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Sleep in seasonal affective disorder

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Abstract

Sleep in seasonal affective disorder (SAD) has been primarily characterized by delayed sleep timing and self-reports of hypersomnolence. It is unclear whether delayed sleep timing is due to circadian or behavioral misalignment and if effective treatments operate independently of the circadian system. Discrepancies between self-report and actigraphic/polysomnographic sleep duration in SAD hinder clarification of hypersomnolence as a cardinal symptom. Previous studies have largely neglected the summer remission period in SAD, which could yield valuable insight to the role sleep disturbances play in the onset and recurrence of winter depressive episodes. Future studies should incorporate multi-method, multi-season assessment of sleep and circadian rhythms to best characterize relevant sleep-circadian phenotypes. Empirically determining sleep phenotypes present in SAD will pave the way for targeted sleep interventions.

Seasonal Affective Disorder (SAD) is a mood disorder characterized by predictable onset of depression in the fall and winter months and spontaneous remission in the spring and summer [1]. SAD affects an estimated 5% of adults in the United States and is associated with around 5 months of functional impairment per year [2]. Sleep disturbance in SAD can be distinguished from nonseasonal Major Depressive Disorder (MDD) in three key domains: (1) delay in the timing of sleep and other circadian rhythms, (2) frequent complaints of hypersomnolence, and (3) inconclusive sleep architecture differences relative to controls. However, there is marked heterogeneity in each of these domains. Interestingly, the seasonal recurrence, circadian rhythm delays, and atypical symptom presentation of hypersomnolence in SAD are more similar to Bipolar Disorder (BD; [3]) than nonseasonal MDD. While, sleep-focused interventions have shown to mitigate depressive symptoms in nonseasonal depression and BD [4,5], no sleep-focused interventions have been employed in SAD. Better characterizing the sleep phenotypes present in SAD will pave the way for targeted sleep interventions. Here, we review recent advances in sleep and circadian disturbances in SAD, and propose avenues for future research.

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Conflict of interest statement

Nothing declared.

Circadian rhythm abnormalities

The seasonality of winter depressive episodes and antidepressant effects of light therapy suggest SAD etiology is related to abnormal responses to low environmental light levels in winter [6]. In healthy individuals, light entrains and synchronizes the circadian clock to the earth's solar day (i.e. circadian photoentrainment) and has downstream effects on sleep regulation [7], alertness [8], and mood [9••] through melanopsin containing intrinsically photosensitive retinal ganglion cells (ipRGCs). In SAD, light treatment and melatonin administration are antidepressant [10], possibly through advancing delayed circadian rhythms and correcting circadian misalignment, which indirectly regulates mood. Such findings support a mechanistic role of circadian rhythm disruption in SAD; however, recent work suggests the therapeutic effect of light on mood may also operate independently of the circadian system [9••,11] through direct central projections of ipRGCs [12].

Circadian misalignment

Multiple existing conceptual frameworks for SAD emphasize circadian rhythm disruption, and have been reviewed elsewhere [13]. The prevailing circadian theory of SAD is the phase-shift hypothesis, which postulates that there is a delay in circadian phase relative to the sleep/wake cycle in SAD (circadian misalignment), and that this delay only occurs during the winter when environmental light levels fall below threshold for circadian photoentrainment [14]. Circadian misalignment often presents as a discrepancy between circadian rhythms of melatonin release and sleep timing and can cause problems with sleep quality and daytime alertness [15]. Evidence for this theory has been mixed, and there has been limited recent work testing this hypothesis in SAD, although evidence supporting the efficacy of light therapy for about half of individuals with SAD is consistent.

Delayed phase

In SAD, delayed circadian timing is thought to manifest behaviorally as early onset insomnia and morning hypersomnolence (i.e. difficulty falling asleep and difficulty waking up the following morning [13,14]). In two large studies, seasonality of depression was associated with later self-reported sleep timing preference [16,17]. Adults with delayed sleep phase syndrome (DSPS) are 3.3 times more likely to meet criteria for SAD than those without DSPS [18]. Though these studies suggest behaviorally delayed sleep timing, prior biological studies of circadian markers have shown that a minority of individuals with SAD are phase advanced [19] and some have circadian timing markers indistinguishable from controls [20]. Recent work on rest activity rhythms (RARs), 24-hour periods of activity and rest, found an increase in early evening settling (downmesor) during the winter months in SAD participants [21•], which was inconsistent with delayed activity timing and reduced inter-daily stability found previously [22]. These discrepancies in circadian timing in SAD might be explained by methodological weaknesses of existing work (e.g. small samples, non-biological circadian measure, lack of multi-season measurements), or could be because not all individuals with SAD are delayed. If present, delayed sleep phase could be indicative of circadian or behavioral misalignment, which have significantly different treatment implications [23•]. Specifically, interventions to advance circadian time will have

no effect in those with intermediate phase, or possibly deleterious effects in those with advanced circadian phase.

Circadian treatments for SAD

If an individual with SAD experiences a delayed sleep phase only in winter, and if the etiology of SAD is delayed circadian photoentrainment, then treatment focused on realigning circadian and behavioral rhythms is indicated. While light therapy has been considered the gold standard treatment in SAD [24] and is effective in about half of individuals with SAD [10], a more recent review has criticized methodological and design issues in previous studies and the paucity of randomized controlled trials of light therapy in SAD within past decade [25••]. Current work supports light therapy as an effective treatment for SAD [26], but suggests that circadian misalignment/delayed phase may be an epiphenomenon of effective treatment and not actually mechanistic [9••]. Despite the relationship between mood symptoms and circadian rhythmicity, the effects of light therapy on mood cannot be fully explained by circadian mechanisms [27].

Hypersomnolence

Hypersomnolence, expressed as either excessive daytime sleepiness and/or increased sleep duration, is an atypical vegetative symptom of depression [28]. Although hypersomnolence is considered a cardinal symptom in the characterization and diagnosis of SAD [1,29], it is a clinically heterogeneous construct that requires further characterization.

Sleep duration

Previous studies have found that approximately 64–80% of SAD participants self-report a winter increase in sleep [1,29,30•,31,32] which varies from 30 min [31] to over two hours longer in duration compared to controls [32]. These studies relied primarily on self-reported changes in sleep duration, typically assessed with retrospective questionnaires or semi-structured clinical interviews. In contrast, behaviorally assessed metrics of sleep do not show significantly lengthened sleep in SAD. Using actigraphy, Winkler *et al.* [33••] found that individuals with SAD slept significantly less than controls in winter, had decreased sleep efficiency, and were phase delayed compared to controls. Those SAD participants slept 7 hours (SD 1:29), which is inconsistent with self-reports from other studies of increased winter sleep duration. Similarly, a meta-analytic study of polysomnographic sleep in psychiatric disorders including three SAD studies found that shorter mean sleep duration in SAD did not statistically differ from controls [34].

These discrepancies between self-reported and actigraphic/polysomnographic sleep duration in SAD could reflect the challenge of accurately quantifying self-reported hypersomnolence in mood disorders [35]. Individuals could be endorsing hypersomnolence due to a subjective need for longer sleep duration in light of excessive daytime sleepiness, or because of depression symptoms including anhedonia and fatigue [29,36], or distorted cognitions about sleep [37]. With only self-report information, individuals reporting hypersomnolence could be experiencing increased time in bed and/or fragmented sleep [38]. Including time in bed variables [29] and incorporating home actigraphy or polysomnography would determine if

hypersomnolence reports stem from increased time in bed, or other behavioral or emotional factors.

Sleep fragmentation

Self-reports of combined insomnia and hypersomnolence in a seasonal depression sample suggests alternating patterns of insomnia and hypersomnolence, which may be obscured when averaging sleep duration across multiple nights [30•]. After a night of difficulty initiating sleep, individuals might compensate by taking daytime naps, getting into bed early, or sleeping in the next morning [39]. This sleep extension may precipitate future nights of insomnia. Co-occurring hypersomnolence and insomnia is observed in unipolar depression and presents as a more severe depressive profile than either alone [40]. Children and adolescents with SAD report mostly insomnia symptoms including shorter sleep durations, delayed sleep times, lower sleep efficiencies, and longer sleep latencies than controls [41•]. Shorter sleep durations and increased sleep need were both associated with increased seasonality in Norwegian adults [42], suggesting multiple subgroups of sleep presentation (i.e. insomnia, hypersomnolence, or combined) [42].

Sleep inertia

Difficulty waking and subsequent daytime sleepiness have been reported in adults [43] and adolescents with SAD [41•,44], indicating that sleep inertia (i.e. decrements in sensory, motor, and cognitive function immediately upon awakening) may be a component of morning hypersomnolence in SAD. However, sleep inertia in mood disorders might be a reflection of mood dysregulation [45] because both healthy controls and those with hypersomnolence and a psychiatric diagnosis did not show abnormalities in the sleep-wake transition during a forced awakening protocol that was present in individuals with primary sleep disorders (e.g. idiopathic hypersomnia or narcolepsy [46]). No studies to date have behaviorally assessed sleep inertia in SAD.

Sleep architecture

To date, there have been only three polysomnographic studies of sleep architecture in SAD, especially compared to nonseasonal MDD. Polysomnography (PSG) involves the simultaneous use of electroencephalography, electromyography, and electrooculography to objectively measure sleep. Visual staging of PSG sleep yields estimates of non-rapid eye movement sleep stages (N1, N2, N3) and rapid eye movement (REM) sleep. A recent meta-analysis of PSG findings across mental disorders concluded no significant differences between SAD and controls for sleep continuity, sleep depth, and REM pressure (defined by shorter REM latency, increased REM density, or longer duration of REM sleep) [34], whereas nonseasonal MDD was associated with the most severe decrements in sleep continuity, sleep depth, and REM sleep pressure alterations [34]. Because only three published PSG studies have been performed in SAD samples, analyses on number of night-time awakenings, total time awake at night, REM density, and duration of N3 (slow-wave sleep; SWS) were not performed. The three SAD PSG samples had a combined sample size of 96, with reported Hedge's $g = 0.12–0.42$, which would require a sample of at least 292 to see significant differences in PSG ($N = 19$ [47]; $N = 31$ [48]; $N = 46$ [49]). The

PSG differences are likely to be small in SAD, but samples of $N = 300$ combined SAD and control participants are feasible to obtain.

Despite recent evidence linking lower SWS in hypersomnolence with or without comorbid nonseasonal depression [50], only one study in SAD found lower SWS in winter [48]. Interestingly, the same study found no difference in SWS between SAD and controls during the summer, suggesting that reduced SWS might be a state marker in SAD. In MDD, findings of reduced SWS are observed in individuals currently in episode, in remission, and in high-risk probands [51]. Future polysomnographic studies in SAD should include both summer and winter assessments in a larger sample to elucidate the role of SWS.

Conclusions and future directions

SAD is characterized by reported delays in sleep onset and offset, and self-reports of hypersomnolence that are not observed in SAD when measured with actigraphy. It is unclear if delayed sleep timing in SAD is due to circadian or behavioral misalignment and whether treatments that advance circadian timing also resolve sleep complaints because such summer assessments are uncommon in SAD. Little is known about sleep architecture in SAD, making comparisons to nonseasonal depression difficult. Overall, hypersomnolence in SAD appears to be a self-reported phenomenon, and there may be sub-groups of individuals with SAD with different sleep patterns, which may be obscured when only group means are reported. Including both home actigraphy or polysomnography measures when assessing sleep in SAD would provide more comprehensive and behavioral or objective estimates of sleep duration.

The characteristics of sleep in SAD during a wintertime depressive episode have not been compared to sleep during summer spontaneous remission. The seasonal recurrence of SAD allows for repeated observation during and between depressive episodes and could yield valuable insight into any role sleep disturbances may play in the onset and recurrence of winter depressive episodes. Sleep disturbances, namely insomnia, are residual symptoms of nonseasonal depression and confer risk of relapse [52]. Hypersomnolence is also a treatment-resistant symptom of depression [53] and has been shown to increase the likelihood of depressive relapse [29,38,54,55]. If either are present during summer remission in SAD, this could contribute to a subsequent winter depressive episode in this highly recurrent depressive disorder [56].

Efforts to treat sleep in nonseasonal depression have been successful [5]. However, the ambiguity of sleep and circadian disruptions in SAD have hindered the development of sleep-focused treatments. We hypothesize four distinct sleep phenotypes in SAD that contribute to the inconsistent findings of delayed circadian phase and hypersomnolence. Subgroups are hypothesized to include (1) a ‘true’ hypersomnolence group as indicated by long sleep durations and/or excessive daytime sleepiness, (2) a phase-delayed group with circadian misalignment, (3) early settlers whose depression presents as fatigue and behavioral disengagement, and (4) an insomnia group with fragmented, non-restorative sleep more typical of nonseasonal depression. Because phenotypic sleep heterogeneity may also be a function of age [41•], sex [42], social role demands, or substance use [57], measuring

such confounds will be critical for subgroup analyses. Although we hypothesize four likely subgroups here, analyses to elucidate latent sleep phenotypic classes could test the validity of our a priori hypothesis. Future studies should incorporate multi-method, multi-season assessments of sleep and circadian rhythms to determine whether sleep variables seen in episode in SAD are either (a) symptoms or correlates of episode status, or (b) pre-existing risk factors present during summer remission and thus possible candidates for intervention to reduce the recurrence of this highly recurrent disorder. Dimensions of sleep health including regularity and subjective sleep quality should also be examined [58], and doing so would facilitate research comparing multiple domains of sleep health in SAD, increasing transdisciplinary understanding of sleep in multiple contexts and disorders. Once distinct sleep phenotypes in SAD are empirically determined, treatments to target-specific sleep phenotypes can be tested as acute and preventive interventions.

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