest-path method, contribution matrices measured the relative contributions of network, direct, and indirect comparisons [45]. Each comparison's level of evidential certainty was rated as high, moderate, low, or extremely low.

2.7. Data analysis

Meta-analyses were conducted using RevMan 5.2 and STATA 12 software, with subgroup analyses stratified by exercise parameters and types of sleep disorders. Data were obtained from the exercise and control groups, including post-intervention means, standard deviations, and sample sizes. In studies with incomplete data, missing values were estimated using standard error conversions, confidence interval extrapolation, or interquartile range transformations, following the recommendations outlined in the Cochrane Handbook (Appendix 13) [46,47]. Continuous variables were expressed as mean differences (MDs), which served as effect size indicators. Random-effects models were employed for the meta-analyses due to the heterogeneity in study designs, demographics of participant, intervention protocols, and measurements of outcomes [48]. Heterogeneity among effect sizes was assessed using the Q statistic and the I^2 index. Heterogeneity was shown by a significant Q statistic ($p < .05$), where I^2 values of 25 %, 50 %, and 75 % were considered low, moderate, and high heterogeneity, respectively. Sensitivity analysis was used to assess how reliable the results were. Publication bias was assessed based on funnel plots symmetry and Egger's regression test. Both visual inspection of funnel plot asymmetry and a statistically significant Egger's test result (p value < 0.05) were considered indicative of potential publication bias.

A Bayesian network meta-analysis (NMA) was implemented to evaluate the efficacy of exercise modalities for adults with sleep disorders. We employed the GeMTC package in R (version 4.1.0) to conduct the NMA. This package employed four Markov Chain Monte Carlo (MCMC) simulations with a burn-in of 20,000 iterations and 50,000 post-burn-in iterations. Model convergence was assessed using residual diagnostic plots, and convergence was considered acceptable when the potential scale reduction factor (PSRF) was <1.05. Random-effects models were employed, and the Hartung-Knapp-Sidik-Jonkman adjustment was executed using the meta package (v.6.2.1, R) for pairwise comparisons [49]. The "netsplit" tool from the "netmeta" package was employed to assess local inconsistencies. The Surface Under the Cumulative Ranking Curve (SUCRA) was employed to rank exercise modalities according to their anticipated efficacy, with SUCRA values defined as 100 % for the most effective treatment and 0 % for the least effective.

3. Results

3.1. Literature results

A total of 4571 records were initially identified through systematic database searches. After duplicates were removed, 3836 articles remained for preliminary screening based on their titles and abstracts. Following a full-text review, 18 RCTs meeting the predefined inclusion criteria were selected for final analysis. The selection process is depicted in Fig. 1. A detailed list of the included studies is available in Appendix 3, while the reasons for exclusion of the other studies are provided in Appendix 4.

3.2. Study characteristics

A total of 18 RCTs involving 1214 participants were included in this study. The mean age of participants was 53.67 ± 11.66 years, with females accounting for 70.02 % of the sample. The included studies targeted four major types of sleep disorders: insomnia (10 studies), OSA (5 studies), poor sleep quality (3 studies), and RLS (2 studies). These studies were conducted across 12 countries and published between 2008 and 2025, with China contributing the highest number (6 studies), followed by the United States (3 studies). In terms of exercise parameters, aerobic exercise was the most used intervention (10 studies), followed by mind-body exercises (5 studies), multicomponent exercise programs (4 studies), and resistance training (2 studies). The most frequent intervention length was 8–12 weeks (13 studies), followed by 13–26 weeks (3 studies) and acute interventions (4 studies), while 1 study of the studies involved interventions lasting ≥26 weeks. Regarding exercise frequency, the most common schedule was 3 times per week (8 studies), followed by 1–2 times per week (7 studies), and ≥4 times per week (5 studies). In terms of session duration, the most frequent duration was 45–60 min (10 studies), followed by less than 45 min (7 studies) and 60 min or more (3 studies). Further details are available in Appendix 3.

3.3. Sensitivity analysis and risk of bias

Sensitivity analyses revealed that the exclusion of each study had no significant impact on the overall results, hence affirming the robustness of the findings (Appendix 7). Egger's test revealed no significant evidence of publication bias ($p > .05$, see Appendix 6). The risk of bias evaluation revealed that 9 studies (50 %) had been categorized as low

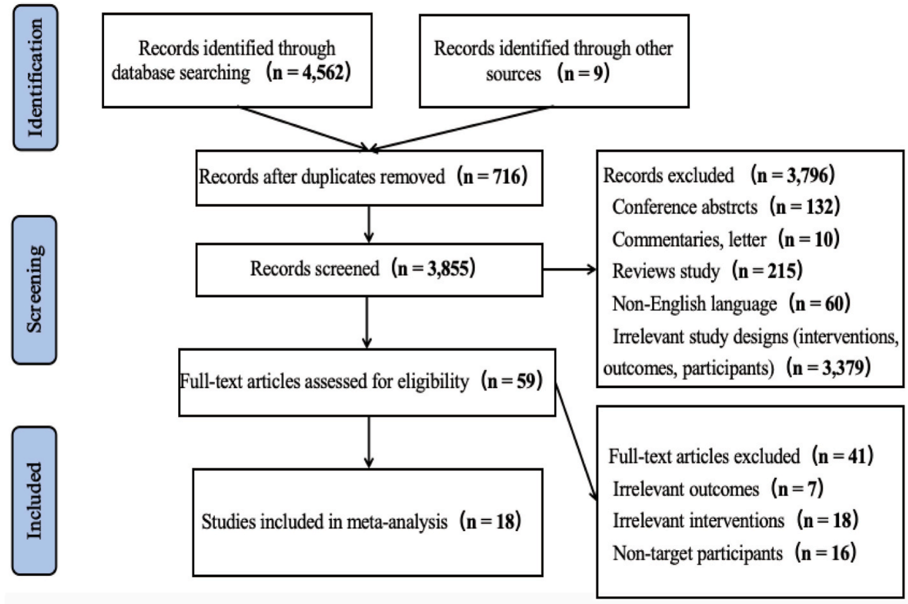

Fig. 1. Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flow diagram of study selection process.

risk, 6 studies (33.33 %) raised some concerns, and 3 studies (16.66 %) were classified as high risk of bias.

3.4. Effects of exercise intervention on sleep architecture

3.4.1. Effect of exercise intervention on SE

As shown in Fig. 2, exercise intervention significantly improved SE in individuals with sleep disorders (MD = 2.85, 95 % CI [0.85, 4.84], p = .005, I^2 = 74 %). Among different types of exercise interventions, aerobic exercise (MD = 5.94, 95 % CI [2.86, 9.02], $p < .001$, I^2 = 62 %) and multicomponent exercise (MD = 4.13, 95 % CI [0.25, 8.01], p = .04, I^2 = 75 %) demonstrated significant improvements in SE. Regarding intervention length, short-term exercise lasting 8–12 weeks yielded the greatest improvement (MD = 2.60, 95 % CI [0.46, 4.75], p = .02, I^2 = 73 %). Exercise performed 3 times per week (MD = 4.73, 95 % CI [2.04, 7.42], $p < .001$, I^2 = 77 %) resulted in the most significant improvement in SE, followed by 1–2 times per week (MD = 3.86, 95 % CI [0.89, 6.83], p = .01, I^2 = 20 %). In terms of exercise duration, 45–60 min per session proved most effective (MD = 3.59, 95 % CI [1.23, 5.94], p = .003, I^2 = 66 %). More information for subgroup analyses can be found in Appendix 8.

3.4.2. Effect of exercise intervention on WASO

As shown in Fig. 3, exercise intervention significantly reduced WASO in individuals with sleep disorders (MD = −10.16, 95 % CI [−15.68, −4.64], $p < .001$, I^2 = 62 %). Among different types of exercise interventions, multicomponent exercise (MD = −16.89, 95 % CI [−32.56, −1.21], p = .03, I^2 = 30 %) and aerobic exercise (MD = −12.25, 95 % CI

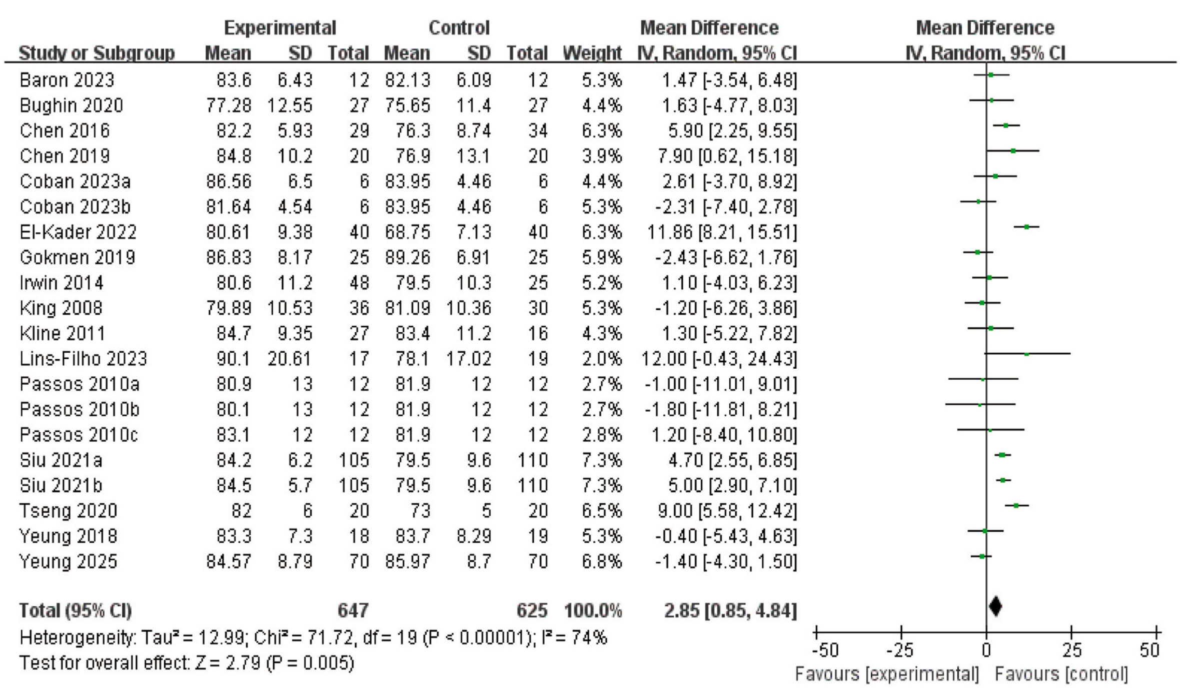

Fig. 2. Forest plot for meta-analysis regarding the effect of exercise intervention on SE.

Study or Subgroup	Experimental Mean	Experimental SD	Experimental Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Random, 95% CI
Baron 2023	58.59	42.87	12	70.1	39.95	12	2.3%	-11.51 [-44.66, 21.64]
Chen 2016	76.5	34.95	29	91.4	40.32	34	5.2%	-14.90 [-33.49, 3.69]
Chen 2019	58.8	52.4	20	91.1	52.6	20	2.3%	-32.30 [-64.84, 0.24]
Coban 2023a	37.39	7.47	29	52.39	17.11	6	7.0%	-15.00 [-28.96, -1.04]
Coban 2023b	63.94	63.94	6	52.39	17.11	6	1.0%	11.55 [-41.41, 64.51]
El-Kader 2022	64.25	6.42	40	83.76	9.81	40	12.0%	-19.51 [-23.14, -15.88]
Irwin 2014	72.5	52.6	48	75.1	43	25	4.1%	-2.60 [-25.08, 19.88]
Kline 2011	63.7	39.99	27	66.1	50.8	16	2.8%	-2.40 [-31.51, 26.71]
Lins-Filho 2023	32.7	3.35	17	41.5	13.7	19	10.9%	-8.80 [-15.16, -2.44]
Passos 2010a	65.3	48	12	60.7	41	12	2.0%	4.60 [-31.12, 40.32]
Passos 2010b	63.3	36	12	60.7	41	12	2.6%	2.60 [-28.27, 33.47]
Passos 2010c	48	34	12	60.7	41	12	2.7%	-12.70 [-42.84, 17.44]
Siu 2021a	70.8	37	105	89.7	48.4	110	8.2%	-18.90 [-30.38, -7.42]
Siu 2021b	68.4	30.5	105	89.7	48.4	110	8.6%	-21.30 [-32.06, -10.54]
Tan 2016	39.1	15.96	17	38.7	11.71	12	8.9%	0.40 [-9.67, 10.47]
Tseng 2020	72.6	27.6	20	94.5	29	20	5.6%	-21.90 [-39.45, -4.35]
Yeung 2018	39.7	26.82	27	34.4	29.63	19	5.9%	5.30 [-11.43, 22.03]
Yeung 2025	41.93	36.07	70	35.99	35.71	70	8.0%	5.94 [-5.95, 17.83]
Total (95% CI)			**608**			**555**	**100.0%**	**-10.16 [-15.68, -4.64]**

Heterogeneity: Tau² = 62.39; Chi² = 44.33, df = 17 (P = 0.0003); I² = 62%
Test for overall effect: Z = 3.61 (P = 0.0003)

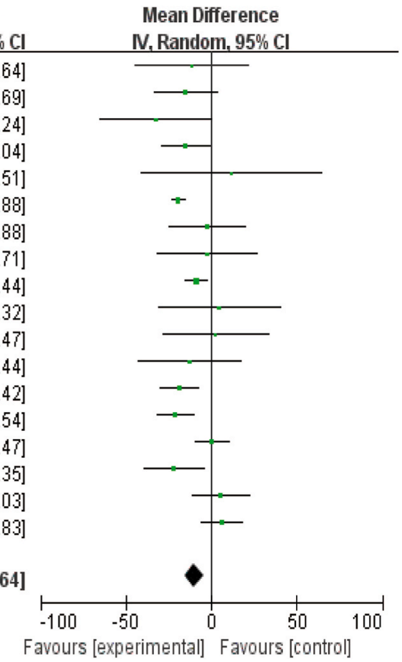

Fig. 3. Forest plot for meta-analysis regarding the effect of exercise intervention on WASO.

[−18.90, −5.60], $p < .001$, $I^2 = 60$ %) demonstrated significant improvements in WASO. The most substantial effect was observed with interventions lasting 8–12 weeks (MD = −10.46, 95 % CI [−16.91, −4.02], $p = .001$, $I^2 = 50$ %). Exercise performed 3 times per week had the most significant effect on WASO (MD = −16.49, 95 % CI [−21.06, −11.93], $p < .001$, $I^2 = 33$ %). In terms of exercise duration, 45–60 min per session (MD = −13.62, 95 % CI [−21.57, −5.67], $p < .001$, $I^2 = 44$ %) was the most effective. Exercise interventions showed the greatest improvement in WASO for individuals with poor sleep quality (MD = −19.18, 95 % CI [−35.32, −3.04], $p = .02$, $I^2 = 0$ %) and OSA (MD = −10.39, 95 % CI [−17.40, −3.39], $p = .004$, $I^2 = 8$ %). More information for subgroup analyses can be found in Appendix 8.

3.4.3. Effect of exercise intervention on SWS

As shown in Fig. 4, exercise intervention significantly increased SWS in individuals with sleep disorders (MD = 2.19, 95 % CI [0.35, 4.03], $p = .02$, $I^2 = 14$ %). Subgroup analysis did not reveal significant differences between exercise types and session duration. Regarding intervention length, 8–12 weeks was the most effective (MD = 3.34, 95 % CI [1.08, 5.60], $p = .004$, $I^2 = 0$ %). Exercise performed ≥4 times per week demonstrated the most significant effect on SWS (MD = 3.83, 95 % CI [1.03, 6.63], $p = .007$, $I^2 = 0$ %). Participants with OSA exhibited the greatest improvement in SWS (MD = 3.24, 95 % CI [1.09, 5.40], $p = .003$, $I^2 = 0$ %). More information for subgroup analyses can be found in Appendix 8.

3.5. Network plot

NMA includes 18 studies, involving seven common exercise interventions: low-intensity exercise, moderate-intensity aerobic exercise (MIAE), vigorous aerobic exercise, resistance training (RT), mind-body exercise, multi-component exercise, Zero-time exercise, and control groups. The network diagram illustrates strong associations between the different interventions, particularly in improving SE and reducing WASO (Fig. 5). The largest sample size is found in MIAE, followed by low-intensity exercise and the control group. The network diagram for SE (Fig. 5A) shows that most studies focused on comparing MIAE and vigorous aerobic exercise with the control group (indicated by thicker lines), followed by comparisons between low-intensity exercise and multicomponent exercise. The network diagram for WASO (Fig. 5B) shows that most studies compared MIAE with the control group, followed by comparisons between vigorous aerobic exercise and the control group, and low-intensity exercise with multicomponent exercise. The leverage plot (Appendix 11), which visualizes the distribution of

Study or Subgroup	Experimental Mean	Experimental SD	Experimental Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Fixed, 95% CI
Bughin 2020	16.6	7.77	27	16.15	7.96	27	19.2%	0.45 [-3.75, 4.65]
Gokmen 2019	17.3	11.6	25	12.96	7.81	25	11.3%	4.34 [-1.14, 9.82]
Gupta 2023	18.5	12.7	19	16.2	9.6	18	6.5%	2.30 [-4.93, 9.53]
Kline 2011	13.2	5.72	27	9.2	6	16	25.5%	4.00 [0.35, 7.65]
Lins-Filho 2023	18.6	9.1	17	12.6	8.4	19	10.3%	6.00 [0.26, 11.74]
Passos 2010a	22	12	12	19.2	7	12	5.5%	2.80 [-5.06, 10.66]
Passos 2010b	15.5	8	12	19.2	7	12	9.4%	-3.70 [-9.71, 2.31]
Passos 2010c	19.4	6	12	19.2	7	12	12.4%	0.20 [-5.02, 5.42]
Total (95% CI)			**151**			**141**	**100.0%**	**2.19 [0.35, 4.03]**

Heterogeneity: Chi² = 8.16, df = 7 (P = 0.32); I² = 14%
Test for overall effect: Z = 2.33 (P = 0.02)

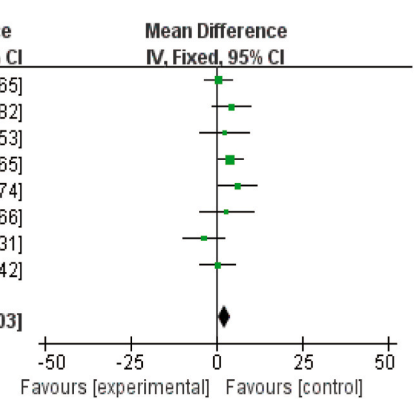

Fig. 4. Forest plot for meta-analysis regarding the effect of exercise intervention on SWS.

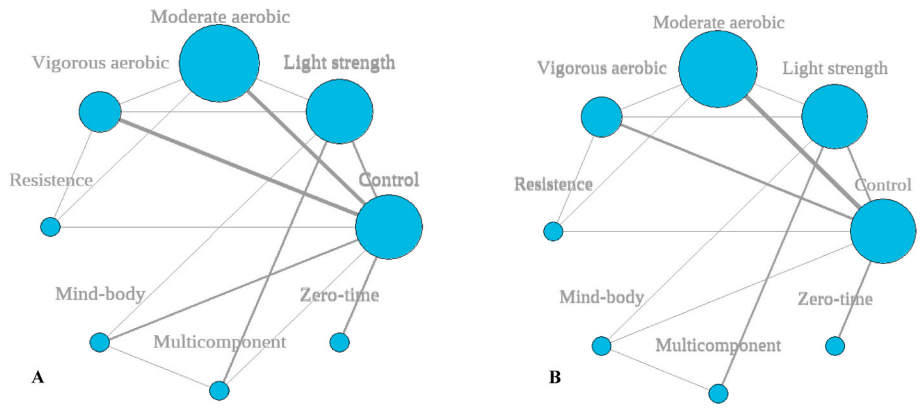

Fig. 5. Network Plot of included comparisons for SE (A) and WASO (B).

study results, reveals a uniform distribution, suggesting good convergence of the Bayesian model. Inconsistency testing using the node-splitting method shows a *p*-value >0.05, suggesting overall good consistency (Appendix 12).

3.6. Network meta-analysis

NMA compares the effects of different exercise interventions on SE and WASO in individuals with sleep disorders (Fig. 6). Moderate-intensity aerobic exercise (MIAE) showed significant improvement in SE compared to the control group (MD = 6.36, 95 % CI [1.26, 10.29]) and was also more effective than low-intensity stretching exercise (MD = 7.22, 95 % CI [0.38, 13.37]). In terms of WASO, MIAE showed a more significant effect compared to the control group (MD = −13.42, 95 % CI [−24.26, −1.67]). No significant differences were found between the other exercise interventions. The SUCRA values (Appendix 10), which measure the relative effectiveness of interventions, indicated that MIAE ranked highest for SE (87.18 %), followed by RT (72.68 %) and vigorous aerobic exercise (70.44 %); for WASO, RT ranked highest (82.56 %), followed by MIAE (68.79 %) and multi-component exercise (68.19 %).

4. Discussion

This review included 18 RCTs and employed both traditional meta-analysis and NMA approaches for evaluating the impact of exercise interventions on sleep architecture in adults with sleep disorders and to identify the most effective exercise modalities. We found that exercise interventions significantly improved SE, reduced WASO, and increased the proportion of SWS. The efficacy of exercise interventions is significantly influenced by exercise types, intervention length, session duration, weekly training frequency and types of sleep disorders. Moreover, NMA revealed modality-specific effects, with MIAE showing superior efficacy for SE improvement and WASO reduction compared to other exercise training types.

Our results align with earlier meta-analyses [50–52], confirming the beneficial effect of exercise on sleep architecture in adults with sleep disorders. WASO and SE, key indicators of perceived sleep quality, are crucial in assessing sleep outcomes [53]. Furthermore, understanding the distribution of time throughout different sleep stages is essential, as SWS is essential for restorative functions, learning, and memory [54]. The review indicated that, in contrast to the control group, exercise interventions markedly enhanced SE, diminished WASO, and elevated the proportion of SWS, underscoring the advantageous effect of exercise on sleep architecture. This discovery has considerable clinical

A

	Light-strength	Moderate-aerobic	Vigorous-aerobic	Resistence	Mind-body	Multicomponent	Zero-time
Control	-0.87 (-5.83, 4.33)	**6.36 (1.26, 10.96)**	3.95 (-0.79, 8.59)	5.37 (-5.28, 16.01)	0.9 (-4.57, 6.25)	1.91 (-4.24, 8.03)	-0.96 (-7.43, 5.57)
Light-strength		**7.22 (0.38, 13.37)**	4.81 (-1.83, 11.16)	6.25 (-5.42, 17.73)	1.78 (-4.48, 7.66)	2.78 (-3.13, 8.42)	-0.08 (-8.44, 8.08)
Moderate-aerobic			-2.41 (-8.65, 4.18)	-0.96 (-11.74, 10.1)	-5.47 (-12.31, 1.86)	-4.46 (-11.71, 3.34)	-7.33 (-15.13, 1.12)
Vigorous-aerobic				1.43 (-9.45, 12.38)	-3.04 (-10.07, 3.98)	-2.03 (-9.48, 5.5)	-4.91 (-12.85, 3.19)
Resistence					-4.47 (-16.4, 7.3)	-3.45 (-15.67, 8.69)	-6.34 (-18.69, 6.14)
Mind-body						1.01 (-5.5, 7.6)	-1.85 (-10.25, 6.71)
Multicomponent							-2.88 (-11.78, 6.12)

B

	Light-strength	Moderate-aerobic	Vigorous-aerobic	Resistence	Mind-body	Multicomponent	Zero-time
Control	0.79 (-15.26, 15.88)	**-13.42 (-24.26, -1.67)**	-11.24 (-27.13, 3.86)	-22.83 (-52.79, 6.86)	-11.26 (-31.55, 10.01)	-13.9 (-36.14, 9.92)	5.72 (-12.55, 24.05)
Light-strength		-14.19 (-30.62, 4.11)	-12.01 (-28.59, 4.66)	-23.61 (-55.36, 8.8)	-12.09 (-30.2, 8.4)	-14.73 (-32.43, 5.62)	4.91 (-18.52, 29.38)
Moderate-aerobic			2.13 (-16.5, 19.1)	-9.5 (-40.22, 20.57)	2.14 (-20.4, 24.83)	-0.51 (-24.6, 24.26)	19.12 (-2.75, 40.07)
Vigorous-aerobic				-11.56 (-41.85, 19.1)	-0.06 (-22.16, 24.35)	-2.7 (-25.86, 22.99)	16.94 (-6.54, 41.36)
Resistence					11.6 (-23.37, 47.46)	8.96 (-26.97, 46)	28.58 (-6.3, 63.5)
Mind-body						-2.66 (-23.45, 18.46)	16.97 (-11.25, 44.09)
Multicomponent							19.6 (-10.62, 48.32)

Fig. 6. League table for SE (A) and WASO (B).
Note: The values in each table denote the mean difference (MD) with a 95 % confidence interval, where each MD value results from a comparison of two interventions.

ramifications, as SE is a consistent determinant of sleep and has been associated with health hazards, including elevated mortality [55]. Consequently, the findings of this study, indicating that exercise enhances SE and diminishes WASO, highlight the positive impact of exercise on sleep continuity and decreased fragmentation. However, a meta-analysis by Gong et al. did not find statistically significant differences, despite evidence suggesting that exercise improves SE and SWS [30]. The discrepancy may stem from the small number of studies included in the review (SE = 6, SWS = 2), limiting the ability to conduct a comprehensive assessment of exercise interventions. This study demonstrates that exercise interventions significantly reduce sleep fragmentation, enhance sleep continuity, and increase the proportion of SWS.

The second purpose of this study was to examine if exercise parameters and the types of sleep disorders moderate their effects on sleep architecture. Our findings indicate that multi-component exercise significantly improves SE, with large and moderate effect sizes observed for multi-component and aerobic exercises, respectively, in reducing WASO. These results emphasize the importance of aerobic exercise in improving sleep architecture. In adults with sleep disorders, short-term exercise interventions (8–12 weeks), with medium frequency (3 sessions per week), significantly improve SE and reduce WASO. These findings are consistent with those of Xie et al. [55], who reported that short-term, medium-frequency exercise prescriptions are more effective for sleep continuity than long-term, high-frequency modalities. By contrast, extending SWS requires high frequency (≥4 times per week), particularly in patients with OSA. Additionally, exercise effects vary across different sleep disorders. For instance, in patients with OSA, exercise significantly improved WASO and SWS. Chen et al. [50] also reported that exercise improved sleep stages in OSA patients. Similarly, Yu et al. [56] found that exercise reduced the apnea-hypopnea index (AHI), stabilizing sleep cycles and optimizing overall sleep architecture. The improvements in WASO and SWS were more pronounced in OSA patients than in those with other disorders, likely because exercise improves respiratory function and reduces awakenings [57]. In conclusion, exercise interventions can improve sleep disorders, with outcomes contingent on both exercise parameters and the types of sleep disorders. Customized exercise prescriptions must be formulated according to each patient's situation to optimize sleep benefits.

The NMA results are partially aligned with prior subgroup studies, emphasizing notable variations in the effects of different exercise modalities on sleep architecture. Specifically, MIAE demonstrated enhanced efficacy in augmenting SE and diminishing WASO relative to the control group and low-intensity stretching activities. Subsequent research utilizing SUCRA rankings revealed that MIAE was the most efficacious in enhancing SE (87.18 %), succeeded by resistance exercise (72.68 %) and vigorous aerobic activity (70.44 %). Regarding WASO reduction, resistance exercise ranked highest in SUCRA (82.6 %), followed by MIAE (68.8 %) and multi-component exercise (68.2 %). However, since only three RCTs support resistance exercise, further verification of the robustness of these results is required. Additionally, exercise modalities that ranked higher in improving SE and reducing WASO were primarily of moderate to high intensity (e.g., 55–90 % of maximal heart rate or 50 % of one-repetition maximum). This suggests that exercise intensity plays a crucial role in enhancing sleep continuity. In conclusion, different exercise modalities significantly affect sleep architecture, with exercise intensity being a key factor in improving sleep architecture. Notably, MIAE showed the most significant effects in improving SE and reducing WASO. Therefore, when designing personalized exercise prescriptions, exercise types, intervention length, session duration and weekly training frequency should be considered holistically to ensure effectiveness.

The neurobiological mechanisms underlying the effects of exercise on improving sleep architecture in adults with sleep disorders are complex. Existing studies show that different exercise intensities lead to varying physiological responses. When exercise intensity is too low, its impact on the cardiovascular and sympathetic nervous systems (SNS) is minimal, thereby limiting its ability to produce significant physiological benefits. Conversely, excessively high exercise intensity may disrupt the balance between pro-inflammatory and anti-inflammatory cytokines, thereby increasing the body's inflammatory burden and elevating the risk of exercise-induced complications [25]. This finding is corroborated by the present study, which identified MIAE as the most effective modality for improving sleep architecture, particularly regarding enhancing SE and extending SWS. Furthermore, exercise modalities that were ranked higher in terms of improving sleep continuity were primarily of moderate to high intensity. These exercise types and intensities may affect sleep architecture through various mechanisms. MIAE improves sleep by increasing daytime energy expenditure, regulating autonomic nervous function, and balancing cortisol levels, all of which contribute to reducing nocturnal awakenings and prolonging deep sleep stages [58]. Additionally, MIAE may enhance brain-derived neurotrophic factor (BDNF) expression, promoting neuroplasticity and alleviating hyperarousal states [16,59,60]. Resistance exercise shows potential for improving sleep by stimulating the skeletal muscle system, enhancing muscle metabolism [61] and stimulating growth hormone secretion [28], which helps reduce WASO and improve overall sleep quality. Further research suggests that short-term, high-frequency exercise interventions (8–12 weeks, 3 times per week, 45–60 min per session or longer) can significantly enhance sleep architecture through acute physiological responses, such as improved circadian rhythm regulation and metabolic shifts. Short-term, high-frequency exercise helps regulate circadian rhythms [31], modulate inflammatory metabolism, and increase the proportion of deep sleep [30,62]. These effects are particularly significant in patients with OSA [57,63]. In conclusion, these physiological responses form a complex neurobiological network through which exercise improves sleep disorders, underscoring the systemic impact of exercise on sleep regulation.

The primary advantage of the review is the combination of conventional meta-analysis with NMA, facilitating a thorough assessment of the impacts of diverse exercise modalities on objective sleep architecture. This methodology enhances the evaluation of interventions by integrating both direct and indirect evidence, resulting in more robust and precise conclusions regarding the impact of exercise modalities on objective sleep architecture across diverse sleep disorders. One more strength is the rigorous inclusion of RCTs and the exclusion of observational or longitudinal studies. This rigorous selection process enhances the reliability of causal inferences concerning the effects of interventions. Nevertheless, this meta-analysis evaluation possesses certain limitations. First, the effect size for each subgroup analysis was based on small sample sizes and unequal numbers of studies (e.g., 3 studies on poor sleep quality and 2 studies on RLS), which may limit the robustness and generalizability of the findings. Future research should focus on understudied sleep disorders (e.g., RLS) and exercise modalities (e.g., resistance training) through long-term interventions to enhance the understanding of their effects and improve the reliability and generalizability of the findings. Second, relying solely on immediate post-intervention outcomes significantly constrains our understanding of the long-term efficacy of exercise interventions. Longitudinal studies incorporating systematic follow-up assessments are essential to determine the sustainability of exercise-induced improvements and establish evidence for durable intervention effects. Third, the high heterogeneity observed in certain outcome measures introduces potential bias that warrants careful consideration. Future studies should employ more standardized assessment protocols and explore potential sources of variability to strengthen the validity of exercise intervention outcomes. Finally, the exclusive inclusion of English-language studies may limit the generalizability of our findings, as it prevents a truly global assessment of exercise interventions for sleep disorders—a significant concern given the worldwide prevalence of sleep-related health challenges. Future research should incorporate multilingual literature searches to better capture cross-cultural variations in treatment efficacy.

5. Conclusion

This review suggests that exercise interventions can improve sleep architecture in adults with sleep disorders, particularly by enhancing SE, reducing WASO, and increasing SWS. The most effective interventions for improving SE and WASO are medium-frequency (3 sessions per week) programs lasting 45–60 min over 8–12 weeks. By contrast, high-frequency interventions (≥4 sessions per week) are more effective in increasing SWS, especially in patients with OSA. Therefore, MIAE has to be prioritized in clinical practice, particularly for enhancing SE and WASO in adults with sleep disorders.

CRediT authorship contribution statement

Peisi Wang: Writing – review & editing, Writing – original draft, Software, Investigation, Conceptualization. **Yanxia Chen:** Writing – review & editing, Investigation, Conceptualization. **Anqi Zhang:** Visualization, Investigation. **Chun Xie:** Software, Conceptualization. **Kun Wang:** Writing – review & editing, Supervision, Project administration.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used ChatGPT 4.0 to improve language and readability. After using this tool, the authors reviewed and edited the content as needed and took full responsibility for the content of the publication.

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We declare that we do not have any commercial or associative interest that represents a conflict of interest in connection with the work submitted.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2025.106680.

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