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Sleep disturbances in individuals with first episode psychosis and clinical high-risk states: A systematic review

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ABSTRACT

Background: Sleep disturbances are increasingly recognized as relevant components of the psychopathology of psychosis, emerging early in the illness trajectory and persisting over time. Indeed, individuals with first-episode psychosis (FEP) commonly experience disruptions in sleep architecture, including reduced sleep efficiency, increased sleep latency, and altered time spent in various sleep stages. These abnormalities are also reported in individuals with Clinical High-Risk (CHR) states, showing a significant correlation with cognitive and affective impairments.

Methods: We conducted a systematic search across four main electronic databases, including PubMed, Web of Science, EMBASE, and PsycINFO, to specifically identify studies examining sleep parameters in FEP and CHR subjects compared to healthy controls. Eligibility criteria included quantitative and qualitative assessments of sleep.

Results: The final selection consisted of 25 studies corresponding to 1255 patients and 342 healthy controls. Increased sleep latency and alterations in slow-wave sleep were frequently reported. These findings highlight the pervasiveness of sleep disturbances in individuals with early psychosis, though further research is needed to clarify their clinical significance. Evidence also suggests bidirectional relationships between sleep disturbances and psychotic symptoms, with sleep disruptions potentially exacerbating cognitive and emotional dysfunctions in FEP and CHR individuals.

Conclusions: Sleep disturbances in FEP/ CHR populations are pervasive and may reflect underlying neurobiological mechanisms implicated in psychosis. These abnormalities represent modifiable targets for early intervention, with the potential to improve clinical and functional outcomes. Future research should explore longitudinal associations and the efficacy of sleep-focused interventions in the early stages of psychotic disorders.

1. Introduction

Sleep disturbances are increasingly recognized as integral to the psychopathology of psychosis, often presenting as early as the prodromal phase and persisting throughout the illness trajectory. These disturbances encompass a broad range of abnormalities, including poor sleep quality, altered sleep architecture, and circadian rhythm disruptions (Reeve et al., 2019; Wulff et al., 2010a, b). Such changes are not merely epiphenomena of psychosis but may contribute to its onset and progression, potentially serving as both risk factors and modifiable treatment targets.

In individuals with a First Episode of Psychosis (FEP), sleep disturbances are frequently reported, including increased sleep latency, reduced total sleep time, and diminished slow-wave sleep (Manoach et al., 2016; Baldini et al., 2025). Similar patterns are observed in individuals with Clinical High-Risk (CHR) states for psychosis. These populations often exhibit sleep disturbances that correlate with cognitive and affective impairments, including heightened emotional

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reactivity, reduced cognitive flexibility, and exacerbated psychotic symptoms (Lunsford-Avery et al., 2017). Such findings suggest that sleep abnormalities could reflect underlying neurobiological dysfunctions associated with psychosis, offering insights into its pathophysiology and potential biomarkers.

Moreover, sleep disruption has been linked to key neurobiological processes relevant to psychosis, such as dysregulated dopamine signaling and impaired sleep-dependent memory consolidation (Wulff et al., 2010a, b). The bidirectional relationship between sleep and psychosis underscores the need to better understand the specific nature and role of sleep disturbances in these clinical populations. Investigating this relationship is particularly relevant in the early stages of psychotic disorders, as interventions targeting sleep may influence long-term outcomes (Blomeyer et al., 2020).

Despite these insights, significant gaps remain in our understanding. Existing studies on sleep disturbances in psychosis-spectrum populations are heterogeneous, varying in their methodologies, samples, and outcome measures. While some authors focused on the prevalence and phenomenology of sleep disturbances, others investigated their longitudinal role as risk factors for psychosis onset or their modulation through pharmacological and behavioral interventions. Moreover, inconsistencies in findings—such as the types of sleep disturbances most strongly associated with psychosis—highlighted the need for a comprehensive synthesis of the evidence to provide clearer insights.

Although previous reviews have explored sleep disturbances in psychosis, they have distinct limitations. Bagautdinova and colleagues provided an overview of sleep alterations in early psychosis but did not systematically compare subjective and objective assessments of sleep dysfunction, leaving gaps in understanding how these measures correlate (Bagautdinova et al., 2023). Indeed, the review by Kaskie focused primarily on chronic schizophrenia, with limited discussion on how sleep disturbances manifest in early-stage psychosis (Kaskie and Ferrarelli, 2020).

A significant contribution of this review is the comparative synthesis of sleep disturbances across various assessment methods, offering insights into both subjective experiences and objective physiological markers of sleep dysfunction. By integrating studies that investigate sleep disruptions in both CHR and FEP populations, we aim to clarify how sleep abnormalities may facilitate the transition from at-risk states to full-blown psychosis. Furthermore, this review critically assesses the methodological heterogeneity in existing studies, addressing inconsistencies in reported findings and highlighting key gaps that future research should explore.

This systematic review addresses these gaps by synthesizing the literature on sleep disturbances in FEP and CHR populations. Specifically, it will examine: (I) the types of sleep disturbances reported in these populations; (II) the relationship between sleep disturbances and clinical outcomes, including symptom severity, transition risk, and functional recovery. Additionally, the Discussion section will highlight potential underlying mechanisms linking sleep disturbances to the onset and progression of psychosis, based on the findings of the reviewed studies.

This review examines the current evidence to better understand the role of sleep in the psychosis continuum and explores potential directions for future research. Gaining deeper insight into the implications of sleep disturbances in these clinical populations could help inform strategies for early detection, prevention, and intervention, potentially contributing to improved outcomes for individuals at risk for or experiencing psychosis.

2. Method

This systematic literature review was conducted and reported according to the preferred reporting items for systematic reviews and meta-analysis PRISMA guidelines (Page et al., 2021). The study protocol was registered in advance on PROSPERO (CRD42024623913).

2.1. Eligibility criteria

We included studies involving participants of any sex with a diagnosis of FEP or CHR based on standardized diagnostic criteria (such as the "Comprehensive Assessment of At-Risk Mental States" [CAARMS], Structural Clinical Interview for DSM (SCID), Structured Interview for Prodromal Syndromes (SIPS), and Psychosis Risk Syndrome Criteria (PSYCHS) were eligible for inclusion (Yung et al., 2005; American Psychiatric Association, 2013). Given the increasing use of SIPS and PSYCHS in identifying individuals CHR for psychosis, studies applying these criteria were also considered to enhance the generalizability of findings to high-risk populations.

Eligibility was restricted to studies that assessed sleep parameters quantitatively or qualitatively in FEP and CHR subjects compared to healthy controls.

Eligible studies compared sleep parameters between individuals with FEP or CHR and Healthy Controls (HC) groups. We considered research assessing a variety of sleep outcomes, including sleep efficiency (SE), total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), and sleep architecture, such as time spent in NREM and REM sleep stages. Both cross-sectional and longitudinal study designs were included. We did not apply a strict minimum sample size threshold for inclusion. Although smaller studies may have limited statistical power, they were included to ensure a thorough synthesis of available evidence.

Quantitative data on sleep in both FEP/ CHR and HC groups were reported. Studies that lacked clearly defined FEP/ CHR or HC groups or did not include relevant sleep data were excluded.

2.2. Search strategy

A systematic search was conducted on PubMed, Web of Science, EMBASE, and PsycINFO from inception up to December 2024 using the following search criteria ("first-episode-psychosis" OR "psychosis" OR "ultra-high risk" OR "at-risk mental state" OR "clinical high risk" OR "early psychosis" OR "prodromal" OR "early intervention") AND ("sleep" OR "insomnia" OR "circadian" OR "dream" OR "actigraphy").

2.3. Selection of the studies

All relevant original research articles were identified based on their titles and abstracts during the screening phase. At this stage, articles without pertinent information—such as those not in English, reviews, case reports/series, conference abstracts, editorials, and viewpoints—were excluded. Further exclusions were applied to studies that did not meet the inclusion criteria regarding study design, sample population, exposure, or outcome of interest. Specifically, studies were excluded if they lacked original data, including reviews, case reports, and editorials. Studies that did not explicitly assess individuals diagnosed with FEP or CHR, such as those focused on chronic schizophrenia, non-psychiatric sleep disorders, or general psychiatric populations without a clear psychosis risk, were also excluded. Additionally, studies that examined sleep variables unrelated to psychosis risk or did not assess sleep disturbances in the context of early psychosis were not considered. Articles that focused exclusively on interventions without analyzing baseline sleep parameters and those that lacked a comparison group or did not include sleep disturbances as a primary or secondary outcome were also excluded. Finally, studies using overlapping or duplicate datasets from the same population were carefully reviewed, and only the most comprehensive or methodologically rigorous study was retained. In addition to database searches, a manual search was conducted to identify any relevant studies that may not have appeared in the initial search results. This process involved screening the reference lists of included articles and reviewing recent systematic reviews on sleep disturbances in psychosis. Through this approach, two additional studies were identified and subsequently assessed against the same eligibility criteria before being included in the final review.

To ensure rigor in study selection, two reviewers (VB and FP) independently assessed each article, and any discrepancies were resolved through discussion with a third reviewer (MM). Full texts were also manually searched to identify additional studies that met the inclusion criteria. No sex or age criteria were applied for article eligibility.

2.4. Data extraction

VB and FP extracted data. VB checked the data extracted on the characteristics of the studies. The following data were extracted: (a) Country, sample (number of participants, mean age, sex), type of publication (i.e., peer-review journal, grey literature, book), year, study design, and study goals; (b) Measures employed to assess FEP/ CHR individuals; (c) other potential relevant concepts were also detected, and research gaps were highlighted.

2.5. Assessment of the quality of studies and risk of bias from the review

Included studies were evaluated with the Newcastle-Ottawa Scale (NOS), which assesses the risk of bias in observational studies on three domains (selection, comparability, and exposure) and provides an overall score ranging from 1 (highest risk of bias) to 9 (lowest risk of bias) (Hartling et al., 2013) Two researchers (VB, FP) assessed the risk independently. Disagreements were discussed with a third researcher (MM).

3. Results

3.1. Flow chart of included studies

Search results are summarized in the Prisma Flow Chart in the Fig. 1. Initially, 5666 studies were identified through the search strategy from the databases. 5437 remained after removing the duplicates. After title and abstract screening, 5116 did not meet our inclusion criteria and, therefore, were excluded. Finally, we included 25 studies in the systematic review (Yung and McGorry, 1996; Tan and Ang, 2001; Iyer et al., 2008; Andriopoulos et al., 2011; Lederman et al., 2017; Subramaniam et al., 2018; Ong et al., 2020; Fekih-Romdhane et al., 2021; Riemann et al., 1995; Poulin et al., 2003; Poulin et al., 2008; Keshavan et al., 2004; Forest et al., 2007; Keshavan et al., 2011; Guénolé et al., 2014; Manoach et al., 2014; Kaskie et al., 2019a, b; Yazıhan and Yetkin, 2020; Denis et al., 2024; Castelnovo et al., 2024; Reeve et al., 2019; Lunsford-Avery et al., 2015; Mayeli et al., 2021; Mayeli et al., 2022; Cohen et al., 2024).

3.2. Ratings of study quality and risk of bias

According to our study quality ratings, 15 studies were of good quality (Iyer et al., 2008; Andriopoulos et al., 2011; Lederman et al., 2017; Subramaniam et al., 2018; Ong et al., 2020; Fekih-Romdhane et al., 2021; Poulin et al., 2003; Poulin et al., 2008; Keshavan et al., 2004; Denis et al., 2024; Castelnovo et al., 2024; Lunsford-Avery et al., 2015; Mayeli et al., 2021; Mayeli et al., 2022; Cohen et al., 2024), 7 were of moderate quality (Tan and Ang, 2001; Yung and McGorry, 1996; Riemann et al., 1995; Poulin et al., 2008; Forest et al., 2007; Guénolé et al., 2014; Manoach et al., 2014) and 3 was of poor quality (Kaskie et al., 2019a, b; Yazıhan and Yetkin, 2020; Reeve et al., 2019). Since most investigations were of moderate quality, the risk of bias from studies included in this review was low.

Two researchers independently performed data extraction on the following variables of the studies: year and place of publication, country of origin, study design, age and sex of participants, and validated

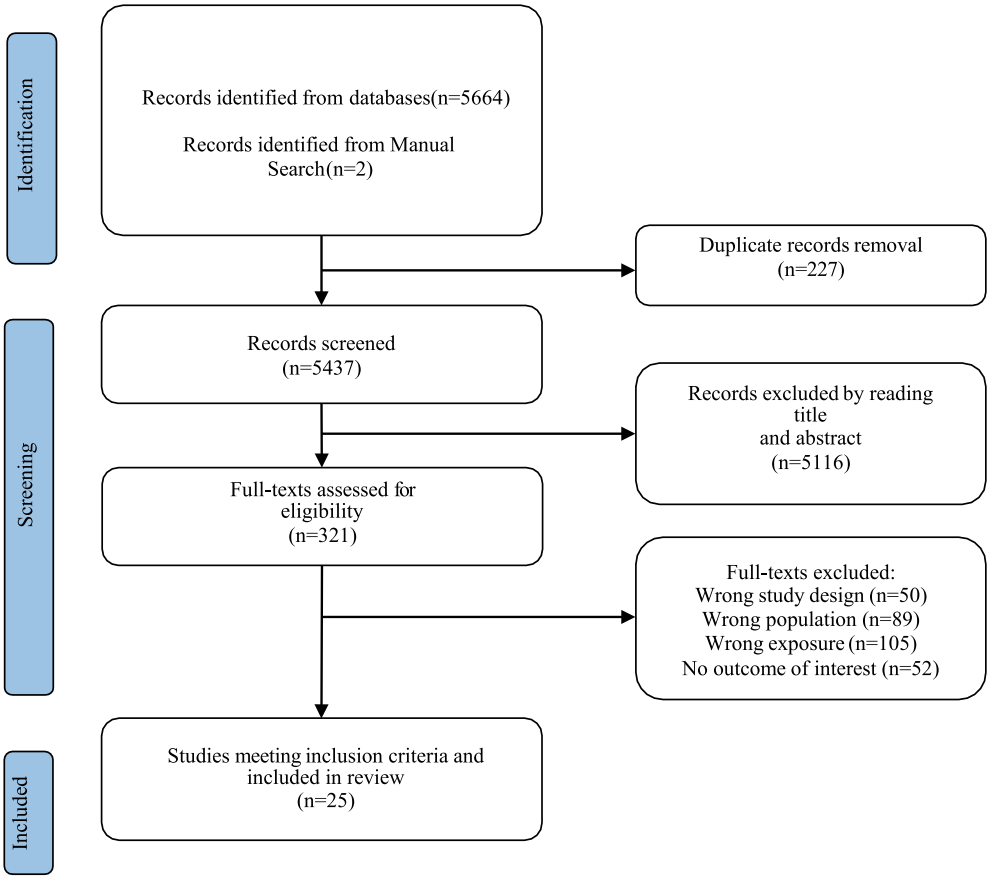

Fig. 1. Flow-chart describing the study selection process.

instruments for evaluating the outcome and authors' interests.

3.3. Characteristics of the included studies

25 studies were included in this systematic review, spanning different geographic regions, including North America, Europe, Asia, and Australia. Study designs were predominantly cross-sectional, with two cohort studies. Sample sizes ranged from 5 to 280 participants, with over 1100 individuals, including participants with FEP and CHR. Sleep disturbances were assessed using self-report questionnaires such as the Pittsburgh Sleep Quality Index (PSQI) and Insomnia Severity Index (ISI), as well as objective measures, including polysomnography (PSG) and actigraphy. Characteristics of the included studies are reported in the Table 1. (Table 2.)

3.4. Sleep architecture in CHR and FEP individuals

3.4.1. Sleep disturbances in CHR individuals

Although studies specifically assessing sleep architecture in CHR individuals are limited compared to those in FEP, emerging evidence suggests that sleep disturbances manifest early in the psychosis spectrum. Lederman et al. (2017) reported that CHR participants experienced greater daytime dysfunction due to sleep disturbances and had a higher need for sleep medications compared to both FEP and HC groups, indicating a clinically significant disruption in sleep-wake regulation.

Yung and McGorry (1996) conducted qualitative interviews with individuals in the prodromal phase of psychosis and found that all participants reported sleep disturbances. Specifically, 10 had decreased sleep, 5 had altered circadian rhythms, and 6 experienced hypersomnia. Similarly, Tan and Ang (2001) observed that 77 % of military recruits with early psychosis reported sleep problems during the prodromal phase.

Iyer et al. (2008) found that 64.8 % of individuals with FEP retrospectively reported sleep problems during the prodromal period, ranking sleep disturbances among the most commonly endorsed early symptoms. Andriopoulos et al. (2011) also found that 33 % of their sample reported pre-onset sleep problems, which were significantly associated with suicidal ideation.

Lunsford-Avery et al. (2015), using actigraphy, reported that CHR adolescents had reduced sleep efficiency, increased WASO, more frequent nocturnal awakenings, and greater nighttime movement compared to controls. These objective sleep disturbances were significantly associated with increased severity of positive symptoms and predicted symptom worsening at 12-month follow-up.

Mayeli et al. (2021) found increased WASO and elevated NREM gamma activity in CHR participants compared to controls. Gamma activity was positively correlated with both WASO and negative symptom severity.

In a follow-up study, Mayeli et al. (2022) observed that CHR participants had reduced sleep efficiency and increased WASO, alongside reduced spindle duration and amplitude. These alterations were associated with poorer verbal memory and slower processing speed.

Cohen et al. (2024) confirmed increased WASO and enhanced NREM gamma activity in CHR individuals, with gamma activity correlating positively with disorganization symptoms.

These findings suggest that both subjective complaints (e.g., insomnia, hypersomnia, circadian rhythm disruption) and objective alterations (e.g., WASO, sleep efficiency, spindle activity) are common and potentially clinically relevant in CHR populations, underscoring the need for further longitudinal and mechanistic research.

3.4.2. Sleep disturbances in FEP individuals

In contrast, sleep architecture in FEP has been more extensively characterized using both subjective and objective assessments. SE was consistently reported as reduced in individuals with FEP (Riemann et al., 1995; Manoach et al., 2014; Kaskie et al., 2019a, b; Yazıhan and Yetkin, 2020; Castelnovo et al., 2024), indicating poorer overall sleep quality. Similarly, SOL was often prolonged, as shown by Poulin et al. (2003) and Kaskie et al. (2019a), while TST was reduced in some studies (Manoach et al., 2014; Kaskie et al., 2019b), though others reported no significant differences (Poulin et al., 2003; Yazıhan and Yetkin, 2020).

Alterations in the distribution of sleep stages were frequently observed. Reduced time spent in REM sleep (Denis et al., 2024; Yazıhan and Yetkin, 2020), decreased N3 (slow-wave sleep), and increased N1 (Castelnovo et al., 2024) were consistently reported. In contrast, Poulin et al. (2003) found a longer duration of REM sleep in FEP subjects, suggesting variability across samples. Sleep microarchitecture also showed notable changes: reduced REM density and prolonged REM onset latency (REMOL) were observed in several studies (Manoach et al., 2014; Kaskie et al., 2019a; Yazıhan and Yetkin, 2020). Sleep spindle density—a key marker of thalamocortical connectivity—was frequently reduced (Manoach et al., 2014; Kaskie et al., 2019b).

Keshavan et al. (2004) and Keshavan et al. (2011) reported that FEP patients exhibited reduced non-linear dynamics in sleep and decreased spindle power, which were associated with poorer cognitive performance. Forest et al. (2007) found that reduced spindle density and stage 4 sleep were linked to slower reaction times, despite no significant differences in gross sleep measures. Denis et al. (2024) found no group differences in sleep architecture, but lower spindle density was linked to impaired memory consolidation.

While some studies screened participants for obstructive sleep apnea (OSA) or periodic limb movement disorder (PLMD), this was not consistent across studies, potentially introducing variability in reported findings.

3.5. Clinical correlates of sleep disturbances in CHR and FEP individuals

3.5.1. Clinical correlates in CHR individuals

Sleep disturbances in CHR individuals have been associated with significant impairments in daily functioning and heightened vulnerability to psychosis onset. Reeve et al. (2019) found that the presence of insomnia and nightmare disorder in CHR individuals was significantly related to increased subthreshold psychotic experiences, depression, anxiety, fatigue, and reduced quality of life. Lederman et al. (2017) observed greater daytime dysfunction and increased need for sleep medications among CHR participants, suggesting functional impairment even before conversion to psychosis.

Yung and McGorry (1996) and Tan and Ang (2001) both reported high rates of sleep disruption during the prodromal period, with Yung et al. noting a variety of complaints including decreased sleep, circadian disruption, and hypersomnia.

Lunsford-Avery et al. (2015) reported that actigraphy-based sleep disruptions in CHR participants were associated with increased positive symptom severity and predicted worsening over time. Mayeli et al. (2021) found that gamma activity during NREM sleep correlated positively with both WASO and negative symptoms. In their 2022 study, reduced spindle duration and amplitude in CHR individuals were associated with deficits in verbal memory and processing speed. Similarly, Cohen et al. (2024) reported that NREM gamma power was positively correlated with disorganization symptoms.

3.5.2. Clinical correlates in FEP individuals

In FEP populations, several studies identified associations between specific sleep parameters and clinical symptoms.

Positive symptoms: Poulin et al. (2003) found that shorter REM latency and reduced REM density were associated with greater positive symptom severity, particularly hallucinations and delusions.

Negative symptoms: Kaskie et al. (2019a, b) reported that reduced sleep spindle density and duration over frontal regions were associated with increased severity of negative symptoms. Castelnovo et al. (2024) also reported a trend toward disrupted slow-wave sleep (N3), potentially relevant to affective flattening and social withdrawal.

Table 1
Study characteristics of included articles.

Article	Country	Study design	Population (N/HC)	Age M ± SD	Male gender	Sleep measure	Diagnostic/ psychotic symptom measure	Main features
Yung and McGorry, 1996	Australia	Cross-sectional	FEP: 21	23.1 ± 4.19	14 (67 %)	Qualitative interview	MAPP	Qualitative interviews to investigate prodromal symptoms prior to FEP. 100 % experienced sleep disturbances. 10 had decreased sleep, 5 had altered circadian rhythms and 6 had hypersomnia
Tan and Ang, 2001	Singapore	Cross-sectional	FEP: 30	20.6 ± 4.5	30 (100 %)	Qualitative semi-structured interview	DSM IV assessment	Investigated prodromal symptoms prior to FEP in military personnel. 77 % reported sleep disturbance during prodromal period.
Iyer et al., 2008	Canada	Cohort	FEP: 128	22.58 ± 3.89	87 (68 %)	CORS	SCID	Qualitative interviews to investigate prodromal symptoms prior to FEP. 64.8 % reported sleep disturbance during this period, making it the third most commonly reported symptom.
Andriopoulos et al., 2011	Greece	Cohort	FEP: 106	29.6 ± 7.5	74 (70 %)	Case note review/ assessment interview/family member report	SCID	Study of prodromal symptoms associated with suicidal behaviour. Sleep disturbances significantly associated with suicidal ideation but not suicide attempts. 33 % total sample reported sleep disturbance prior to FEP.
Lederman et al., 2017	Australia	Cross-sectional	FEP: 10 HC: 10 CHR: 10	FEP: 21.6 ± 1.83 HC: 22 ± 0.94 CHR: 19.5 ± 0.62	FEP: 8 (80 %) HC: 7 (70 %) CHR: 8 (80 %)	Self-report questionnaire (PSQI)	DSM V assessment	Study on the prevalence of modifiable cardiometabolic risk factors in CHR. Day dysfunction due to sleepiness and the need for specific medications to improve sleep was significantly greater among CHR participants compared to HC and FEP participants
Subramaniam et al., 2018	Singapore	Cross-sectional	FEP: 280	25.8 ± 6.2	142 (51 %)	Self-report questionnaire (ISI)	SCID/PANSS, GAF	Study on the prevalence of insomnia in FEP and on the relationship between insomnia and socio-demographic and clinical variables. The prevalence of clinical insomnia was 22.6 %. Insomnia was associated with significant decreases in all QOL domains.
Lunsford-Avery et al., 2015	USA	Prospective longitudinal observational study	CHR: 36 HC: 31	16.5 ± 2.2	41 (61 %)	Actigraphy, PSQI	SIPS	CHR participants showed reduced sleep efficiency, increased WASO, number of awakenings, and movement during sleep. Objective sleep disturbances predicted worsening of positive symptoms at 12 months, outperforming subjective measures.
Ong et al., 2020	Singapore	Cross-sectional	FEP: 280	25.8 ± 6.2	142 (51 %)	Self-report questionnaire (PSQI)	SCID/PANSS, GAF	Investigated the association of poor sleep quality and its components with domains of QOL in FEP. 62.9 % reported reduced sleep quality. Poor sleep quality was associated with significantly lower scores in all domains of QOL.
Fekih-Romdhane et al., 2021	Tunisia	Cross-sectional	FEP: 54 HC: 61	FEP: 26.8 ± 6.1 HC: 27.9 ± 5.5	FEP: 35 (65 %) HC: 44 (72 %)	Self-report questionnaire (PSQI)	DSM V assessment/ PANSS, GAF	Sleep parameters examined in FEP, compared to their unaffected siblings and HC. 96 % of patients reported reduced sleep quality. Clinically significant daytime sleepiness was noted in 57.4 % of patients.
Riemann et al., 1995	Germany	Cross-sectional	FEP: 10 HC: 10	FEP: 15.8 ± 1.8 HC: 16.6 ± 1.9	FEP: 4 (40 %) HC: 7 (70 %)	PSG	SCID	Sleep EEG examined in FEP, compared to participants with depression and healthy controls. FEP group significantly reduced sleep efficiency compared to HC.
Poulin et al., 2003	Canada	Cross-sectional	FEP: 11 HC: 11	FEP: 29.6 ± 15.8 HC: 25.3 ± 11.3	FEP 6: (55 %) HC: 8 (73 %)	PSG	DSM IV-R assessment/ BPRS	Inpatient FEP sample. Shorter REM latency associated with greater positive symptom severity. Reduced REM duration and density associated with greater symptom severity overall.
Keshavan et al., 2004	USA	Cross-sectional	FEP: 10 HC: 10	FEP: 20.5 ± 4.4	FEP: 4 (40 %)	PSG	SCID/SAPS, SANS	Lower non-linearity scores during wake and reduced symbolic dynamics and largest Lyapunov exponent (LLE)

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Table 1 (*continued*)

Article	Country	Study design	Population (N/HC)	Age M ± SD	Male gender	Sleep measure	Diagnostic/ psychotic symptom measure	Main features
				HC: 20.3 ± 5.4	HC: 6 (60 %)			values during sleep compared to controls. Inverse relationship between symbolic dynamics and executive functioning.
Forest et al., 2007	Canada	Cross-sectional	FEP: 8 HC: 8	FEP: 31 ± 19.9 HC: 21.4 ± 4.9	FEP: 6 (75 %) HC: 6 (75 %)	PSG	DSM IV-R assessment/ PANSS	No differences in sleep variables between groups. Reduced spindle density and % stage 4 sleep associated with longer RT in psychosis group.
Poulin et al., 2008	Canada	Cross-sectional	FEP: 10 HC: 30	FEP: 24.6 ± 10.0 HC: 24.5 ± 7.4	FEP: 5 (50 %) HC: 16 (53 %)	PSG	DSM IV-R assessment/ BPRS	No differences in absolute spectral amplitudes between groups. Analysis of relative spectral amplitude showed reduced alpha over frontal, central and temporal regions and increased beta over occipital regions in psychosis group.
Keshavan et al., 2011	USA	Cross-sectional	FEP: 27	27.2 ± 7.3	18 (67 %)	PSG	SCID/SAPS, SANS	Spindle power in relation to psychotic symptoms and neurocognition. No relationship between spindles and symptoms. Decreased spindle power associated with poorer cognitive performance but not IQ.
Guénolé et al., 2014	Canada	Cross-sectional	FEP: 10 HC: 10	FEP: 24.9 ± 11.1 HC: 23 ± 8.0	FEP: 5 (50 %) HC: 6 (60 %)	PSG	BPRS	Examined relationship between sleep instability (using microstructural EEG) and BPRS scores. No differences in sleep instability between psychosis participants and healthy controls.
Manoach et al., 2014	USA	Cross-sectional	sFEP: 15 oFEP: 11 HC: 25	sFEP: 28 ± 8 oFEP: 27 ± 7 HC: 27 ± 7	sFEP: 11 (73 %) oFEP: 6 (55 %) HC: 16 (64 %)	PSG	SCID/SAPS, SANS	Sleep spindles, cognition and psychotic symptoms in 'schizophrenia' FEP (sFEP)and 'other psychosis' (oFEP), compared to HC. sFEP participants showed reduced spindle density compared to oFEP and HC.
Mayeli et al., 2022	USA	Cross-sectional	CHR: 24 HC: 24	CHR: 21.2 ± 4.5 HC: 22.3 ± 4.0	CHR: 13 (54 %) HC: 14 (58 %)	hd-EEG, PSG	SIPS/SOPS	CHR participants showed increased WASO, reduced sleep efficiency, and reduced spindle duration and amplitude. Sleep spindle abnormalities were associated with worse verbal memory and processing speed.
Mayeli et al., 2021	USA	Cross-sectional	CHR: 22 HC: 20	CHR: 20.3 ± 4.6 HC: 20.6 ± 4.2	CHR: 10 (45 %) HC: 9 (45 %)	hd-EEG, PSG	SIPS/SOPS	CHR showed increased WASO and higher NREM gamma power, especially in fronto-parieto-occipital areas. Gamma power was positively correlated with WASO and SOPS negative symptoms.
Kaskie et al., 2019a, b	USA	Cross-sectional	FEP: 27 HC: 23	FEP: 23.2 ± 5.8 HC: 24.7 ± 5.7	FEP: 17 (63 %) HC: 16 (70 %)	hd-EEG	SCID/PANSS	Study on the sleep architecture in FEP. FEP patients had significantly increased sleep latency as well as reduced total sleep time. Additionally, they showed reduced spindle duration and density, but not in spindle amplitude. These spindles reductions predicted the severity of FEP patients' negative symptoms.
Yazıhan and Yetkin, 2020	Turkey	Cross-sectional	FEP: 21 HC: 21	FEP: 22.66 ± 2.39 HC: 22.71 ± 3.45	FEP: 21 (100 %) HC: 21 (100 %)	PSG	DSM IV assessment/ PANSS	Examined sleep characteristics, sleep spindles, and neuropsychological profiles of the drug-naive patients with FEP. The patient group's percentage of stage N2 sleep and sleep efficiency index was lower than in the control group. Spindle density was found to be reduced in the patient group. Different percentage of stage N1 and N3 was associated with different severity of negative symptoms.
Denis et al., 2024	USA	Cross-sectional	FEP: 17 HC: 27	FEP: 21 ± 4 HC: 24 ± 4	FEP: 9 (53 %) HC: 14 (52 %)	PSG	SCID/ K-SADS-PL/PANSS	Evaluation of sleep architecture and sleep spindles in FEP. No group differences in sleep quality and architecture. Reduced sleep spindle density and a correlated deficit in overnight memory consolidation in

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Table 1 (*continued*)

Article	Country	Study design	Population (N/HC)	Age M ± SD	Male gender	Sleep measure	Diagnostic/ psychotic symptom measure	Main features
								minimally medicated early course schizophrenia.
Cohen et al., 2024	USA	Cross-sectional	CHR: 12 HC: 16	CHR: 21.2 ± 4.4 HC: 23.6 ± 4.2	CHR: 7 (58 %) HC: 8 (50 %)	PSQI, EEG spectral analysis	SIPS/SOPS	CHR showed increased WASO and elevated NREM gamma activity, especially in fronto-parietal areas. Gamma activity was positively associated with disorganization symptoms.
Castelnovo et al., 2024	Italy	Cross-sectional	FEP: 5 HC: 5	FEP: 24.4 ± 3.5 HC: 24.0 ± 3.32	FEP: 5 (100 %) HC: 5 (100 %)	hd-EEG, PSQI, ESS, SBAQ	PANSS/ LSHS-R/BACS	Investigated sleep characteristics and sleep slow wave traveling in FEP. FEP patients showed a trend for an increase in wake after sleep onset and NREM stage 1 (%), and for a decrease in NREM stage 3 and sleep efficiency. Slow wave analysis showed lower slow wave density at baseline in FEP.
Reeve et al., 2019	England	Cross-sectional	FEP: 60	23.7 ± 3.2	FEP 39 (65 %)	ACT	DSM V assessment/ DISP	Study on the assessment of sleep disorders and their correlates in patients with early psychosis. 80 % had at least one sleep disorder, with insomnia and nightmare disorder being the most common. Sleep disorders were significantly associated with increased psychotic experiences, depression, anxiety, fatigue, and lower quality of life.

BPRS: brief psychiatric rating scale; CORS: circumstances of onset and relapse schedule; DSM: diagnostic and statistical manual of mental disorders; FEP; first episode psychosis; HC: healthy controls; MAPP: multidimensional assessment of psychotic prodrome; PANSS: positive and negative syndrome Scale; PSG: polysomnography; SAPS/SANS: scale for the assessment of positive symptoms/scale for the assessment of negative symptoms; SCID: structured clinical interview for DSM-IV; SIPS: structured Interview for prodromal symptoms; PSQI = Pittsburgh sleep quality index; ISI: insomnia severity index; hd-EEG = high-density electroencephalography; ACT = actigraphy; DISP = diagnostic interview for sleep patterns and disorders; SBAQ = stop-bang apnea questionnaire.

Table 2
Sleep architecture in early psychosis compared to healthy controls.

	N	N (HC)	Nights recorded	Screened for OSA/PLMD	SE	SOL	WASO	TST	% time spent in each sleep stage	REM density	REMOL	Spindle density
Riemann et al., 1995	10	10	3	No	Reduced in FEP	NS	NS	NS	NS	NS	NS	–
Poulin et al., 2003	11	10	3	Yes	NS	Longer in FEP	–	NS	NS	NS	Longer in FEP	NS
Forest et al., 2007	8	8	2	Yes	NS	NS	NS	NS	NS	NS	NS	NS
Manoach et al., 2014	26	25	2	No	Reduced in FEP	NS	Increased in FEP	Reduced in FEP	–	NS	NS	Reduced in FEP
Mayeli et al., 2022	24	24	1	Yes	Reduced in CHR	NS	Increased in CHR	NS	NS	–	–	NS
Mayeli et al., 2021	22	20	1	Yes	NS	NS	Increased in CHR	NS	–	–	–	–
Lunsford-Avery et al., 2015	36	31	6	No	Reduced in CHR	–	Increased in CHR	–	–	–	–	–
Kaskie et al., 2019a	20	20	1	No	Reduced in FEP	Longer in FEP	NS	Reduced in FEP	NS	NS	–	–
Kaskie et al., 2019b	27	23	1	No	–	Longer in FEP	NS	Reduced in FEP	NS	NS	–	Reduced in FEP
Yazıhan and Yetkin, 2020	21	21	2	No	Reduced in FEP	NS	NS	NS	N2 reduced in FEP	NS	NS	Reduced in FEP
Denis et al., 2024	17	27	2	No	NS	NS	NS	NS	REM reduced in FEP	NS	NS	Reduced in FEP
Castelnovo et al., 2024	5	5	1	Yes	Reduced in FEP	–	Increased in FEP	NS	N1 increased and N3 reduced in FEP	–	NS	–

FEP = first episode of psychosis; CHR = Clinical High-Risk; HC = healthy controls; OSA = obstructive sleep apnea; PLMD = periodic limb movement disorder; SE = sleep efficiency; SOL = sleep onset latency; WASO = wake after sleep onset; TST = total sleep time; REM = rapid eye movement; REMOL = rapid eye movement onset latency; NS = non-significant; – = not studied.

Neurocognitive functioning: Denis et al. (2024) found that reduced sleep spindle density was linked to impaired overnight memory consolidation. Keshavan et al. (2011) demonstrated a correlation between spindle power and executive function performance, though not IQ. Forest et al. (2007) observed reduced reaction time in FEP patients with diminished spindle activity.

Suicidal ideation: Andriopoulos et al. (2011) found that 33 % of patients reported sleep disturbances prior to psychosis onset, and these were significantly associated with suicidal ideation, though not attempts.

Quality of life: Subramaniam et al. (2018) observed that 22.6 % of FEP patients met criteria for clinical insomnia, which was associated with significantly lower scores in all quality of life domains. Similarly, Ong et al. (2020) found poor sleep quality (as measured by the PSQI) was linked to reduced psychological, physical, and social functioning.

4. Discussion

This systematic review provides an overview of the literature investigating sleep disturbances in individuals experiencing FEP or CHR. Our findings underscore the pervasiveness of sleep disturbances across both CHR and FEP populations, reinforcing their role as potential early indicators of psychosis. The integration of both subjective and objective measures provides a nuanced perspective, suggesting that while self-reported sleep disturbances correlate with symptom severity and functional impairments, objective findings (e.g., PSG-derived alterations in sleep architecture) offer insights into underlying neurophysiological mechanisms.

The mechanisms underpinning the relationship between sleep disturbances and psychosis remain an area of active investigation. Emerging evidence suggests that sleep disruptions may exacerbate psychotic symptoms, especially through their impact on emotional regulation, cognitive functioning, and neuroinflammation (Krystal et al., 2013). Additionally, REM sleep abnormalities—crucial for memory consolidation and emotional processing—may contribute to the persistence and intensity of psychotic symptoms.

The relationship between sleep disturbances and psychosis is likely multifactorial. Dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, impaired melatonin secretion, and disruptions in slow-wave sleep have all been proposed as contributing mechanisms. Furthermore, genetic studies indicate that sleep and psychosis may share overlapping pathways, particularly those involving CLOCK genes and dopaminergic signaling (Walker and Stickgold, 2006; Arguello and Gogos, 2006).

It is also possible that sleep disturbances contribute to psychosis through their impact on cognitive functioning and emotional regulation. For instance, REM sleep abnormalities, frequently observed in the reviewed studies, have been linked to impaired memory consolidation and emotional processing, which are critical for maintaining psychological resilience (Nishida et al., 2009).

Given the central role of sleep disturbances in FEP and CHR populations, addressing these issues should be a priority in clinical practice. Non-pharmacological approaches such as cognitive-behavioral therapy for insomnia (CBT—I) have demonstrated effectiveness in reducing sleep-onset latency and improving sleep continuity (Myers et al., 2011; Davies et al., 2017). These improvements in sleep quality may alleviate psychotic symptoms, including delusions and paranoia. Similarly, chronotherapy, which involves realigning disrupted circadian rhythms through light exposure and structured sleep scheduling, has shown potential for stabilizing mood and reducing symptom severity in these populations (Wirz-Justice et al., 2005). The integration of tools like polysomnography, actigraphy, and self-reported sleep diaries into routine clinical evaluations can facilitate the early identification of significant sleep disturbances (Kaplan and Harvey, 2013). Prioritizing interventions that address cognitive, behavioral, and environmental factors contributing to poor sleep is essential to achieving sustainable outcomes without undue reliance on medication. While these findings provide important insights into sleep disturbances in early psychosis, several areas remain insufficiently explored and merit further investigation.

4.1. Future directions

Among the most relevant areas for future investigation is the role of pharmacological treatments in managing sleep disturbances. Pharmacological treatments also play a role in managing sleep disturbances. Melatonin receptor agonists and antipsychotics with sedative properties can improve sleep outcomes by enhancing sleep architecture and reducing the time needed to fall asleep (McClung, 2013). However, these interventions must be carefully managed to avoid side effects such as daytime sedation or metabolic complications (Sateia et al., 2017). While antipsychotics may help regulate sleep patterns in FEP patients by promoting sedation and enhancing sleep continuity, their effects often target symptoms rather than the underlying mechanisms of sleep disturbances Krystal et al., 2008). For instance, these medications may improve sleep architecture, such as increasing total sleep time or reducing nocturnal awakenings, but they do not directly address the cognitive, behavioral, or environmental factors that contribute to disrupted sleep. As a result, reliance on their sedative properties may create a superficial sense of improvement while underlying issues—such as maladaptive thought patterns, irregular sleep-wake schedules, or environmental disruptions—remain unresolved. This masking effect can complicate long-term management, as it may delay the identification and treatment of root causes, potentially leading to persistent sleep problems once the medication is reduced or discontinued. Therefore, while antipsychotics can be a valuable tool for acute symptom management, they should be used in conjunction with non-pharmacological strategies to ensure a more comprehensive and sustainable approach to treating sleep disturbances in FEP patients (Laskemoen et al., 2019). Future research should aim to determine the optimal balance between pharmacological and non-pharmacological interventions, tailoring strategies to individual needs while minimizing adverse effects. Integrating sleep-focused interventions into standard care could improve overall treatment outcomes and enhance the quality of life for individuals with FEP and CHR.

The findings of this review highlight the critical need for an interdisciplinary approach to addressing sleep disturbances in psychosis. Collaboration between psychiatrists, sleep specialists, and neuroscientists can foster the development of integrative care models. Such models might combine behavioral therapies, pharmacological interventions, and lifestyle modifications tailored to individual patients' needs. Additionally, integrating sleep-focused strategies into existing early psychosis intervention programs could enhance overall treatment efficacy.

Understanding the socio-environmental factors influencing sleep in early psychosis populations is another area requiring attention. Variables such as housing instability, which may disrupt sleep routines and create chronic stress, employment challenges that contribute to irregular schedules and financial stress, and the role of social support networks in providing emotional and practical resources, could profoundly impact sleep quality and psychosis trajectories (Marwaha and Johnson, 2004). For instance, individuals experiencing housing instability might face noise disturbances or unsafe environments that compromise sleep. At the same time, those lacking social support might struggle with isolation, exacerbating sleep and mental health issues (Cohen and Wills, 1985). Addressing these contextual factors through psychosocial interventions, such as housing assistance programs, vocational rehabilitation, and community-based support networks, may complement direct sleep-focused treatments. Future research should prioritize exploring how these factors intersect with biological vulnerabilities in early psychosis to guide patient-centered care.

Despite the robust findings, this review reveals critical gaps in the literature. Most studies utilized cross-sectional designs, which limit

causal inferences about the relationship between sleep disturbances and psychosis. Longitudinal studies are needed to determine whether sleep disturbances precede psychosis onset and whether their resolution alters disease progression.

Methodological inconsistencies in sleep assessment also present a challenge. While self-report measures like PSQI are valuable for large-scale studies, they are prone to biases and lack the objectivity of tools like PSG or actigraphy. Future research should prioritize integrating objective and subjective measures to provide a comprehensive understanding of sleep disturbances in psychosis.

This review has several limitations. First, we did not distinguish between help-seeking and non-help-seeking individuals when determining eligibility. Since help-seeking status may affect clinical and sleep-related outcomes, future research should investigate its potential moderating effects.

Secondly, some of the studies included had small sample sizes, with as few as five participants. Although these studies may provide valuable preliminary insights, their limited statistical power requires cautious interpretation of the findings.

Lastly, although circadian rhythm dysregulation has been implicated in psychosis, we did not include studies specifically assessing circadian markers such as melatonin rhythms or actigraphy-based phase shifts. Our focus was on sleep disturbances and architecture rather than circadian parameters. Future studies should examine how circadian disruptions contribute to sleep alterations in early psychosis, as this remains a critical yet underexplored area.

Another avenue for exploration is the neurobiological basis of sleep disruptions in psychosis. Advanced neuroimaging techniques, such as functional MRI (fMRI), high-density EEG, and diffusion tensor imaging (DTI), could help delineate the neural circuits underlying these disturbances (Keihani et al., 2025). For example, thalamocortical dysconnectivity has been implicated in both disrupted sleep spindles and cognitive impairments in psychosis (Ferrarelli et al., 2007), while alterations in limbic circuits may contribute to the heightened emotional reactivity and sleep disturbances observed in these populations (Walker and van der Helm, 2009). Similarly, prefrontal cortex dysfunction, a hallmark of psychosis, could impair the regulation of sleep-wake cycles through disruptions in executive function and circadian rhythms (Wulff et al., 2010a, b). Researchers can better understand the interplay between sleep disruptions and psychopathology by identifying specific network alterations. These insights may ultimately guide the development of targeted interventions, such as neuromodulation techniques (e.g., transcranial magnetic stimulation) or pharmacological approaches tailored to specific neural deficits.

5. Conclusion

This systematic review provides a comprehensive synthesis of sleep disturbances in early psychosis, bridging gaps between subjective reports and objective physiological markers. By addressing methodological heterogeneity and inconsistencies in prior literature, we offer a refined understanding of the role of sleep in the progression of psychosis. Given the increasing recognition of sleep disturbances as modifiable treatment targets, future research should focus on developing tailored interventions that address both subjective sleep complaints and neurophysiological disruptions. Integrating sleep-focused interventions into early psychosis care could represent a promising avenue for improving long-term clinical outcomes.

CRediT authorship contribution statement

Valentina Baldini: Writing – original draft, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Francesco Pasquino:** Writing – original draft, Formal analysis, Data curation. **Diana De Ronchi:** Writing – original draft, Validation, Supervision. **Giuseppe Plazzi:** Writing – review & editing, Validation, Supervision. **Lorenzo Pelizza:** Writing – review & editing, Writing – original draft, Validation, Supervision. **Marco Menchetti:** Writing – review & editing, Writing – original draft, Validation, Supervision.

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Declaration of competing interest

The authors declare no other conflicts of interest.

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