





Recent Advances in Sleep and Circadian Characteristics in Eating Disorders: A Systematic Review of the Last Five Years (2020–2025)

Giulia Cera ¹, Serena Scarpelli ^{1,2}, Silvamaria Mastrocola³, Margherita Boltri³, Federico Brusa⁴, Alberto Scalia⁴, Maurizio Gorgoni ^{1,2}, Carolina Lombardi^{5,6}, Leonardo Mendolicchio^{4,*}, Luigi De Gennaro ^{1,*}

¹Department of Psychology, Sapienza University of Rome, Rome, Italy; ²Body and Action Lab, I.R.C.C.S. Fondazione Santa Lucia, Rome, Italy; ³Department of Psychology, Università Cattolica Del Sacro Cuore, Milano, Italy; ⁴I.R.C.C.S. Istituto Auxologico Italiano, Experimental Laboratory for Metabolic Neurosciences Research, Piancavallo, VCO, Italy; ⁵Department of Cardiology, S. Luca Hospital, IRCCS, Istituto Auxologico Italiano, Milan, Italy; ⁶School of Medicine and Surgery, Università di Milano – Bicocca, Milan, Italy

*These authors contributed equally to this work

Correspondence: Giulia Cera, Department of Psychology, Sapienza University of Rome, Rome, Italy, Email giulia.cera@uniroma1.it

Purpose: A high number of people affected by eating disorders also show severe sleep disturbances, as well as circadian rhythm disruptions. However, research is relatively limited, often yielding conflicting results, especially when comparing clinical populations to healthy controls. Few interventions currently integrate sleep and circadian considerations into ED treatment. This review aims to synthesize recent evidence on sleep and circadian alterations in EDs and to identify research and clinical priorities.

Methods: A systematic research through four academic databases was conducted during September 2025, seeking studies on the theme published between 2020–2025. Eligible articles were narratively synthesized to provide a comprehensive and recent overview of the state of the art. Quality appraisal tools were used according to the studies' design.

Results: Eleven studies met inclusion criteria. Sleep disturbances were most consistently reported in individuals with anorexia nervosa, including poor sleep quality. Evidence for bulimia nervosa and binge-eating disorder was limited and inconclusive, since only a study was included. Five case-control studies compared clinical populations to healthy controls and three studies assessed the effects of ED-focused treatments on sleep. Two studies evaluated a sleep-specific intervention, while one employed bright light therapy. Objective sleep measures were rarely employed.

Conclusion: Sleep and circadian disturbances represent an underexplored but clinically relevant dimension of EDs. This review provides a systematically organized synthesis of recent evidence, clarifies diagnosis-specific patterns, and identifies methodological and intervention gaps. Integrating sleep and circadian considerations into assessment and treatment may enhance rehabilitation outcomes and inform the development of more effective, targeted interventions.

Keywords: eating disorders, sleep, circadian rhythms, anorexia nervosa, bulimia nervosa, binge-eating disorder, chronotherapy, sleep-related interventions, eating-related interventions

Introduction

Introducing the Core Symptomatic Features of Eating Disorders

The main feature of eating disorders (EDs) is a persistent disruption in eating-related habits, which negatively impacts the individual's physical health and psychosocial functioning.¹ Anorexia nervosa (AN) is mainly characterized by severe food restriction and an intense and pervasive fear of gaining weight, while bulimia nervosa (BN) and binge-eating disorder (BED) are defined by recurrent binge-eating episodes – which are, specifically, brief periods during which the individual consumes large amounts of food while experiencing a loss of control.¹ In BN, these episodes are followed by inappropriate compensatory behaviors (e. g., self-induced vomiting, laxative use) to prevent weight gain, whereas people diagnosed with BED only present binge episodes, not followed by any form of compensation.¹ As a result, the latter individuals typically present overweight or are affected by different degrees of obesity.

Sleep and Circadian Features in Eating Disorders

As well as nutrition, sleep is a powerful biological drive, and these processes mutually influence each other. Indeed, impairments in either sleep quality and quantity lead to dysfunctions in several physiological processes, such as those pertaining to cardiovascular, immune, metabolic and cognitive systems,^{2–5} as well as affecting food preferences, increasing the consume of highly processed or caloric foods.^{6,7} Moreover, sleep contributes to regulating body weight through several processes, such as duration, synchronization of the biological clock and hormonal secretion.^{8,9} On the other hand, even the type of consumed food, as well as the timing of intake, may impact on sleep duration and quality.^{6,10} For example, diets rich in carbohydrates are characterized by shorter sleep latency, less deep sleep and higher amounts of REM sleep, while other specific foods, such as fish, fruit and vegetables, might enhance sleep processes.^{6,11}

First, sleep plays a crucial role in the regulation of body weight. Indeed, both sleep duration and the synchronization of the biological clock are necessary to obtain the energy required for daily activities, and hormonal secretion is strongly associated to weight regulation.^{8,9} Since sleep and eating are mutually exclusive behaviors, the regulation of energy homeostasis is closely connected to sleep regulation.^{12,13} In a bidirectional manner, food intake also contributes to the regulation of sleep.^{8,14}

Despite the fact that several psychopathological conditions – such as depressive and anxiety disorders^{15,16} – have already been studied in relation to sleep disturbances, the relationship between sleep and EDs still remains insufficiently investigated.^{3,17}

Even circadian rhythms – which may be defined as an internal biological system that is responsible for 24-hour cyclic processes – may contribute to the relationship between sleep and nutrition, by regulating them.^{18–20} Indeed, although circadian rhythms are regulated by several peripheral oscillators, our main timer is the hypothalamic suprachiasmatic nucleus (SCN), which is responsible for the synchronization of our inner circadian rhythm with that of the outside world, as for almost all physiological functions.^{21–25} Briefly, research showed that lesions of the SCN and gene mutations disrupt circadian feeding rhythm^{26,27} – which is normally distributed over a 24-hour period in humans^{28–30} – as well as influence even drinking behavior,³¹ sucrose assumption and preference.^{23,32} Therefore, evidence suggests that feeding behaviors are partially controlled by the SCN, through projections to hypothalamic brain centers implicated in feeding habits – for example, via the dorsomedial and the lateral hypothalamus^{23,33,34} – as well as by his interactions with peripheral oscillators, which are sensible to timing and rhythm of food intake.²¹ In other words, feeding mistiming is associated with chronodisruption – defined as an incompatibility between the rhythms of eating habits, regulated by SCN, and mealtimes.²¹ According to the biobehavioral circadian model,³⁵ both fasting and bingeing misconducts may serve as “zeitgebers” (i.e., external cues that entrain circadian rhythms), disrupting circadian rhythms. On the other hand, even circadian disruptions maintain disrupt food intake timing, thus influencing each other.^{35,36} For example, impairments in sleep quality (i.e., poorer sleep efficiency) – considered as an alteration of circadian rhythms – has been linked with decreased morning appetite,^{35,37} while sleep restriction seems to enhance food – especially those high-in-fat – intake.^{35,38} Moreover, a recent review³⁹ supported the link among binge-eating episodes and delayed food intake patterns – especially, higher consumption of caloric food later in the day – even if, according to the authors, there may be a contribution of both chronobiological and non-chronobiological variables (i.e., social factors and work-related schedule restrictions).

In addition to abnormal eating behavior, individuals affected by EDs may also present varying degrees of sleep disturbances.^{40–45} In a prevalence study, Kim et al⁴² reported that approximately half of those who suffer from an ED may experience sleep problems, such as difficulties falling asleep, midsleep and early morning awakenings, parasomnias, and hypersomnia. Accordingly, in a case-control study on patients diagnosed with EDs (i.e., AN, BN and BED), De la Torre et al⁴⁶ found more complaints about daytime sleepiness and sleep disturbances, as well as a greater use of medications, with respect to healthy controls. Regarding circadian disruptions, patients with EDs seem to present disruptions in circadian rhythms.^{47–50} Specifically, the presence of circadian disruptions in EDs has been linked to the relationship between the SCN and the metabolic and hedonic structures implicated in altered eating behaviors,²³ resulting in a desynchronization associated with the chronotypes in this clinical population.²¹

Overview of the Literature Exploring Sleep and Circadian Features of Eating Disorders

Although the presence of sleep alterations and circadian disruptions in EDs has been widely established, research in this field is still insufficient or inadequate, thus causing a crucial lack of knowledge in the field of rehabilitation from these psychiatric conditions.^{40,51–53} Indeed, one of the main issues of the studies on this theme is that they mostly employ subjective methods and rely on cross-sectional designs. As a consequence, research is not able to deeply examine objective sleep and circadian parameters, as well as provide useful insights into the relationship between circadian rhythms, sleep patterns, and eating habits. Moreover, these studies usually rely on small sample sizes, thus limiting the validity of their results.^{41,54,55}

To date, several studies have been conducted comparing patients with EDs with the general population, but their results appear to be contradictory when taken all together.

Regarding AN, research has usually reported worse sleep efficiency,^{41,56–58} lower sleep time^{55,57} and more,^{58–60} longer⁵⁶ awakenings, as well as greater wake after sleep onset^{57,58,60} and arousal index.^{41,58,60} Several studies showed even that this clinical population complains more about greater sleep difficulties.^{61,62} However, other studies^{63–65} did not find significant differences compared with healthy controls. Findings about sleep architecture – especially regarding sleep stages – are more controversial. Overall, research suggests that individuals diagnosed with AN present impairments either in slow-wave^{54,57,58,60} or in REM sleep.^{41,54,56,59} By contrast, while in three studies^{55,57,59} this clinical population showed increased time spent in stage 1 sleep, another one⁵⁸ found a reduction of the same stage. Similarly, it is still unclear whether patients with AN reported an increase in stage 2⁵⁴ or 3⁵⁶ sleep or, differently, a reduction in stage 2⁵⁵ or 4⁵⁹ sleep.

Research about BN is scarce and inconclusive, as several studies did not find any significant difference between clinical and control samples.^{55,57,64} On the other hand, other research found reduced sleep efficiency⁶³ and time spent in stage 1,⁶⁶ as well as delayed wake-up and bedtime.⁶⁷ Also, while Della Marca et al⁴¹ observed either a reduction in total REM sleep time or an increase in REM latency in individuals with BN compared to healthy controls, another study⁶⁸ found shorter REM latency. However, patients with BN complained more about worse sleep quality, difficulties to fall or return to sleep, greater daytime sleepiness and use of medications.^{67,69}

Similarly to BN, studies conducted so far on samples of patients with BED showed conflicting results, as one⁶⁷ reported greater sleep difficulties – such as lower sleep efficiency, less total and continuous sleep time (i.e., without awakenings), and more nocturnal awakenings – while another⁷⁰ did not reveal any significant difference compared to healthy controls. Similarly, compared to overweight subjects (i.e., not diagnosed with an ED and/or BED), some studies^{71,72} found no significant differences in sleep measures in the clinical population, while another⁷³ reported greater sleep time, better sleep efficiency and shorter sleep latency – particularly, according to the authors,⁷³ these latter improvements may be considered due to the multidisciplinary treatment that their sample was following. Subjectively, individuals affected by BED complained more about poor sleep quality^{71,74} and snoring.⁷⁰ Two studies^{72,73} reported dysfunctions in the rest-activity cycle in patients with BED.

Even broader correlational and cross-sectional literature investigated the relationships among sleep measures and ED symptoms. Linnaranta et al⁷⁵ found that later and variable sleep phasing, assessed through actigraphy, associated with irregular eating pattern and eating frequency among individuals affected by EDs. Sleep disturbances have been linked to malnutrition in AN,^{51,76} since associations were found among sleep parameters and measures and body mass index,^{41,55,58,60} rather than with eating symptoms.^{8,58,77,78} However, sleep impairments may also be associated with AN through anxiety and depression,⁷⁹ as suggested by several studies.^{80–82} Accordingly, Ralph-Nearman et al⁷⁹ showed that sleep and anxiety symptoms are core features of AN and both correlated with severity of eating symptoms. A study comparing individuals diagnosed with AN, BN or BED on several clinical measures found that patients with BN reported higher sleep impairments, as well as a relationship among insomnia symptoms and affective (i.e., depression, anxiety, stress) and cognitive (i.e., memory functioning) parameters.⁸³ Finally, sleep measures in the context of binge-eating seem to be associated either with cognitive, behavioural and affective symptomatology⁸⁴ as well as with depression symptoms.⁸⁵ Despite these consistent associations, the correlational literature has important limitations, as previously observed.⁸⁶ Indeed, most of these studies employ mainly subjective measures of sleep or mixed objective/subjective metrics without control groups, limiting comparability across samples. Moreover, they frequently investigated heterogeneous samples with EDs, with high variability in assessment tools. Consequently, while correlational evidence highlights consistent relations between sleep/circadian variables and ED features, it underscores the need for more rigorous, controlled, and longitudinal research to clarify underlying mechanisms.

Regarding circadian rhythms, research is even more limited, and still no significant differences have been found in ED cohorts and amongst specific diagnostic groups when compared to the general population.^{49,69} Overall, broader literature suggests that EDs are often associated with a delay in circadian rhythms.^{47–50} While restrictive habits have been specifically correlated to a morningness tendency,¹⁹ binge-eating behaviors showed an association with eveningness preferences.^{49,87–91}

In recent years, several reviews and meta-analyses investigated both the relationships and the alterations associated with sleep and circadian rhythms in patients with EDs, even though with different primary focuses. For instance, Grimaldi et al⁹² examined the associations between sleep parameters and anthropometric, nutritional, and ED-related variables, whereas another study⁹³ explored similar relationships in non-clinical populations. Additional contributions have focused on specific diagnostic groups, such as AN⁸ and binge-eating symptoms.⁹⁴

In 2016, Allison et al⁵¹ explored the presence of sleep disturbances and their associations with EDs in a clinical sample of adolescents, synthesizing evidence from the preceding decade. Subsequently, a 2020 review⁴⁰ and a 2023 scientometric review¹⁷ further examined sleep alterations in ED populations. Also in 2020, Kim and Lee⁹⁵ carried out a review on sleep and circadian rhythms disruptions in the context of EDs. More recently, a review and meta-analysis by Degaspero et al³ provided evidence from case-control studies on sleep and circadian alterations in EDs and examined the effects of ED treatments on sleep parameters and circadian measures. Specifically regarding circadian rhythms disruptions in EDs, reviews were carried out in 2019⁴⁷ and in 2022,³⁹ this latter with a specific focus on binge-eating habits. Menculini et al⁴⁷ confirmed the presence of circadian rhythms alterations in EDs, while Romo-Nava et al³⁹ further suggested the evening preference in patients with binge-eating behaviors.

Gaining a deeper comprehension of the connection between sleep, circadian rhythms, and EDs, could, however, allow for the development of more effective treatment paths for patients, thus enabling a more comprehensive and multi-dimensional approach to care.^{96,97}

Rehabilitation Trajectories in Eating Disorders: The Neglected Role of Sleep and Circadian Features

Psychological interventions are recommended as first line treatments for EDs.^{98,99} In particular, the American Psychiatric Association recommends: a) eating disorder-focused psychotherapies, such as cognitive-behavioral, focal psychodynamic, or interpersonal therapy, in the case of adults with AN and family-based treatments for adolescents, b) cognitive-behavioral therapy for bulimic adults and family-based treatments for adolescents, and c) cognitive-behavioral therapy or interpersonal therapy for individuals with BED.¹⁰⁰ However, neither in this guideline nor in reviews about the effectiveness of rehabilitation treatments for EDs¹⁰¹ sleep or circadian rhythms are specifically addressed as an outcome of interest, even if their persistence was found to be associated with poorer treatment outcomes.^{40,43,102} As a result, the promising integration of sleep and circadian rhythms-related treatments into rehabilitation trajectories for EDs seems far from being fully achieved, thus causing a gap into clinical practices.

Aims of the Present Work

Our aim is to provide a comprehensive update of the studies conducted from 2020 to 2025 among patients with EDs – especially, in those diagnosed with AN, BN, and BED – investigating: a) sleep and circadian features in the clinical sample compared to the general population, b) ED-related interventions' psychopathological effects on sleep and circadian parameters, and c) sleep and circadian rhythms-related interventions' psychopathological effects.

Building on the review by Degaspero et al,³ this work provides a research update by systematically examining the most recent case-control studies. By including only studies with control groups, we offer a methodologically consistent synthesis of between-group differences, extending prior evidence with newly available data that were not captured in earlier reviews. A systematic evaluation of sleep and circadian rhythm alterations identified in the most recent literature, particularly when compared with earlier studies, would help to clearly define the current state of the art, distinguishing between well-established evidence and areas requiring further investigation.

The decision to focus on the 2020–2025 period was driven by the observation that, while earlier experimental literature was largely descriptive and observational, recent years have marked the initial translation of this knowledge into targeted clinical applications. Although methodological limitations remain substantial, these contributions represent a shift from purely observational research toward early-stage therapeutic integration. By focusing on this recent time frame, the present review aims to critically evaluate this emerging translational phase, identifying both its potential and its limitations, and clarifying the current state of the art to inform future research directions and clinical development.

Importantly, the review also focuses on recent intervention studies targeting sleep, representing a novel and original contribution. Sleep-focused practices in EDs are very recent, and their integration into treatment protocols is still in an early phase, whereas circadian research continues to build on previously established findings. By reviewing these emerging sleep interventions, the present study provides new insights that were not addressed in previous reviews, highlighting both their potential and the limitations of the current empirical support.

By synthesizing both past contributions and recent studies within a single, coherent framework, the review reinterprets existing knowledge in light of the newest evidence. This approach allows us to critically examine the current research–practice gap, identify areas where clinical enthusiasm may have outpaced empirical validation, and outline priorities for future investigations. Overall, the review aims to offer a unified, multidimensional perspective on the interaction between sleep, circadian rhythms, and disordered eating behaviors, supporting the development of more rigorous research and more informed clinical applications in this emerging field.

Method

Study Design

The methodological model of the study (e.g., design, analysis, and reporting) followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.¹⁰³ Completed PRISMA checklists, respectively, for the entire article and the abstract, are provided within the [Supplementary Material 2](#) and [3](#). A systematic literature search was conducted throughout four academic databases. Consequently, relevant studies on the theme were extracted, and their methodological quality was evaluated with appropriate tools. The study protocol was registered in the PROSPERO International Prospective Register of Systematic Reviews under ID code: CRD420251104939.¹⁰⁴

Search Strategy

Relevant studies on the topic were identified through a literature search carried out during the second week of June 2025 on four academic databases, PubMed, Web of Science, Scopus, and PsycInfo. To complement and integrate the findings of previous reviews while capturing recent methodological and clinical developments, the search was restricted to studies published between January 2020 and September 2025.

The following keywords were used as search terms, tailoring the strategy to each database:

((eating disorders) OR (anorexia nervosa) OR (bulimia nervosa) OR (binge eating disorder) OR (disordered eating) OR (binge eating)) AND ((sleep) OR (circadian rhythms) OR (circadian preference)) AND ((insomnia) OR (parasomnia) OR (hypersomnia)) AND ((intervention) OR (psychotherapy*) OR (treatment) OR (therap*) OR (rehabilitat*) OR (chronotherapy) OR (bright light therap*) OR (cognitive treatment) OR (cognitive behavioral)).

As presented, sleep- and circadian-related terms (e.g., “sleep”, “insomnia”, “hypersomnia”, “parasomnia”, “circadian rhythm”, “circadian preference”) were combined using the Boolean operator OR to maximize sensitivity within this domain and subsequently combined with eating disorder terms using AND. Intervention-related terms were also combined using OR and then linked to the main search string through AND, in accordance with the predefined aims of the review.

In the PubMed database, the selected filters were “Languages” and “Last 5 years”; in Web of Science “Date (i.e., 2020–2025)”, “Languages” (i.e., refine), and “Article” (i.e., refine); in PsycInfo “Languages”, “Last 5 years”, “Academic Publications”; and in Scopus “Languages”, “Date”, and “Review” and “Book chapters” (i.e., exclude).

Inclusion and Exclusion Criteria

Studies were selected based on the population, intervention, comparison/comparator, outcome, and study design (PICO) approach. The inclusion criteria were:

1. Participants: Inclusion of a sample of adolescents or adults diagnosed with EDs – specifically, AN, BN, or BED;
2. Intervention: Assessment of physiological parameters and/or subjective features of sleep processes and/or of circadian preferences;
3. Comparison: Inclusion of a control group, given by individuals without any diagnosis of neurological or psychopathological disorders;
4. Outcome: Physiological sleep parameters (measured through polysomnography (PSG) or actigraphy) and subjective data (collected through diaries or questionnaires);
5. Study Design: Case-control design for studies investigating sleep and circadian features in ED samples, while quasi-experimental design for interventions on ED samples;
6. Further criteria: Articles written in English, Italian, Spanish, German and French and conducted among 2020–2025.

The exclusion criteria were:

1. Reviews, meta-analysis, case reports and series, book chapters, commentaries, and letters to the editor;
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, or, if the studies had the same number of subjects, the most recent one was included.

Study Selection

The search was performed independently and blindly by three authors (GC, SS and SM) and the results from the literature search were uploaded to Rayyan.¹⁰⁵ After removing duplicates, the studies were scrutinized based on their title and abstract. Reviews, meta-analysis, case reports, book chapters, commentaries, and letters to the editor were excluded, as well as articles that were not written in English, Italian, Spanish, German or French. Only among the studies which had examined sleep and circadian parameters among individuals with EDs, did we search for case-control studies.

After screening and selecting the studies for inclusion, two members of the team (GC and SS) extracted several pieces of information from the texts. Disagreements were resolved through discussion.

Risk of Bias Appraisal

Two members of the research team (GC and SS) independently assessed the risk of bias using several quality appraisal tools, according to the design of each study. Case-control studies^{84,106–109} were assessed using the NIH Quality Assessment Tool for Case-Control Studies, evaluating risk of bias in selection, comparability, and measurement of exposure and outcomes. The non-randomized controlled before–after study¹¹⁰ was appraised using the JBI Critical Appraisal Checklist for Quasi-Experimental Studies,¹¹¹ focusing on group comparability, confounding factors, and outcome assessment. Before–after studies without a control group^{112,113} were appraised using the NIH Quality Assessment Tool for Before–After Studies With No Control Group, assessing intervention description, outcome measurement, and statistical analyses. Finally, the single-case design study was evaluated using the What Works Clearinghouse (WWC) Tool.¹¹⁴

Data Extraction and Management

After screening, selecting, and evaluating the quality of selected studies, two members of the research (GC and SS) extracted several information within the texts, including the first author's name, year of publication, country of the study, features (sample size and type, female percent, mean age and body-mass index (BMI)) of both clinical and control groups, type of sleep assessments, outcomes, type of assessment of EDs, and main findings, in order to provide

a narrative synthesis of the studies. The three phases of the study selection, quality assessment, and data extraction were carried out independently by the research team. Disagreements were resolved through discussion.

Results

Study Screening and Selection Process

The literature search carried out through the four databases retrieved 576 studies: PubMed (n = 199), PsycInfo (n = 206), Scopus (n = 74), and Web of Science (n = 97), from which 181 duplicates were removed. A total of 395 papers were screened based on title and abstract information. Finally, 29 articles were selected as potentially eligible and their full texts were examined. From this process, eleven studies met the eligibility criteria and were included in the review. [Figure 1](#) shows the search process based on the PRISMA flowchart.

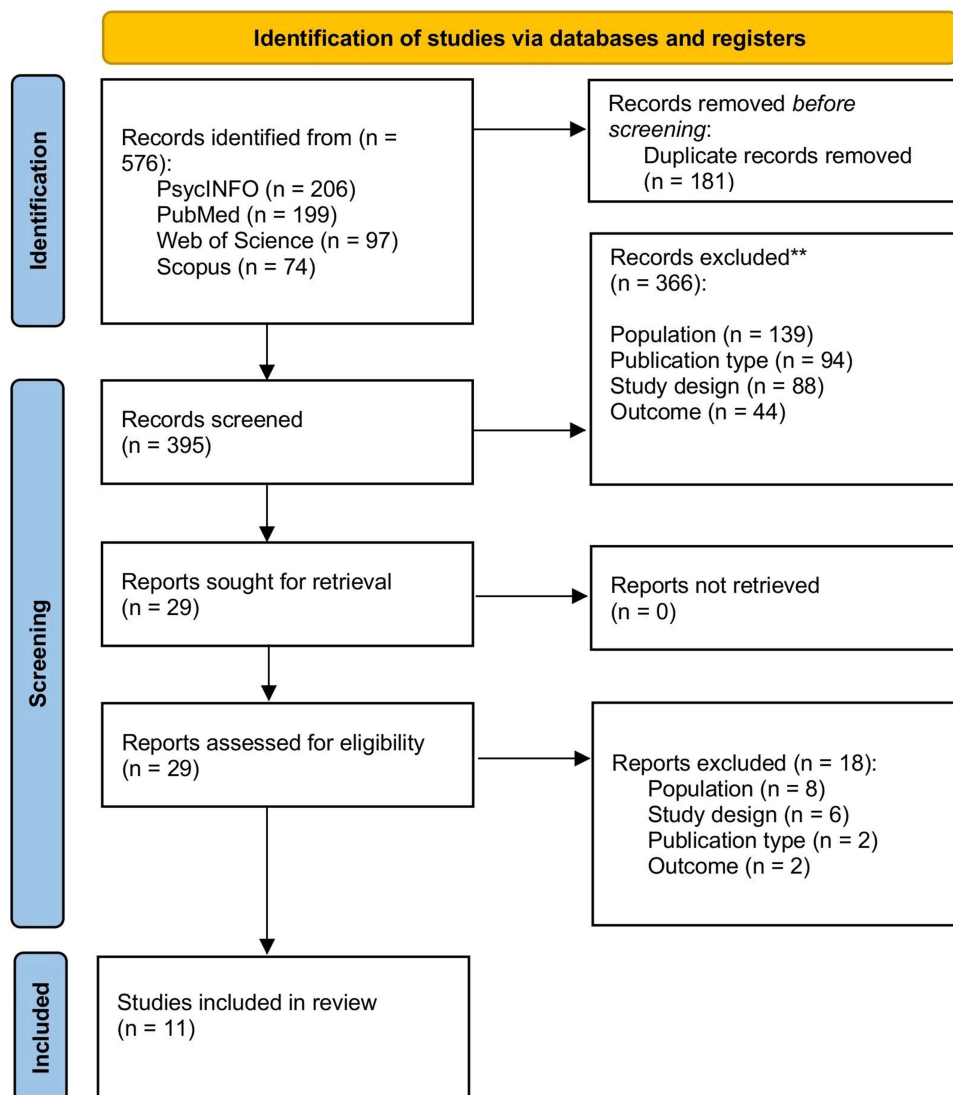


Figure 1 PRISMA flowchart.

Quality Assessment

Eleven studies were appraised within their methodological quality. This process is available within the [Supplementary Materials 4](#). All the studies met the authors' judgment of fair quality.

Study Description

[Table S1](#) in the [Supplementary Material 1](#) shows a narrative synthesis of the included studies, including the following features: first author, publication year, country of the study, study design, which diagnosis of EDs was taken into account, type of sleep parameters/circadian rhythms/clinical assessment.

Out of the eleven studies included in the review, five focused exclusively on patients with AN, two examined mixed cohorts of individuals with all three ED diagnoses, one investigated only patients with BED, and three explored non-low-weight EDs (i.e., samples with either BN or BED). Among the diagnostic categories, AN was the most frequently examined (5 studies; 45.4%), followed by BED (1 study; 9.1%). The remaining five studies (45.4%) explored ED more broadly; in three cases, they explored non-low-weight EDs.

In order to summarize our findings, the included studies were divided into the following categories: case-control studies investigated sleep and circadian features in EDs (ie, [Table S2](#) in the [Supplementary Material 1](#)), research exploring the efficacy of ED treatments on sleep and circadian features (ie, [Table S3](#) in the [Supplementary Material 1](#)) and interventions targeting sleep and circadian features in ED samples (ie, [Table S4](#) in the [Supplementary Material 1](#)).

Sample Description

Overall, 3528 individuals affected by EDs and 1159 healthy controls were included in the final synthesis. In the clinical group, 3319 (94.1%) patients were female and 209 (5.9%) were male. In the control group, 1027 (88.6%) individuals were female and 132 (11.4%) were male.

Among the patients for whom a specific diagnosis was reported (3342), 2077 had a diagnosis of AN (62.1%), 775 of BN (23.2%), 24 (0.7%) of BED and 229 (6.8%) had other specified Eating Disorders (OSFED).

On average, the healthy control group had an age of 29.53 years ($SD = 9.03$ years; min–max = 19.0–42.5 years), while the clinical ED population of 25.20 years ($SD = 4.89$ years; min–max = 14.3–36.2 years). Among individuals affected by AN, the mean age was of 17.23 years ($SD = 3.69$ years; min–max = 14.3–27.86 years), while by BED of 26.42 years ($SD = 4.27$ years; minimum and maximum could not be provided since there was only one study focusing exclusively on BED). Overall, individuals with AN had the lowest mean age, while the highest for individuals diagnosed with BED. Controls for patients with AN had on average an age of 24.33 years ($SD = 5.92$ years; min–max = 19.0–30.70 years), while for BED of 32.17 years ($SD = 7.85$ years; minimum and maximum could not be provided since there was only one study focusing on BED).

The clinical population on average had a body mass index (BMI) of 26.12 kg/m^2 ($SD = 5.98$ kg/m^2 ; min–max = 15.34–33.15 kg/m^2), while the control group of 22.58 kg/m^2 ($SD = 1.88$ kg/m^2 ; min–max = 20.98–25.10 kg/m^2). On average, individuals diagnosed with AN had a BMI of 16.83 kg/m^2 ($SD = 1.05$ kg/m^2 ; min–max = 15.34–20.16 kg/m^2), and with BED of 33.15 kg/m^2 ($SD = 3.74$ kg/m^2 ; minimum and maximum could not be provided since there was only one study focusing exclusively on BED). As expected, individuals affected by AN showed the lowest BMI, while the highest was found for individuals with BED. On average, controls for patients with AN had a BMI of 23.77 kg/m^2 ($SD = 2.06$ kg/m^2 ; min–max = 20.98–25.10 kg/m^2), and for those with BED of 21.33 kg/m^2 ($SD = 1.96$ kg/m^2 ; minimum and maximum could not be provided since there was only one case-control study focusing on BED).

Assessment and Interventions Description

Among the five included case-control studies, four^{84,106,108,109} (80%) have examined sleep parameters or circadian preferences through subjective methods, and only one¹⁰⁷ with objective instruments (20%). None of them combined both. Among the first, all (100%) employed the Pittsburgh Sleep Quality Index, while, respectively, the Epworth Sleepiness Scale and the Sleep, Circadian Rhythm and Mood Questionnaire were used only in one study (20%). The Pittsburgh Sleep Quality Index is therefore the most used subjective method. The only study that objectively investigated sleep characteristics employed actigraphy.

Six studies^{110,112,113,115–117} evaluated the effects of different interventions on several clinical parameters: three of them^{112,113,116} explored the efficacy of ED treatments on sleep variables, while the other three^{110,115,117} focused on different programs targeting sleep variables or circadian features.

Sleep and Circadian Features in Eating Disorders

[Table S2](#) in the [Supplementary Material 1](#) provides a synthesis of the case-control studies investigating sleep and circadian features in individuals with EDs compared to healthy controls.

Only one study¹⁰⁶ subjectively assessed sleep parameters among individuals affected by AN, BN and BED – without differentiating between diagnoses –, reporting that patients diagnosed with EDs perceived poorer sleep quality compared to healthy controls [see [Table S2](#), Kenny (2025) – main findings].

Regarding AN, this finding is supported by two additional studies,^{108,109} which show consistent results: in both, patients reported more sleep difficulties compared to the control group. In particular, Malcolm et al¹⁰⁸ further observed that these disturbances might result in more severe daytime impairments, in addition to a greater use of medications [see [Table S2](#), Malcolm (2022) – main findings]. Furthermore, patients with AN included in the study by Romigi et al¹⁰⁹ perceived their sleep as less efficient, shorter, and qualitatively poorer, than healthy controls [see [Table S2](#), Romigi (2022) – main findings]. Only one study¹⁰⁷ objectively examined sleep features using actigraphy [see [Table S2](#), Kleppe (2023) – main findings]. The authors found that individuals with AN experienced longer duration of awakenings but shorter wake after sleep onset, compared to healthy individuals.

No research focused specifically on BN. Therefore, the findings reported by Kenny et al¹⁰⁶ cannot be directly compared with other studies.

Using subjective methods, only one investigation⁸⁴ assessed sleep parameters in individuals affected by BED, thus confirming the worse sleep quality perceived by patients compared to normal-weight controls [see [Table S2](#), Antunes (2020) – main findings]. However, since only a study regarding sleep features in BED was included, these findings should be interpreted with care.

Another study¹⁰⁸ assessed circadian preferences in patients diagnosed with EDs, especially amongst individuals affected by AN. However, no significant differences were found between the clinical group and healthy controls [see [Table S2](#), Malcolm (2022) – main findings].

Eating Disorder-Related Interventions' Effects on Sleep and Circadian Parameters

In the last five years, three studies^{112,113} evaluated the effects of multidisciplinary treatments on sleep features amongst people affected by EDs. Based on the included studies, no research has evaluated the effects of such interventions on circadian rhythms. [Table S3](#) in the [Supplementary Material 1](#) shows detailed information about the included studies.

In the study by Martínez -Sánchez et al¹¹² the intervention was conducted by a variety of specialized professionals in EDs, such as nurses, psychiatrists, endocrinologists, occupational therapists, and social workers, ranging from 18 to 20 weeks of duration, according to each patient clinical profile. The treatment, based on a cognitive-behavioral approach, aimed at reducing dysfunctional eating symptoms and was delivered mainly in a group format (i.e., 8–10 participants), paralleled by individual sessions with the psychiatrist and the medical staff.¹¹² Sleep measures were collected at baseline (i.e., about 49 days after admission to the hospital) and after 10 weeks. From the baseline, patients with AN showed an increase in sleep latency, a decrease in sleep efficiency and increased number and duration of night perturbations [see [Table S3](#), Martínez -Sánchez (2020) – main findings].

Differently, Meule et al¹¹³ found a significant improvement from admission to discharge of sleep quality following a psychosomatic treatment program, which also included cognitive-behavioral individual and group therapy sessions, based on the German psychosomatic approach¹¹⁸ [see [Table S3](#), Meule (2023) – main findings]. As mentioned elsewhere,¹¹⁹ psychosomatic care programs are multimodal treatments oriented on current guidelines and evidence-based practices.

Pacella et al¹¹⁶ carried out a treatment trial, exploring the relationship among binge-eating and insomnia symptoms, before, during and after an ED treatment delivered through guided self-help versions of Cognitive Behavioral Therapy-Enhanced (i.e., CBT-gsh), called BEST-U (i.e., Building Healthy Eating and Self-Esteem Together for University Students). As mentioned also in previous works,^{120,121} the ED intervention consisted of 14 short modules, delivered over 11 weeks through a mobile

phone-app, based on second and third wave-CBTs.¹¹⁶ Each module had an instructional content about a specific theme (i.e., weight, eating and compensatory behaviors, cognitive distortions, interpersonal skills, body image) combined with interactive components, such as behavioral exercises.¹²¹ Moreover, the BEST-U app monitored participants daily, weekly and monthly, thus tracking the ED psychopathological trajectory and was supplemented with weekly telehealth coaching sessions by a trainee or licensed provider.^{116,121} In their study on participants with non-low-weight EDs, Pacella et al¹¹⁶ found that the ED treatment was effective in reducing insomnia symptoms, even if this was not the main target. Moreover, about a half of the sample (i.e., 44.94%) reported no clinically significant insomnia at baseline or at the end of the treatment and 22.47% remitted from insomnia post-treatment, while 25.84% did not achieve remission and 6.74% developed significant insomnia symptoms by post-treatment. Thus, the study suggests that the ED treatment was effective in remitting clinically significant insomnia in about a half of participants [see [Table S3](#), Pacella (2025) – main findings].

Sleep and Circadian Rhythms-Related Interventions' Effects

Based on the included research, 3 studies¹¹⁰ evaluated the effects of interventions targeted at sleep parameters or circadian features of samples with EDs. [Table S4](#) in the [Supplementary Material 1](#) shows detailed information about these longitudinal studies.

In particular, Crevits et al¹¹⁰ investigated the impact of a Cognitive-Behavioral Therapy intervention for Insomnia (CBT-I) on sleep, eating, and psychopathological-related outcomes amongst subjects with AN (i.e., CBT-I was an adjunctive treatment to ED care), by comparing them to another clinical group that followed a classic intervention program for the psychopathology (i.e., either partial or full hospitalisation as ED care). CBT-I consisted of behavioral techniques (i.e., sleep restriction therapy and stimulus control), cognitive therapy and sleep-related psychoeducation and the program was delivered through four sessions of one hour each in group format (i.e., eight patients per group). ED care included re-nutrition and therapeutic groups for patients and their families. Both groups were evaluated at the same times: before therapy (T0) and after therapy (i.e., after five weeks) (T1). Each intervention lasted five weeks. Sleep parameters were assessed through subjective measures, such as self-report questionnaires and sleep diaries.

During this time, the clinical group following a classic program showed a significant improvement only in BMI, while patients treated adjunctively with CBT-I, as well as a significant weight-gain, presented improvements even in sleep latency, sleep efficiency and total wake time. Moreover, there was also a significant positive change in their physical wellness. Eating-related symptomatology did not change in both groups [see [Table S4](#), Crevits (2024) – main findings].

Pacella et al¹¹⁵ employed a single-case multiple-baseline design to investigate the efficacy of Brief Behavioral Treatment for Insomnia (BBTI) as a follow-up to ED treatment. The BBTI intervention, even if similar to CBT-I – for example, involving psychoeducation as well as stimulus control and sleep restriction – is more focused on behavioral processes rather than cognitive mechanisms. The program was carried out on a sample of individuals diagnosed with non-low-weight EDs who previously underwent an ED protocol (i.e., BEST-U) and lasted four weeks (ie, four sessions, each one delivered weekly). After being considered eligible, participants completed a minimum baseline period of three weeks during which they were assessed with the Insomnia Severity Index (ISI) and the Eating Pathology Symptom Inventory (EPSI) weekly. When the ISI score demonstrated insomnia severity stability (i.e., similar ISI scores during the baseline period) – or a worsening trend -, participants reached the intervention phase. During the treatment, subjects were asked to complete daily sleep diaries and weekly the ISI and the EPSI. After four weeks of BBTI, individuals were re-called at the one and three-month follow-ups to evaluate stability of treatment effects, through the completion of several psychopathological indices.

A significant decrease in insomnia severity was observed at post-treatment, with the majority of participants (i.e., five out of six) reporting ISI scores below the established cut-off for clinical insomnia. This latter result was maintained even at the follow-ups, thus suggesting that BBTI effects still persisted one and three months after the intervention. Even sleep efficiency and daytime fatigue significantly improved after treatment, with the latter remaining below initial levels at both the follow-ups. No change in ED symptoms and anxiety levels was observed, while ED-related clinical impairment and depressive symptoms significantly improved after BBTI, persisting at the 1-month follow-up, but returning to baseline levels three months post-intervention [see [Table S4](#), Pacella (2025) – main findings].

Kambanis et al¹¹⁷ tested the effects of bright light therapy on individuals with BN and BED, randomizing individuals to one of two full spectrum light therapy conditions (i.e., normal light first or bright light first). Each participant received both

conditions, even if in different orders. They were told to use a therapy lamp condition for 30 minutes per day for 10 days; then, they would return to the laboratory, complete the assessments and exchange their lamp setting for other 10 days. Only patients with BN or BED characterized either by high dietary restraint or negative affects showed significant reductions following bright light therapy compared to individuals at other combinations [see [Table S4](#), Kambanis (2023) – main findings].

Discussion

Research Over the Last Five Years: A Substantial Improvement or a Persistent Gap?

This review aimed to provide an update on the most recent research investigating sleep characteristics and circadian preferences amongst individuals diagnosed with EDs compared to healthy individuals. The five included studies span the years 2020 to 2025, thus contributing to an up-to-date synthesis of the literature regarding this topic. Overall, it can be observed that research on this theme is still limited, relying mainly on subjective methods – such as questionnaires – and focusing primarily on specific diagnostic categories – with AN still being the most studied, while BED and, especially, BN remain largely unexplored. The greater emphasis on AN in the present review reflects the current state of the literature, rather than a methodological imbalance of the review process, as studies specifically addressing BN remain notably scarce.

Only one study¹⁰⁶ investigated a heterogeneous ED sample, thus supporting the findings of Asaad Abdou⁷⁶ and De La Torre Luque et al.⁴⁶ To the best of our knowledge, only an investigation⁷⁶ had previously examined through objective instruments (i.e., polysomnography) patients affected by mixed EDs (e.g., AN and BN). In accordance with the subjective sleep complaints reported in other studies,^{46,106} the clinical sample showed reduced sleep efficiency, longer sleep latency, and increased levels of arousal. Overall, as currently reported by literature, these findings are consistent with the presence of sleep alterations in individuals with EDs.

AN remains the most extensively studied ED to date: 5 of the 11 included studies exclusively focused on it. In line with previous findings,^{61,62} the studies by Malcolm¹⁰⁸ and Romigi et al¹⁰⁹ also report greater self-perceived sleep difficulties in patients with AN compared to healthy controls. Previous works by Burger⁸ and by Degasperi et al³ did not provide a meta-analytical evidence about sleep quality in individuals diagnosed with AN, since the included studies were only a few. However, in both cases narrative findings suggest that this clinical population perceives a poor sleep quality, thus confirming our results.

Only one of the included studies employed objective assessment methods, thus limiting the strength of the conclusions that can be drawn. Actigraphy was used only by Kleppe et al,¹⁰⁷ who found that individuals with AN experienced longer durations of awakenings and, in contrast to previous studies,^{57,58,60} shorter wake after sleep onset, compared to healthy individuals. In their study, Burger et al⁸ concluded that patients affected by AN generally experience lower total sleep time, higher wake after sleep onset and lower sleep efficiency compared to healthy controls. These results were further confirmed by Degasperi et al,³ who further added that this clinical population presents even reduced slow-wave sleep and increased time of stage 1 sleep, in accordance with previous studies.^{55,57,59} Therefore, our results neither do add new insights on sleep alterations nor clarify the discrepancies about sleep architecture parameters in AN.

By contrast, not a single study focused exclusively on BN, thus not allowing to compare the findings from previous research. As previously mentioned, only 9 studies have investigated sleep disruptions in individuals diagnosed with BN in comparison with healthy subjects.^{41,55,57,63,64,66–69} Nevertheless, several of them^{55,57,64} did not find any significant difference between groups, whilst other objective sleep results,^{41,55,63,66,67} taken together, are controversial. Even the meta-analysis by Degasperi et al³ did not detect any significant difference in sleep measures among patients with BN and the general population.

The worse sleep quality perceived by subjects diagnosed with BED in the study carried out by Antunes et al⁸⁴ supports previous findings.^{70,71,74} Other studies^{72,73} that employed objective methods have previously suggested that sleep alterations in patients with BED may be more strongly linked to obesity per se rather than to the ED itself. Compared to normal-weight controls, individuals affected by BED showed lower sleep efficiency, shorter total and continuous (i.e., without awakenings) sleep time, and more awakenings.^{71,72} Differently, Degasperi et al³ concluded that there was an insufficient number of studies to reach any conclusive evidence. According to our results, Da Luz et al⁹⁴ previously found that recurrent binge eating was associated with poor overall sleep quality, greater daytime sleepiness, insomnia and difficulty falling asleep.

Only one study¹⁰⁸ investigated circadian rhythms, showing no significant differences in the clinical population compared to healthy controls. In their review, Degasperi et al³ included only a work⁴⁹ investigating circadian rhythms in a sample of

patients with EDs, thus concluding that this clinical population may exhibit an eveningness preference. This evidence is supported even by other research.^{47,48} Recently, strong association has been found in the works by Romo-Nava et al³⁹ and Kim and Lee⁹⁵ between binge-eating conducts and evening chronotype.

Targeting Sleep and Circadian Features: From Research Gaps to Clinical Opportunities

While the research field still has several methodological and conceptual limitations, the clinical practice shows a renewal interest for the evaluation of the effects of multidisciplinary treatments, as well as sleep or circadian rhythms-related interventions among patients with EDs.

With respect to the effects of ED treatments on sleep measures, Degasperi et al³ already observed that research on this theme was scarce and provided contradictory results, basing on the included case-control studies^{77,122,123} in their review. Moreover, this literature has been focusing mostly on treatments for AN, as represented in the work by Burger et al.⁸ Our review aimed at providing an update and integration of previous findings, in the light of more recent approaches.

Regarding AN treatments and their effects on sleep measures, the most extensively studied has been weight restoration.^{112,122,124–126} The improvements of sleep measures – especially, those regarding sleep duration – after weight gain has been reported in several studies,^{77,122,124,125} which further suggested that sleep disturbances in AN may be linked to malnutrition. This observation was further encouraged by associations among sleep duration measures – such as total sleep time – and patients' body mass index.⁷⁷ Other studies^{112,126} did not reach similar evidence, founding that even with re-feeding patients with AN still presented sleep disturbances. Moreover, in another research,⁶⁵ the patients' body mass index was negatively associated with sleep duration measures, reinforcing the contradiction of findings about the effects of weight restoration on objective sleep measures. Although, perceived sleep quality showed improvement after weight restoration in other studies.^{113,126}

A renewal interest for the effects of treatments targeting binge-eating behaviors on sleep parameters may be seen in the inclusion of two studies^{113,116} conducted within the last five years. Specifically, Meule et al¹¹³ examined the efficacy of a psychosomatic-based intervention on the subjective sleep quality of a sample with EDs – including patients with binge-eating symptoms. More recently, another research¹¹⁶ investigated specifically the effects of an ED treatment – CBT-gsh based – on insomnia symptoms, as well as this latter association with binge-eating conducts, in a sample of non-low-weight EDs. The ED intervention – which did not target specifically sleep disturbances – showed partial efficacy in reducing insomnia symptoms, since half of participants at the end of treatment still reported clinically significant insomnia.

Therefore, we argue that the integration of interventions more directly targeting sleep could offer a promising direction, as shown by Crevits et al,¹¹⁰ who demonstrated the efficacy of a CBT-I intervention in AN. Specifically, after treatment, participants showed several improvements not only in objective sleep parameters – such as sleep latency, total wake time and sleep efficiency – but also in other clinically relevant measures, such as the perception of physical wellness.

Both the other two included studies^{115,117} focused on sample of participants with non-low-weight EDs. Similarly to the study by Crevits et al,¹¹⁰ Pacella et al¹¹⁵ investigated the efficacy of a protocol employing Brief Behavioral Treatment for Insomnia (BBTI). Even if similar to CBT-I, BBTI, as the authors¹¹⁵ argued, seems to be preferable as a follow up for current ED treatments. The intervention showed preliminary efficacy on sleep measures – such as insomnia symptoms, daytime fatigue and sleep efficiency – as well as on depression symptomatology. Accordingly with a recent review,¹²⁷ Kambanis et al¹¹⁷ employed bright light therapy on patients diagnosed with BN and BED, reporting efficacy in reducing ED-related and mood symptoms in participants who presented either high levels of dietary restraint and negative affect. De Young and Bottera³⁷ also proposed the effectiveness of combining scheduled regular eating and sleep/wake times with bright morning light exposure, suggesting that they may affect diurnal appetite rhythms through the regulation of circadian rhythms. Finally, Natale et al⁴⁹ found that a shift towards morningness during a psychological treatment was associated with an improvement of ED-related symptoms. As suggested by another review,²¹ chronotherapeutic approaches – such as bright light therapy – may be a valid and promising treatment option for individuals affected by EDs. However, mechanisms underlying these effects are still unclear, as most of the included studies were not adequately controlled or their designs did not allow drawing causal explanations.¹²⁷

Recommendations for Future Research and New Practices: A Research and Clinical Agenda

By providing a comprehensive overview of the most recent research and interventions on sleep and circadian rhythms in EDs, we want to highlight current inconsistencies to promote a more effective approach towards this area of study.

To conclude, our review highlighted the following gaps:

- Lack of studies employing objective sleep measurements – such as polysomnography or actigraphy – and longitudinal designs;
- Employment of small samples and, therefore, limited generalizability;
- Inadequate control of potential confounding variables, due to the study designs that did not allow to draw any causal explanation – such as participants' education level, socioeconomic status, age – and psychiatric comorbidities (i.e., anxiety and depression levels), which may be an alternative explanation for the frequent co-occurrence of EDs and sleep disturbances;
- Paucity of studies evaluating the effects of multidisciplinary treatments on sleep and circadian rhythms of patients diagnosed with EDs;
- Lack of studies and interventions that combine sleep and circadian rhythms-focused techniques with traditional ED programs.

Therefore, to overcome these gaps, we propose the following recommendations:

1. Conduct rigorous research on sleep and circadian rhythms among individuals affected by EDs, employing both subjective and objective instruments and combining them with other psychopathological assessments (i.e., eating-related symptoms, body image outcomes, physical activity levels, anxiety and mood symptomatology), as well as clinical outcomes (i.e., hormone levels), even with longitudinal designs. Such studies may help to recognize mechanisms underlying the presence of sleep alterations and circadian disruptions among people with disordered eating.
2. Study the effects of key socio-demographic features in ED care – such as the age at the disease onset and illness duration – in conjunction with disturbances of wake-sleep cycle and circadian rhythms, in order to identify effective trajectories for chronic patients with EDs.
3. Focus more extensively on the investigation of under-represented ED diagnoses, such as BN and BED. Even less prevalent diagnoses, such as Avoidant-Restrictive Food Intake Disorder and/or Other Specified Feeding and Eating Disorders (i.e., Atypical Anorexia Nervosa), should be fully taken into consideration.
4. Convene researchers, clinicians and other experts in ED care to promote the knowledge and implication of sleep processes and circadian rhythms, as well as their research approaches.
5. Inform clinicians of the importance of addressing sleep and circadian features in the rehabilitation from ED and promote the integration of specific interventions into evidence-based treatments.

Limitations of the Study

As an intrinsic limitation of the adopted methodology, the quality of the review depends on the quality of the included studies. While all selected studies were of acceptable or good quality, they were predominantly case-control or quasi-experimental studies, which carry inherent methodological weaknesses and threats to validity, such as the presence of potential confounding variables (e.g., participants' education level, socioeconomic status, age) – which were not adequately controlled in the included studies – and limited generalizability. Specifically, the control group included in the current review was notably older than the clinical sample. Consequently, even the age mismatch may be a potential confounding factor. Moreover, the methodological nature of these studies does not allow to draw even causality explanations for the relationship among sleep alterations and EDs, as well as for the effects of interventions; longitudinal clinical research, such as randomized controlled trials, is therefore needed.

Furthermore, some sample features (such as socioeconomic status or age) were not adequately controlled among the clinical and the control group – thus potentially influencing the findings – or were not reported. For example, in two research^{106,108} details about the diagnostic EDs process were not provided, since diagnoses were self-reported by participants. In Malcolm et al study,¹⁰⁸ age significantly differed among groups; also, in AN sample the prevalence of other psychiatric comorbidities or clinically relevant sleep disorders was not established. Additionally, an alternative explanation for the frequent co-occurrence of EDs and sleep disturbances could lie in the presence of comorbid depressive or anxious symptoms, which are sometimes not adequately controlled for in these studies. Future research should therefore systematically account for these potential confounding factors.

Beyond study-level limitations, some methodological constraints pertain to the review itself. First, the selection of studies was restricted to publications in specific idioms (i.e., English, Italian, Spanish, French and German), which may have introduced language bias and led to the exclusion of relevant evidence published in other languages. This is particularly relevant given that research on EDs and sleep is conducted across diverse cultural contexts. Second, although the search strategy was designed to be comprehensive, reliance on predefined search terms and keywords may have influenced study identification. Variability in terminology may have resulted in potentially eligible studies not being captured. In addition, despite efforts to retrieve all identified records, not all studies were accessible in full text, which may have further limited the completeness of the synthesis. Furthermore, by excluding correlational studies, the breadth of evidence considered may have been reduced. We acknowledge this as a limitation and highlight it as an important direction for future research, emphasizing the need for complementary study designs to fully explore the complex interactions between sleep, circadian rhythms, and disordered eating behaviors.

Finally, while this review provides an updated and integrative overview of the literature on sleep, circadian rhythms, and EDs, a more explicit comparison with recent prior systematic reviews would further clarify its specific contribution within the existing body of evidence. Future work synthesizing findings across reviews may help delineate areas of convergence, methodological gaps, and priorities for future investigation.

Conclusion

Encouraging research on EDs that goes beyond variables directly tied to eating symptomatology – and that includes factors such as sleep and circadian rhythms – is first and foremost an ethical imperative. It also represents a valuable opportunity to enrich the treatments we offer as clinicians, enabling more individuals to achieve stable and effective rehabilitation. By systematically synthesizing recent studies from the past five years and integrating them with findings from prior reviews, this work provides a unified and updated framework of current evidence. In particular, the review clarifies distinctions between general sleep characteristics, ED-focused interventions, and sleep- or circadian-specific treatments, while mapping methodological strengths, gaps, and emerging directions.

This structured overview offers concrete guidance for either researchers or clinicians. Future investigations are encouraged to adopt longitudinal and interventional designs, broaden diagnostic representation across ED populations, and incorporate objective sleep and circadian assessments. Clinically, recognizing and targeting sleep and circadian processes within assessment and treatment planning could enhance rehabilitation outcomes and inform more personalized care strategies.

Overall, this review not only highlights the clinical and ethical relevance of sleep and circadian rhythms in EDs but also provides a practical roadmap for advancing research and intervention. We hope that our synthesis and recommendations will support the development of evidence-informed approaches, ultimately contributing to more effective, comprehensive, and patient-centered care for individuals affected by EDs.

Data Sharing Statement

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

Author Contributions

Giulia Cera: conceptualization, data curation, investigation, methodology, formal analysis, writing – original draft, writing – review & editing; Serena Scarpelli: conceptualization, data curation, formal analysis, writing – original draft;

Silvamaría Mastrocola: data curation, formal analysis, writing – review & editing; Margherita Boltri: methodology, visualization, writing – review & editing; Federico Brusa: methodology, visualization, writing – review & editing; Alberto Scalia: methodology, visualization, writing – review & editing; Maurizio Gorgoni: investigation, supervision, writing – review & editing; Carolina Lombardi: conceptualization, supervision, writing – original draft, writing – review & editing; Leonardo Mendolicchio: conceptualization, supervision, writing – original draft, writing – review & editing; Luigi De Gennaro: conceptualization, supervision, writing – original draft, writing – review & editing. Luigi De Gennaro and Leonardo Mendolicchio share the last authorship. All authors took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This work was not funded.

Disclosure

The authors declare that they have no competing interests.

References

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders* (5th edition, text revision). 2022. doi:10.1176/appi.books.9780890425787.
2. Alvarez GG, Ayas NT. The impact of daily sleep duration on health: a review of the literature. *Prog Cardiovasc Nurs*. 2004;19(2):56–59. doi:10.1111/J.0889-7204.2004.02422.X
3. Degasperis G, Meneo D, Curati S, Cardi V, Baglioni C, Cellini N. Sleep quality in eating disorders: a systematic review and meta-analysis. *Sleep Med Rev*. 2024;77:101969. doi:10.1016/j.smrv.2024.101969
4. Klinzing JG, Niethard N, Born J. Mechanisms of systems memory consolidation during sleep. *Nat Neurosci*. 2019;22(10):1598–1610. doi:10.1038/S41593-019-0467-3
5. Kuna K, Szewczyk K, Gabrylska A, et al. Potential role of sleep deficiency in inducing immune dysfunction. *Biomedicine*. 2022;10(9):2159. doi:10.3390/B10M092159
6. Bazzani A, Faraguna U. Eat well, sleep well: exploring the association between eating behavior and sleep quality. *Nutrients*. 2025;17(17):2908. doi:10.3390/nu17172908
7. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. *Sleep*. 2010;33(5):585–592. doi:10.1093/sleep/33.5.585
8. Burger P, Bos RW, Maas J, Simeunovic-Ostojic M, Gemke RJB. Sleep disturbances in anorexia nervosa. *Eur Eat Disord Rev*. 2025;33(2):318–342. doi:10.1002/ERV.3148
9. Leger D, Bayon V, de Sanctis A. The role of sleep in the regulation of body weight. *Mol Cell Endocrinol*. 2015;418:101–107. doi:10.1016/j.mce.2015.06.030
10. Andreeva VA, Perez-Jimenez J, St-Onge MP. A systematic review of the bidirectional association between consumption of ultra-processed food and sleep parameters among adults. *Curr Obes Rep*. 2023;12(4):439–452. doi:10.1007/s13679-023-00512-5
11. St-Onge MP, Mikic A, Pietrolungo CE. Effects of diet on sleep quality. *Adv Nutr*. 2016;7(5):938–949. doi:10.3945/an.116.012336
12. Gamble KL, Berry R, Frank SJ, Young ME. Circadian clock control of endocrine factors. *Nat Rev Endocrinol*. 2014;10(8):466–475. doi:10.1038/NREND0.2014.78
13. Hebebrand J, Denecke S, Antel J. The role of leptin in rodent and human sleep: a transdiagnostic approach with a particular focus on anorexia nervosa. *Neurosci Biobehav Rev*. 2023;149. doi:10.1016/j.neubiorev.2023.105164
14. Binks H, Vincent GE, Gupta C, Irwin C, Khalesi S. Effects of diet on sleep: a narrative review. *Nutrients*. 2020;12(4):936. doi:10.3390/NU12040936
15. Baglioni C, Nanovska S, Regen W, et al. Sleep and mental disorders: a meta-analysis of polysomnographic research. *Psychol Bull*. 2016;142(9):969–990. doi:10.1037/BUL0000053
16. Pandi-Perumal SR, Monti JM, Burman D, et al. Clarifying the role of sleep in depression: a narrative review. *Psychiatry Res*. 2020;291. doi:10.1016/j.psychres.2020.113239
17. Carollo A, Zhang P, Yin P, et al. Sleep profiles in eating disorders: a scientometric study on 50 years of clinical research. *Healthc*. 2023;11(14):2090. doi:10.3390/HEALTHCARE11142090
18. Takahashi JS, Hong HK, Ko CH, McDearmon EL. The genetics of mammalian circadian order and disorder: implications for physiology and disease. *Nat Rev Genet*. 2008;9(10):764–775. doi:10.1038/NRG2430
19. Wilcox H, Paz V, Saxena R, Winkelman JW, Garfield V, Dashti HS. The role of circadian rhythms and sleep in anorexia nervosa. *JAMA Network Open*. 2024;7(1):E2350358. doi:10.1001/JAMANETWORKOPEN.2023.50358
20. Zee PC, Attarian H, Videnovic A. Circadian rhythm abnormalities. *Continuum*. 2013;19(1 Sleep Disorders):132–147. doi:10.1212/01.CON.0000427209.21177.AA
21. Kandeger A, Egilmez U, Selvi Y. Feeding and eating disorders in the context of circadian rhythms. *Alpha Psychiatry*. 2021;22(6):278–284. doi:10.1530/ALPHAPSYCHIATRY.2021.21151

22. Selvi Y, Aydin A, Atli A, Boysan M, Selvi F, Besiroglu L. Chronotype differences in suicidal behavior and impulsivity among suicide attempters. *Chronobiol Int.* 2011;28(2):170–175. doi:10.3109/07420528.2010.535938
23. Mendoza J. Food intake and addictive-like eating behaviors: time to think about the circadian clock(s). *Neurosci Biobehav Rev.* 2019;106:122–132. doi:10.1016/j.neubiorev.2018.07.003
24. Welsh DK, Takahashi JS, Kay SA. Suprachiasmatic nucleus: cell autonomy and network properties. *Annu Rev Physiol.* 2010;72(72):551–577. doi:10.1146/annurev-physiol-021909-135919
25. Mohawk JA, Green CB, Takahashi JS. Central and peripheral circadian clocks in mammals. *Annu Rev Neurosci.* 2012;35(35):445–462. doi:10.1146/annurev-neuro-060909-153128
26. Nagai K, Nishio T, Nakagawa H, Nakamura S, Fukuda Y. Effect of bilateral lesions of the suprachiasmatic nuclei on the circadian rhythm of food-intake. *Brain Res.* 1978;142(2):384–389. doi:10.1016/0006-8993(78)90648-0
27. Van Den Pol AN, Powley T. A fine-grained anatomical analysis of the role of the rat suprachiasmatic nucleus in circadian rhythms of feeding and drinking. *Brain Res.* 1979;160(2):307–326. doi:10.1016/0006-8993(79)90427-X
28. de Castro JM. Circadian rhythms of the spontaneous meal pattern, macronutrient intake, and mood of humans. *Physiol Behav.* 1987;40(4):437–446. doi:10.1016/0031-9384(87)90028-X
29. Poirel C, Larouche B. Murine circadian patterns of eating and drinking behaviors revisited: rhythmometric analyses and neuro-ethological comments. *Chronobiologia.* 1986;13(4):345–350.
30. Possidente B, Birnbaum S. Circadian rhythms for food and water consumption in the mouse, *Mus musculus*. *Physiol Behav.* 1979;22(4):657–660. doi:10.1016/0031-9384(79)90226-9
31. Stephan FK, Zucker I. Circadian rhythms in drinking behavior and locomotor activity of rats are eliminated by hypothalamic lesions. *Proc Natl Acad Sci.* 1972;69(6):1583–1586. doi:10.1073/pnas.69.6.1583
32. Bainier C, Mateo M, Felder-Schmittbuhl MP, Mendoza J. Circadian rhythms of hedonic drinking behavior in mice. *Neuroscience.* 2017;349:229–238. doi:10.1016/j.neuroscience.2017.03.002
33. Vujovic N, Gooley JJ, Zhou TC, Saper CB. Projections from the subparaventricular zone define four channels of output from the circadian timing system. *J Comp Neurol.* 2015;523(18):2714–2737. doi:10.1002/cne.23812
34. Moore J. JD. Insurers, AHA-IRI (AHA Insurance Resource Inc.) to develop products. *Mod Healthc.* 1995;25(25):88.
35. De Young KP, Bottera AR. A biobehavioral circadian model of restrictive eating and binge eating. *Int J Eat Disord.* 2022;55(10):1291–1295. doi:10.1002/EAT.23758
36. Challet E. The circadian regulation of food intake. *Nat Rev Endocrinol.* 2019;15(7):393–405. doi:10.1038/s41574-019-0210-x
37. De Young KP, Bottera AR, Kambanis PE. Sleep/waketime preference and delayed diurnal eating rhythms are associated through light exposure timing and modified by sleep efficiency. *Appetite.* 2022;170:105904. doi:10.1016/j.appet.2021.105904
38. McNeil J, Forest G, Hintze LJ, Brunet JF, Doucet É. The effects of partial sleep restriction and altered sleep timing on olfactory performance. *Eur J Clin Nutr.* 2017;71(12):1471–1472. doi:10.1038/ejcn.2017.109
39. Romo-Nava F, Guerdjikova AI, Mori NN, et al. A matter of time: a systematic scoping review on a potential role of the circadian system in binge eating behavior. *Front Nutr.* 2022;9:978412. doi:10.3389/fnut.2022.978412
40. Cooper AR, Loeb KL, McGlinchey EL. Sleep and eating disorders: current research and future directions. *Curr Opin Psychol.* 2020;34:89–94. doi:10.1016/j.copsyc.2019.11.005
41. Della Marca G, Farina B, Mennuni GF, et al. Microstructure of sleep in eating disorders: preliminary results. *Eat Weight Disord.* 2004;9(1):77–80. doi:10.1007/BF03325049
42. Kim KR, Jung YC, Shin MY, Namkoong K, Kim JK, Lee JH. Sleep disturbance in women with eating disorder: prevalence and clinical characteristics. *Psychiatry Res.* 2010;176(1):88–90. doi:10.1016/j.psychres.2009.03.021
43. Lombardo C, Battagliese G, Venezia C, Salvemini V. Persistence of poor sleep predicts the severity of the clinical condition after 6months of standard treatment in patients with eating disorders. *Eat Behav.* 2015;18:16–19. doi:10.1016/j.eatbeh.2015.03.003
44. Lundgren JD, O'Reardon JP, Allison KC, Spesser CD. Sleep and quality of life in eating disorders. In: Verster JC, Pandi-Perumal SR, Streiner DL editors. *Sleep and Quality of Life in Clinical Medicine*. Humana Press; 2008:281–289. doi:10.1007/978-1-60327-343-5
45. Padez-Vieira F, Afonso P. Sleep disturbances in anorexia nervosa. *Adv Eat Disord.* 2016;4(2):176–188. doi:10.1080/21662630.2016.1175958
46. de la Torre-Luque A, Miró E, Martínez Narváez MP. Calidad subjetiva de sueño en pacientes con Trastornos de la Conducta Alimentaria. *Apunt Psicol.* 2013;31(1):11–20. doi:10.55414/se2tjk24
47. Menculini G, Brufani F, Del Bello V, Moretti P, Tortorella A. Circadian rhythms disruptions and eating disorders: clinical impact and possible psychopathological correlates. *Psychiatry Danub.* 2019;31(Suppl 3):497–502.
48. Zou H, Zhou H, Yan R, Yao Z, Lu Q. Chronotype, circadian rhythm, and psychiatric disorders: recent evidence and potential mechanisms. *Front Neurosci.* 2022;16. doi:10.3389/FNINS.2022.811771
49. Natale V, Ballardini D, Schumann R, Mencarelli C, Magelli V. Morningness-eveningness preference and eating disorders. *Pers Individ Dif.* 2008;45(6):549–553. doi:10.1016/J.PAID.2008.06.014
50. Maukonen M, Kanerva N, Partonen T, et al. Chronotype differences in timing of energy and macronutrient intakes: a population-based study in adults. *Obesity.* 2017;25(3):608–615. doi:10.1002/oby.21747
51. Allison KC, Spaeth A, Hopkins CM. Sleep and eating disorders. *Curr Psychiatry Rep.* 2016;18(10). doi:10.1007/s11920-016-0728-8
52. Christensen KA, Short NA. The case for investigating a bidirectional association between insomnia symptoms and eating disorder pathology. *Int J Eat Disord.* 2021;54(5):701–707. doi:10.1002/EAT.23498
53. Irish LA, Bottera AR, Manasse SM, Christensen Pacella KA, Schaefer LM. The integration of sleep research into eating disorders research: recommendations and best practices. *Int J Eat Disord.* 2024;57(9):1816–1827. doi:10.1002/EAT.24241
54. Neil JF, Merikangas JR, Foster FG, Merikangas KR, Spiker DG, Kupfer DJ. Waking and all-night sleep eeg's in anorexia nervosa. *Clin Electroencephalogr.* 1980;11(1):9–15. doi:10.1177/155005948001100102
55. Walsh BT, Goetz R, Roose SP, Fingerth S, Glassman AH. EEG-monitored sleep in anorexia nervosa and bulimia. *Biol Psychiatry.* 1985;20(9):947–956. doi:10.1016/0006-3223(85)90191-X
56. Delvenne V, Kerkhofs M, Appelboom-Fondu J, Lucas F, Mendlewicz J. Sleep polygraphic variables in anorexia nervosa and depression: a comparative study in adolescents. *J Affect Disord.* 1992;25(3):167–172. doi:10.1016/0165-0327(92)90002-N

57. Levy AB, Dixon KN, Schmidt H. Sleep architecture in anorexia nervosa and bulimia. *Biol Psychiatry*. 1988;23(1):99–101. doi:10.1016/0006-3223(88)90112-6
58. Nobili L, Baglietto MG, De Carli F, et al. A quantified analysis of sleep electroencephalography in anorectic adolescents. *Biol Psychiatry*. 1999;45(6):771–775. doi:10.1016/S0006-3223(98)00107-3
59. Burbiel I, Köppen U, Thome A, Stein G. Schlafuntersuchungen bei Patienten mit Anorexia Nervosa im Vergleich zu Patienten anderer humanstruktureller Diagnosegruppen. [Sleep research with patients suffering from anorexia nervosa compared to other human-structural diagnosis groups]. *Dynamische Psychiatr*. 1991;24(1–2):34–55.
60. Nobili L, Baglietto MG, Beelke M, et al. Impairment of the production of delta sleep in anorectic adolescents. *Sleep*. 2004;27(8):1553–1559. doi:10.1093/SLEEP/27.8.1553
61. Latzer Y, Tzischinsky O, Epstein R. Sleep-wake monitoring in women suffering from anorexia nervosa. *Eat Disord*. 2001;9(2):159–166. doi:10.1080/10640260127713
62. Sauchelli S, Jiménez-Murcia S, Sánchez I, et al. Orexin and sleep quality in anorexia nervosa: clinical relevance and influence on treatment outcome. *Psychoneuroendocrinology*. 2016;65:102–108. doi:10.1016/j.psyneuen.2015.12.014
63. Lauer C, Zully J, Krieg JC, Riemann D, Berger M. EEG sleep and the cholinergic REM induction test in anorexic and bulimic patients. *Psychiatry Res*. 1988;26(2):171–181. doi:10.1016/0165-1781(88)90072-8
64. Lauer C, Krieg JC, Zully J, Riemann D, Berger M. Der Schlaf bei Anorexia nervosa, Bulimia nervosa und depressiven Erkrankungen: Eine polysomnographische Vergleichsstudie. *Fortschritte der Neurol Psychiatr*. 1989;57(10):403–410. doi:10.1055/S-2007-1001136
65. Lehmann CS, Hofmann T, Elbelt U, et al. The role of objectively measured, altered physical activity patterns for body mass index change during inpatient treatment in female patients with anorexia nervosa. *J Clin Med*. 2018;7(9):289. doi:10.3390/JCM7090289
66. Hudson JI, Pope HG, Jonas JM, et al. Sleep EEG in bulimia. *Biol Psychiatry*. 1987;22(7):820–828. doi:10.1016/0006-3223(87)90081-3
67. Latzer Y, Tzischinsky O, Epstein R, Klein E, Peretz L. Naturalistic sleep monitoring in women suffering from bulimia nervosa. *Int J Eat Disord*. 1999;26(3):315–321. doi:10.1002/(SICI)1098-108X(199911)26:3<315::AID-EAT9>3.0.CO;2-6
68. Waller DA, Hardy BW, Pole R, et al. Sleep EEG in bulimic, depressed, and normal subjects. *Biol Psychiatry*. 1989;25(5):661–664. doi:10.1016/0006-3223(89)90233-3
69. Tu CY, Meg Tseng MC, Chang CH. Night eating syndrome in patients with eating disorders: is night eating syndrome distinct from bulimia nervosa? *J Formos Med Assoc*. 2019;118(6):1038–1046. doi:10.1016/j.jfma.2018.10.010
70. Tzischinsky O, Latzer Y. Sleep-wake cycles in obese adolescents with and without binge eating episodes. *Eur Eat Disord Rev*. 2006;14(2):111–117. doi:10.1002/ERV.674;REQUESTEDJOURNAL:JOURNAL:10990968;WGROU:STRING:PUBLICATION
71. Tzischinsky O, Latzer Y, Epstein R, Tov N. Sleep-wake cycles in women with binge eating disorder. *Int J Eat Disord*. 1999;27(1):43–48. doi:10.1002/(sici)1098-108x(200001)27:1<43::aid-eat5>3.0.co;2-z
72. Roveda E, Montaruli A, Galasso L, et al. Rest-activity circadian rhythm and sleep quality in patients with binge eating disorder. *Chronobiol Int*. 2018;35(2):198–207. doi:10.1080/07420528.2017.1392549
73. Galasso L, Montaruli A, Mulè A, et al. The multidisciplinary therapy in binge eating disorder is able to influence the interdaily stability and sleep quality? *Chronobiol Int*. 2019;36(10):1311–1315. doi:10.1080/07420528.2019.1650059
74. Vardar E, Caliyurt O, Arikan E, Tuglu C. Sleep quality and psychopathological features in obese binge eaters. *Stress Heal*. 2004;20(1):35–41. doi:10.1002/SMI.992
75. Linnaranta O, Bourguignon C, Crescenzi O, et al. Late and instable sleep phasing is associated with irregular eating patterns in eating disorders. *Ann Behav Med*. 2020;54(9):680–690. doi:10.1093/ABM/KAAA012
76. Asaad Abdou T, Esawy HI, Abdel Razek Mohamed G, et al. Sleep profile in anorexia and bulimia nervosa female patients. *Sleep Med*. 2018;48:113–116. doi:10.1016/j.sleep.2018.03.032
77. El Ghoch M, Calugi S, Bernabè J, et al. Sleep patterns before and after weight restoration in females with anorexia nervosa: a longitudinal controlled study. *Eur Eat Disord Rev*. 2016;24(5):425–429. doi:10.1002/ERV.2461
78. Lim M, Kou R, Esposito G, Jawed A, Dimitriou D, Mangar SA. Exploring the relationship between disordered sleep and mood in male anorexia nervosa: an actigraphy study. *Nutrients*. 2023;15(9):2176. doi:10.3390/nu15092176
79. Ralph-Nearman C, Williams BM, Ortiz AML, Smith AR, Levinson CA. Pinpointing core and pathway symptoms among sleep disturbance, anxiety, worry, and eating disorder symptoms in anorexia nervosa and atypical anorexia nervosa. *J Affect Disord*. 2021;294:24–32. doi:10.1016/j.jad.2021.06.061
80. Bat-Pitault F, Da Silva C, Charvin I, et al. Rationale for the application of exposure and response prevention to the treatment of anorexia nervosa. *Int J Eat Disord*. 2011;26(2):1845–1852. doi:10.1007/s40519-020-01033-z
81. Bat-Pitault F, Da Silva C, Charvin I, Da Fonseca D. Sleep disturbances in anorexia nervosa subtypes in adolescence. *Eat Weight Disord*. 2021;26(6):1845–1852. doi:10.1007/s40519-020-01033-z
82. Lloyd EC, Sallis HM, Verplanken B, Haase AM, Munafò MR. Understanding the nature of association between anxiety phenotypes and anorexia nervosa: a triangulation approach. *BMC Psychiatry*. 2020;20(1):495. doi:10.1186/s12888-020-02883-8
83. Özcan ÖÖ, Çevreli B. A comparative neuropsychological assessment of anorexia nervosa, bulimia nervosa, binge eating disorder and healthy control: a cross-sectional study in female university students. *BMC Psychol*. 2025;13(1):1209. doi:10.1186/s40359-025-03515-0
84. Antunes LC, Elkfury JL, Parizotti CS, et al. Longer cortical silent period length is associated to binge eating disorder: an exploratory study. *Front Psychiatry*. 2020;11:11. doi:10.3389/FPSYT.2020.559966
85. Velimirović M, Campione M, Manwaring J, et al. Sleep disturbances, night eating, and depression in patients with binge-eating disorder. *J Psychiatr Res*. 2025;189:488–494. doi:10.1016/j.jpsychires.2025.07.008
86. Lauer CJ, Krieg JC. Sleep in eating disorders. *Sleep Med Rev*. 2004;8(2):109–118. doi:10.1016/S1087-0792(02)00122-3
87. Dashti HS, Chen A, Daghlas I, Saxena R. Morning diurnal preference and food intake: a Mendelian randomization study. *Am J Clin Nutr*. 2020;112(5):1348–1357. doi:10.1093/ajcn/nqaa219
88. Garaulet M, Vizmanos B, Muela T, et al. Evening types as determined by subjective and objective measures are more emotional eaters. *Obesity*. 2023;31(5):1192–1203. doi:10.1002/OBY.23749
89. Greeno CG, Wing RR, Marcus MD. Nocturnal eating in binge eating disorder and matched-weight controls - Greeno - 1995. *Int J Eat Disord*. 1995;18(4):343–349. doi:10.1002/1098-108X(199512)18:4<343::AID-EAT2260180407>3.0.CO;2-P

90. Rand CSW, Macgregor AMC, Stunkard AJ. The night eating syndrome in the general population and among postoperative obesity surgery patients. *Int J Eat Disord.* 1998;22(1):65–69. doi:10.1002/(sici)1098-108x(199707)22:1<65::aid-eat8>3.0.co;2-0
91. Sun X, Gustat J, Bertisch SM, Redline S, Bazzano L. The association between sleep chronotype and obesity among black and white participants of the Bogalusa Heart Study. *Chronobiol Int.* 2020;37(1):123–134. doi:10.1080/07420528.2019.1689398
92. Grimaldi M, Bacaro V, Natale V, Tonetti L, Crocetti E. The longitudinal interplay between sleep, anthropometric indices, eating behaviors, and nutritional aspects: a systematic review and meta-analysis. *Nutrients.* 2023;15(14):3179. doi:10.3390/nu15143179
93. Opitz MC, Trompeter N, Rabelo-da-Ponte FD, et al. The association between disordered eating and sleep in non-clinical populations—a systematic review and meta-analysis. *J Sleep Res.* 2025;35(2). doi:10.1111/jsr.70117
94. da Luz FQ, Sainsbury A, Salis Z, et al. A systematic review with meta-analyses of the relationship between recurrent binge eating and sleep parameters. *Int J Obes.* 2023;47(3):145–164. doi:10.1038/S41366-022-01250-9
95. Kim S, Lee HJ. Sleep and circadian rhythm disturbances in eating disorders. *Chronobiol Med.* 2020;2(4):141–147. doi:10.33069/cim.2020.0027
96. Abad VC, Guilleminault C. Sleep and psychiatry. *Dialogues Clin Neurosci.* 2005;7(4):291–303. doi:10.31887/DCNS.2005.7.4/vabad
97. Ivanenko A, Johnson K. Sleep disturbances in children with psychiatric disorders. *Semin Pediatr Neurol.* 2008;15(2):70–78. doi:10.1016/j.spen.2008.03.008
98. Bryson C, Douglas D, Schmidt U. Established and emerging treatments for eating disorders. *Trends Mol Med.* 2024;30(4):392–402. doi:10.1016/j.molmed.2024.02.009
99. Himmerich H, Lewis YD, Conti C, et al. World Federation of Societies of Biological Psychiatry (WFSBP) guidelines update 2023 on the pharmacological treatment of eating disorders. *World J Biol Psychiatry.* 2023;24(8):643–706. doi:10.1080/15622975.2023.2179663
100. Crone C, Fochtmann LJ, Attia E, et al. The American psychiatric association practice guideline for the treatment of patients with eating disorders. *Am J Psychiatry.* 2023;180(2):167–171. doi:10.1176/APPI.AJP.23180001
101. Monteleone AM, Pellegrino F, Croatto G, et al. Treatment of eating disorders: a systematic meta-review of meta-analyses and network meta-analyses. *Neurosci Biobehav Rev.* 2022;132:142. doi:10.1016/j.neubiorev.2022.104857
102. Bartlett D, Jackson ML. The bidirectional nature of sleep problems and psychopathology. *Med Today.* 2016;17(3):23–28.
103. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ.* 2021;372. doi:10.1136/bmj.N71
104. Cera G, Scarpelli S. Recent advances in sleep and circadian characteristics in eating disorders: a comprehensive short systematic review of the last five years. 2025.
105. Ouzzani M, Hammady H, Fedorowicz Z, Elmagarmid A. Rayyan-a web and mobile app for systematic reviews. *Syst Rev.* 2016;5(1). doi:10.1186/S13643-016-0384-4
106. Kenny S, Robillard R, Saad M, et al. The association between sleep and eating disorders in Canada before and during the first wave of the COVID-19 pandemic. *J Affect Disord Reports.* 2025;19:100860. doi:10.1016/J.JADR.2024.100860
107. Kleppe MM, Kessler U, Rekkedal GÅ, Skjåkødegård HF, Danielsen YS. Differences in sleep patterns between patients with anorexia nervosa and healthy controls: a cross-sectional study. *J Eat Disord.* 2023;11(1). doi:10.1186/S40337-023-00799-8
108. Malcolm A, Toh WL, Crocker K, Phillipou A. The contribution of sleep to anorexia nervosa severity. *Eat Weight Disord.* 2022;27(4):1563–1568. doi:10.1007/S40519-021-01286-2
109. Romigi A, Mercuri NB, Caccamo M, et al. Subjective sleep disorders and daytime sleepiness in patients with restrictive type anorexia nervosa and effects on quality of life: a case-control study. *Sleep Biol Rhythms.* 2022;20(2):181–189. doi:10.1007/S41105-021-00352-7
110. Crevits L, Silva C, Bat-Pitault F. Preliminary study: cognitive behavioural therapy for insomnia in adolescents with anorexia nervosa. *Eat Weight Disord.* 2024;29(1). doi:10.1007/S40519-023-01634-4
111. Barker TH, Habibi N, Aromataris E, et al. The revised JBI critical appraisal tool for the assessment of risk of bias for quasi-experimental studies. *JBI Evid Synth.* 2024;22(3):378–388. doi:10.11124/JBIES-23-00268
112. Martínez-Sánchez SM, Martínez-García TE, Munguía-Izquierdo D. Clinical, psychopathological, physical, and sleep evolution in adolescents with restrictive anorexia nervosa participating in a day hospital program. *Psychiatry Invest.* 2020;17(4):366–374. doi:10.30773/PI.2020.0016
113. Meule A, Riemann D, Voderholzer U. Sleep quality in persons with mental disorders: changes during inpatient treatment across 10 diagnostic groups. *J Sleep Res.* 2023;32(2). doi:10.1111/jsr.13624
114. Kratochwill TR, Hitchcock JH, Horner RH, et al. Single-case intervention research design standards. *Remedial Spec Educ.* 2013;34(1):26–38. doi:10.1177/0741932512452794
115. Christensen Pacella KA, Bottera AR, Forbush KT, Isquith J, De Young K. Feasibility and acceptability of brief behavioral treatment for insomnia after eating disorder treatment: the TIRED study. *Int J Eat Disord.* 2025;58(12):2451–2464. doi:10.1002/eat.24540
116. Christensen Pacella KA, Chen Y, Bottera AR, Forbush KT. Changes in insomnia and binge-eating symptom severity before and after treatment of eating disorders in individuals with non-low-weight binge-spectrum disorders. *J Sleep Res.* 2025;34(3):e14378. doi:10.1111/jsr.14378
117. Kambanis PE, Bottera AR, De Young KP. Responses to bright light exposure in individuals with binge-spectrum eating disorders characterized by high dietary restraint and negative affect. *Int J Eat Disord.* 2023;56(12):2250–2259. doi:10.1002/eat.24054
118. von Uexküll T, Wesiack W. *Kapitel 1 - Integrierte Medizin als Gesamtkonzept der Heilkunde: Ein bio-psycho-soziales Modell.* Adler RH, Herzog W, Joraschky P, eds. *Uexküll – Psychosomatische Medizin.* Urban & Fischer; 2011:3–40. doi:10.1016/B978-3-437-21831-6.10001-2
119. Zipfel S, Herzog W, Kruse J, Henningsen P. Psychosomatic medicine in Germany: more timely than ever. *Psychother Psychosom.* 2016;85(5):262–269. doi:10.1159/000447701
120. Bohrer BK, Chen Y, Christensen KA, et al. A pilot multiple-baseline study of a mobile cognitive behavioral therapy for the treatment of eating disorders in university students. *Int J Eat Disord.* 2023;56(8):1623–1636. doi:10.1002/eat.23987
121. Forbush KT, Christensen Pacella KA, Thomeczek ML, et al. The building healthy eating and self-esteem together for university students mobile app to treat eating disorders: user-centered research design and feasibility study. *JMIR Form Res.* 2023;7:e43504. doi:10.2196/43504
122. Lindberg N, Virkkunen M, Tani P, Appelberg B, Rimón R, Porkka-Heiskanen T. Growth hormone-insulin-like growth factor-1 axis, leptin and sleep in anorexia nervosa patients. *Neuropsychobiology.* 2003;47(2):78–85. doi:10.1159/000070013
123. Lauer CJ, Krieg JC. Weight gain and all-night EEG-sleep in anorexia nervosa. *Biol Psychiatry.* 1992;31(6):622–625. doi:10.1016/0006-3223(92)90250-4

124. Crisp AH, Stonehill E, Fenton GW. An aspect of the biological basis of the mind-body apparatus: the relationship between sleep, nutritional state and mood in disorders of weight. *Psychother Psychosom.* 1970;18(1):161–175. doi:10.1159/000286075
125. Lacey JH, Crisp AH, Kalucy RS, Hartmann MK, Chen CN. Weight gain and the sleeping electroencephalogram: study of 10 patients with anorexia nervosa. *Br Med J.* 1975;4(5996):556. doi:10.1136/BMJ.4.5996.556
126. Pieters G, Theys P, Vandereycken W, Leroy B, Peuskens J. Sleep variables in anorexia nervosa: evolution with weight restoration. *Int J Eat Disord.* 2004;35(3):342–347. doi:10.1002/EAT.10256
127. Beauchamp MT, Lundgren JD. A systematic review of bright light therapy for eating disorders. *Prim Care Companion CNS Disord.* 2016;18(5). doi:10.4088/PCC.16R02008

Nature and Science of Sleep

Publish your work in this journal

Nature and Science of Sleep is an international, peer-reviewed, open access journal covering all aspects of sleep science and sleep medicine, including the neurophysiology and functions of sleep, the genetics of sleep, sleep and society, biological rhythms, dreaming, sleep disorders and therapy, and strategies to optimize healthy sleep. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit <http://www.dovepress.com/testimonials.php> to read real quotes from published authors.

Submit your manuscript here: <https://www.dovepress.com/nature-and-science-of-sleep-journal>

Dovepress
Taylor & Francis Group