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Biological rhythms in premenstrual syndrome and premenstrual dysphoric disorder: a systematic review

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Abstract

Background Women with premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD) typically experience a range of psychological and physiological symptoms that negatively affect their quality of life. Disruption in biological rhythms, including alterations of the sleep–wake cycle, have been implicated in PMS/PMDD, though literature is still growing to substantiate these findings. The objective of this study is to systematically review the available literature on biological rhythms disruption in PMS/PMDD.

Methods A literature search was conducted on four databases (Pubmed, Embase, Medline, and Web of Science) on December 3rd, 2021. This search yielded a total of 575 articles that assessed the relationship between biological rhythms and PMS/PMDD/premenstrual symptoms.

Results After the exclusion of irrelevant articles and hand-searching references, 25 articles were included in this systematic review. Some studies showed that women with PMS/PMDD present lower melatonin levels, elevated nighttime core body temperature, and worse subjective perception of sleep quality when compared to women without PMS/PMDD. Other biological rhythms parameters showed either no differences between groups (wrist actimetry) or conflicting results (objective sleep parameters, cortisol, prolactin, and thyroid stimulating hormone).

Conclusion Current research demonstrates that women with PMS/PMDD experience lower melatonin levels, higher body temperature, and worse subjective perception of sleep quality. This review outlines some possible mechanisms behind these findings and proposes recommendations for future research. This systematic review was registered in the International Prospective Register of Systematic Reviews (PROSPERO) under registration number CRD42020149921.

Keywords Premenstrual syndrome, Premenstrual dysphoric disorder, Premenstrual symptoms, Biological rhythms, Circadian rhythms

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Introduction

Women experience great variability in the severity of premenstrual symptoms, ranging from mild to severe and incapacitating. Around 80–90% of women experience at least one mild premenstrual symptom and 30–40% experience premenstrual syndrome (PMS), which can cause significant distress and functional impairment [1]. A recent worldwide systematic review identified the prevalence for provisional PMDD to be 7.7%, whereas the prevalence for confirmed PMDD, which requires two months of prospective assessments, was 3.2% [2]. Due to the high heterogeneity in samples and methodology, restricting the data to community-based samples revealed a point prevalence of 1.6% [2]. PMDD has also been associated with increased suicidal behavior: a recent meta-analysis revealed that women with PMDD are 7×at higher risk of suicide attempts and 4×more likely to exhibit suicidal ideation when compared to women without premenstrual disturbances [3]. An increased risk of suicidal ideation, but not attempt, was also found in women with PMS [3].

The symptoms of both PMDD and PMS must occur in the luteal phase and subside within the first few days of menses, negatively affecting one's quality of life. According to the DSM-5-TR [4], PMDD is a depressive disorder that is diagnosed by at least five symptoms present during the luteal phase (LP), categorized into two main criteria. The first criterion addresses affect, including symptoms such as: affect lability; increase in irritability, anger, or interpersonal conflicts; depression, hopelessness, or self-deprecating thoughts; and greater anxiety/tension. The second criterion describes somatic and cognitive symptoms: insomnia or hypersomnia; lethargy, fatigue, or lack of energy; loss of interest; difficulty concentrating; changes in appetite or cravings; feeling overwhelmed; and physical symptoms such as muscle and joint pain, bloating, weight gain, or breast tenderness/swelling. These symptoms must be confirmed with two months of prospective charting to meet diagnostic criteria for PMDD. On the other hand, though PMS shares common symptoms to PMDD, its diagnostic definition is varied. According to the American College of Obstetricians and Gynecologists, the criteria for PMS diagnosis involves endorsement of at least one of the above symptoms with 2–3 months of prospective charting [5]. However, there is much variance in the evaluation of PMS in clinical and research environments and is often assessed using methods such as modified questionnaires stemming from DSM criteria for PMDD (some examples include: [6–8]).

The neurobiological mechanisms underlying PMS/PMDD are active areas of investigation and current research indicates a multifactorial etiology [9, 10]. An excess or deficit of ovarian hormones has not been supported—rather, emerging research shows that women with PMS/PMDD may have an altered sensitivity to predictable levels of hormonal fluctuations [11–13]. A recent longitudinal study outlined three subtypes of PMDD according to symptom trajectory: severe in full luteal phase, severe in premenstrual week (with late offset), and moderate in premenstrual week [14]. This promising work supports the benefit of further exploring the timing and interplay of physiological, psychological, and psychosocial factors to uncover the etiology of PMS/PMDD.

Disruption in biological rhythms has been implicated in PMDD [15], which describe changes in biological processes that occur at regular intervals in an attempt to optimize physiological performance [16]. When these rhythms oscillate every 24 h, they are known as circadian rhythms [17], and are largely regulated by the central pacemaker of the circadian system, the suprachiasmatic nucleus (SCN). The SCN regulates the physiological cascades of numerous systems, such as the hormone melatonin, which acts as a cue for the sleep–wake cycle and follows a cyclical pattern of peaking in the first half of the night and reaching its decline in the early morning [18]. The hormone prolactin (PRL), which plays an important role in regulating hormones that trigger egg development and ovulation, follows a similar circadian pattern [19]. Body temperature also dips to its lowest in the early morning and peaks in the afternoon and evening [20]. It is essential in maintaining physiological thermal homeostasis and regulating the menstrual cycle, whereby it peaks shortly after ovulation has occurred [21]. A similar pattern is seen in the thyroid stimulating hormone (TSH), which aids in regulating the menstrual cycle by modulating the release of thyroid hormones [22]. Similarly, cortisol, which is involved in the synchronization of reproductive hormones, peaks in the early morning and declines throughout the day [23, 24]. The circadian system is thereby directly and indirectly involved in the regulation of numerous physiological processes and is a pillar of communication between these systems.

The most frequently studied and discussed aspect of circadian rhythms is the sleep–wake cycle, as it is largely dictated by the light–dark cycle and has great influence over numerous other circadian processes. Sleep complaints are common in women, and even more so in women who suffer from premenstrual complaints [15]. Disruptions in sleep and non-sleep related parameters are also reported in other mood disorders such as major depressive disorder (MDD) and bipolar disorder (BD) [25–27]. When compared to healthy controls, individuals with MDD experience a longer sleep onset latency, increased nocturnal body temperature, and dampened rhythms of body temperature, activity, melatonin,

cortisol, and TSH [27, 28]. Individuals with BD show longer total sleep time, lower levels of urinary melatonin, and dampened rhythms [27, 28]. Furthermore, one's perception of circadian rhythms is subjectively-rated as more disturbed by individuals with MDD and BD [28–30]. Considering the marked differences in biological rhythms of certain parameters in MDD and BD, it is pertinent to investigate how these rhythms may present in other depressive disorders like PMDD. Given the hormonal fluctuations during the menstrual cycle, it is crucial to understand how circadian parameters may change across the cycle, especially in individuals with heightened sensitivity to changes in reproductive hormones. However, there is a gap in the literature concerning disturbances in biological rhythms beyond sleep in PMS/PMDD, and to our knowledge, there are no systematic reviews on this subject.

This systematic review aims to synthesize the current research on PMS/PMDD and biological rhythms. As explained above, we selected markers of the endogenous circadian rhythm that typically display robust 24-h patterns and categorized them as sleep-related (objective and subjective sleep measures, melatonin, and wrist actimetry) and non-sleep-related (body temperature, cortisol, PRL, and TSH).

Methods

This systematic review was registered in the International Prospective Register of Systematic Reviews (PROSPERO) under registration number CRD42020149921 and structured under the guidelines of The Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) [31].

Search strategy

The article search was conducted on December 3rd, 2021 with no year or language restrictions, generating 575 articles from four databases: Pubmed = 214, Embase = 166, Medline = 105, Web of Science = 90. The search criteria used were (biological rhythm OR biological rhythms OR circadian rhythm OR circadian rhythms OR sleep disruption OR melatonin) AND (PMDD OR premenstrual dysphoric disorder OR PMS OR premenstrual syndrome OR premenstrual syndromes).

Our screening criteria required that articles: (1) compared women with PMS/PMDD and healthy controls; and (2) compared groups at baseline if the study involved an intervention. After removing duplicate articles, we excluded all articles that were: (1) case reports; (2) reviews or meta-analyses; (3) clinical trials with no baseline reports; and (4) studies that did not control for other primary psychiatric disorders. The articles were first screened by title, abstract, then full text. All articles were independently evaluated by two blinded reviewers (AN, LC) to determine if they met the inclusion criteria, and any discrepancies in the assessments were resolved by consensus between three reviewers (AN, LC, TC).

Given the limited number of studies in this area, studies that included samples of both provisional and confirmed diagnoses of PMDD were eligible to be included in our systematic review. Studies with PMS samples were eligible as long as they provided a detailed description of their assessment of PMS. Regarding healthy controls, studies were eligible if the healthy controls included in the study did not meet criteria for PMS/PMDD diagnosis or any Axis I psychiatric disorders.

Data extraction

Data were extracted from the final articles by four researchers (AN, LC, TC, JS). The first author, year, and country were identified, as well as the type of study design and the aim of the study. Sample characteristics were also extracted, including number of participants, ages, and diagnoses. Additionally, methodological data were extracted, such as the menstrual phase, instruments used for neuropsychological evaluation, questionnaires, and methods of obtaining samples. Lastly, the most relevant findings were summarized alongside the conclusion of an association between groups.

Quality assessment

Quality assessments of the final selection of articles were independently performed by three researchers (AN, LC, TC) using the Newcastle–Ottawa Quality Assessment Scale adapted for cross-sectional studies [32]. Any discrepancies in assessments were resolved by consensus. According to the DSM-5-TR [4], prospective daily ratings of at least two symptomatic cycles are necessary to confirm the diagnosis of PMDD. Studies that included this criterion, in addition to the provisional diagnosis detected through the clinical interview, received the maximum score in the *Ascertainment of Exposure* section of the quality control.

Results

The literature search yielded 575 articles. Once duplicates were removed, 394 articles remained. We excluded 341 articles based on the title/abstract and 32 based on full-text screening, for a total of 21 articles included in the systematic review. We hand-searched references of the included studies and found 4 additional articles, for a final number of 25 articles (Fig. 1).

Characteristics of included studies

Characteristics of the 25 included studies are shown in Table 1. Studies were published between 1989 and

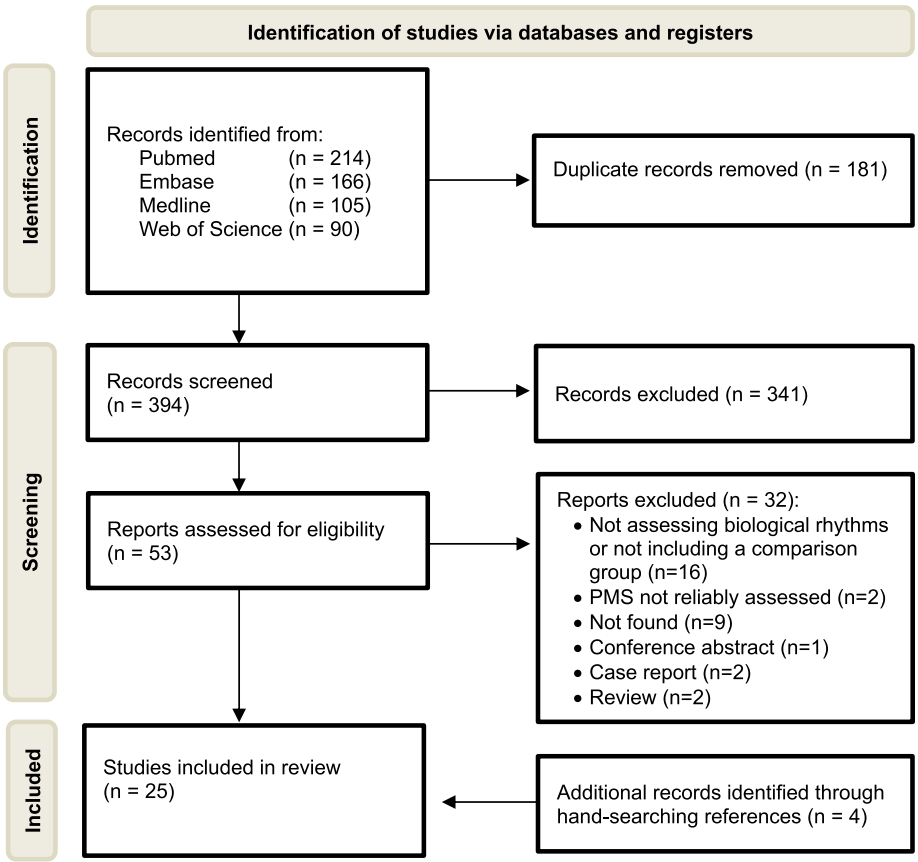

Fig. 1 Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) flow diagram for the articles included in the systematic review

2022 in the United States of America ($n=16$), Canada ($n=3$), Australia ($n=1$), Sweden ($n=1$), Japan ($n=1$), Slovakia ($n=1$), Germany ($n=1$), and one that included samples from both Canada and Brazil. The total sample size ranged from 12 to 660 and ages ranged from 18 to 45 years. Most of the studies assessed PMDD ($n=15$) or PMS ($n=7$), and some had mixed samples of PMS and PMDD ($n=3$). Due to the limited number of studies available, all samples will be referred to as "women with PMS/PMDD" and specific sample breakdowns can be found in Table 1. Any studies that used the DSM-III-R criteria for late luteal phase dysphoric disorder (LLPDD) will be referred to as PMDD, given the similarity in criteria when the change from LLPDD to PMDD was made in the DSM-IV [33, 34]. Most of the studies diagnosed PMDD using the DSM-III-R ($n=9$) and DSM-IV ($n=9$) were all prospectively-assessed for 2–3 months, with the exception of Beddig et al., (2019) (see Table 1 for details of each study). Three of these studies had a combined PMS and PMDD diagnosis: Severino et al., (1991) assigned PMDD diagnosis if additional criteria were met regarding symptom timing and change of severity from follicular to luteal phase; Baker et al., (2007) and Baker et al., (2012) assigned PMDD diagnosis if at least five DSM-IV PMDD symptoms were rated as severe. Studies that diagnosed PMS used Premenstrual Syndrome Self-Rating Scale [8] ($n=2$, not prospectively-assessed), the Menstrual Distress Questionnaire [35] ($n=1$, not prospectively-assessed), a modified version of the Premenstrual Symptoms Screening Tool (PSST) [7] ($n=1$, not prospectively-assessed), or questionnaires developed in-lab ($n=3$, one prospectively-assessed).

Quality assessments of included studies

The quality assessment scores ranged from 3 to 7 (maximum 10), with a mean of 5.84. The main factors impacting the quality of studies were sample selection bias, whereby most studies did not include a sample representative of the population, justify their sample size, or report response rate and characteristics between responders and non-responders (Table 2).

Sleep assessments

Seventeen out of 25 studies assessed sleep-related outcomes, which included polysomnography, melatonin

Table 1 Data extracted from studies in this systematic review

Author (year) Country	Relevant aim for the systematic review	Menstrual phase	Study design	Demographic	PMS / PMDD Assessment	Biological Rhythm Assessment	Association?	Relevant Findings
Mauri, Reid, and MacClean (1988) Canada [50]	To assess premenstrual sleep complaints in a clinical sample of women with PMS, and non-clinical samples of women with (HC+) and without (HC-) premenstrual symptoms	LP	Cross-sectional	PMS: $n=14$, mean age: 35.4±6.5 HC+: $n=11$, mean age: 34.2±4.8 HC-: $n=15$, mean age 32.2±8.2	PMTS	PSI	Yes	When compared to both HC+ and HC- groups, women with PMS reported: i) worse sleep quality, ii) frequent awakenings, iii) a long time going back to sleep after awakening, iv) feeling more tired in the morning
Mortola, Girton, and Yen (1989) USA [58]	To analyze pulsatile activity of serum cortisol in women with PMS and HC during	FP LP	Cross-sectional	PMS: $n=16$ HC: $n=16$ Age range of whole sample: 21–36	PMS criteria developed in lab COPE	24-h serum cortisol	No	No differences between PMS and HC in cortisol secretion levels, number of pulses, time of nadir, mean amplitude, and mean duration
Parry et al. (1989) USA [39]	To examine sleep parameters using EEG, as well as parameters of prolactin, cortisol, wrist actimetry, and body temperature in women with PMS and HC	EFP LFP ELP LFP	Cohort	PMS: $n=8$ HC: $n=8$ Mean age of whole sample: 30, range 26–45	DSM-III criteria for major depressive episode during the week before menses Daily sleep and mood ratings for 2 months	1) Sleep EEG 2) Serum cortisol 3) Wrist actimetry for activity and temperature	Yes	1) PMS experienced more Stage 2 sleep and less REM sleep than HC across all menstrual phases 2) No differences between PMS and HC in cortisol levels 3) No differences between PMS and HC in actimetry or temperature
McIntyre et al. (1990) Australia [45]	To examine the production of urinary 6-sulphatoxy melatonin in women with PMS and HC	EFP LLP	Cohort	PMS: $n=5$ HC: $n=15$ Age range: 22- 45	MDQ	Urinary 6-sulphatoxy melatonin	No	No differences between PMS and HC in mean levels of urinary 6-sulphatoxy melatonin at neither EFP nor LLP

Table 1 (continued)

Author (year) Country	Relevant aim for the systematic review	Menstrual phase	Study design	Demographic	PMS / PMDD Assessment	Biological Rhythm Assessment	Association?	Relevant Findings
Parry et al. (1990) USA [42]	To investigate circadian phase and amplitude of melatonin secretion in PMS and HC	EFP-MFP LFP MLP LLP	Cohort	PMS: $n=8$, mean age: 31 ± 1.7 HC: $n=8$, mean age: 30 ± 1.0	DSM-III-R Daily mood ratings HAM-D BDI	Serum melatonin	Yes	When compared to HC at all menstrual phases, PMS showed: i) earlier offset time of melatonin secretion, ii) shorter secretion duration, iii) a decreased area under curve (AUC, which is used as a more accurate estimate of circadian amplitude for melatonin, as opposed to the peak, when a cosine fit is not appropriate for the data)
Severino et al. (1991) USA [49]	To examine body temperature and wrist actimetry in women with PMS/PMDD and HC	EFP LFP MLP LLP	Cross-sectional	PMS/PMDD: $n=10$, mean age: 37.2 ± 3.0 HC: $n=6$, mean age: 31.5 ± 4.1	DSM-III-R Daily mood ratings	1) Ambulatory rectal temperature 2) Wrist activity monitor	Yes	1) The nocturnal temperatures of the women with PMS/PMDD (mean, maximum, minimum) were significantly higher than HC across the entire menstrual cycle 2) No differences between PMS/PMDD and HC in activity

Table 1 (continued)

Author (year) Country	Relevant aim for the systematic review	Menstrual phase	Study design	Demographic	PMS / PMDD Assessment	Biological Rhythm Assessment	Association?	Relevant Findings
Parry et al. (1994) USA [53]	To examine circadian profiles of cortisol, prolactin, TSH, and core body temperature in women with PMDD and HC	MFP LLP	Clinical trial	PMDD: $n=20$, mean age: 36 ± 1.5 HC: $n=11$, mean age: 36 ± 0.9	DSM-III-R Daily mood ratings HAM-D BDI	1) Serum cortisol 2) Serum prolactin 3) Serum TSH 4) Rectal/vaginal core body temperature		1) Cortisol peak significantly delayed for HC and advanced in PMDD in LLP compared to MFP 2) Prolactin amplitude and peak higher and acrophase earlier in PMDD when compared to HC in both MFP and LLP 3) No differences in TSH amplitude, acrophase, mesor, peak, time of peak, nadir, or time of nadir 4) PMDD had greater temperature amplitudes during MFP and LLP when compared to HC
Parry et al. (1996) USA [60]	To explore disturbances in prolactin and TSH in PMDD patients and HC	MFP LLP	Clinical trial	PMDD: $n=23$ HC: $n=18$ Ages not reported	DSM-III-R Daily mood ratings HAM-D BDI	1) Serum prolactin 2) Serum TSH	Yes	1) Prolactin peak higher in PMDD when compared to HC across the menstrual cycle. Amplitude tended to be higher and acrophase earlier in PMDD when compared to HC (group differences did not reach statistical significance) 2) TSH acrophase and peak time earlier in PMDD when compared to HC across the menstrual cycle

Table 1 (continued)

Author (year) Country	Relevant aim for the systematic review	Menstrual phase	Study design	Demographic	PMS / PMDD Assessment	Biological Rhythm Assessment	Association?	Relevant Findings
Parry et al., (1997a) USA [43]	To compare the melatonin secretion patterns in women with PMDD compared to HC	MFP LFP MLP LLP	Clinical trial	PMDD: $n=21$ HC: $n=11$ Ages not reported	DSM-III-R DSM-IV Daily mood ratings HAM-D BDI	Plasma melatonin	Yes	Differences in melatonin parameters between PMDD and HC: i) Melatonin onset time was delayed in PMDD and advanced in HC during LLP compared to the MFP ii) AUC, Amplitudes 1 and 3, and mean levels were significantly lower in PMDD than in HC throughout the menstrual cycle, as well as when comparing LLP to MFP iii) Midpoint concentration increased in PMDD but decreased in HC in LLP when compared to MFP
Parry et al. (1997b) USA [48]	To assess whether the circadian rhythm of core body temperature is altered in PMDD as compared to HC	MFP LLP	Clinical trial	PMDD: $n=23$ HC: $n=18$ Ages not reported	DSM-III-R DSM-IV Daily mood ratings HAM-D BDI	Core body temperature	No	No differences between PMDD and HC in mean core body temperature, nor any circadian parameters (mesor, acrophase, time of minimum and maximum values)
Parry et al. (1997c) USA [54]	To compare melatonin levels and circadian rhythm between women with PMDD and HC	MFP LLP	Clinical trial	PMDD: $n=8$, mean age: 39.3 ± 5.0 HC: $n=5$, mean age: 40.4 ± 2.7	DSM-III-R Daily mood ratings HAM-D BDI	Plasma melatonin	No	No significant differences between PMDD and HC in overall melatonin levels, onset and offset time, peak concentration, duration, or AUC

Table 1 (continued)

Author (year) Country	Relevant aim for the systematic review	Menstrual phase	Study design	Demographic	PMS / PMDD Assessment	Biological Rhythm Assessment	Association?	Relevant Findings
Parry et al. (1999) USA [36]	To examine sleep EEG measures between PMDD and HC	MFP LLP	Clinical trial	PMDD: $n=14$ (full sample $n=23$, mean age: 36.0 ± 4.1, range 29—43) HC: $n=9$ (full sample $n=18$, mean age: 37.2 ± 5.8, range 24—45)	DSM-IV DSM-III-R Daily mood ratings HAM-D BDI	Sleep EEG	Yes	HC experienced more Stage 1 sleep (minutes and percent) than PMDD in LLP when compared to MFP
Parry et al. (2000) USA [56]	To examine cortisol circadian rhythms in PMDD patients and HC	MFP LLP	Clinical trial	PMDD: $n=15$, mean age: 36.0 ± 4.1 HC: $n=15$, mean age: 37.2 ± 5.8	DSM-III-R	Serum cortisol	Yes	Significant differences between PMDD and HC in acrophase: cortisol acrophase earlier in LLP vs. MFP for HC, but not for PMDD
Baker et al. (2007) USA [40]	To investigate sleep quality and sleep composition using conventional and quantitative EEG analyses in women with severe PMS as compared to HC	FP LLP	Cross-sectional	Severe PMS/PMDD: $n=9$ Mild/no PMS: $n=12$ Age range of whole sample: 18–40	DSM-IV PSST	1) PSG 2) PSD	Yes	1) There were group differences in EEG measures in all menstrual phases, including decreased delta incidence and increased theta incidence and amplitude in women with PMS, suggesting the possibility of sleep EEG trait markers in women with PMS 2) Women with severe PMS reported a significantly poorer subjective sleep quality during the LLP

Table 1 (continued)

Author (year) Country	Relevant aim for the systematic review	Menstrual phase	Study design	Demographic	PMS / PMDD Assessment	Biological Rhythm Assessment	Association?	Relevant Findings
Lamarche et al. (2007) Canada [41]	To examine daytime sleepiness and alertness, as well as nocturnal sleep parameters, in women with significant premenstrual symptoms (PS) and women with minimal PMS	FP LLP	Cross-sectional	Significant PMS: $n=10$ Minimal PMS: $n=9$ Mean age of whole sample: 26 (range 20–37)	PMTS	1) PSG 2) Core body temperature	Yes	1) Women with significant PMS were sleepier and less alert during the LLP 2) Women with significant PMS had a higher mean nocturnal temperature across the menstrual cycle when compared to women with minimal PMS
Parry et al. (2008) USA [46]	To assess if melatonin measures change differentially between PMDD and HC	MFP LLP	Clinical trial	PMDD: $n=13$, mean age: 36 ± 4.1, range 29–43 HC: $n=12$, mean age: 37.2 ± 5.8, range 24–45	DSM-IV HDRS	Plasma melatonin	No	No differences between groups in melatonin parameters
Parry et al. (2010) USA [47]	To assess plasma melatonin levels PMDD and HC	MFP LLP	Clinical trial	PMDD: $n=10$, mean age: 37.83 ± 6.16, range 30–44 HC: $n=13$, mean age: 37.73 ± 5.14, range 23–44	DSM-IV HDRS BDI	Plasma melatonin	No	No differences between groups in melatonin parameters
Parry et al., (2011) USA [44]	To assess differences in melatonin levels and timing between PMDD and HC	MFP LLP	Clinical trial	PMDD: $n=17$, mean age: 34.71 ± 7.6 HC: $n=14$, mean age: 37.50 ± 5.4	DSM-IV HDRS BDI	Plasma melatonin	Yes	AUC of melatonin decreased more in PMDD than in HC as a consequence of the transition from MFP to LLP
Baker et al. (2012) USA [37]	To evaluate sleep quality subjectively and objectively using PSG and quantitative EEG measures in women with severe PMS and HC	FP LLP	Cross-sectional	PMS: $n=18$, mean age: 30.5 ± 7.6 HC: $n=18$, mean age: 29.2 ± 7.3	DSR	1) PSG 2) PSD	Yes	1) Women with PMS had less Stage 1 sleep, more slow-wave sleep and slow-wave activity than HC at LLP and FP 2) Women with PMS reported poorer subjective sleep quality in LLP

Table 1 (continued)

Author (year) Country	Relevant aim for the systematic review	Menstrual phase	Study design	Demographic	PMS / PMDD Assessment	Biological Rhythm Assessment	Association?	Relevant Findings
Shechter et al. (2012) Canada [38]	To investigate nocturnal PSG sleep in women with PMDD and HC	EFP MFP LFP OV ELP MLP LLP	Cross-sectional	PMDD: $n=7$, mean age: 32 ± 5.72 HC: $n=5$, mean age: 30.4 ± 8.20	DSM-IV PRISM VAS	1) PSG 2) Core body temperature 3) 6-Sulfatoxy-melatonin concentration	Yes	1) Slow-wave sleep was significantly increased in PMDD when compared to HC across the menstrual cycle 2) PMDD had significantly decreased melatonin levels than HC at all menstrual phases 3) No differences between PMDD and HC in melatonin levels
Segebladh et al. (2013) Sweden [59]	To assess differences in diurnal variation of circulating cortisol in PMDD patients and HC	LLP	Interventional study	PMDD: $n=26$, mean age: 37.8 ± 6.9 HC: $n=30$, mean age: 37.6 ± 6.2	DSM-IV CDS	Salivary and serum cortisol	No	No significant difference of diurnal cortisol secretion between HC and PMDD patients
Bedding et al. (2019) Germany [55]	To compare cortisol levels in women with and without PMDD	Menses FP OV LP	Case–control	PMDD: $n=61$, mean age: 29.4 ± 5.8 HC: $n=61$, mean age: 29.5 ± 5.1	DSM-IV PSST	Salivary cortisol	Yes	When compared to HC, women with PMDD showed a delayed cortisol awakening response peak and a flattened diurnal cortisol slope, regardless of menstrual phase
Miura and Honma (2019) Japan [51]	To explore daytime sleepiness in college students in correlation with severity of premenstrual symptoms	Premenstrual period	Cross-sectional	PMDD: $n=47$ Moderate-to-severe PMS: $n=79$ No or mild PMS: $n=257$ Mean age: 21 ± 2	PMDD scale developed by Japanese researchers, modified from the PSST	Japanese version of the ESS	Yes	The severity of daytime sleepiness in women is positively correlated with the severity of premenstrual symptoms in the premenstrual period
Izakova et al. (2021) Slovakia [57]	To assess cortisol levels in women with PMS and HC	FP ELP LLP	Case–control	PMS: $n=49$, mean age: 26.7 ± 7.5 HC: $n=50$, mean age: 29.5 ± 7.6	Self-reporting questionnaire for premenstrual symptoms based on DSM-5 criteria for PMDD	Salivary cortisol	No	No differences between PMS and HC in any cortisol parameters

Table 1 (continued)

Author (year) Country	Relevant aim for the systematic review	Menstrual phase	Study design	Demographic	PMS / PMDD Assessment	Biological Rhythm Assessment	Association?	Relevant Findings
El Dahr et al. (2022) Canada [52]	Study 1: To investigate biological rhythm disruption in women with PMDD vs. HC Study 2: To investigate biological rhythm disruption and subjective sleep quality in women with PMDD when compared to HC	Study 1: Entire cycle Study 2: MFP LLP	Cross-sectional	Study 1: PMDD: *n* = 104, mean age: 27 (IQR 24–28) HC: *n* = 556, mean age: 26 (IQR 24–27) Study 2: PMDD: *n* = 19, mean age: 33 (IQR 25–39.5) HC: *n* = 25, mean age: 26 (23–28)	Study 1: DSM-IV criteria for PMDD Study 2: DSM-IV criteria for PMDD	Study 1: BRIAN Study 2: 1) BRIAN 2) PSQI	Yes	Study 1: PMDD had greater overall biological rhythm disruption than HC. Specifically, they had overall greater disruption in all four domains of the BRIAN: sleep, social, activity, and eating rhythms Study 2: 1) PMDD had greater rhythm disruption than HC in sleep at LLP, activity at MFP and LLP, and social patterns in LLP. There were no differences between groups in eating patterns at either stage of the menstrual cycle 2) There were no differences between groups in subjective sleep quality

AUC Area under the curve, *BDI* Beck Depression Inventorym, *BRIAN* Biological Rhythms Interview of Assessment in Neuropsychiatry, *CDS* Daily ratings on the Cyclicity Diagnoser Scale, *COPE* Calendar of Premenstrual Experiences, *DSR* Penn Daily Symptom Rating Form, *EEG* Electroencephalogram, *EFP* Early follicular phase, *ELP* Early luteal phase, *ESS* Epworth Sleepiness Scale, *FP* Follicular phase, *HAM-D/HDRS* Hamilton Rating Scale for Depression, *HC* Healthy controls, *LFP* Late follicular phase, *LLP* Late luteal phase, *LP* Luteal phase, *MDQ* Menstrual Distress Questionnaire, *MFP* Mid follicular phase, *MLP* Mid luteal phase, *OV* Ovulation, *PMDD* Premenstrual Dysphoric Disorder, *PMS* Premenstrual Syndrome, *PMTS* Premenstrual Tension Syndrome Self Rating Scale / Premenstrual Syndrome Self-Rating Scale, *PRISM* Prospective Record of the Impact and Severity of Menstrual Symptom, *PSST* Premenstrual Symptoms Screening Tool, *PSD* Pittsburgh Sleep Diary, *PSG* Polysomnography, *PSI* Post-Sleep Inventory, *PSQI* Pittsburgh Sleep Quality Index, *REM* Rapid eye movement, *TSH* Thyroid stimulating hormone, *VAS* Visual Analogue Scale

Table 2 Quality of assessment of the studies included assessed using the Newcastle–Ottawa Quality Assessment Scale

Author (year)	Representativeness of the sample (Selection bias)	Sample size (Selection bias)	Non-respondents (Selection bias)	Ascertainment of exposure (Selection bias)	Comparability (Comparability bias)	Assessment of outcome (Outcome bias)	Statistical test (Outcome bias)	Total score
Mauri et al., 1988 [50]				1	1	1		3/10
Mortola et al., 1989 [58]				2	2	2	1	7/10
Parry et al., 1989 [39]				2	1	2	1	6/10
McIntyre et al., 1990 [45]				2	1	2		5/10
Parry et al., 1990 [42]				2	1	2	1	6/10
Severino et al., 1991 [49]				2	2	2	1	7/10
Parry 1994 MISSING [53]				2	1	2	1	6/10
Parry et al., 1996 [60]				2	1	2	1	6/10
Parry et al., 1997a [43]				2	1	2	1	6/10
Parry et al., 1997b [48]				1	1	2	1	5/10
Parry et al., 1997c [54]				2	1	2	1	6/10
Parry et al., 1999 [36]				2	1	2	1	6/10
Parry et al., 2000 [56]			1	2	1	2	1	7/10
Baker et al.,2007 [40]	1			2	1	2	1	7/10
Lamarche et al., 2007 [41]				2	2	2	1	7/10
Parry et al., 2008 [46]				2	1	2	1	6/10
Parry et al., 2010 [47]				2		2	1	5/10
Parry et al., 2011 [44]				2	1	2	1	6/10
Baker et al.,2012 [37]	1			2	1	2	1	7/10
Shechter et al., 2012 [38]				2	1	2	1	6/10
Segebladh et al., 2013 [59]				1	1	2	1	5/10
Beddig et al., 2019 [55]	1			1	1	1	1	5/10
Miura et al., 2019 [51]				1	1	1	1	4/10
Izakova et al., 2021 [57]	1			1	2	1	1	6/10

Table 2 (continued)

Author (year)	Representativeness of the sample (Selection bias)	Sample size (Selection bias)	Non-respondents (Selection bias)	Ascertainment of exposure (Selection bias)	Comparability (Comparability bias)	Assessment of outcome (Outcome bias)	Statistical test (Outcome bias)	Total score
El Dahr et al., 2022 Study 1 [52]	1	1		1	1	1	1	6/10
El Dahr et al., 2022 Study 2 [52]	1			2	1	1	1	6/10

secretion, wrist actimetry, and self-reported clinical questionnaires.

Polysomnography (PSG)

Five out of six studies assessing sleep using PSG found at least one sleep alteration in women with PMS/PMDD as compared to controls. Replicated findings supported by two independent studies show that women with PMS/PMDD exhibited lower stage 1 sleep than healthy controls in the luteal phase [36] and across the menstrual cycle [37]. In addition, two independent studies showed that women with PMS/PMDD had increased slow-wave sleep (SWS) at both menstrual phases [37, 38]. Other findings reported by single studies include: (1) women with moderate-to-severe "premenstrual depression" displayed greater Stage 2 and less rapid eye movement (REM) sleep at both menstrual phases [39]; and (2) women with PMS had a longer REM sleep latency and a trend of poorer sleep efficiency at both menstrual phases [40]. This study also found that women with PMS had lower delta incidence in non-REM (NREM) sleep, higher theta incidence, and higher theta amplitude than controls at both menstrual phases, which is hypothesized to be indicative of deficiencies in sleep-regulatory processes [40]. In a more detailed exploration of sleep architecture, it was found that women with PMS had more delta power in NREM sleep, associated with more time in the delta band, and waveforms of greater amplitude during NREM sleep [37], contradicting some of the earlier findings by Baker et al. [40].

Lamarche et al., (2007) did not find any differences in sleep characteristics between groups across the menstrual cycle [41].

Melatonin

Four out of eight studies found differences in melatonin secretion between women with PMS/PMDD and controls. The most consistent finding was supported by three studies, for which it was not possible to determine if the samples were the same, different, or overlapping. These studies showed a decreased area under the curve (AUC) of plasma melatonin in women with PMS/PMDD [42–44], indicative of a lower circadian amplitude of melatonin secretion. Parry et al., (1990) observed this difference across the menstrual cycle for PMDD and healthy controls [42], as did Parry et al., (1997), and further observed that women with PMS/PMDD had significantly lower melatonin AUC in the luteal phase [43]. Parry et al., (2011) observed significantly lower melatonin AUC for both groups in the luteal phase, with the PMS/PMDD group having significantly lower levels than controls [44]. In addition, women with PMS/PMDD had lower mean melatonin levels in the luteal phase, as well as lower Amplitude 1 and 3, which represent the mean of the highest point and three highest points of melatonin secretion, respectively [43]. These results were accompanied by a delayed onset time and an increased midpoint of melatonin secretion, only in the luteal phase. Finally, one study [42] noted an earlier offset time of melatonin secretion and a shorter secretion duration across the menstrual cycle, and another [44] observed a significantly lower peak of melatonin secretion in women with PMS/PMDD in the luteal phase. One study found that levels of urinary 6-sulphatoxy-melatonin, the main metabolite of melatonin, was significantly lower in women with PMS/PMDD across the menstrual cycle [38], while another did not find differences between groups at the follicular or luteal phases [45].

Three studies did not find significant differences in melatonin levels or timing of secretion between women with PMS/PMDD and controls [46–48].

Wrist actimetry

Two studies using wrist actimetry found no differences between women with PMS/PMDD and controls in 24-h

activity levels [39] or in-bed nocturnal activity levels [49] across the menstrual cycle.

Self-report assessments

All six studies assessing subjective perception of sleep showed differences between women with PMS/PMDD and controls. Women with PMS/PMDD report poorer sleep quality, often accompanied by more frequent awakenings, as well as increased tiredness and decreased alertness in the mornings [37, 40, 41, 50]. Although some studies observed this result across all menstrual phases [37, 40, 50], one study observed this effect only in the late luteal phase [41]. One study found that severity of daytime sleepiness correlated with severity of premenstrual symptoms [51]. The perception of disruption of biological rhythms was assessed in one study with two different samples using the Biological Rhythms Interview of Assessment in Neuropsychiatry (BRIAN) [52]. Women with PMS/PMDD reported greater perceived disruption of sleep rhythms in both samples. In the sample of 19 women with prospectively-diagnosed PMDD, this association was significant only in the luteal phase. In the sample of 104 women with a provisional diagnosis of PMDD, the PMDD sample reported greater perceived disruption of sleep rhythms across the menstrual cycle.

Biological rhythms assessments

Twelve out of 25 studies assessed the rhythmicity of body temperature, cortisol, PRL, and TSH levels.

Body temperature

Significant changes in body temperature rhythms were observed in three out of six studies. In two studies, women with PMS/PMDD had a higher mean nocturnal [41, 49] and 24-h [53] temperature across the menstrual cycle. The remaining three studies found no differences in body temperature between groups [38, 39, 54].

Cortisol

Three out of seven studies on cortisol found contradicting differences between women with PMS/PMDD and controls. An advanced cortisol peak was observed in women with PMS/PMDD in the luteal phase [53], but in another study, a delayed cortisol peak upon awakening and a flattened diurnal cortisol trajectory was observed [55]. In another contradictory finding, women without PMS/PMDD experienced an advance in cortisol peak in the late luteal phase, whereas women with PMS/PMDD did not [56]. The remaining four studies did not find differences between groups in cortisol levels or timing [39, 57–59].

PRL and TSH

In two studies, PRL displayed higher amplitude and peak with earlier acrophase (peak timing) in women with PMS/PMDD across the menstrual cycle [53, 60]. Regarding TSH, one study observed earlier acrophase and peak time in women with PMS/PMDD across the menstrual cycle [60] whereas another showed no differences [53].

Discussion

This systematic review showed that women with PMS/PMDD experience abnormal profiles of some (but not all) parameters that typically display rhythmic circadian patterns. The most consistent abnormalities observed in women with PMS/PMDD were lower melatonin secretion, higher body temperature, and worse subjective sleep quality. Conflicting findings were observed for PSG-related sleep parameters, cortisol, PRL, and TSH, whereas activity patterns from wrist actimetry results were not altered in women with PMS/PMDD.

Lower nocturnal [42–44] and morning urinary melatonin levels [38] suggest there may be a blunted melatonin secretion in women with PMS/PMDD. Parry et al. [42] and Shechter et al. [38] observed these patterns in both the symptomatic LP and asymptomatic follicular phase (FP), indicating that low melatonin levels may serve as a trait marker of the disorder. The latter study was conducted in a subgroup of women with PMDD with severe insomnia, which may limit the generalization of results to all women with PMDD. Melatonin is often considered an indicator of endogenous circadian pacemaker functioning, as it is regulated by the suprachiasmatic nucleus of the hypothalamus, the central circadian rhythm generator [18, 61]. As such, a blunted melatonin profile may reflect dampened strength of the circadian pacemaker. In a sample of healthy women, Rahman et al. [62] found that reproductive hormones, as well as melatonin, displayed significant 24-h rhythms only in the FP, suggesting that the fluctuation of reproductive hormone levels in the LP exerts a stronger influence on melatonin than in the FP. Alternatively, a reciprocal relationship may also be possible, whereby melatonin may exert modulatory effects on reproductive physiology [63]: melatonin receptors have been found in the uterus, and ovarian follicular fluid has been shown to have high concentrations of melatonin, which can reduce oxidative stress during the ovulation process. The lower melatonin levels seen in women with PMS/PMDD may also be related to mood disturbances through the serotonin cycle. Women with PMS/PMDD exhibit a dysregulated serotonergic system, such as abnormal whole blood serotonin levels, lower platelet uptake, abnormal responses to serotonergic triggers, and worsening of symptoms after intentional

serotonin depletion [64–66]. Furthermore, selective serotonin reuptake–inhibiting antidepressants have been established as the first-line treatment for PMDD [67, 68]. Considering that serotonin is a precursor of melatonin, an abnormal serotonergic system may affect melatonin levels. Alternatively, lower melatonin levels may act as a signal to increase the production of melatonin and may disturb the serotonergic pathway through increased demand.

Core body temperature (Tc) typically displays a clear circadian pattern of a diurnal maximum and nocturnal minimum [69] In women with PMS/PMDD, elevated nocturnal Tc [41, 49] and 24-h Tc [53] were observed in half of the included studies across the menstrual cycle, not just in the symptomatic LP, suggesting that elevated Tc may be a trait marker of the disorder. In studies of thermoregulation across the menstrual cycle, Tc is shown to be influenced by reproductive hormones, whereby the thermoregulatory setpoint is decreased by estrogen and increased by progesterone [70]. Studies on body temperature in affective disorders are scarce, though one study found that patients with MDD have increased body temperature [71], which the authors link to the inflammation hypothesis of MDD [72]. There is also speculation that women with PMS/PMDD may have an increased inflammatory response [73–75], with increased C-reactive protein levels associated with worsened mood symptoms [76, 77], though evidence is scarce in this area as well and is not directly linked to an increase in Tc.

It is worth noting that four studies on melatonin [45–48] and three on body temperature [38, 39, 54] showed no differences between women with PMS/PMDD and controls.

This review also found that women with PMS/PMDD consistently reported worse perceived sleep quality, though this is not clearly reflected in objective sleep parameters. The most consistent results among studies assessing objective sleep were increased SWS [37, 38] and decreased Stage 1 sleep [36, 37] in women with PMS/PMDD, indicating that more time is spent in restorative "deep sleep", as characterized by SWS, and less time in light sleep. One study that reported both increased SWS and decreased Stage 1 sleep [37] also observed a lower number of awakenings. However, women with PMS/PMDD subjectively reported a greater number of perceived awakenings and feeling less refreshed and alert the next day. Sleep complaints are common in women, often during the premenstrual phase and accompanied by pains, changes in mood, and physical symptoms—poor sleep quality then has negative effects on mood, essentially establishing a bidirectional relationship between sleep and mood [78]. It is possible that the effect of changes in reproductive hormones on melatonin and body temperature may affect the subjective experience of sleep without affecting sleep architecture [15]. These discrepancies between objective and subjective sleep measures are frequently reported in mood disorders [79, 80] and in healthy populations [81, 82], suggesting that the perception of sleep differs in patient populations, which may be influenced by other lifestyle factors that may not relate directly to sleep architecture. The lack of consistency between objective and subjective sleep reports may be attributable to numerous factors, including the vague language used in subjective sleep quality assessments [83]. These sleep parameters could benefit from longitudinal assessments, possible through actigraphy, that will allow mapping of the trajectory of sleep patterns across the menstrual cycle. To be able to detect these patterns, longitudinal assessments are considerably beneficial for cyclical disorders such as PMS/PMDD.

In this population, however, wrist actigraphy is understudied and current literature prevents us from drawing specific conclusions. Other domains considered in this review (cortisol, PRL, and TSH) are also understudied in PMS/PMDD and deserve greater consideration considering the robust findings in other mood disorder populations. Cortisol is an essential regulator of the sleep–wake cycle and has an inverse pattern to melatonin release: cortisol levels peak in the morning and are at their lowest at night, allowing melatonin to signal timing for sleep. Flatter diurnal cortisol rhythms (indicating lack or lessening of a morning peak) have been associated with worse depression [84, 85], consistent with the findings of one study included in this review [55]. Both hormones of the pituitary gland, PRL and TSH are theorized to act synergistically in their influence on female biology [86]. High levels of PRL (hyperprolactinemia) and both high and low levels of TSH (hyper/hypothyroidism) are often associated with depressive symptoms [87–89].

In comparison to other mood disorders, PMS and PMDD are largely understudied. The lifetime comorbidity of PMS/PMDD with other psychiatric disorders is high, particularly with MDD, BD, and anxiety disorders [90]. Specifically, MDD is the most prevalent lifetime psychiatric disorder in women with PMDD [91]. Disruptions of biological rhythms have been associated with the precipitation and perpetuation of mood episodes in patients with MDD and BD [29, 30]: both disorders have also been associated with altered sleep and activity variables measured through actigraphy [28]. Furthermore, disturbance of biological rhythms was found to be an independent predictor of functional impairment in patients with mood disorders [92]. Considering the classification of PMDD as a depressive disorder, as well as the fact that PMDD is often comorbid with other mood disorders, there may be overlapping mechanisms of biological rhythm disruption

in the experience of mood symptoms in a similar way as in MDD and BD. This review highlights the scarcity of studies in this area and calls for further investigation of how disruptions of biological rhythm across multiple domains are associated with symptoms of PMS/PMDD.

Limitations of included studies

The study of biological rhythms in PMS and PMDD is still in its infancy, as indicated by limited studies that, for the most part, have not been independently replicated. Importantly, due to the heterogeneity among samples and protocols between the studies, a meta-analysis was not possible.

First, an important limitation is the lack of consideration for the heterogeneity in symptom profiles of PMS/PMDD. Clinical subtypes of PMS or PMDD have been classified as predominantly physical, predominantly psychological, or both [93] based on the nature of symptoms, and temporal subtypes of PMDD have been theorized based on the trajectory of the disorder [15]. Clustering participant samples would allow for a more detailed understanding of biological rhythm disruptions at different stages of the menstrual cycle and the behavioural/physiological manifestations within subgroups. A second limitation is the lack of standardized protocols, which could potentially lead to masking of the endogenous rhythms by external factors [94]. Through implementing standardized bedtime routines, light exposure, and mealtimes during the data collection period, the masking effects of external influences on the internal oscillator could be reduced. However, the opposing argument can be made for ecological validity by assessing patients in their natural living conditions [95]. A third limitation of this review is that, despite multiple communication attempts, we were unable to determine if there was overlap among the samples in twelve studies included in this review [36, 39, 42–44, 46–48, 53, 54, 56, 60]. This is an important limitation because it hinders our ability to discern whether the results are truly replicated across independent samples or simply a characteristic of a single sample analyzed and presented in different ways.

Recommendations for future research

There is a necessity for longitudinal tracking of biological rhythms across menstrual cycles in women with PMS/PMDD in order to elucidate whether the observed disruptions are consistent within individuals and provide insights into the potential role of hormonal fluctuations. Considering the diagnosis of PMDD requires prospective assessment for at least 2 menstrual cycles, longitudinal tracking will aid in understanding intra-individual variability of biological rhythms in terms of magnitude (e.g., the amount of melatonin secreted at night or the degree of decrease in body temperature) or timing (e.g. chronotype-related differences in sleep timing) of physiological and behavioral variables [96–99]. This variability is likely a result of the interplay of individual genetic characteristics with environmental regulators like work schedules and lifestyle choices. Therefore, prospective studies make it possible to observe individual variations rather than group distinctions.

Additionally, future studies should aim to use standardized protocols in assessments of biological rhythms that

Table 3 Recommendations for future studies

Recommendation	Explanation and examples
Use of Diagnostic Criteria	Ensure that studies adhere to recognized diagnostic criteria for PMS and PMDD, outlined in the DSM-5-TR, including 2-month prospective symptom charting
Longitudinal Objective and Subjective Measures	Collect longitudinal assessments of both objective and subjective measures of sleep and other biological rhythm parameters
Comorbid psychiatric disorders	Explore how comorbid psychiatric disorders (e.g., mood disorders) and the severity of PMS/PMDD symptoms interact with disruptions in biological rhythms. Investigate whether these factors moderate or mediate the relationship between biological rhythms and symptom severity
Influence of Lifestyle Factors	Examine the influence of lifestyle factors, such as diet, physical activity, and stress, on biological rhythms in women with PMS/PMDD. These factors may contribute to both symptom severity and the disruption of rhythms
Incorporate Multidisciplinary Approaches	Encourage multidisciplinary collaboration among researchers from psychology, psychiatry, endocrinology, and chronobiology to gain a comprehensive understanding of the biological rhythms in PMS/PMDD
Biological Mechanisms	Investigate the underlying biological mechanisms linking disruptions in biological rhythms (such as melatonin, core body temperature) to mood disturbances in PMS/PMDD. This could include exploring phase-angle differences of circadian markers and reproductive hormones to understand the effect of timing
Clinical Subtyping	Consideration of clinical subtypes of PMS and PMDD based on symptom profiles and temporal patterns (e.g. clustering analyses). This could provide insights into how specific biological rhythms disruptions may vary among subgroups of women with PMS/PMDD

balance ecological validity and controlled conditions. This balance will address the challenge of external influences on the internal oscillator without sacrificing real-world relevance, ultimately improving the comparability of results across different studies. To enhance reliability and generalizability of results, replication studies with appropriate sample sizes are essential. Finally, we encourage open science frameworks, including data sharing and collaboration within the research community, to facilitate data pooling, large collaborative studies, and meta-analyses. Table 3 outlines further recommendations for study designs and focus of research areas.

Conclusions

This systematic review highlights that some domains seem to differ between individuals with PMS/PMDD and controls, such as lower melatonin levels, higher core body temperature, and worse subjective sleep quality, whereas others showed no differences (wrist actimetry), or conflicting results (objective PSG sleep reports, cortisol, PRL, and TSH parameters). Future studies assessing biological rhythmicity in PMS/PMDD should aim to follow standardized protocols such that masking of endogenous bodily rhythms by the external environment is minimized.

Abbreviations

AUC	Area under the curve
BD	Bipolar Disorder
BRIAN	Biological Rhythms Interview of Assessment in Neuropsychiatry
FP	Follicular phase
LLPDD	Late luteal phase dysphoric disorder
LP	Luteal phase
MDD	Major Depressive Disorder
NREM	Non-rapid eye movement
PMS	Premenstrual syndrome
PMDD	Premenstrual dysphoric disorder
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-analysis
PRL	Prolactin
PROSPERO	Prospective Register of Systematic Reviews
PSG	Polysomnography
REM	Rapid eye movement
SCN	Suprachiasmatic nucleus
SWS	Slow-wave sleep
Tc	Core body temperature
TSH	Thyroid-stimulating hormone

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Authors' contributions

The concept and design of this systematic review were created by A.N., L.C., T.A.C., and B.N.F. Abstracts and full-text screenings were completed by A.N. and L.C., and conflicts were resolved by T.A.C. Quality assessments were completed by A.N., L.C., and T.A.C., and conflicts were resolved by consensus. Data extraction and writing of the manuscript were completed by A.N., L.C., T.A.C., J.S.S., and A.C.T. Critical revisions of the manuscript and interpretation of data were provided by B.N.F. and A.C.T. All authors approved the final version of the manuscript.

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Availability of data and materials

The datasets analysed during the current study are not publicly available due to the nature of a systematic review but may be available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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