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Effects of morning versus evening exercise on sleep: a systematic review of comparative studies with a meta-analysis of randomized controlled trials

Maher Souabni [a,b,c], Giovanna C. Del Sordo [d], Maïtena Saurel [a], Paul Peyrel [e], Magali Giacomoni [a], Mehdi J. Souabni [c], Damien Davenne [f], Brendan M. Gabriel [g], Marie-Pierre St-Onge [h,i], Oussama Saidi [a,*]

[a] Laboratory Youth - Physical Activity, Sports and Health (J-AP2S), Toulon University, Toulon, F-83041, France
[b] LBEPS, Univ Evry, IRBA, Université de Paris-Saclay, Evry, France
[c] Interdisciplinary Laboratory in Neurosciences, Physiology and Psychology Physical Activity, Health and Learning (LINP2), UFR STAPS (Faculty of Sport Sciences), UPL, Paris Nanterre University, Nanterre, France
[d] Psychology Department, New Mexico State University, 1780 E University Blvd, Las Cruces, NM, 88003, USA
[e] Sleep and Cardiometabolic Health Laboratory, Pennington Biomedical Research Center, Baton Rouge, LA, USA
[f] Inserm U1075, COMETE, PFRS, Caen Normandie University, 2, rue des Rochambelles, Caen, 14032, France
[g] Aberdeen Cardiovascular and Diabetes Centre, The Rowett Institute of Nutrition and Health, University of Aberdeen, Aberdeen, UK
[h] Sleep Center of Excellence, Department of Medicine, Columbia University Irving Medical Center, New York, NY, USA
[i] Division of General Medicine, Department of Medicine, Columbia University Irving Medical Center, New York, NY, USA

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ABSTRACT

This systematic review synthesized evidence from comparative studies examining the effects of morning versus evening exercise on sleep, with a meta-analysis restricted to randomized controlled trials. Of the 1111 records identified, 25 studies met the eligibility criteria, including five observational studies, two non-randomized intervention studies, and 18 randomized controlled trials (RCTs), 16 of which were included in the meta-analysis. At the group level, the available evidence does not indicate a clear overall advantage of morning over evening/nighttime exercise for most sleep outcomes. Evening/nighttime exercise was associated with higher wake after sleep onset (WASO) (SMD = −0.51, 95% CI [−0.89, −0.12]), indicating a modest effect on sleep continuity. However, this finding should be interpreted with caution, as potential small-study effects were identified. Subgroup analyses suggested that timing-related differences were more apparent at higher exercise intensities. Overall, these findings support flexible exercise timing that prioritizes adherence and individual constraints, while recognizing that effects vary with intensity, proximity to sleep, and recovery, and that evening moderate-to-vigorous exercise may impair sleep continuity. Future research should identify the exercise-related and individual-level factors that moderate sleep responses to exercise timing to inform more personalized chrono-exercise strategies.
Registration: PROSPERO (CRD42021277708).

1. Introduction

Chronomedicine has introduced a key concept: the effectiveness of an intervention depends not only on its nature but also on the timing of its administration [1–3]. Exercise is no exception [4,5]. Research on exercise and health has long examined parameters such as intensity, volume, and type (e.g., aerobic versus resistance training), which shape physiological adaptations and influence cardiovascular, metabolic, and psychological outcomes [6]. Recently, increasing attention has shifted toward the temporal dimension of exercise, with growing evidence that the time of day at which it is performed modulates its physiological effects [7–9]. Optimizing exercise timing has therefore been proposed as a strategy to complement therapeutic approaches in diverse clinical contexts, including metabolic and cardiovascular disease management [8]. This perspective, viewing health through the lens of biological timing, is rooted in circadian biology, the body's internal clock that regulates sleep–wake cycles, hormone secretion, metabolism, and immune function. Just as circadian rhythms modulate the effects of pharmacological treatments such as chemotherapy or antihypertensive drugs

* Corresponding author. Laboratory Youth-Physical Activity, Sport and Health (J-AP2S), University of Toulon, CS 60584, 83041, Toulon, Cedex, France.
E-mail address: oussama.saidi@univ-tln.fr (O. Saidi).

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Abbreviations

DLMO	Dim light melatonin onset
ESS	Epworth Sleepiness Scale
HR_{max}	Maximum heart rate
HRR	Heart rate reserve
MCTQ	Munich Chronotype Questionnaire
MEQ	Morningness-Eveningness Questionnaire
METs	Metabolic equivalents
PICO	Population, Intervention, Comparison, Outcomes
PRISMA 2020	Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020
PROSPERO	International Prospective Register of Systematic Reviews
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
RCT	Randomized controlled trial
REM	Rapid eye movement
RoB-2	Revised Cochrane Risk of Bias tool for randomized trials
SE	Sleep efficiency
SMD	Standardized mean difference
SOL:	Sleep onset latency
TIB	Time in bed
TST	Total sleep time
VO_{2peak}	Peak oxygen uptake
VT1	First ventilatory threshold
WASO	Wake after sleep onset
WHIIRS	Women's Health Initiative Insomnia Rating Scale
ΔEX_T	Temporal separation between evening and morning exercise sessions

[10–12], they have also been demonstrated to influence exercise responses [13].

Sleep disturbances are pervasive in modern societies [14,15], contributing to heightened risks of obesity, diabetes, cardiovascular disease, and cognitive decline [16–18]. These adverse health consequences have intensified interest in exercise as a non-pharmacological strategy to improve sleep. However, evidence from experimental and epidemiological studies remains inconsistent [19–21]. Some investigations report beneficial effects [22], whereas others find negligible or detrimental impact on sleep parameters [23,24]. These discrepancies likely reflect limited understanding of underlying mechanisms, interindividual variability, and differences in exercise characteristics (e.g., frequency, intensity, duration, timing). However, the specific role of exercise timing in shaping sleep outcomes remains unclear, particularly when morning and evening exercise are directly compared.

Traditionally, morning exercise has been regarded as more conducive to restorative sleep, whereas evening exercise has raised concerns about impaired sleep due to heightened physiological arousal at bedtime [23,25,26]. Yet, accumulating evidence over the past two decades has brought this notion into question, suggesting that evening exercise may represent a viable option for sleep promotion rather than an inherent risk [27,28]. In addition, the late afternoon and early evening are often associated with more favorable neuromuscular and metabolic conditions for exercise, leading some individuals to prefer this time window to optimize training performance [29]. Beyond these considerations, the relevance of exercise timing must also be understood in the context of contemporary lifestyles. Long working hours, commuting demands, and family obligations frequently restrict opportunities for daytime physical activity, effectively limiting exercise to morning or evening windows [30,31]. Under such constraints, time availability, rather than individual preference, often determines when exercise can be performed [30]. As a result, morning and evening represent the most realistic and accessible periods for exercise, prompting increasing interest in whether these time windows differentially influence sleep outcomes, an issue that remains insufficiently clarified despite its practical relevance [31].

This systematic review and meta-analysis aimed to: (i) synthesize all current original evidence from comparative studies on the effects of morning versus evening exercise on sleep parameters, (ii) identify, based

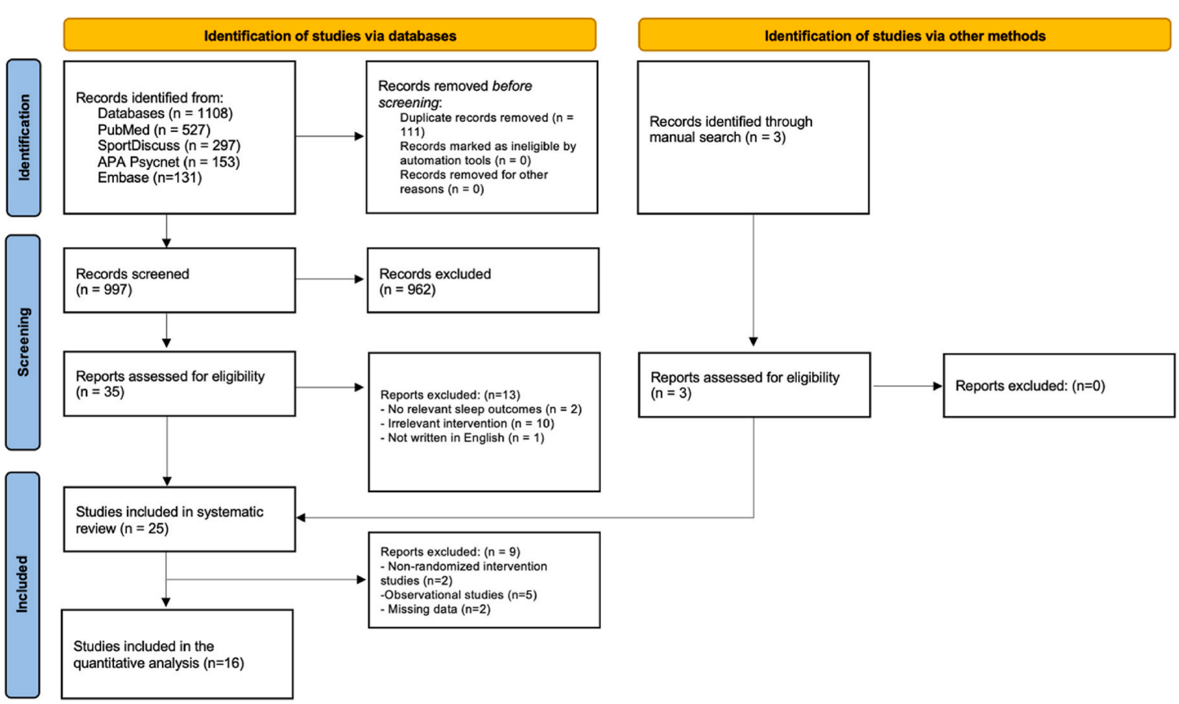

Fig. 1. PRISMA 2020 flow diagram of study selection.

on data from randomized controlled trials (RCTs), moderators and methodological limitations that may explain variability in timing effects, with the objective of identifying future research directions required to move toward personalized exercise-timing recommendations for better sleep.

2. Methods

2.1. Design and registration

This systematic review was conducted in accordance with PRISMA 2020 guidelines [32] (see Supplementary file) and prospectively registered in PROSPERO (CRD42021277708).

2.2. Data sources and search strategy

The academic databases SPORTDiscus, Embase, PubMed, and APA PsycNet were searched from 2000 to April 2024 to find peer-reviewed English-language primary research on the impact of exercise timing on sleep. Studies were identified by combining 3 sets of keywords linked to: (1) exercise, (2) timing, and (3) sleep (See Table S1 for Database search strategies). In addition, related citations were retrieved via Google Scholar, and the authors' personal files were examined to identify studies not captured by the initial search terms. To ensure the review remained current, the search was updated in January 2026, allowing identification, screening, and inclusion of studies published after April 2024.

2.3. Eligibility criteria/selection criteria

Eligibility was defined using PICO criteria: adults (>18 years), interventions comparing at least two exercise timing conditions (morning vs. evening/nighttime), and at least one post-intervention sleep outcome (objective or self-reported). No restrictions were applied regarding study design, methodological quality, or geographical location for the systematic review; however, only randomized controlled trials (RCTs) were included in the meta-analysis. Exclusion criteria were non-primary research (e.g., reviews, case reports, editorials, conference papers), animal or cell studies, abstracts, and non-English articles.

2.4. Study selection

Records were imported into EndNote (Clarivate, London, UK) and then into Cadima for systematic review management [33]. Study selection is illustrated in Fig. 1. After duplicate removal, two reviewers (P.*P. and* G.D.S.) performed a consistency check on 5% of studies ($\kappa = 0.66$), followed by independent screening of titles and abstracts. Discrepancies were resolved by discussion or a third reviewer (O.S.). Full texts were then assessed independently by two reviewers (P.*P. and* O.S.) against the eligibility criteria. Additional studies identified after April 2024 were screened using the same procedure.

2.5. Data extraction

Data were independently extracted by two reviewers (P.*P. and* M.S.) using a standardized form, with discrepancies resolved by discussion or a third reviewer (O.S.). Extracted data included the country in which each study was conducted, study characteristics, participant characteristics, study design, exercise protocols (type, intensity, and timing), sleep assessment methods, and main outcomes. For the meta-analysis, when the required data were unavailable, the corresponding authors were contacted. If the requested data could not be obtained and the available data were insufficient to perform the meta-analysis, the study was not included in the final analyses.

2.6. Quality assessment

Two reviewers (M.SA. and M.J.S.) performed quality assessments independently, with disagreements resolved by a third reviewer (O.S.). Risk of bias in included RCTs was assessed using the Cochrane Risk of Bias 2 tool (RoB-2) [34]. The RoB-2 tool evaluates five domains: randomization process, missing outcome data, deviations from intended interventions, outcome measurement, and selection of reported results, with judgments categorized as low risk, some concerns, or high risk. Given the inclusion of both parallel and crossover designs, the RoB-2 tool was applied using the design-specific variants, with an additional domain assessing potential period and carryover effects in crossover trials. The Risk of Bias In Non-Randomized Studies of Interventions (ROBINS-I) tool was used for non-randomized interventional studies [35] and the Risk of Bias In Non-Randomized Studies of Exposure (ROBINS-E) was used for observational studies [36]. In accordance with the ROBINS-I and ROBINS-E instructions for use, assessment criteria and ideal versions were predefined for each of the seven bias domains and the individual assessment of each study and item was then carried out. Quality of evidence was assessed using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) guidelines [37]. These guidelines apply a set of predetermined domains that either increase or decrease the level of confidence in the evidence. Domains that reduce confidence in the evidence are risk of bias, inconsistency of results, indirectness of evidence, imprecision, and publication bias. On the other hand, a large magnitude of effect, confounding that increases effect magnitude, and a dose-response gradient can increase confidence. Two researchers (M.S. and M.J.S.) discussed the domains for each outcome until consensus was reached. Publication bias was assessed using funnel plots and Egger's test when ≥10 effect sizes were available [38].

2.7. Statistical analysis

Meta-analyses were conducted in R (version 4.3.2; R Core Team), with results displayed using forest plots. Meta-analyses were conducted only for outcomes supported by at least five independent comparisons ($k \geq 5$, with k referring to the number of independent comparisons, not the number of studies), where this threshold was not met, pooled estimates were calculated for descriptive purposes only and interpreted with caution. Statistical significance was set at $p < 0.05$ for all analyses. Standardized mean differences (Hedges' g) with 95% confidence intervals were calculated to quantify differences between exercise timing conditions. Effect sizes were interpreted as trivial (<0.10), small (0.20), medium (0.50), or large (0.80). Because several primary studies contributed multiple comparisons (e.g., multiple time points or intensity conditions), effect sizes within the same study are statistically dependent. To account for this clustering, three-level random-effects models were used, with comparisons (level 2) nested within studies (level 3) [39]. This approach partitions total variance into within-study and between-study components, avoiding the underestimation of uncertainty that arises when dependent effect sizes are treated as independent. Variance components were estimated using restricted maximum likelihood (REML). The proportion of total variance attributable to each level was quantified using the I^2 statistic adapted for multilevel models [40], computed as the ratio of total between-study variance to total variance plus typical within-study sampling variance. Subgroup analyses examined health status, participants' physical activity level, exercise intensity, exercise exposure and sleep assessment method. Each subgroup was modelled using the same three-level structure; where the number of studies within a subgroup precluded multilevel estimation, a standard random-effects model with Knapp-Hartung adjustment was used as a fallback [39]. Meta-regression assessed the influence of ΔEX_T (temporal separation between morning and evening/nighttime exercise sessions). Heterogeneity was evaluated using Cochran's Q ($p < 0.10$) and I^2 statistics [41,42]. Sensitivity analyses included leave-one-out

Table 1
Characteristics of studies included in the systematic review and meta-analysis.

Study	Participants	Design	Study timeframe	Exercise characteristics	Intensity	Timing (morning vs evening)	Sleep assessment	Main findings
Alley et al., 2015 *United States*	24 healthy adults 18–25 years Chronotype: NR	RCT, cross-over	Acute	Single resistance exercise session 3×10 at 65% 10RM (9 exercises)	Moderate	4 conditions: • Control • 7 a.m. • 1 p.m. • 7 p.m. $\Delta EX_T = 12$ h	EEG	• Morning: ↑ SOL vs afternoon/evening; ↓ awakenings vs control • Evening: ↓ WASO; ↓ awakenings • Morning vs evening: Evening exercise → better sleep continuity; no effect on sleep architecture
Azharuddin et al., 2022 *India*	20 adults with poor sleep 18–30 years Chronotype: NR	RCT, parallel groups	Acute	Single exercise session Knee extension (4×7 at 70% 1RM) + 10 min cycling at 90% HRmax	High	2 groups: • 7 a.m. (n = 10) • 7 p.m. (n = 10) $\Delta EX_T = 12$ h	PSG Questionnaire (SMHSQ)	• Morning: ↑ sleep efficiency; ↑ subjective sleep quality • Evening: ↑ REM sleep; marginal ↑ sleep efficiency • Morning vs evening: Greater improvement in sleep efficiency and subjective sleep quality after morning exercise
Fairbrother et al., 2014 *United States*	20 prehypertensive adults 30–60 years Chronotype: NR	RCT, cross-over	Acute	Single aerobic session 30 min treadmill at 65% HR at VO_2peak	Moderate	3 conditions: • 7 a.m. • 1 p.m. • 7 p.m. $\Delta EX_T = 12$ h	EEG	Morning vs evening: Morning exercise → ↓ SOL; ↑ deep sleep; ↓ awakenings; ↓ WASO; no effect on TST
Goldberg et al., 2024 *France*	13 recreational runners 27.7 ± 7.2 years Chronotype: Intermediate (MEQ)	RCT, cross-over	Acute	Single run 45–60 min at 70% maximal aerobic velocity	Moderate	Conditions: • Control • Morning (30 min–2 h post-wake) • Evening (2 h–30 min pre-sleep) ΔEX_T: NR	EEG; Questionnaire (Spiegel Sleep Inventory)	• Morning & evening: ↑ NREM (N2) • No timing effect on TST, SOL, WASO, SE
Ramos-Campo et al., 2019 *Spain*	14 ultra-endurance runners 27.6 ± 7.4 years Chronotype: Intermediate	RCT, cross-over	Acute	Moderate and vigorous running sessions	Moderate/ High	10 a.m. vs 8 p.m. $\Delta EX_T = 10$ h	Accelerometer; Karolinska; diary	• Morning vs evening: Morning training consistently associated with ↑ objective sleep quality across intensities. Overall, exercise intensity appeared to have a stronger impact on sleep than time of day, with moderate morning exercise showing the most favorable sleep profile.
Vitale et al., 2017 *Italy*	23 collegiate soccer players 21.7 ± 2.72 years Chronotype: MEQ	RCT, cross-over	Acute	HIIT running (4 × 4 min at 90–95% HRmax)	High	8 a.m. vs 8 p.m. $\Delta EX_T = 12$ h	Accelerometer	• Evening high-intensity exercise impaired objective sleep quality selectively in morning-type athletes, whereas morning HIIT had no detrimental effect regardless of chronotype.
Morita et al., 2017 *Japan*	43 older adults (DIS/EMA/ controls) 55–65 years Chronotype: NR	Non-randomized cross-over	Acute	Single aerobic session 4 × 10 min step at 59% HRR	Moderate	Conditions: 9:30 a.m. vs 5:30 p.m.	PSG; PGI-C	• Morning vs evening: Morning exercise benefited nocturnal sleep stability (late-night;

(continued on next page)

Table 1 (*continued*)

Study	Participants	Design	Study timeframe	Exercise characteristics	Intensity	Timing (morning vs evening)	Sleep assessment	Main findings
								sleep-onset difficulty); evening exercise showed no clear benefit.
Lee et al., 2018 *South Korea*	241 older adults (±insomnia) 61.2 years Chronotype: NR	Observational	1-week monitoring	Unrestricted exercise modalities	NR	Groups: No Exercise Morning Afternoon Evening	Accelerometer; Questionnaires (PSQI; ESS)	• Morning: Less favorable subjective sleep • Afternoon/ evening: ↓ PSQI • Outdoor afternoon EX → best sleep quality
Chahine et al., 2022 *Lebanon*	92 healthy sedentary students 19.3 ± 1.2 years Chronotype: NR	RCT, parallel groups	Short-term	4 consecutive exercise sessions 1 h stretching + aerobics	Moderate	3 groups: • Control • 8 a.m. • 8 p.m. $\Delta EX_T = 12$ h	Spiegel Sleep Inventory	• Morning EX: ↑ sleep quality; ↑ melatonin; ↓ evening cortisol • Evening EX: Limited improvement • Timing: Morning superior
Thomas et al., 2020 *United States*	52 sedentary adults 18–45 years Chronotype: MEQ	RCT, parallel groups	Short-term (5 consecutive days)	Exercise sessions 30 min at 70% VO_2peak	High	Groups: Relative to DLMO $\Delta EX_T = 10$ h	Accelerometer	• Morning: Circadian phase advance • Sleep quality/ duration: ↔
Yamanaka et al., 2015 *Japan*	22 healthy young males 22.0 ± 1.8 years Chronotype: NR	RCT, parallel groups	Short-term (4 consecutive days)	2-h intermittent cycling 4 consecutive days	Moderate	Conditions: Control (no EX); Morning EX (ZT3–ZT5, ~3–5 h post-wake); Evening EX (ZT10–ZT12, ~10–12 h post-wake)	PSG; Accelerometer	• Morning EX: ↔ sleep architecture; ↔TST • Evening EX: ↓ REM; ↑ nocturnal HR; attenuated CBT decline
Carrillo et al., 2024 *United Kingdom*	18 adults with type 2 diabetes 61 ± 8.2 years Chronotype: NR	RCT, cross-over	Medium-term (6 weeks)	Chronic aerobic exercise 30 min walking every other day	Moderate	Conditions: •Morning (07:00–10:00) •Evening (16:00–19:00) $\Delta EX_T \approx 8–9$ h	Wrist accelerometry (Garmin Vivosmart 4); MCTQ	• Sleep duration & architecture: ↔ • Timing: No effect
Seol et al., 2021 *Japan*	60 healthy older adults 71.0 ± 3.9 years Chronotype: NR	RCT, parallel groups	Medium-term (8 weeks)	Low-intensity aerobic exercise 30 min/day step-up	Low	Groups: Morning vs Evening ΔEX_T: NR	Accelerometer; PSQI; Sleep diaries	• Evening EX: ↓ SOL; ↑ satisfaction • Morning EX: No significant change
Saidi et al., 2021 *France*	28 sedentary overweight/obese adults 30–65 years Chronotype: MEQ	RCT, parallel groups	Medium-term (12 weeks)	Combined aerobic + resistance training	Moderate	2 groups: • 9 a.m. • 6:30 p.m. $\Delta EX_T = 9.5$ h	Accelerometer; PSQI; ESS	• Sleep quality: ↑; Duration: ↔; Timing: advance (morning), delay (evening)
Brooker et al., 2023 *Australia*	97 inactive adults with overweight/ obesity 39 ± 11 years Chronotype: NR	RCT, parallel groups	Medium-term (12 weeks)	250 min/week moderate-to-vigorous exercise	Moderate	3 groups: • Control • 6 a.m. • 4 p.m. $\Delta EX_T = 10$ h	MARCA	• Morning: ↑ sleep duration (~+36 min/day) • Evening: ↑ sleep duration (~+20 min/day) • Timing: No differential effects
Creasy et al., 2022 *United States*	33 adults with overweight/ obesity 18–56 years Chronotype: NR	RCT, parallel groups	Medium-term (15 weeks)	Exercise training 4 × 54 min/ week at 70–80% HRmax	High	2 groups: • 6 a.m. • 3 p.m. $\Delta EX_T = 9$ h	Accelerometer	• Morning: Slight ↓ sleep duration • Evening: ↑ sleep duration (~+25–30 min/day) • Timing: Divergent effects
Teo et al., 2021 *Australia*	40 overweight/ obese adults 51 ± 13 years Chronotype: NR	RCT, parallel groups	Medium-term (12 weeks)	Combined aerobic + resistance training	High	2 groups: • 8 a.m. • 5 p.m. $\Delta EX_T = 9$ h	Accelerometer; PSQI; ESS	• Both timings: ↑ sleep quantity • Timing: No difference
Shen et al., 2025 *China*	58 sedentary males 18–28 years Chronotype: NR	RCT, parallel groups	Medium-term (12 weeks)	Chronic aerobic exercise ≥150 min/week	Moderate	Groups: Morning vs Evening vs Control $\Delta EX_T = 12$ h	MCTQ; PSQI	• Morning & evening: ↓ sleep latency; ↑ sleep quality • Morning only: Circadian phase advance

(*continued on next page*)

Table 1 (*continued*)

Study	Participants	Design	Study timeframe	Exercise characteristics	Intensity	Timing (morning vs evening)	Sleep assessment	Main findings
Passos et al., 2011 *Brazil*	19 sedentary adults with chronic primary insomnia 45.0 ± 1.9 years Chronotype: NR	RCT, parallel groups	Long-term (6 months)	Exercise training 3 × 50 min/ week treadmill running at VT1	Moderate	2 groups: • 10 a.m. • 6 p.m. $\Delta EX_T = 8$ h	PSG; Sleep diary	• Morning & evening: ↓ SOL; ↓ WASO; ↑ SE • Timing: No meaningful difference; slight subjective advantage for morning exercise.
Tworoger et al., 2003 *United States*	173 postmenopausal overweight/obese women 50–75 years Chronotype: NR	RCT, parallel groups	Long-term (1 year)	Aerobic + resistance exercise	Moderate	Timing groups: Morning vs Evening	WHIIRS	• Morning high-volume EX: ↓ SOL • Evening high-volume EX: ↑ SOL
Küüsmaa-Schildt et al., 2019 *Finland*	52 healthy adults 32.4 ± ~5.5 years	Non-randomized cohort study	Long-term (24 weeks)	Combined aerobic + resistance training	High	3 groups: • Control • Morning (6–10 a.m.) • Evening (4–8 pm) ΔEX_T: NR	PSQI	• Sleep duration stable (7–8 h) • Timing: No effect
Marchesano et al., 2023 *Uruguay*	17 dancers 18–27 years	Non-randomized cross-over	Long-term (1 year)	Habitual training schedule shift	NR	Conditions: •Morning (8:30 a.m.) •Evening (8:00 p.m.) ΔEX_T: NR	MCTQ	• Timing shift: ↔ sleep duration
Buman et al., 2014 *United States*	1000 adults 41.95 ± 11.1 years Chronotype: NR	Cross-sectional survey	Habitual	Habitual free living physical activity	Self-reported light/ moderate/ vigorous exercise	• Morning (>8 h before bedtime) • Evening (<4 h before bedtime) ΔEX_T: Categorized relative to bedtime	Self-reported sleep quality; TST; SOL; Waking unrefreshed	• Morning EX: ↑ sleep quality; ↓ waking unrefreshed • Evening EX (<4 h): Not associated with poorer sleep • Timing: No effect on SOL
Mecenas et al., 2023 *Brazil*	2050 adults 18–65 years Chronotype: Early/ Intermediate/Late (MSFsc)	Observational study	Habitual	Self-reported physical exercise Frequency & weekly duration assessed	Moderate-to-vigorous (self-reported)	Morning (5:00–12:00) Afternoon (12:00–18:00) Evening (>18:00)	Self-reported sleep duration Sleep latency Nocturnal awakenings Sleep quality Insomnia diagnosis	• Physical exercisers → better sleep parameters • Morning exercise → shorter sleep latency • Chronotype interaction: – Early types: Evening exercise → ↑ awakenings & ↑ insomnia – Late types: Morning exercise → ↓ sleep latency • Timing effects depended on chronotype
Bailey et al., 2023 *United States*	24 healthy adults 18–25 years Chronotype: NR	Observational study	1-week monitoring	Unrestricted exercise modalities	NR	6 post-evaluation groups: • No EX • Varied EX times • Early morning • Late morning • Afternoon • Evening/night ΔEX_T: NR	MOS Sleep Scale; Sleep Quality Score; Ultra-Short Munich Chronotype Questionnaire	• Morning: ↔ self-reported sleep • Evening: ↔ self-reported sleep • Timing: No association with sleep outcomes

Abbreviations: ΔEXT, temporal separation between morning and evening exercise sessions; DIS, difficulty initiating sleep; DLMO, dim light melatonin onset; EEG, electroencephalography; EMA, early morning awakening; ESS, Epworth Sleepiness Scale; EX, exercise; HIIT, high-intensity interval training; HRmax, maximal heart rate; HRR, heart rate reserve; HRV, heart rate variability; IPAQ, International Physical Activity Questionnaire; MARCA, Multimedia Activity Recall for Children and Adolescents; MCTQ, Munich Chronotype Questionnaire; ME, morning exercise; MEQ, Morningness–Eveningness Questionnaire; NREM, non–rapid eye movement sleep; NR, not reported; PGI-C, Patient Global Impression of Change; PSG, polysomnography; PSQI, Pittsburgh Sleep Quality Index; RCT, randomized controlled trial; REM, rapid eye movement sleep; RM, repetition maximum; SE, sleep efficiency; SOL, sleep onset latency; SMHSQ, St. Mary's Hospital Sleep Questionnaire; TIB, time in bed; TST, total sleep time; VO_2max/VO_2peak, maximal/peak oxygen uptake; VT1, first ventilatory threshold; WASO, wake after sleep onset; WHIIRS, Women's Health Initiative Insomnia Rating Scale; ZT, Zeitgeber time.
Studies included in the meta-analysis are indicated in bold.

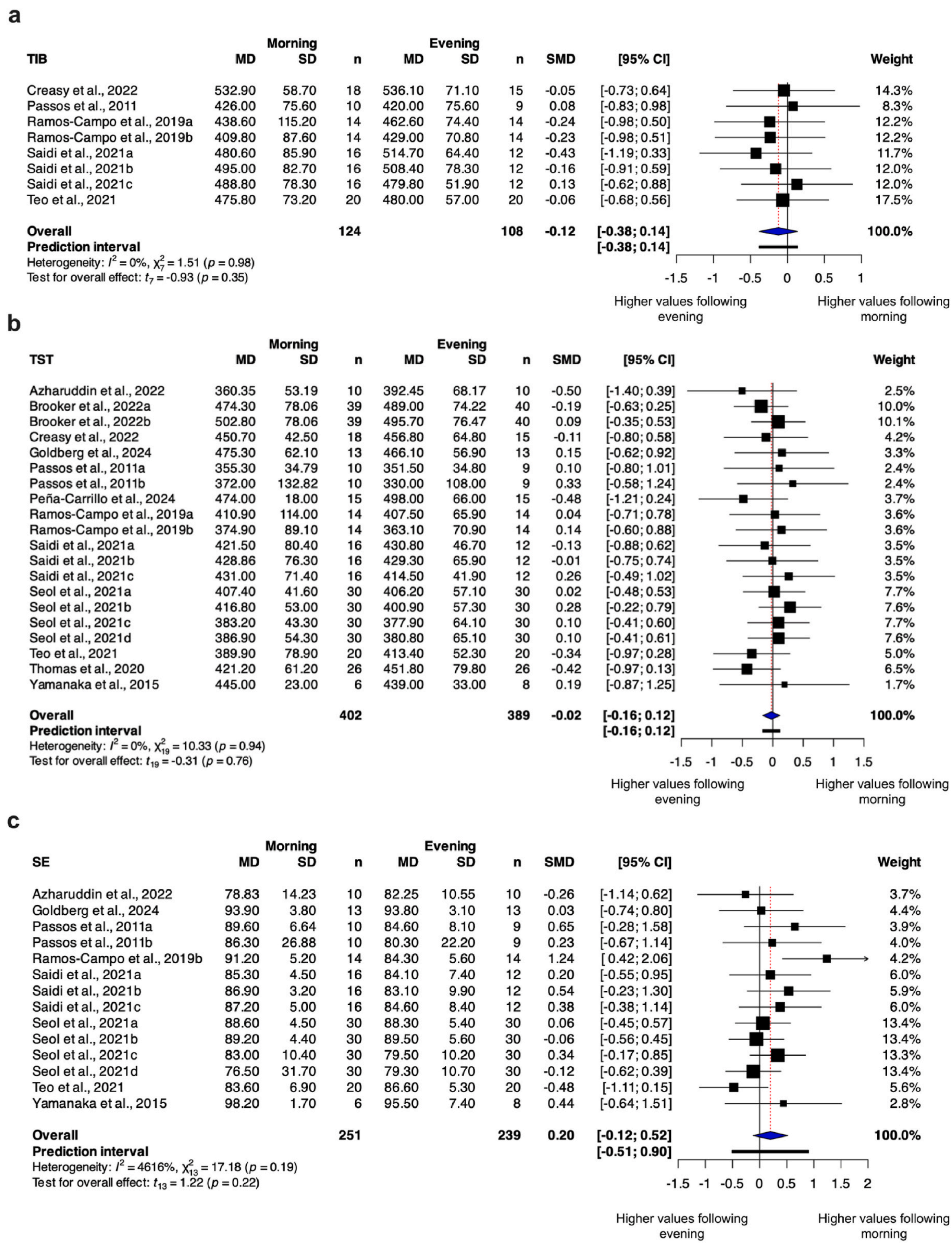

Fig. 2. Effects of exercise timing (morning vs evening/nighttime) on sleep outcomes.

procedures, in which each comparison was systematically removed and the three-level model re-estimated on the remaining data (see Table S7) [43]. This confirmed the stability of pooled estimates across all outcomes. Outliers were identified using externally standardized residuals (threshold: $|z| > 1.96$) and excluded from final models; results are reported both with and without the identified outliers (see Table S8).

3. Results

3.1. Study selection

The study selection process is outlined in Fig. 1. Database searches identified 1108 records (PubMed n = 527; SPORTDiscus n = 297;

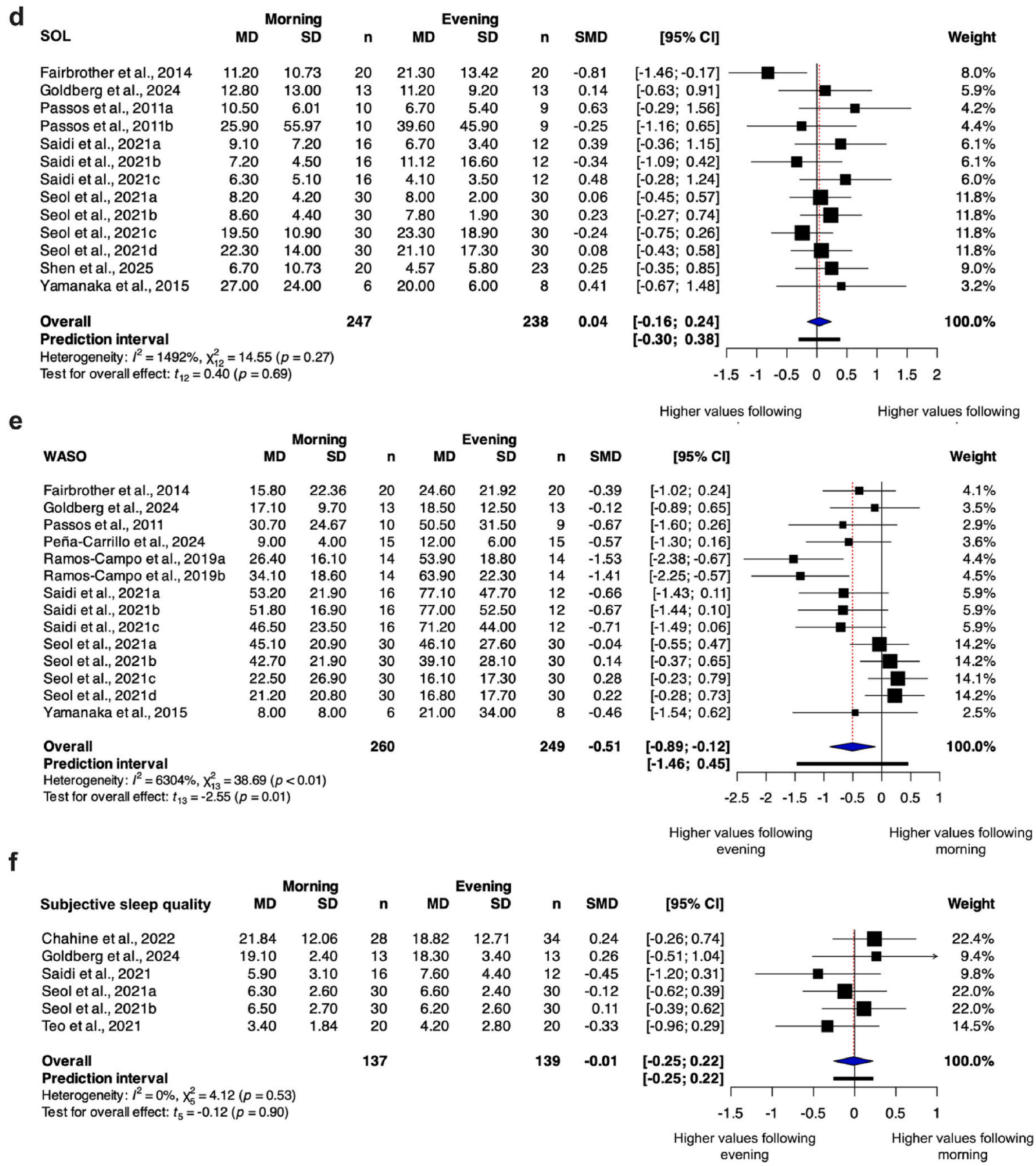

Fig. 2. (*continued*).

Embase n = 131; APA PsycNet n = 153). After duplicate removal and title/abstract screening, 35 articles underwent full-text assessment. Thirteen were excluded (two for missing sleep outcomes, ten for irrelevant interventions, and one non-English). Two studies published after April 2024 and one identified through other sources were added. The review included 25 studies (2003–2025), comprising 4233 participants (range: 13–2050). Eighteen were RCTs, with the remainder including non-randomized interventions (n = 2) [44,45] and observational studies (n = 5) [46–49]. Sixteen RCTs (n = 611) were included in the meta-analysis; two were excluded due to unavailable outcome data despite author contact [50,51].

3.2. Characteristics of the included studies

Study characteristics and main findings are summarized in Table 1. Of the included studies, 15 examined healthy populations [45–48,50,52–61], whereas 10 focused on clinical populations [22,31,44,49,51,62–66], primarily individuals with obesity (n = 6) or sleep disorders (n = 3). Participant ages ranged from 18 to 79 years. Samples were mixed but predominantly male, with some single-sex studies (e.g., all-male athletes or post-menopausal women).

Among all studies, protocols included acute (single bout; n = 8) and repeated exercise interventions (≥5 days; n = 17), spanning 5 days to 12 months. Exercise modality was predominantly aerobic (n = 13), with fewer studies using combined training, resistance training, or high-intensity interval training, limiting modality-specific analyses. Among included RCTs, exercise intensity, classified according to American College of Sports Medicine guidelines [67], was low (n = 1), moderate (n = 8), high (n = 6), or mixed (n = 1), and prescribed using heterogeneous metrics (e.g., maximum heart rate [HRmax], heart rate reserve

Table 2
Subgroup analyses of exercise timing (morning vs evening) effects on sleep outcomes.

Outcome	Moderator (subgroup)	Subgroup level	k	SMD [95%CI]	Test of subgroup difference	Magnitude	I^2
TIB	Health status	Healthy	2	−0.24 [-0.28; −0.20]	$\chi^2 = 0.24, p = 0.625$	Small	0%
		Clinical	6	−0.09 [-0.38; 0.21]		Trivial	0%
	Activity status	Active	2	−0.24 [-0.28; −0.20]	$\chi^2 = 0.24, p = 0.625$	Small	0%
		Sedentary	6	−0.09 [-0.38; 0.21]		Trivial	0%
	Exercise intensity	High	3	−0.11 [-0.50; 0.29]	$\chi^2 = 0.01, p = 0.905$	Small	0%
		Moderate	5	−0.14 [-0.48; 0.21]		Small	0%
	Exercise exposure	Acute	2	−0.24 [-0.28; −0.20]	$\chi^2 = 0.24, p = 0.625$	Small	0%
		Repeated	6	−0.09 [-0.38; 0.21]		Trivial	0%
	Method	Objective	7	−0.14 [-0.41; 0.13]	$\chi^2 = 0.20, p = 0.651$	Small	0%
		Subjective	1	0.08 [-0.83; 0.98]		Trivial	0%
TST	Health status	Healthy	10	0.00 [-0.22; 0.23]	$\chi^2 = 0.47, p = 0.493$	Trivial	9.3%
		Clinical	10	−0.07 [-0.27; 0.13]		Trivial	0%
	Activity status	Active	4	0.12 [-0.28; 0.52]	$\chi^2 = 0.57, p = 0.451$	Small	0%
		Sedentary	16	−0.05 [-0.21; 0.11]		Trivial	4.5%
	Exercise intensity	High	5	−0.26 [-0.56; 0.04]	$\chi^2 = 6.22, p = 0.045$	Small	0%
		Moderate	11	−0.01 [-0.21; 0.19]		Trivial	0%
		Low	4	0.13 [-0.05; 0.30]		Small	0%
	Exercise exposure	Acute	5	0.01 [-0.35; 0.38]	$\chi^2 = 0.05, p = 0.830$	Trivial	0%
		Repeated	15	−0.03 [-0.19; 0.12]		Trivial	2%
	Method	Objective	15	−0.06 [-0.23; 0.12]	$\chi^2 = 0.37, p = 0.540$	Trivial	0%
		Subjective	5	0.03 [-0.19; 0.26]		Trivial	0%
SE	Health status	Healthy	8	0.26 [-0.22; 0.74]	$\chi^2 = 0.13, p = 0.722$	Small	61%
		Clinical	6	0.13 [-0.42; 0.68]		Small	50%
	Activity status	Active	3	0.57 [-0.19; 1.33]	$\chi^2 = 2.07\ p = 0.150$	Medium	56%
		Sedentary	11	0.08 [-0.20; 0.35]		Trivial	24%
	Exercise intensity	High	3	0.15 [-0.91; 1.21]	$\chi^2 = 0.29, p = 0.863$	Small	83%
		Moderate	7	0.33 [0.02; 0.65]		Medium	0%
		Low	4	0.05 [-0.26; 0.37]		Trivial	0%
	Exercise exposure	Acute	3	0.02 [-0.49; 0.63]	$\chi^2 = 0.32, p = 0.571$	Trivial	0%
		Repeated	11	0.28 [-0.19; 0.75]		Small	66%
	Method	Objective	11	0.20 [-0.15, 0.56]	$\chi^2 = 0.02, p = 0.887$	Small	50%
		Subjective	3	0.13 [-0.22, 0.47]		Small	6%
SOL	Health status	Healthy	8	−0.03 [-0.41; 0.34]	$\chi^2 = 0.66, p = 0.417$	Trivial	49%
		Clinical	5	0.18 [-0.21; 0.56]		Small	12%
	Activity status	Active	2	0.23 [-0.40; 0.85]	$\chi^2 = 0.35, p = 0.554$	Small	0%
		Sedentary	11	0.02 [-0.20; 0.24]		Trivial	24%
	Exercise intensity	Moderate	9	0.06 [-0.27; 0.40]	$\chi^2 = 0.00, p = 0.993$	Trivial	40%
		Low	4	0.03 [-0.28; 0.35]		Trivial	0%
	Exercise exposure	Acute	3	−0.16 [-0.92; 0.59]	$\chi^2 = 2.24, p = 0.135$	Small	62%
		Repeated	10	0.10 [-0.10; 0.29]		Small	0%
	Method	Objective	9	0.09 [-0.21, 0.38]	$\chi^2 = 0.22, p = 0.637$	Trivial	37%
		Subjective	4	−0.02 [-0.31, 0.27]		Trivial	0%
WASO	Health status	Healthy	9	−0.44 [-1.04; 0.16]	$\chi^2 = 0.25, p = 0.616$	Small	77%
		Clinical	5	−0.65 [-1.01; −0.30]		Medium	0%
	Activity status	Active	4	−0.73 [-1.60; 0.14]	$\chi^2 = 0.89, p = 0.346$	Large	68%
		Sedentary	10	−0.36 [-0.76; 0.04]		Small	54%
	Exercise intensity	High	1	−1.41 [-2.24; −0.58]	$\chi^2 = 24.69, p < 0.001$	Large	-
		Moderate	9	−0.62 [-0.88; −0.36]		Medium	0%
		Low	4	0.15 [-0.07; 0.37]		Small	0%
	Exercise exposure	Acute	5	−0.65 [-1.22; −0.01]	$\chi^2 = 0.46, p = 0.496$	Large	61%
		Repeated	9	−0.37 [-0.85; 0.12]		Small	62%
	Method	Objective	12	−0.53 [-0.90, −0.16]	$\chi^2 = 1.44, p = 0.230$	Medium	55%
		Subjective	2	0.25 [-0.09, 0.60]		Small	0%
Self-reported sleep quality	Health status	Healthy	4	0.10 [-0.17; 0.37]	$\chi^2 = 2.88, p = 0.090$	Small	0%
		Clinical	2	−0.38 [-0.86; 0.10]		Small	0%
	Activity status	Active	1	0.26 [-0.51; 1.04]	$\chi^2 = 0.55, p = 0.459$	Small	-
		Sedentary	5	−0.04 [-0.29; 0.21]		Trivial	0%
	Exercise intensity	High	1	−0.33 [-0.95; 0.29]	$\chi^2 = 1.02, p = 0.601$	Small	-
		Moderate	3	0.07 [-0.33; 0.48]		Trivial	14%
		Low	2	0.00 [-1.46; 1.46]		Trivial	0%
	Exercise exposure	Acute	2	0.25 [0.17; 0.68]	$\chi^2 = 2.17, p = 0.140$	Small	0%
		Repeated	4	−0.14 [-0.51; 0.24]		Small	0%
	Method	Objective	5	−0.09 [-0.36, 0.18]	$\chi^2 = 1.27, p = 0.259$	Trivial	0%
		Subjective	1	0.24 [-0.26, 0.74]		Small	-

Note. k indicates the number of independent comparisons. SMDs are expressed such that negative values indicate higher sleep parameter values following evening exercise, whereas positive values indicate higher values following morning exercise. Subgroup differences were evaluated using χ^2 tests under random-effects models; heterogeneity is presented as I^2 (%). Subgroup analyses were conducted at the study level and should be interpreted cautiously. Several subgroup categories were partially overlapping across studies and should not be considered independent. Subgroups with $k < 3$ are reported for descriptive purposes only. Effect magnitude classifications should be interpreted cautiously, particularly when confidence intervals are wide or include zero.
TIB: Time in bed; TST: Total sleep time; SE: Sleep efficiency; SOL: Sleep onset latency; WASO: Wake after sleep onset; SMD: Standardized mean difference; I^2: Heterogeneity statistic.

[HRR], peak oxygen uptake [VO_2peak], metabolic equivalents [METs], or one-repetition maximum [1RM]). In studies included in the meta-analysis, morning sessions occurred between 06:00 and 12:00 (08:14 ± 01:16 h) and evening/nighttime sessions between 16:00 and 23:00 (18:22 ± 01:19 h). This corresponded to a ΔEX_T of 7–12 h (typically ≥9 h). Habitual bedtime was reported in fewer than one-third of studies, limiting quantification of exercise–sleep proximity. Moreover, some studies defined timing relative to wake time or dim light melatonin onset (DLMO), limiting comparability. Sleep outcomes were assessed using polysomnography (n = 5), electroencephalography (n = 3), accelerometry (n = 7), or self-reported measures (n = 10). Chronotype was reported in only four of the 16 meta-analyzed studies, typically using the Morningness–Eveningness Questionnaire [68] or Munich Chronotype Questionnaire [69], with extreme chronotypes often excluded, precluding subgroup analyses. Only five studies monitored physical activity outside prescribed exercise, with the remaining studies relying on self-report or no control, introducing potential residual confounding in the interpretation of timing effects.

3.3. Methodological quality and risk of bias

Risk of bias assessments are detailed in Table S2 for parallel-group RCTs, Table S3 for crossover RCTs, and Table S4 and Table S5 for non-randomized studies. In parallel-group RCTs, none were rated as having an overall low risk of bias. Most studies (n = 10, 91%) were judged as presenting some concerns, while one study (9%) was classified as having a high risk of bias. In crossover RCTs, all studies (n = 5, 100%) were rated as presenting some concerns, with no study classified as low or high risk of bias. Across both designs, risk of bias was most frequently driven by concerns related to deviations from intended interventions, missing outcome data, and selection of the reported results, whereas the randomization process and outcome measurement were generally judged as low risk.

In non-randomized studies, none were rated as having an overall low risk of bias. Risk-of-bias assessment indicated some concerns for all ROBINS-E studies (n = 5, 100%) and moderate risk for all ROBINS-I studies (n = 2, 100%). Across the observational studies, the main concerns were related to confounding, exposure measurement, outcome measurement, participant selection, and selection of the reported result. In the intervention studies, exercise timing and intervention classification were clearly defined, and deviations from intended interventions were judged to be low risk. However, the overall judgement remained moderate because of residual concerns related to confounding, outcome measurement, missing data in one study, and selection of the reported result. No included study was rated as serious or critical risk of bias.

3.4. Meta-analysis

3.4.1. Effect of exercise timing on sleep outcomes

The overall effects of exercise timing (morning vs evening/nighttime) on sleep outcomes are presented in Fig. 2. Pooled effects were small across most sleep outcomes, indicating that morning versus evening/nighttime exercise results in limited differences in sleep. Time in bed (TIB) showed no clear difference between conditions (Fig. 2a–Table S6; N = 232, k = 8, SMD = −0.12, 95% CI [−0.38, 0.14], p = 0.351; I^2 = 0%; GRADE = Very low), although estimates slightly favored longer TIB following evening/nighttime exercise. Total sleep time (TST) did not differ between conditions (Fig. 2b–Table S6; k = 20, SMD = −0.02, 95% CI [−0.16, 0.12], p = 0.757; I^2 = 0%; GRADE = Low). Sleep efficiency (SE) showed a small, non-significant effect favoring morning exercise (Fig. 2c–Table S6; k = 14, SMD = 0.20, 95% CI [−0.12, 0.52], p = 0.223; I^2 = 46%; GRADE = Low), suggesting differences in sleep continuity rather than sleep initiation. For TST, funnel plot (Fig. S1) revealed no publication bias, confirmed by Egger's test (t = 0, p = 1.000), while SE demonstrated moderate funnel plot asymmetry with a non-significant Egger's test (t = 1.563, p = 0.144).

Sleep onset latency (SOL) showed no timing-related effect (Fig. 2d, Table S6; k = 13, SMD = 0.04, 95% CI [−0.16; 0.24], p = 0.351; I^2 = 14%; GRADE = Low) with no evidence of publication bias (Fig. S1; Egger's test: t = 0.681, p = 0.510), whereas wake after sleep onset (WASO) was the only outcome showing a statistically significant timing effect, with higher WASO following evening/nighttime exercise (Fig. 2e–Table S6; k = 14, SMD = −0.51, 95% CI [−0.89, −0.12], p = 0.011; I^2 = 63%; GRADE = Very low). However, Egger's test indicated potential small-study effects for WASO (Fig. S1; t = −4.55, p < 0.001), suggesting that this effect may be influenced by asymmetry in the evidence base and should therefore be interpreted with caution. Self-reported sleep quality did not differ according to exercise timing (Fig. 2f; Table S6; N = 276, k = 6, SMD = −0.01, 95% CI [−0.25, 0.22], p = 0.900; I^2 = 0%; GRADE = Very low), although the limited number of studies warrants cautious interpretation. Quantitative synthesis of sleep architecture outcomes (e.g., sleep stages) and daytime sleepiness was not performed, as fewer than five independent comparisons were available for each outcome, precluding meta-analysis based on our predefined criteria (k ≥ 5).

3.4.2. Subgroup analyses and meta-regression

Subgroup analyses were performed only for sleep outcomes with k ≥ 5 independent comparisons (Table 2). As this criterion was not met for self-reported sleepiness, moderator analyses for this outcome were not conducted.

- Exercise intensity

Effects of exercise timing on several sleep outcomes differed across exercise intensity subgroups (Table 2). No evidence of intensity-related effect was observed for TIB between the high and moderate-intensity conditions (χ^2 = 0.01, p = 0.905), however low-intensity exercise was not represented in the TIB subgroup analyses. In contrast, TST showed significant moderation by exercise intensity (Fig. S2; χ^2 = 10.06, p = 0.045), with directionally inconsistent effects: high-intensity exercise tended to favor longer TST following evening/nighttime exercise (SMD [95%CI] = −0.26 [−0.56; 0.04]), whereas low-intensity exercise showed the opposite pattern (SMD [95%CI] = 0.13 [−0.05; 0.30]), with higher TST following morning exercise; no meaningful timing-related difference was observed at moderate intensity. WASO showed statistically significant differences across exercise intensity subgroups (Fig. S3; χ^2 = 39.82, p < 0.001), with a consistent pattern at moderate intensity (SMD [95%CI] = −0.62 [−0.88; −0.36]) indicating higher WASO following evening/nighttime exercise, whereas only a small timing-related differences were observed at low intensity (SMD [95% CI] = 0.15 [−0.07; 0.37]) in the opposite direction. Although a larger effect was observed at high intensity (SMD [95%CI] = −1.41 [−2.24; −0.58]), this estimate was based on a single study and should therefore be interpreted with caution. SE did not show significant differences across exercise intensity subgroups (χ^2 = 0.29, p = 0.863), with a small pooled effect favoring morning exercise at moderate intensity (SMD [95%CI] = 0.33 [0.02; 0.65]) no clear differences at low intensity (SMD [95%CI] = 0.05 [−0.26; 0.37]), and heterogeneous estimates at high intensity (SMD [95%CI] = 0.15 [−0.91; 1.21], I^2 = 83%), largely driven by a single study conducted in athletes [54].

- Exercise exposure

Exercise exposure (acute vs repeated) shows no significant timing-related differences with repeated interventions compared with single-day protocols (Table 2). For TIB, no subgroup differences were observed (χ^2 = 0.24, p = 0.625), with single-day interventions showing slightly longer TIB following evening/nighttime exercise, whereas repeated interventions showed no meaningful timing-related differences. TST showed no evidence of subgroup differences (χ^2 = 0.05, p = 0.830), with trivial effects. Similarly, SOL was not moderated by exercise exposure ($\chi^2 = 2.24$, p = 0.135), indicating comparable timing effects on sleep initiation. For WASO, no statistically significant subgroup differences were detected, but slightly higher WASO following evening/nighttime exercise was observed in single-day interventions compared with repeated protocols ($\chi^2 = 0.46$, $p = 0.496$). SE showed a small, non-significant effect favoring morning exercise in repeated compared with single-day interventions ($\chi^2 = 0.32$, p = 0.571).

- Individual characteristics

Subgroup analyses by health status (healthy vs clinical) and physical activity status (active vs sedentary) did not show meaningful differences in the effects of exercise timing on sleep (Table 2). Across TIB, TST, and SOL, effects were small and largely overlapping between subgroups. Sleep efficiency showed small effects favoring morning exercise, but without significant subgroup differences. Larger WASO estimates were observed in some subgroups, particularly clinical samples, although these findings were based on limited data and should be interpreted cautiously.

- Sleep assessment method

Subgroup analyses by sleep assessment method (objective vs subjective) did not show meaningful differences in the effects of exercise timing on sleep (Table 2). Across TIB, TST, SE, SOL, WASO and self-reported sleep quality, between-subgroup differences were trivial to small.

- Temporal separation between morning and evening/nighttime exercise (ΔEXT)

Although timing-related differences were observed for some sleep outcomes, meta-regression did not indicate that ΔEX_T (temporal separation between sessions) explained variability in timing effects across sleep outcomes (Fig. S4). No significant associations were observed for objective (TIB, TST, SE, SOL, WASO). These findings should be interpreted with caution, as the range of ΔEX_T across studies was relatively constrained, which may have limited the ability to detect moderating effects.

4. Discussion

This systematic review and meta-analysis examined whether exercise timing meaningfully influences sleep when morning and evening/nighttime sessions are directly compared. Overall, the findings suggest that exercise timing exerts modest and outcome-specific effects on sleep. No clear differences were observed for sleep duration, whereas small but more consistent effects emerged for sleep continuity, with higher WASO following evening/nighttime exercise. These results indicate that exercise timing is likely a conditional factor whose effects depend on individual and exercise characteristics. The present results align with recent systematic reviews and meta-analyses indicating that evening/nighttime exercise does not meaningfully impair sleep in most individuals and may warrant reconsideration of traditional sleep hygiene recommendations [27,70,71]. Across these reviews, evening/nighttime exercise was generally associated with minimal adverse effects compared with no-exercise control conditions and, in some cases, modest improvements in sleep, particularly when not performed close to bedtime. The current findings extend prior work and raise the hypothesis that timing-related differences are unlikely to have meaningful practical implications at the population level, while providing no evidence of a systematic advantage of morning over evening exercise for sleep.

The present findings are best interpreted within a conditional perspective in which the effects of exercise timing arise from the interaction between physiological activation, recovery processes, and sleep regulation [72]. Exercise induces acute thermoregulatory, autonomic, and metabolic responses that must resolve prior to sleep onset [57,73,74]. When exercise is performed in the evening, incomplete downregulation of these processes may persist into the initial sleep period. Residual elevations in core body temperature, sympathetic activity, and metabolic rate may increase physiological arousal and contribute to sleep fragmentation as shown in previous literature. The meta-analysis by Stutz et al. reported that elevated bedtime core body temperature following evening exercise was associated with lower sleep efficiency and higher WASO [27]. Similarly, vigorous evening exercise may attenuate parasympathetic activity at bedtime, reflecting delayed autonomic recovery and promoting fragmented sleep [23]. This interpretation is consistent with the higher WASO observed following evening exercise, suggesting that timing-related effects are more apparent for sleep continuity than for sleep duration. At the same time, the absence of meaningful differences in either total sleep time or time in bed does not provide evidence of the compensatory sleep response previously reported in the literature [75]. Individuals may extend sleep window in response to perceived or actual sleep disruption [76,77]. Moreover, although no significant difference in TIB was observed, scheduling constraints may still influence sleep opportunity, as morning exercise often requires earlier wake times, thereby restricting sleep opportunity time. This pattern has been reported in athletic populations, where early training schedules reduce total sleep time primarily through curtailed time in bed [78–81]. Circadian factors may also contribute, as exercise can act as a zeitgeber, and late-day exercise may delay circadian phase. In contrast, morning exercise may better align with the circadian system temporal organization and facilitate the accumulation of homeostatic sleep pressure. Exercise timing has been shown to influence circadian phase [57], and delay sleep timing following evening exercise has been reported in previous studies [64]. However, sleep timing outcomes (e.g., bedtime) were infrequently reported, and heterogeneity in study settings, including fixed versus habitual sleep windows, limited quantitative synthesis and interpretation. Taken together, these findings are compatible with a dual-process account in which physiological activation influences sleep continuity, while behavioral adjustments influence sleep opportunity. These interpretations remain hypothetical and should be viewed as consistent with, rather than directly demonstrated by, the present data.

The modest average effects observed across studies likely reflect substantial heterogeneity in the conditions under which exercise is performed. Exercise intensity emerged as the most consistent moderator. Timing-related differences were minimal at low-to-moderate intensities but more apparent at higher intensities, particularly for sleep continuity outcomes. This pattern is consistent with the notion that higher-intensity exercise induces larger and more sustained perturbations in thermoregulatory, autonomic, and neuroendocrine systems [82–87]. When performed in the evening, these responses may extend into the early sleep period, increasing vulnerability to sleep fragmentation. In contrast, lower-intensity exercise is associated with transient physiological responses that are more likely to resolve prior to sleep onset, thereby limiting potential disruption. Recent large-scale observational data support this interpretation. Leota et al. (2025) reported a dose-response relationship between evening physical activity strain, its timing relative to sleep onset, and sleep outcomes, with later and more intense physical activity associated with delayed sleep onset, reduced sleep duration, lower sleep quality, elevated nocturnal heart rate, and suppressed heart rate variability [24]. Importantly, exercise ending $\geq$4 h before sleep onset was not associated with measurable disruption, suggesting a critical recovery window. Similarly, a recent network meta-analysis indicated that effects on WASO are intensity-dependent, with low- and moderate-intensity exercise ranked more favorably than high-intensity protocols [71]. Notably, these effects did not extend to SOL. This dissociation suggests that increased sleep pressure following strenuous exercise may facilitate sleep initiation despite heightened physiological activation, while still compromising sleep continuity.

Whether late exercise delays actual bedtime under free-living conditions remains to be determined. By contrast, when high-intensity exercise is performed in the morning, the longer recovery interval before bedtime likely allows these physiological disturbances to resolve, thereby attenuating their impact on sleep continuity.

Although evidence supporting a moderating effect of exercise exposure remained limited, adaptive responses to regular training reported in the literature, including enhanced autonomic recovery and improved thermoregulation [84], provide a plausible biological basis for an attenuation of the time-related effects of exercise on sleep. It is possible that the limited number of studies, the uneven distribution across exposure subgroups, and the heterogeneity of the included populations reduced the ability to detect meaningful exposure-related differences. Furthermore, the small effect favoring morning exercise in repeated versus single-day interventions on SE is consistent with emerging evidence suggesting that repeated morning exercise may enhance circadian alignment through phase-advancing effects, improve metabolic regulation, reduce cortisol concentrations after awakening, and ultimately promote better overall sleep quality [59,88]. In contrast, subgroup analyses based on health status and physical activity level showed largely overlapping effects, suggesting that these factors play a less prominent role than exercise characteristics in shaping timing-related differences. Similarly, meta-regression analyses did not support a role for temporal separation between exercise sessions (ΔEX_T), although the relatively narrow range of values limits firm conclusions.

4.1. Implications for exercise timing and sleep health

From a practical perspective, the present findings do not support rigid recommendations regarding the optimal time of day to exercise for sleep. Instead, they suggest that exercise timing can remain flexible for most individuals, although this flexibility should be guided by exercise intensity and proximity to bedtime, since evening exercise appears less likely to impair sleep when performed at low-to-moderate intensity and sufficiently before bedtime. Evening exercise does not appear to systematically impair sleep, particularly when performed at low-to-moderate intensity and with sufficient time before bedtime. However, high-intensity exercise performed close to sleep onset may increase the likelihood of sleep fragmentation, indicating that recovery time is a critical determinant of sleep responses. In this context, the interval between exercise cessation and sleep onset may be more relevant than clock time alone. These findings align with a broader shift toward individualized approaches to behavioral health. The emerging concept of chrono-exercise emphasizes the alignment between exercise timing, circadian phase, and individual constraints [9,56]. Within this perspective, optimizing exercise timing involves balancing physiological load, recovery processes, and feasibility, rather than adhering to fixed temporal prescriptions.

4.2. Methodological considerations

The present findings should be interpreted considering several methodological limitations. Although restricting the meta-analysis to RCTs strengthens causal inference, it reduces statistical power for several outcomes. Furthermore, some outcomes, including sleep architecture and subjective sleepiness, could not be meta-analyzed due to the limited number of available studies. Accordingly, non-significant findings should not be interpreted as evidence of absence of effect. Second, substantial heterogeneity exists across studies in exercise protocols, timing definitions, and sleep assessment methods. Exercise timing was defined using both clock time and relative circadian markers, and sleep window was not consistently standardized. Timing was often reported using broad intervals, such as 6:00–10:00 a.m. and 3:00–7:00 p.m. [65], "30 min to 2 h after waking" and "2 h to 30 min before sleep" [53], or "between wake time and 12:00 p.m." and "between 6:00 p.m. and bedtime" [58]. This reporting heterogeneity prevented meaningful analyses of exercise proximity to sleep and limited our ability to determine whether narrower timing windows produce distinct sleep responses. In addition, early-morning sessions (e.g., 6:00–7:00 a.m.) may carry sleep deprivation risk for individuals who are not extreme morning chronotypes, an effect that studies scheduling exercise at 8:00 a.m. or later would not share. Further, although some studies controlled sleep windows, others, particularly ecological protocols, allowed participants to adjust their sleep timing. Such variability may lead to uncontrolled interactions between exercise timing and sleep opportunity, thereby contributing to heterogeneity in observed effects. Third, sleep was assessed using several methods including polysomnography, accelerometry, and self-reported measures. Although standardized mean differences allow pooling across methods, differences in measurement sensitivity may obscure outcome-specific effects. Fourth, key individual characteristics were insufficiently assessed. Primarily, chronotype was rarely reported, making it a major limitation, and extreme chronotypes were often excluded, thereby limiting the evaluation of circadian phenotype as a moderator. Previous work suggests that the effects of exercise timing on sleep parameters are unlikely to follow a one-size-fits-all pattern and may vary according to individual circadian characteristics [76,89]. Morning-type, for instance, were reported to experience poorer sleep-quality compared to evening-type after the evening exercise, whereas no significant changes in the sleep quality of the two chronotypes after the morning exercise was observed [50]. However, such interindividual variability could not be meaningfully addressed in the present study given the available evidence base. Moreover, exercise timing may influence circadian phase in a chronotype-dependent manner [56,64]. While morning exercise generally induces phase advances, reflected by earlier bedtimes and mid-sleep times, evening exercise tends to delay these markers; therefore, late chronotypes, who are particularly prone to circadian misalignment, may benefit from appropriately timed exercise, whereas evening exercise may worsen circadian misalignment in early chronotypes. Taken together, these observations support the potential value of chronotype-dependent exercise timing prescriptions. Similarly, sex-specific effects could not be examined. The absence of these variables, together with the heterogeneity of the included populations, particularly the classification of athletes as a healthy population despite well-documented alterations in their sleep [90], may partly explain the small average effects observed, as pooling heterogeneous individuals can obscure meaningful interindividual variability. Finally, only a minority of studies objectively monitored physical activity outside prescribed exercise sessions, leaving residual confounding from daily movement behaviors and environmental factors (e.g., light exposure).

4.3. Future directions

Future research should move beyond simple comparisons of morning versus evening exercise and instead examine how timing interacts with exercise intensity, duration, and proximity to bedtime. A priority will be the integration of circadian phenotype into study designs. Assessing chronotype and circadian phase markers will allow evaluation of whether alignment between exercise timing and individual biological rhythms influences sleep responses. In addition, improved standardization of reporting is needed, including precise definitions of exercise timing, explicit characterization of sleep opportunity, and consistent reporting of the interval between exercise cessation and sleep onset. Future studies should also incorporate both objective and subjective sleep measures and monitor daily movement behaviors to better isolate the specific effects of exercise timing. Importantly, while sleep duration, continuity, efficiency, and quality were quantitatively extracted and analyzed, the limited availability of data regarding sleep timing, regularity, sleep architecture precluded meaningful synthesis of these outcomes and limited broader characterization of the effects of exercise timing on overall sleep health dimensions. Therefore, to better characterize the effects of exercise timing on sleep health, future studies should

Practice points

- Exercise timing can remain flexible, provided that regular exercise is maintained, although, evening/nighttime moderate-to-vigorous exercise may compromise sleep continuity.
- Evening/nighttime exercise was associated with slightly higher WASO compared with morning exercise, with differences more apparent at higher exercise intensities.
- The effects of exercise timing depend on exercise intensity, proximity to sleep, and recovery dynamics.

Research agenda

- Move beyond timing comparisons: Examine how exercise timing interacts with intensity, duration, circadian phase, and sleep context.
- Standardize key variables: Clearly define exercise timing, sleep opportunity, and the interval between exercise and sleep onset.
- Integrate circadian phenotype: Systematically assess chronotype.
- Examine dose–timing interactions: Investigate combined effects of intensity, duration, and timing within unified models. Greater standardization and precision in reporting exercise timing are also needed to enable robust analyses of exercise proximity to bedtime and its potential influence on sleep.
- Prioritize longitudinal designs: Determine whether timing-related effects persist or attenuate with training adaptation.
- Improve measurement: Combine objective and subjective sleep assessments and monitor 24-h movement behaviors, including light exposure and feeding patterns as additional assessments.

adopt a multidimensional approach by assessing effects on sleep regularity, sleep timing, sleep architecture, social jetlag, and daytime alertness. For instance, young athletes have been shown to exhibit greater night-to-night sleep variability than age-matched non-athletes. However, whether this variability is primarily attributable to training schedules, exercise timing, or other sport-related factors remains to be determined [91]. Furthermore, sociocultural and lifestyle factors represent another source of heterogeneity, as work schedules, social obligations, and environmental influences are known to shape sleep–wake patterns, sleep regularity, social jetlag, and overall sleep health [92–95]; although direct evidence remains limited, these factors may also plausibly moderate the relationship between exercise timing and sleep [96], and should therefore be systematically considered in future research. Finally, analytical approaches that capture interindividual variability, including individual participant data meta-analyses and models of response heterogeneity, will be essential to identify subgroups for whom exercise timing meaningfully influences sleep. Such approaches may help move the field toward more personalized exercise timing recommendations that account for individual differences in sleep responses.

5. Conclusion

Exercise timing exerts modest effects on sleep that are contingent on exercise and individuals' characteristics. Morning exercise does not appear systematically superior to evening/nighttime exercise for most sleep outcomes. Rather, timing-related effects seem conditional and more apparent under specific circumstances, particularly at higher exercise intensities. The observed increase in WASO following evening/nighttime moderate-to-vigorous exercise suggests a potential impact on sleep maintenance, though not sleep duration. These findings indicate that exercise timing should not be considered in isolation, but as part of a broader interaction between physiological activation, recovery processes, and behavioral constraints. Given the broader benefits of regular exercise for overall health [97], taken together, the current findings suggest that recommendations should avoid being overly restrictive regarding evening exercise. Rather, they should encourage regular physical activity while considering individual preferences, exercise characteristics, and sufficient recovery time before sleep.

Contributions

OS conceived and designed the study and its methodology. GDS, PP, and OS performed study screening. MSA, MJS, MS and OS conducted data extraction and quality assessment. MS, OS, MG, and BMG drafted the manuscript. DD and MPS critically reviewed the manuscript. OS revised the manuscript. All authors approved the final version.

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Appendix A. Supplementary data

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