Sleep Medicine Reviews 77 (2024) 101969

Contents lists available at ScienceDirect

Sleep Medicine Reviews

journal homepage: www.elsevier.com/locate/smrv

Sleep quality in eating disorders: A systematic review and meta-analysis

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ARTICLE INFO

Handling Editor: M Vitiello

Keywords:
Eating disorders
Anorexia nervosa
Bulimia nervosa
Binge eating disorder
Sleep
Sleep disorders
Circadian rhythms

ABSTRACT

Eating disorders (ED) are psychological disorders characterized by dangerous eating behaviours, including protracted fasting and binge eating. Mental disorders comorbidities (e.g., anxiety and depression), as well as sleep difficulties, are common and might interfere with treatment response. This work investigated sleep quality, circadian preferences, and sleep disorders in ED patients compared to healthy controls (HC) and the impact of ED treatment on patients' sleep.

A literature search on Pubmed, Web of Science, Medline, and PsychInfo included 27 studies. Random effect analyses were performed (sample eating disorders = 711; sample healthy controls = 653) and subgroup analyses were calculated based on the ED subgroups: Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder. Whole sample analyses showed poorer physiological and subjective sleep quality in patients. Subgroup analyses showed that poorer physiological sleep was present only in anorexia nervosa. Two studies reporting circadian preferences and sleep disorders showed higher evening preference in patients and no differences in apnea prevalence between patients and healthy controls, respectively. Some studies suggested that specialized eating disorder treatments (e.g., Cognitive Behavioural Therapy for ED) can improve sleep quality in patients.

Although these findings highlight poorer sleep in patients with ED compared to healthy controls, the mechanisms underlying sleep alterations in eating disorders remain to be identified.

AN: anorexia nervosa
BED: binge eating disorder
BMI: body mass index
BN: bulimia nervosa
DSM-5: Diagnostic and statistical manual of mental disorders fifth edition
ED: eating disorder
MEQ: morningness-eveningness questionnaire
N1: sleep stage 1
N2: sleep stage 2
PICOs: population, intervention, comparison/outcome and study design
PRISMA: preferred reporting items for systematic reviews and meta-analyses
PSG: polysomnography
PSQI: pittsburgh sleep quality index
RE: random effect
REM: rapid eye movement sleep
REMD: rapid eye movement sleep density
REML: rapid eye movement sleep latency
RoB: risk of bias
SE: sleep efficiency
SMD: Standardized mean difference

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(*continued*)

SOL: sleep onset latency
SPT: sleep period time
SWS: slow wave sleep
TST: total sleep time
WASO: wake after sleep onset

1. Introduction

Sleep has attracted the attention of scientists for decades and has a fundamental role in human well-being. Good sleep quality can be defined as the individual's satisfaction with all aspects of the sleep process, including the ability to fall asleep, the timing, and the duration. It is characterised by feelings of rest and restoration following a night's sleep. Good sleep quality sustains a multitude of processes, including cognitive functioning, emotion regulation [1,2], and immune system

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https://doi.org/10.1016/j.smrv.2024.101969
Received 20 February 2024; Received in revised form 4 June 2024; Accepted 12 June 2024
Available online 18 June 2024

functions [3]. Therefore, it is not surprising that clinical research documents relevant impairments in sleep quality among clinical populations. While sleep impairments are well established in some conditions, such as depression, bipolar disorder, and anxiety disorders [4,5], the role played by sleep difficulties in eating disorders is less clear [6].

Eating disorders are mental disorders primarily affecting young female adolescents (male to female ratio between 0.3 and 0.6) [7]. Patients use unhealthy eating behaviours, including extended fasting periods or loss of control over eating, and tend to attribute excessive importance to the control of weight and body shape for self-evaluation [8]. According to the Diagnostic and statistical manual of mental disorders fifth edition (DSM-5; [8]), eating disorders include anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED). AN is characterized by an extreme reduction of caloric intake which leads to severe underweight. Patients with AN might also report binge-eating episodes (i.e., consuming an amount of food that is significantly larger than what most people would consume within the same period of time, accompanied by a sense of lack of control over eating during the episode) followed by purging behaviours, such behaviours include self-induced vomiting or the misuse of laxatives, diuretics, or enemas. Patients with BN report binge-eating episodes and purging behaviours and their weight tend to be within the normal or elevated range [9]. BED is characterized by binge-eating episodes in the absence of purging behaviours and patients' body mass index (BMI) is often in the overweight or obese range.

Sleep and eating patterns are highly related [10]. Food restriction, i.e., restriction of quantity and quality of foods that a person would need to keep healthy, and hunger can negatively affect sleep quality. Intense physical distress might cause several nocturnal awakenings and lead to a scarcely restful sleep. One neuropeptide, orexin, could have a role in the relationship between sleep and dysfunctional eating pattern. The activation of orexin-producing neurons in the lateral hypothalamus has been observed in fasting, thus indicating a potential involvement in the promotion of food-seeking behaviour [11]. Moreover, a recent study suggested that orexin should be considered primarily as an inhibitor of REM and, to a lesser extent, as a wake promoter [12]. Under normal physiological circumstances, REM sleep tends to be most prevalent during the latter part of the primary nighttime sleep cycle. Given that nocturnal sleep extends for several hours, it coincides with a physiological fasting period, with the substantial occurrence of REM sleep towards the final hours of the night serving as a natural appetite suppressant [13]. As a result, REM sleep could potentially serve an anti-obesity role [14]. At the same time, a reduction of REM sleep due to increased orexin, particularly in short sleepers, could promote appetite and contribute to weight gain. In addition, given that food choices are often influenced by emotional factors, and since REM sleep has been associated with emotional processing and regulation, some authors speculated that REM sleep may influence the formation of food preferences and aversions (see Ref. [13]).

Binge-eating episodes could also impair sleep quality. As they frequently occur in the evening [15], the laborious digestive process could interfere with falling asleep. On the cognitive level, rumination, frequently reported by patients with binge-eating episodes [16], may be associated with sleep difficulties. As rumination is associated with heightened arousal, it may cause a state of agitation that prevents the individuals both from falling asleep and maintaining sleep.

Therefore, it seems plausible that some of the core aspects of the eating disorder psychopathology may be associated with altered sleep patterns. Some findings indicated sleep impairments in eating disorders [17–19] or no differences between patients and healthy controls [20,21]. One recent meta-analysis on sleep difficulties in clinical samples included only five published manuscripts on eating disorders, all of which focused on anorexia nervosa [4], therefore an update is needed.

The overall goal of this study was to conduct a systematic review and meta-analysis of studies assessing sleep in patients with eating disorders. In particular, differences between patients with an eating disorder and healthy controls were summarised with reference to: 1) sleep parameters and circadian preferences assessed using i) physiological and ii) self-report measures; 2) changes in sleep quality and circadian preferences following eating disorders treatment; 3) the extent of sleep disorders in eating disorder patients.

2. Method

2.1. Search strategy and study selection

This meta-analysis was conducted in accordance with the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines [22]. The protocol for the present work was pre-registered on PROSPERO (ID: CRD42022350845).

Study eligibility was assessed using the population, intervention, comparison/outcome, and study design (PICOs) approach and the inclusion criteria were:

1. **P**articipants: Inclusion of a sample of participants with a diagnosis of eating disorders (AN, BN, or BED), as assessed by clinicians based on standardized criteria;
2. **I**ntervention: Assessment of subjective and/or physiological sleep parameters and/or circadian preference;
3. **C**ontrol: Inclusion of a healthy control group, defined as individuals with no self-reported diagnoses of psychological or neurological diseases;
4. **O**utcome: Physiological sleep parameters were measured using polysomnography (PSG) or actigraphy. When both measurements were available, PSG data were preferred. Subjective data were collected through diaries or questionnaires and diaries were preferred over questionnaires;
5. **S**tudy Design**:** Case-control studies;
6. *Further criteria*: Manuscript written in English, Italian, German, or Spanish language.

The exclusion criteria were:

1. Narrative reviews, meta-analysis, or case reports;
2. Overlap of samples across studies: for each outcome, in cases in which the same sample was used across different studies, we selected the study with a larger sample size. If the studies had the same number of subjects, the most recent one was included.

A literature search was conducted in December 2023 using the following databases: Web of Science, Pubmed, PsycINFO, and Medline without any data publication constraint. The following keywords - adapted for each database - were used:

"eating disorders" OR "anorexia nervosa" OR "bulimia nervosa" OR "binge eating disorder" OR "disordered eating" OR "binge eating" AND "sleep" OR "insomnia" OR "circadian rhythms" OR "circadian preference" OR "Pittsburgh Sleep Quality Index" OR "PSQI" OR "Morningness–Eveningness questionnaire" OR "MEQ" OR "stage 1″ OR "stage 2" OR "stage 3" OR "slow wave sleep" OR "REM" OR "apnea" OR "narcolepsy" OR "parasomnias" OR "hypersomnia" OR "TST" OR "WASO" OR "SE" OR "SOL".

The results of the search were uploaded on CITAVI 6 software (https://www.citavi.com). Two authors (G.D. and D.M.) independently screened the items by title and abstract and duplicates were removed. The full texts of the included studies and of those studies that raters were unsure to include were assessed. References of included papers were checked for additional published manuscripts. Disagreements were solved through discussion and reasons for exclusion were recorded. When full texts were not available, corresponding authors were contacted a maximum of two times, one week apart; in case of no response after three weeks from the first e-mail, the item was marked as

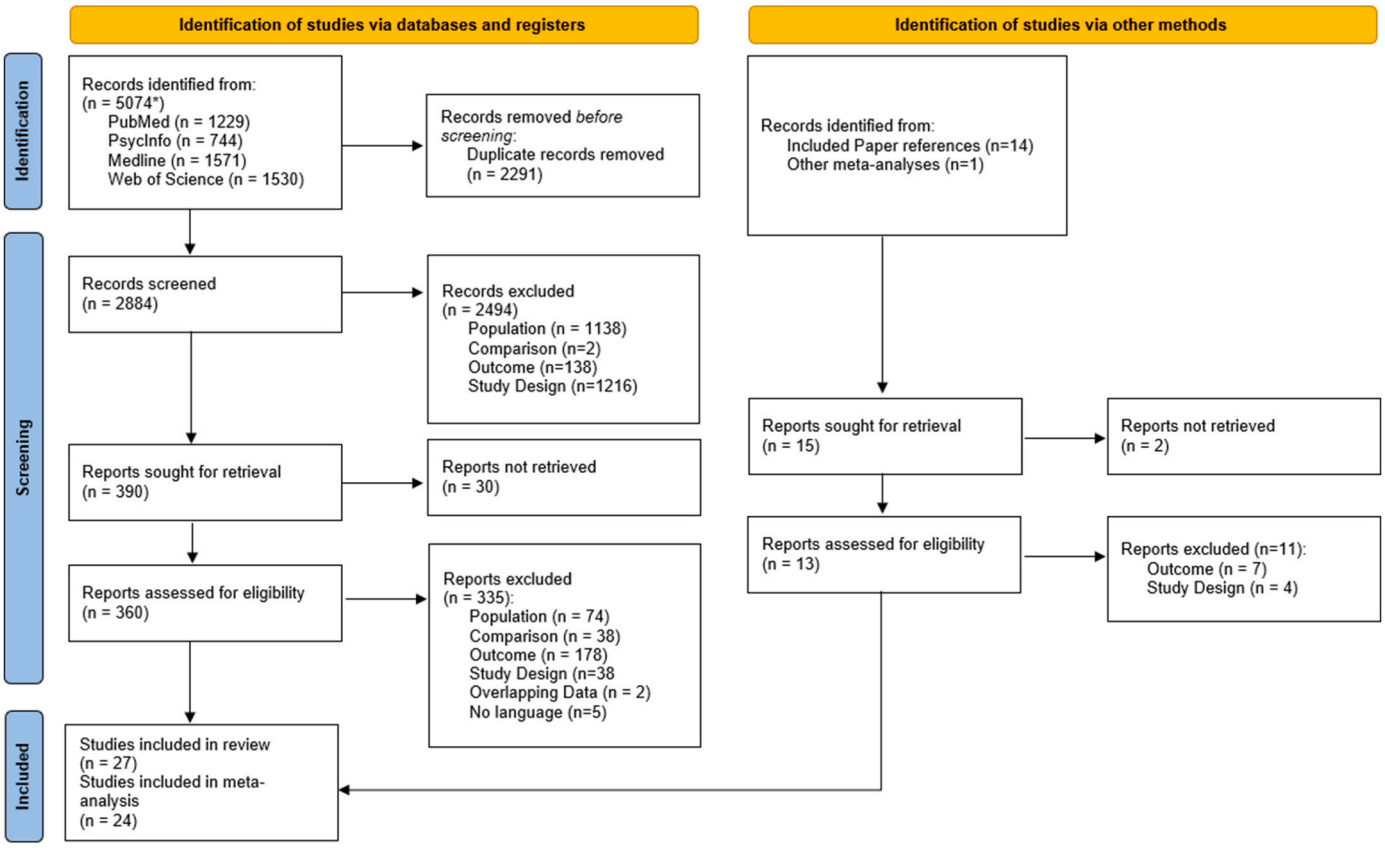

Fig. 1. Flow chart of the study screening process. PRISMA 2020 flow diagram including searches of databases, registers, and other sources. * 101 records had been automatically added to the total by Citavi. This is not an error, but a difference in how Citavi stores related references. Citavi differentiates between parent and child references and divides them into different reference types. For example, when Citavi imports a citation related to a contribution in an Edited book. Therefore, the number of records screened is slightly higher than the one found through the Databases (https://www1.citavi.com/sub/manual5/en/importing_from_other_reference_management_programs.html).

unavailable and excluded. For all included studies, the risk of bias (RoB) was assessed using section A of the critical appraisal skills programme tool for case-control studies (https://casp-uk.net/casp-tools-checklists/). We followed a decision-by-consensus procedure, and in the event of disagreement between the two raters, a discussion was held with all authors.

2.2. Outcome definition

Based on a previous meta-analysis [4], the physiological outcomes (i.e. measured by actigraphy or PSG) were grouped into three macro outcomes:

1) "Sleep continuity":
   - Sleep efficiency index (%, SE): the ratio of the time spent asleep (TST) to the time in bed x 100 or the time from falling asleep to time of awakening (SPT);
   - Sleep onset latency (min, SOL): time spent to fall asleep;
   - Total sleep time (min, TST): the total time spent asleep during the recording night or SPT;
   - Wake after sleep onset (min, WASO): the duration of wake during the night.
2) "Sleep depth":
   - Stage 1 (%, N1): the duration of N1 reported as the percentage of SPT or TST;
   - Stage 2 (%, N2): the duration of N2 reported as the percentage of SPT or TST;
   - Slow wave sleep (%, SWS): the duration of N3 or the sum of the duration of stages 3 and 4 reported as the percentage of SPT or TST.
3) "REM pressure":
   - Rapid eye movement sleep (%, REM): the duration of REM reported as the percentage of SPT or TST;
   - REM latency (REML): the interval between sleep onset and the onset of the first REM;
   - REM density (REMD): the frequency of rapid eye movements during REM sleep.

For sleep stages (N1, N2, N3, REM) only papers reporting data in percentage were considered.

The total scores of the Pittsburgh sleep quality index (PSQI) [23], the Morningness-eveningness questionnaire (MEQ) [24], the MEQ reduced version (MEQ-r) [25], and sleep diaries were used to obtain self-assessment data of sleep quality and circadian preference.

2.3. Data extraction

Two authors (G.D. and D.M.) extracted descriptive information, including age, gender, BMI, diagnosis, and comorbidities. For all sleep variables, means and standard deviations were extracted. Doubts about data extraction were resolved through discussion. If a paper did not include sufficient data, further information was requested from the authors. Two e-mails were sent one week apart. After three weeks after the first e-mail, if the author did not reply, the article was considered excluded for lack of information. CITAVI 6 software (https://www.citavi.com) was employed for the bibliography categorization.

Table 1
Characteristics of included studies: patients vs healthy controls.

Study	Eating disorders patients					Healthy controls				Outcome measure	Country
	N	%F	Age (sd)	BMI	diagnosis	N	%F	Age (sd)	BMI		
Asaad Abdou et al., 2018 [19]	23	100 %	30 (6)	>15	AN & BN	20	–	–	–	PSG	Egypt
Antunes et al. , 2020 [54]	13	100 %	26.42 (4.27)	33.15	BED	14	100 %	32.17 (7.85)	21.33	PSQI	Brazil
Burbiel et al., 1991 [55]	9	–	–	–	AN	41	–	–	–	PSG	Germany
De la Torre Luque et al., 2013 [32]	13	100 %	23.38 (7.50)	22.46	AN & BN & not specified	15	100 %	31.98 (10.31)	21.9	PSQI, sleep diary	Spain
Della Marca et al., 2004 [18] (1)	6	100 %	20.67 (1.86)	16.17	AN	6	–	21.67 (1.75)	20.77	PSG	Italy
Della Marca et al., 2004 [18] (2)	6	100 %	21 (2.45)	20.75	BN	6	–	21.67 (1.75)	20.77	PSG	Italy
Delvenne et al., 1992 [56]	11	100 %	18 (3)	–	AN	11	100 %	18 (3)	–	PSG	Belgium
El Ghoch et al., 2016 [34]	50	100 %	24.6 (8.9)	14.5	AN	25	100 %	26.3 (10.6)	21.5	SWA	Italy
Hudson et al., 1987 [57]	11	100 %	25.2 (5.6)	–	BN	20	100 %	28.4 (5.6)	–	PSG	USA
Kleppe et al., 2023 [29]	20	100 %	19.5 (8.0)[a]	16.7[a]	AN	23	100 %	19.00 (7.0)[a]	22.9[a]	ACT	Norway
Latzer et al., 1999 [58]	25	100 %	22.2 (8.2)	21.7	BN	21	–	24 (8.1)	20.3	ACT	Israel
Latzer et al., 2001 [59]	20	100 %	18.7 (3.5)	16.8	AN	16	–	19.4 (3.9)	20	ACT	Israel
Lauer et al., 1988 [60] (1)*	20	95 %	21 (3.1)	–	AN	10	–	23.1 (3.1)	–	PSG	Germany
Lauer et al., 1988 [60] (2)*	10	100 %	23.2 (3.1)	–	BN	10	–	23.1 (3.1)	–	PSG	Germany
Lauer et al., 1989 [61] (1)	20	95 %	21 (3.1)	–	AN	10	90 %	23.1 (3.1)	–	PSG	Germany
Lauer et al., 1989 [61] (2)	10	100 %	23.2 (3.1)	–	BN	10	–	23.1 (3.1)	–	PSG	Germany
Lehmann et al., 2018 [21]	50	100 %	25 (-)	14.4	AN	30	100 %	26 (-)	21.3	SWA	Germany
Levy et al., 1988 [17] (1)	9	100 %	27.4 (8.8)	–	AN	10	–	23.2 (2.4)	–	PSG	USA
Levy et al., 1988 [17] (2)	9	100 %	25.3 (2.4)	–	BN	10	–	23.2 (2.4)	–	PSG	USA
Lindberg et al., 2003 [36]	11	100 %	19.7 (1.1)[b]	13.3	AN	11	–	20.9 (0.8)[b]	21.4	ACT & PSG	Finland
Natale et al., 2008 [33] (1)	41	100 %	31.23 (11.34)	–	AN	124	100 %	31.98 (10.31)	–	MEQ-r	Italy
Natale et al., 2008 [33] (2)	48	100 %	31.23 (11.34)	–	BN	124	100 %	31.98 (10.31)	–	MEQ-r	Italy
Natale et al., 2008 [33] (3)	57	100 %	31.23 (11.34)	–	BED	124	100 %	–	–	MEQ-r	Italy
Neil et al., 1980 [62] (1)	10	–	–	–	AN (normal EEG)	10	–	–	–	PSG	USA
Neil et al., 1980 [62] (2)	7	–	–	–	AN (abnormal EEG)	10	–	14 (2)	–	PSG	USA
Nobili et al., 1999* [63]	10	100 %	14 (2)	–	AN	10	–	14 (2)	102	PSG	Italy
Nobili et al., 2004 [39]	20	100 %	13.9 (2)	–	AN	12	–	23.3 (6.53)	–	PSG	Italy
Romigi et al., 2022 [37]	34	100 %	23.3 (6.53)	16.5	AN	34	100 %	60 (12.4)	20.98	PSQI	Italy
Roveda et al., 2018 [31]	8	100 %	55.7 (15.6)	31.3	BED	8	100 %	–	31.6	ACT	Italy
Sauchelli et al., 2016 [38]	48	100 %	–	18.5	AN	98	100 %	26.6 (7.6)	–	PSQI	Spain
Tu et al., 2019 [30]	41	95.1 %	26.1 (7.3)	21.2	BN	31	90.30 %	29.3 (5.6)	22.1	PSQI	Taiwan
Waller et al., 1989 [64]	11	100 %	25.6 (7.3)	–	BN	19	60 %	26.6 (5.0)	–	PSG	USA
Walsh et al., 1985 (1) [65]	8	–	26.9 (6.9)	–	AN	14	100 %	26.6 (5.0)	–	PSG	USA
Walsh et al., 1985 (2) [65]	14	–	26.1 (5.9)	–	BN	14	–	31.98 (10.31)	–	PSG	USA

Notes: ACT: Actigraphy; AN: Anorexia Nervosa; BED: Binge Eating Disorder; BMI: Body Mass Index; BN: Bulimia Nervosa; MEQ-r: Morningness-Eveningness Questionnaire – reduced version; NR: not reported; PSG: Polysomnography; PSQI: Pittsburgh Sleep Quality Index; SD: standard deviation; SWA: Sense Wear Armband. - no data available.
*Only considered for one sleep parameter.
[a] Median (interquartile range).
[b] Standard Error.

2.4. Statistical analyses

Analyses were performed by a professional statistician (S.C.) using SAS Software version 9.4 with SAS/STAT version 14.1 (SAS Institute Inc., Cary, NC, USA), and the meta-analytical calculations for sleep domains were performed using the statistical software package R "meta" (http://www.r-project.org; version: 4.1.1). Differences in sleep outcomes between patients with eating disorders and healthy controls were calculated using the Standardized mean difference (SMD) as a measure of effect size. The Random effects (RE) model [26] was used to estimate the average effect sizes. Using the RE Model for each variable, a separate meta-analysis (measure with at least 3 studies available) was conducted to calculate the size of effects as standardized mean difference (Cohen's d) using exact formulae [27]. For each diagnosis and each variable, a separate meta-analysis was conducted if at least three studies were available for the calculation of the effect size. Each study could then contribute multiple times to the same domain, based on the number of variables reported in the domain. Analyses were first performed including the whole sample of patients with eating disorders and subsequently specifically for patients with AN, BN, or BED when data were available. Heterogeneity was tested using Chi-square tests and I^2 metrics. Heterogeneity was noted when the test showed a p-value <0.20 and an $I^2 \geq 50$ %. Sensitivity analyses were conducted when a paper or a parameter in a paper was a specific source of heterogeneity. Potential publication bias was assessed by plotting the effect size against standard error (funnel plot) and then by graphical inspection for asymmetry, as publication bias may lead to asymmetrical funnel plots.

3. Results

3.1. Study selection

The literature search yielded 5074* articles, from which 2291 duplicates were removed. 2494 items were excluded based on abstract information and 30 articles were not retrieved. 360 full-texts were examined and 25 were included. By screening the references of the included papers and other meta-analyses, 15 papers were found of which 2 were selected. A total of 27 studies were included in the meta-

Table 2
Characteristics of included studies: pre-vs post-treatment.

Study	N	Drop-out rate	%F	Age (sd)	ΔBMI	diagnosis	Outcome	Country
El Ghoch et al., 2016 [34]	50	0.16	100 %	24.6 (8.9)	4.3	AN	SWA	Italy
Lauer & Krieg, 1992 [35]	10	0	100 %	20.9	–	AN	PSG	Germany
Lindberg et al., 2003 [36]	5	0	100 %	21.2 (1.1)	2.2	AN	ACT & PSG	Finland

Notes: AN: Anorexia Nervosa; ΔBMI: Body Mass Index increase from pre-to post-treatment; SD: standard deviation; SWA: Sense Wear Armband; - no data available.

Table 3
Metanalytic elaborations of single outcomes for the whole sample.

Single Outcome	k	ES	95 % CI	SE	z	p-value	τ^2	I^2	H	Q	Q p-value
SE	20	−0.78	[-1.1914; −0.3658]	0.41	−3.70	<0.0001	0.68	76 %	2.06	80.43	<0.0001
TST	18	−0.38	[-0.5682; −0.1893]	0.19	−3.92	<0.0001	0.03	28 %	1.18	23.55	0.1323
SOL	13	0.13	[-0.2620; 0.5260]	0.39	0.66	0.512	0.36	71 %	1.86	41.68	<0.0001
WASO	12	0.38	[-0.0220; 0.7799]	0.40	1.85	0,064	0.35	70 %	1.83	36.89	0.0001
N1 %	12	0.55	[-0.0908; 1.1798]	0.64	1.68	0.093	1.05	82 %	2.33	59.53	<0.0001
N2 %	14	0.26	[-0.1918; 0.7130]	0.45	1.13	0.259	0.55	74 %	1.97	50.43	<0.0001
SWS %	15	−0.44	[-0.8558; −0.0157]	0.42	−2.03	0.042	0.49	73 %	1.93	52.41	<0.0001
REM %	15	−0.47	[-0.9645; 0.0247]	0.50	−1.86	0.063	0.75	79 %	2.17	66.20	<0.0001
REM L	16	−0.10	[-0.4310; 0.2276]	0.33	−0.61	0.545	0.26	59 %	1.55	36.13	<0.002
REM D	10	−0.17	[-0.5852; 0.2409]	0.41	−0.82	0.414	0.27	61 %	1.60	22.99	<0.0100
PSQI	4	0.95	[0.5244; 1.3716]	0.42	4.4	<0.0001	0.11	66 %	1.71	8.74	0.0329
WASO[a]	11	0.49	[0.1552; 0.8259]	0.34	2.87	0.004	0.17	52 %	1.45	21.00	0.0211

Notes.
[a] Sensitivity analysis; N1%: Sleep Stage 1 (%); N2%: Sleep Stage 2 (%); PSQI: Pittsburgh Sleep Quality Index; REM%: Rapid Eye Movement; REML: REM latency; REMD: REM Density; SE: Sleep Efficiency; SWS%: Slow Wave Sleep (%); SOL: Sleep Onset Latency; TST: Total Sleep Time; WASO: Wake After Sleep Onset.

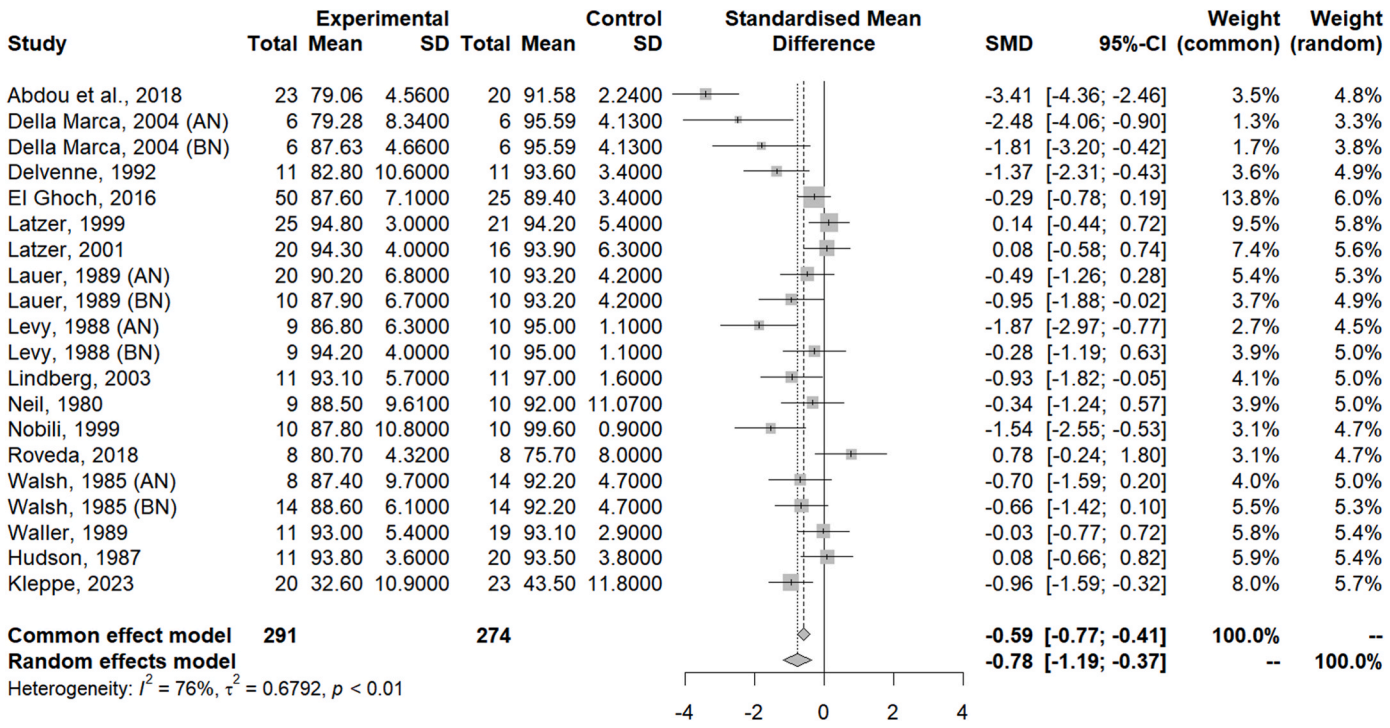

Fig. 2. Forest plot of SE of the whole sample. SD: standard deviation; SMD: standardised mean difference.

analysis [28] (Fig. 1).

* 101 records had been automatically added to the total by Citavi. This is not an error, but a difference in how Citavi stores related references. Citavi differentiates between parent and child references and divides them into different reference types. For example, when Citavi imports a citation related to a contribution in an Edited book. Therefore, the number of records screened is slightly higher than the one found through the Databases. (https://www1.citavi.com/sub/manual5/en/importing_from_other_reference_management_programs.html)

3.2. Studies characteristics

The eligible studies involved young female patients with AN mostly, while BED was particularly underrepresented. Most studies had a sample size including less than 20 participants and employed PSG or actigraphy. Subjective measures were less often used, especially the MEQ-r (N = 1).

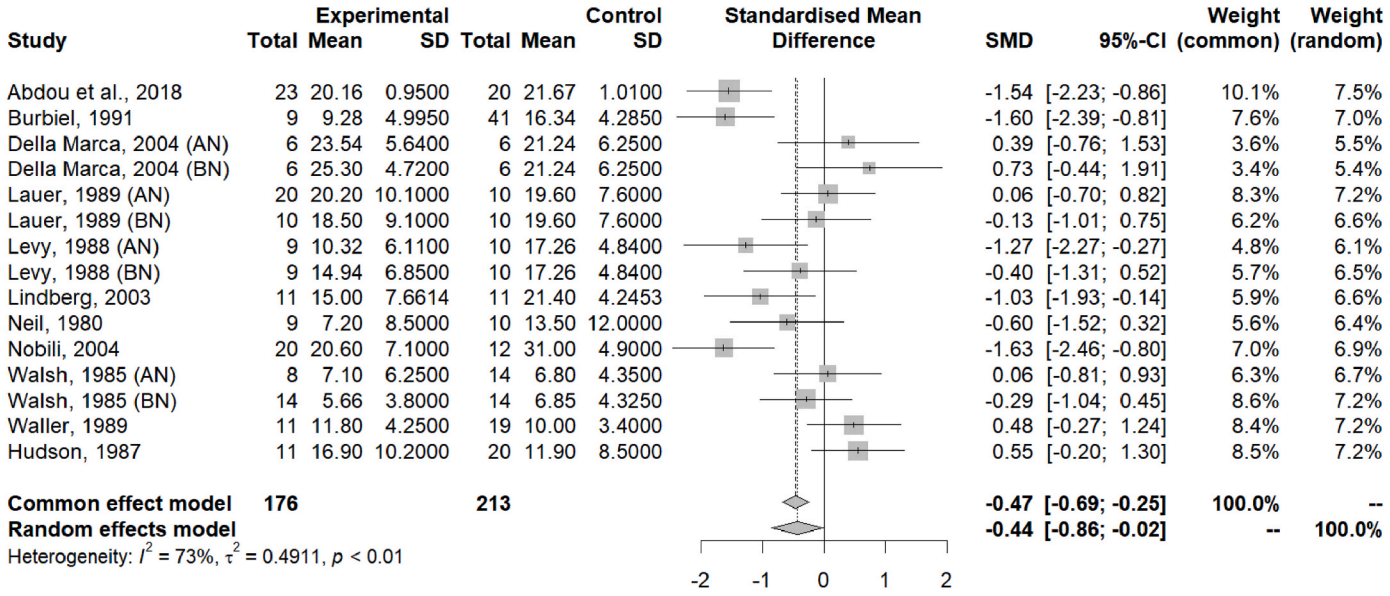

Fig. 3. Forest plot of SWS of the whole sample. SD: standard deviation; SMD: standardised mean difference.

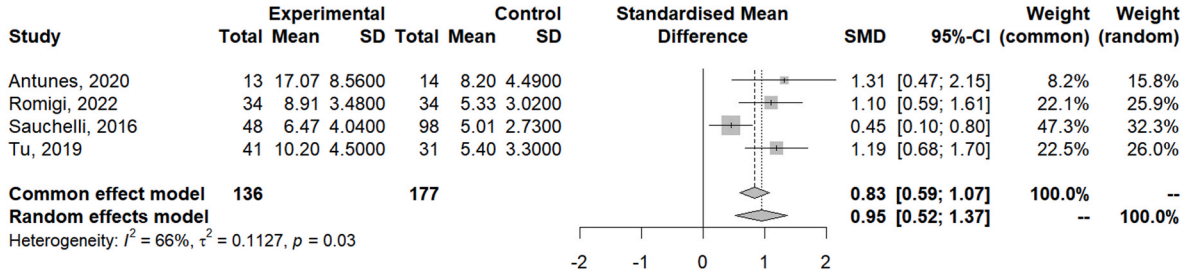

Fig. 4. Forest plot of PSQI of the whole sample. SD: standard deviation; SMD: standardised mean difference.

No studies assessed symptoms of insomnia. A more detailed summary of the characteristics of the studies (N = 26) with differences between samples is shown in Table 1. The characteristics of the studies comparing pre- and post-treatment variables are reported in Table 2 (N = 3).

3.3. Risk of bias

All studies were assessed as at a low RoB for questions related to the clarity of the aims and the validity of the methods. Some uncertainty was found in the selection of the control group, while a high RoB was found due to possible confounding variables, such as education or socio-economic background. Further information is shown in Figs. S1 and S2 and the supplementary file ("Supplementary_RoB").

3.4. Physiological sleep parameters: eating disorders vs. healthy controls

For the whole sample (AN, BN, and BED), the summary of the meta-analytic elaboration of the single parameters is reported in Table 3. SE, TST, and SWS were significantly lower in the eating disorders group compared to the healthy control group. Effect sizes were larger for SE (d = −0.78, p < 0.001, I^2 = 76 %, Fig. 2) and medium for TST (d = −0.38, p < 0.001, I^2 = 28 %) and SWS (d = −0.44, p < 0.001, I^2 = 73 %, Fig. 3). The eating disorder group also reported poorer sleep quality, as assessed by the PSQI, with a large effect size (d = 0.95, p < 0.001, I^2 = 66 %, Fig. 4). The other parameters did not show any significant between-group difference (p > 0.05).

For WASO, sensitivity analyses were conducted excluding the work of Kleppe and colleagues (2023) [29] since, as also reported by Kleppe and colleagues, the automatic algorithm they used to score actigraphy-derived WASO might have been unreliable, as the accelerometer may mistake normal movement for wake periods. This could also explain why the direction of the effect is completely the opposite compared to the other studies, as shown in Fig. S11. Our analyses showed a significantly increased duration of WASO in patients compared to controls (d = 0.49, p < 0.005, I^2 = 52 %).

Table 4
Metanalytic elaborations on macro outcome of the whole sample.

Macro Variable	Single Outcome	Abs (ES)	SE	ES	SE
Sleep continuity	SE	0.78	0.41	0.42	0.35
	TST	0.38	0.19		
	SOL	0.13	0.39		
	WASO	0.38	0.40		
Sleep depth	N1%	0.545	0.635	0.41	0.50
	N2%	0.261	0.452		
	SWS%	0.436	0.420		
REM pressure	REM%	0.470	0.495	0.25	0.41
	REM L	0.102	0.329		
	REM D	0.172	0.413		

Notes. N1%: Sleep Stage 1 (%); N2%: Sleep Stage 2 (%); REM%: Rapid Eye Movement; REML: REM latency; REMD: REM Density; SE: Sleep Efficiency; SWS %: Slow Wave Sleep (%); SOL: Sleep Onset Latency; TST: Total Sleep Time; WASO: Wake after Sleep Onset.

The results for the macro variables ("sleep continuity", "sleep depth", and "REM pressure") are reported in Table 4. Larger effect sizes were found for Sleep Continuity (d = 0.42, SE = 0.35) and Sleep Depth (d = 0.41. SE = 0.50) compared to REM Pressure (d = 0.25, SE = 0.41).

Forest plots and funnel plots are reported in supplements

(Figs. S3–S14). The graphical inspection of funnel plots showed an asymmetry for SE and PSQI, suggesting possible publication bias.

3.5. Differences in physiological sleep parameters: subgroup analysis

3.5.1. Anorexia nervosa

As reported in Table S1, patients suffering from AN show a reduced SE (Cohen d' = −0.90, p-value <0.001, I^2 = 61 %), TST (Cohen d' = −0.51, p-value<0.0001, I^2 = 24 %), and SWS (Cohen d' = −0.74, p-value <0.01, I^2 = 72 %) compared to healthy controls. They also showed a nearly significant increase of N1 (Cohen d' = 1.06, p-value = 0.052, I^2 = 86 %). All the other outcomes were not statistically different between groups (all p-values >0.05). As studies related to PSQI were few (N = 2), quantitative analyses were not computed. Sensitivity analyses for WASO, excluding the work of Kleppe and colleagues (2023) [29], showed also an enhanced duration of WASO (Cohen d' = 0.67, p-value <0.01, I^2 = 61 %).

Analyses of macro variables are reported in Table S2. Differences between samples in Sleep Continuity (d = 0.48, SE = 0.48) and Sleep Depth (d = 0.61, SE = 0.67) showed medium effect sizes, while REM Pressure showed a small size (d = 0.29. SE = 0.56).

Forest and funnel plots for the outcomes are reported in the supplementary materials (Figs. S15–S25). Based on the graphical inspection of the funnel plots, SE presented an asymmetry, suggesting a possible publication bias.

3.5.2. Bulimia nervosa

As shown in Table S3, patients affected by BN and healthy controls did not display any significant differences across the outcomes (all p-values>0.05). Only one of the included studies assessed differences in subjective sleep quality [30] and found poorer sleep quality in patients.

As reported in Table S4, the effect size of Sleep Continuity was small (d = 0.23. SE = 0.36), while the other two variables (Sleep Depth and REM Pressure) did not show any differences between groups.

Forest and funnel plots are reported in the supplementary material (Figs. S26–S35).

3.5.3. Binge eating disorder

There was an insufficient number of studies (N = 1) to assess sleep outcomes in patients with BED. The only paper available found no differences between patients and healthy controls [31].

3.6. Narrative findings

The number of published manuscripts on i) self-reported sleep quality and circadian preferences; ii) the effect of the treatment on sleep quality; and iii) sleep disorders in patients with eating disorders was insufficient (<3) for a meta-analysis to be calculated. Therefore, findings were narratively discussed.

3.6.1. Self-reported sleep quality and circadian preference

Only one study assessed sleep quality, such as sleep efficiency and sleep onset latency, through a sleep diary [32]. The results showed no significant differences between patients and healthy controls in the diary parameters, although lower subjective sleep quality in patients with eating disorders tended to be significant (p = 0.06). The researchers also reported differences in some subscales of the PSQI, finding worse sleep quality and greater use of hypnotic drugs in patients with an eating disorder regardless of diagnosis. They also found higher levels of daytime sleepiness in patients.

Only one study [33] assessed circadian preferences and secondary outcomes (i.e., the effect of the treatment on sleep quality). The authors found significantly lower MEQ-r scores in the clinical sample, including patients with AN, BN, or BED, indicating an evening preference. However, no differences between diagnoses emerged. A negative correlation between MEQ-r and perfectionism and ineffectiveness was found. This study also monitored the change in circadian preferences as a function of the change in eating symptomatology over time in a group of patients undergoing cognitive-behavioural therapy. The results showed that a reduction in eating psychopathology over time was associated with an increased MEQ-r, (i.e., participants who reported a clinical improvement had a shift toward a morning chronotype).

3.6.2. Effect of treatment on sleep quality

Only three published manuscripts reported the effect of a treatment focused on eating disorders symptomatology (i.e., Cognitive Behavioural Therapy for Eating Disorders, behavioural therapy, and nutritional therapy) on sleep quality, all of which included patients with AN. El Ghoch and colleagues (2016) [34] found a significant increase in TST after treatment, whereas TST and SOL levels were similar to healthy controls. WASO and SE, which were comparable to healthy controls before treatment, slightly worsened after treatment and became significantly different from healthy controls. In another study, Lauer & Krieg (1992) [35] observed a decrease in WASO and Stage 3 and a slight increase in Stage 4 after treatment, although this was not significant. In a third study [36], a decrease in N1 and N2 and an increase in SWS were reported.

3.6.3. Sleep disorders in patients with an eating disorder

To our knowledge, only one study analysed sleep apnea in patients with an eating disorder (AN or BN). The authors reported no difference in the percentage of sleep apnea between patients and healthy controls [19].

4. Discussion

To the best of our knowledge, the present work is the largest systematic review and meta-analysis to date on the relationship between sleep features and circadian preferences in patients with eating disorders. Results highlighted that patients suffering from an eating disorder present poor and inadequate physiological sleep quality. In particular, they show reduced sleep duration, sleeping on average about 21 min less than controls and experiencing a lighter and more fragmented sleep, with an average of 0.6 % less deep sleep (corresponding to approximately 6 min less) and about 15 min more nocturnal wake (excluding Kleppe and colleagues, 2023 [29]), which inevitably leads to reduced sleep efficiency (on average about 3.6 % less). Although this loss of sleep may seem insignificant, it becomes more important in the long term. In an average month, a patient with an eating disorder sleeps about 630 min less, spends 180 min less in deep sleep, and is awake 450 min longer than the general population. Not surprisingly, these results are consistent with the subjective experience of patients, who complain of poorer sleep quality than healthy controls.

Subgroup analyses showed that the sleep of patients suffering from AN is not only inadequate in duration but also less restorative. Sleep continuity parameters, in particular WASO, clearly show that sleep is highly fragmented and therefore characterized by frequent and prolonged nocturnal awakenings. This is consistent with the finding of a reduced SE, which reduces as WASO increases, in AN compared to healthy individuals. Furthermore, the PSG studies seem to support the idea that patients with AN are characterized by a reduction of deep sleep and an increase in lighter sleep. The quality of sleep perceived by patients with AN is coherent with the picture that emerged from the physiological findings. Indeed, in the two studies using PSQI in AN [37, 38] patients reported poorer sleep quality than healthy controls. Overall, these results might suggest that, on the one hand, the light sleep that characterizes this subgroup may favour nocturnal awakenings and, on the other hand, frequent awakenings might prevent individuals from reaching a deep sleep state. As a result, patients with AN may feel that night-time rest is inadequate for their well-being. We can speculate that this feeling of inadequate sleep may reduce patients' ability to cope with daily cognitive, physical, and social demands (including treatment

efforts).

A reasonable explanation for the sleep impairments observed in patients with AN could be linked to malnutrition and underweight. This hypothesis is supported by studies showing an association between BMI and sleep quality [17,34,39]. However, it is worth remembering that the severity of reduction in BMI is an index of the severity of the disorder itself. Therefore, the differentiation between the influence of BMI and the severity of psychopathology is still unknown and could be an interesting topic for future studies. The dangerous reduction of BMI could also explain why the only significant results were found in patients with AN, whereas patients with BN or BED appear to have comparable sleep quality to healthy individuals. However, it is important to note that the number of studies focusing on AN is higher than those involving BN and especially BED, where only one study was found reporting no difference between patients and controls. In addition, patients suffering from AN have pervasive, severe, and potentially chronic physical issues, such as bone and heart problems [40], which may indirectly affect sleep quality. Moreover, some studies also suggest that this population may be characterized by brain tissue atrophy [41], which might negatively impact sleep regulation. Future studies should try to better understand the aetiology of sleep impairments in patients with AN.

This systematic work also revealed the paucity of research on this particular aspect of eating disorders, especially in the last two decades. The majority of studies with PSG were conducted in the past century, and recent literature has produced only a few studies using actigraphy [31,34]. The lack of more recent studies on the topic is surprising, since i) eating disorders prevalence has increased over the last 20 years [7], and ii) the results of the current meta-analysis showed a clear difficulty of the eating disorder population in sleep continuity and reduction of SWS. Surprisingly, studies evaluating sleep disorders, particularly insomnia, are also lacking. This is an area that requires further attention, considering that insomnia is a risk factor for mood disorders and relapses.

The effect of orexin seems to play an important role in eating disorders. Neurons in the lateral hypothalamus containing orexin seem to be activated in the condition of fasting, therefore orexin may be associated with food-seeking behaviours [11]. However, orexin seems also to be able to suppress REM sleep besides being a wake promoter [12]. Speculatively, it could be hypothesised that food restriction, associated with a higher level of orexin, could induce REM sleep impairments and general poor sleep quality. Although we did not find any difference in REM between eating disorders patients and healthy controls, we may hypothesise that an impairment of REM may be observed only in patients with a longstanding eating disorder, where prolonged fasting might have affected brain processes.

Orexin may also be involved in eating disorders via a complex interplay with other neuropeptides and neurotransmitter systems involved in appetite regulation, such as the neuropeptide Y, an appetite stimulator, and melanin-concentrating hormone, which is involved in the appetite suppression [42,43]. Given the pivotal involvement of orexin in regulating both feeding behaviour and sleep patterns, future studies may explore the potential therapeutic efficacy of orexin modulation for the treatment of eating disorders, as observed in animal models [44], particularly in those individuals showing sleep disturbances and REM sleep alterations. When binge-eating episodes are present, a reduction of orexin level may support both appetite and sleeping processes by decreasing the sense of hunger and facilitating physiological REM sleep.

More recently, self-report assessments suggested that patients with ED experience poorer sleep quality compared to healthy controls [37]. This finding deserves attention considering that subjective assessment of sleep quality is not secondary to the physiological one and the diagnosis of some sleep disorders, such as insomnia, is made based on subjective assessment. Therefore, the complaint of poor sleep quality alone should draw clinical attention and specific interventions.

Circadian patterns have been particularly poorly investigated, at least in patients with an eating disorder (but see Zou et al., 2022 for a review also on eating behaviours [45]). The only study we were able to find seems to suggest that eating disorder patients have an evening preference compared to healthy controls [33]. Also, Natale and colleagues [33] found that more extreme evening preferences were associated with higher levels of ineffectiveness and perfectionism. Interestingly, the evening preference in eating disorders patients is consistent with the observation of a generalized delayed shift of circadian rhythms in psychiatric disorders [45].

Overall, we observed a significant heterogeneity among the studies included in the meta-analysis, due to variables that were not measured or reported. Future studies should control for variables that might act as confounders of the variables of interest (e.g., purging behaviour and BMI). Furthermore, comorbidities can affect the findings. Depression, for instance, could interact with both eating psychopathology and sleep alterations [5] and modify the relationship. However, some studies have found that sleep alterations transcend mood disorders [18,19] and are at least partially independent of it. Interestingly, although a classic feature of depressed patients is the increase in REM sleep [46], we did not find any REM sleep alteration in eating disorders patients. Nevertheless, further studies are needed to understand the role of depression in mediating the relationship between eating pathology and sleep impairments, as already suggested by Kenny and colleagues (2018) [47]. Future studies should try to clarify the mechanisms underlying both sleep and eating disorders. A promising candidate is self-regulation and emotional regulation in particular. Poor sleep impairs emotional regulation, potentially exacerbating eating symptoms. This is because the inability to cope with negative emotions could contribute to triggering dysfunctional eating behaviours, such as food avoidance and binge eating [13]. Another potential mediator between perceived sleep quality and eating disorders is body image representation. A preliminary study by Akram et al. (2021) [48] suggested that certain cognitive processes that affect body image disturbances can be negatively influenced by insomnia symptoms. Future studies could further investigate whether difficulties with sleep could impact how individuals perceive their bodies and consequently exacerbate symptoms of eating disorders. A further approach for investigating mediators could be to consider insomnia and sleep quality as mediators of the effect of other variables, such as internalising symptoms [49] and exercise addiction [50] on disordered eating behaviours. Such an approach, which incorporates the dynamics of several factors, could provide insight into the complex mechanisms underlying the relationship between eating and sleeping patterns.

Also, the software used for the scoring of physiological data might be a source of heterogeneity, as in the case of Kleppe and colleagues (2023) [29]. As the authors themselves stated, for the detection of WASO they used an automatic software that seems to be not perfectly reliable for this parameter, especially when not supported by sleep diaries to distinguish wake time from normal movements. In fact, when only considering WASO, they observed reduced WASO in patients with AN, a result inconsistent with the rest of the literature. To overcome the limitation related to the software, the authors employed manual scoring for further analyses related to longer awakening (>5 min). Using this manual procedure, they found that the duration of awakenings of at least 5 min was longer in patients with AN than in healthy controls. This latter result is in line with the literature.

Studies on the effect of eating disorder interventions on sleep quality are particularly scarce and provide contradictory results. Some works, including those not considered for this work as they did not meet inclusion criteria, seem to suggest that perceived sleep quality increases with recovery [51], while another study reported a worsening of some sleep parameters [52]. A recently published study found that perceived sleep quality improves after treatment, but the authors pointed out that it still does not reach an adequate level of satisfaction [53]. These results are insufficient to have a clear idea of the impact of eating disorder treatment on sleep outcomes; however, it seems plausible that eating

Practical points

1. Eating disorders patients complain of poor sleep quality;
2. Physiological sleep in eating disorders patients is shorter, more fragmented, and less efficient;
3. Circadian preference and sleep disorders in eating disorders patients have been poorly investigated;
4. Sleep-targeted intervention could benefit eating disorders patients' recovery.

Research agenda

In future, researchers need to understand which:

1. Aspects of the eating psychopathology are associated with sleep alterations, and vice versa;
2. Consequences sleep disruptions have on the eating pathology, through the design longitudinal studies;
3. Specific sleep-centred interventions are most suitable to address the poor quality of sleep.

disorder treatment might remediate sleep difficulties to some extent. This is a proposal that warrants further investigation.

Future studies might address some of the methodological limitations in the current literature on sleep problems in eating disorders. For instance, it would be informative to compare findings across different eating disorder groups (e.g., AN, BN, BED) and assessment measures (e.g., physiological or self-reported measures) to highlight possible overlap and discrepancies. To enhance methodological rigour, it would be beneficial to include a group of healthy individuals as a comparison and control for physiological distress, such as depression and anxiety.

4.1. Limitations

The results of this work should be taken in light of some limitations. First of all, an intrinsic limit of reviews and meta-analyses is that their quality depends on the quality of the included studies. As the RoB analysis indicated, there are some possible biases in the selection of the control group and the presence of possible confounding variables (education or socio-economic background). Secondly, the scarcity of studies in the literature prevented us from performing some meta-analytical calculations (e.g., analysis of circadian preferences) and it may have affected the reliability of the results. More research is needed on the relationship between eating disorders and sleep and circadian rhythms.

4.2. Conclusions

The current study showed that patients suffering from an eating disorder are characterized by a physiological sleep quality impairment, in particular reduced sleep continuity and sleep depth compared to healthy controls. Eating disorder patients also report lower sleep quality. This work highlights the need for more specific, rigorous, and controlled studies to overcome the aforementioned limits. Moreover, the studies conducted so far are relatively few and old. There is a need to design more rigorous experimental paradigms to test whether a well-defined symptom is related to a specific sleep parameter and vice versa.

Longitudinal studies, for instance, could be used to assess causality in the relationship between sleep quality and eating disorder symptomatology. For example, these studies could assess whether poor sleep quality has a causal effect on the severity of eating pathology or on treatment effectiveness. This approach could be particularly beneficial for clinicians to understand whether sleep-centred treatments may affect patients' recovery.

Declaration of competing interest

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.smrv.2024.101969.

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