

The Influence of Circadian Rhythm on Mood Disorders

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Abstract: Mood disorders are among the leading causes of disability; their etiology is recognized to involve chronobiological dysregulation. The circadian system, governed by a central pacemaker in the suprachiasmatic nucleus (SCN), orchestrates daily rhythms in sleep, hormone secretion, and neurotransmission. Circadian rhythm disruption plays a central role in the onset and maintenance of mood disorders, including major depressive disorder (MDD), bipolar disorder (BD), seasonal affective disorder (SAD), and anxiety. Disruption of these rhythms through environmental factors such as light exposure or genetic vulnerabilities can trigger or exacerbate emotional dysregulation. Clinical studies indicate that treatments like bright light therapy, melatonin receptor agonists, and social rhythm therapy are effective in restoring circadian coherence and improving symptoms. This review synthesizes evidence across molecular, endocrine, and behavioral levels, highlighting how disruptions in melatonin and cortisol secretion, sleep-wake cycles, and clock gene expression contribute to the development of psychopathology. It suggests that future directions should emphasize precision approaches based on genetic and circadian biomarkers, aiming to personalize interventions. Moreover, bridging chronobiology with psychiatric care offers promising pathways toward sustainable, rhythm-informed mental health treatments.

Keywords: Mood disorder, Circadian rhythm, Light therapy, Chronotherapy, Clock genes

1. Introduction

As an endogenous biological rhythm system, circadian rhythms exhibit autonomic oscillations of nearly 24 hours (the actual measured mean is 24.2 hours) in mammals [1]. This complex system precisely coordinates key physiological functions such as the sleep-wake cycle, neuroendocrine activity (including the nighttime peak of melatonin secretion), and basic metabolic processes through a multilevel regulatory network. The central pacemakers located in the hypothalamic suprachiasmatic nucleus (SCN) play a central role in receiving ambient light signals via specific photosensitive retinal ganglion cells (ipRGCs), which in turn synchronize peripheral tissue clocks via neural and humoral pathways [2]. Studies have shown that light stimulation can significantly affect the activity pattern of SCN neurons and then affect further regulation of melatonin secretion dynamics in the pineal gland, diurnal fluctuations in core body temperature (normal amplitude about 0.5-1.0°C), and morning peak cortisol secretion [3]. These indicators have become important biomarkers for clinical assessment of circadian function.

Clinical observation and experimental studies have jointly confirmed that circadian rhythm disturbance is significantly associated with various mood disorders. In patients with major depressive disorder (MDD), it is characterized by a delayed sleep phase (meaning delay of 1.5-2 hours) and a

delayed melatonin secretion rhythm. Patients with bipolar disorder (BD) exhibit significantly reduced rhythm amplitude (especially flattening of body temperature rhythms) during manic episodes. The pathophysiological basis of this association may involve several levels: interaction of clock gene polymorphisms (e.g., PER3 rs57875989) with the monoaminergic neurotransmitter system, abnormal cortisol rhythm due to impaired negative feedback regulation of the HPA axis, and decreased SCN output function due to defective light signal transduction [1]. These findings not only help us understand how mood disorders develop but also support the idea of using treatments based on biological rhythms, like light therapy and melatonin receptor modulators.

Mood disorders are a class of mental diseases characterized by persistent affective disorders, among which major depressive disorder (MDD) and bipolar affective disorder (BD) have the most significant clinical impact. Studies have shown that MDD patients generally have a characteristic abnormal sleep rhythm, including prolonged sleep latency (an average increase of 40-60 minutes), early waking phenomenon (waking up 1.5-2 hours earlier than normal), and the phase delay of melatonin secretion peak (an average delay of 2-3 hours). In contrast, BD patients exhibit a more complex pattern of disrupted circadian rhythms, with significantly reduced sleep requirements common during manic episodes (< 4 hours/day and still energetic) and extreme instability of sleep rhythms during depressive episodes (up to 68% incidence of circadian inversion) [4].

Seasonal affective disorder (SAD) is a special subtype of MDD and BD, and its pathogenesis is closely related to photoperiodic changes. The reduction of daylight duration in winter can affect mood regulation in two ways: on the one hand, it inhibits the signaling of the retina-hypothalamus pathway, leading to a decrease in serotonin synthesis (about 25-30% lower in winter); on the other hand, the melatonin secretion phase of the pineal gland is prolonged (an average increase of 1.8-2.5 hours) [5]. Molecular genetic studies further found that the carrying rate of CLOCK gene polymorphisms was significantly increased in SAD patients, especially the PER2, CRY2, and clock alleles [6]. Together, this evidence supports the gene-environment interaction hypothesis that genetically predisposed individuals are more likely to develop mood symptoms associated with circadian dysregulation under certain photoperiodic conditions.

The interaction between the circadian rhythm system and mood disorders involves complex mechanisms at multiple levels. At the molecular level, core CLOCK genes (including the CLOCK, BMAL1, PER, and CRY families) influence emotional states by regulating the rhythmic expression of neurotransmitter systems. Specifically, rs1801260 polymorphisms in the CLOCK gene were significantly associated with manic episodes in bipolar disorder, while PER2 and CRY2 variants increased the risk of developing seasonal affective disorder by 32% and 28%, respectively [7]. These gene variants lead to a dissynchronization of the affective regulatory network by altering the diurnal firing patterns of dopaminergic neurons and serotonergic neurons.

The hypothalamic-pituitary-adrenal axis (HPA axis) is the core regulatory system of stress response, and its activity shows significant diurnal fluctuations. In healthy individuals, cortisol secretion shows a typical pattern of high morning and low night concentrations (about 15 µg/dl at 06:00, dropping to 3-5 µg/dl at night). However, patients with depression often show a flattening of the circadian rhythm of cortisol (reduced amplitude by 40-60%) and abnormally elevated evening levels (concentrations > 7 µg/dl at 22:00), and this abnormality is significantly associated with reduced hippocampal volume (mean reduction of 8-12%) and impaired cognitive function [8]. Patients with bipolar disorder show a more dynamic disturbance of cortisol rhythm, with increased cortisol arousal response (CAR) in manic periods (> 50% increase) and delayed rhythm phase (peak delay of 2-3 hours) in depressive periods.

Light, as the most important zeitgeber, regulates the synthesis of emotion-related neurotransmitters through the retina-hypothalamus pathway. Reduced sunlight in winter resulted in increased serotonin transporter (SERT) binding in the prefrontal cortex (about 18-22%) and a longer duration of

melatonin secretion in the pineal gland (2.5 hours more on average in winter than in summer) in patients with seasonal emotional disorder [5]. More notably, artificial light sources at night (especially blue light at 480nm) increased the risk of depression by 33-41% by suppressing melatonin secretion (50-60% after 1 hour of exposure) and delaying circadian clock phase (0.5-1.2 hours per night) [6]. These findings provide an important basis for the development of preventive interventions based on light environment.

The bidirectional mechanism of circadian rhythm and mood disorders provides an important opportunity to develop novel treatment strategies. Available clinical evidence suggests that timing therapy has unique advantages in improving symptoms and preventing recurrence: Bright light therapy (BLT, usually with 10,000 lux white light for 30-60 minutes in the morning) can lead to a 65-72% response rate for seasonal emotional disorder (response rate is 2.3 times higher than placebo); the melatonin receptor agonist agomelatine (25-50 mg/day) not only shortened the sleep latency of MDD patients by 40-50 minutes but also increased the proportion of patients with $\geq 50\%$ reduction in HAM-D-17 scores [9]. In particular, social rhythm therapy (SRT), designed for bipolar disorder, reduces the risk of mood relapse within 12 months by stabilizing daily activity patterns. The common feature of these interventions is to reconstruct the circadian rhythm of neurotransmitter secretion by correcting the out-of-sync of the internal biological clock with the external environment (with an average phase adjustment of 1.5-2.8 hours) [5]. This review aims to consolidate current understanding of the molecular, hormonal, and environmental mechanisms linking circadian rhythms with mood disorders and to explore how rhythm-based interventions may inform future personalized treatment strategies.

2. Circadian dysregulation in mood disorders

2.1. Sleep disturbances and mood disorders

The dysregulation of the circadian rhythm system affects sleep homeostasis and emotional regulation through multilevel mechanisms (Figure 1). A number of controlled studies have confirmed that patients with bipolar disorder exhibit characteristic rapid eye movement sleep (REM) abnormalities, including significantly shortened latency [7] and reduced proportion of slow-wave sleep, while patients with major depression generally exhibit sleep continuity disorders. It is characterized by prolonged sleep latency and difficulty in sleep maintenance [4]. This disruption of sleep structure may exacerbate the pathological process of emotional regulation disorders by affecting the functional connectivity of the prefrontal limbic system. As an important regulator of the endogenous timing system, the secretion of melatonin shows a specific pattern in different mood disorders. Patients with bipolar disorder during a manic episode showed increased amplitude of nocturnal melatonin secretion [5], while patients with a depressive phase showed delayed secretion. Winter-specific hypersecretion of melatonin in patients with seasonal affective disorder [10] further confirmed the important role of photoperiodic dysregulation in the pathogenesis of mood disorders. These findings provide a theoretical basis for the development of time-specific intervention strategies. Moreover, recent genetic studies have revealed the mechanism of association between circadian clock gene variation and mood disorders. CLOCK gene polymorphism was significantly associated with sleep-wake cycle disturbance in bipolar disorder [11], while postmortem brain tissue analysis of depressed patients showed that PER2 gene expression rhythm was significantly weakened. Epigenetic studies further found that there is a dose-effect relationship between hypermethylation in the promoter region of the BMAL1 gene and increased risk of depression [8], suggesting that environmental factors may exacerbate functional abnormalities of clock genes through epigenetic modification. Existing evidence supports the idea of the circadian system as a new target for the treatment of mood disorders. The development of an individualized chronobiological intervention scheme based on the

chronobiological characteristics of different clinical subtypes may provide a new way to improve the existing therapeutic effect. Future studies need to further elucidate the interaction mechanism between the circadian clock gene regulatory network and emotion-related neural circuits to lay a foundation for the development of precise diagnosis and treatment strategies.

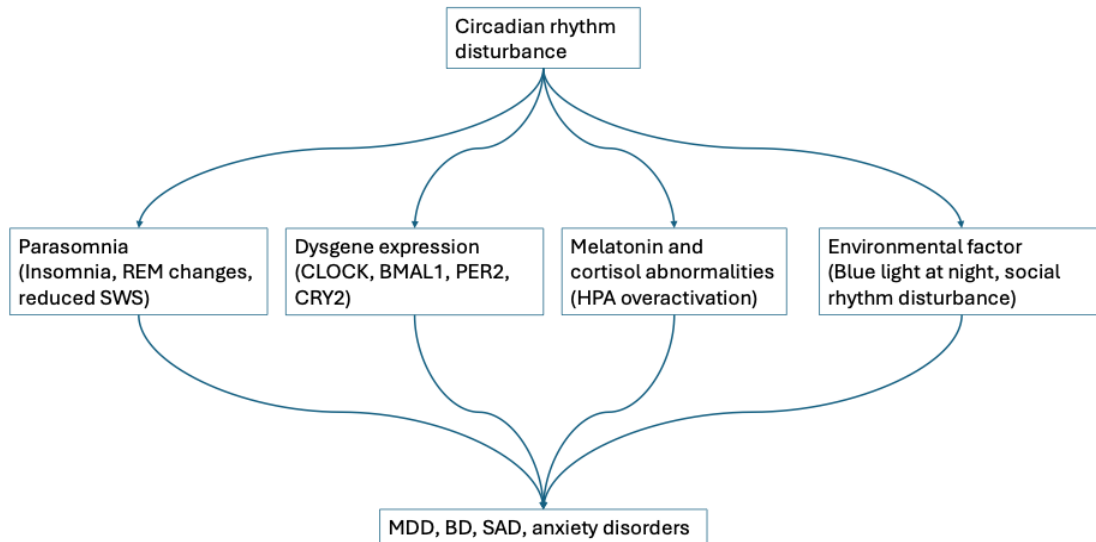


Figure 1: Mechanisms linking circadian dysregulation to mood disorders

2.2. Other factors and mood disorders

Circadian misalignment has been shown to profoundly affect the regulation of the hypothalamic-pituitary-adrenal (HPA) axis, a system critical to modulating both stress responses and affective stability. Among individuals with mood disorders—especially those diagnosed with MDD or BD—cortisol rhythms frequently deviate from the norm. A typical feature is an exaggerated cortisol awakening response (CAR), in which morning cortisol levels surge more than usual. This phenomenon has been linked to heightened vulnerability to stress in anxiety-related conditions and is frequently observed in depressive states as well [8, 12]. Moreover, the rhythmicity of gene expression in key brain regions is significantly reduced in patients with depression, especially the oscillations between the prefrontal cortex and the amygdala, which are out of sync. This time-phase separation across brain regions may lead to impaired integration of emotion-related neural circuits, thus explaining the dysregulation of emotions common in patients with circadian rhythm disorders. Furthermore, neurotransmitters involved in mood regulation—namely serotonin, dopamine, and norepinephrine—also follow circadian fluctuations that appear disrupted in psychiatric populations [4]. Reduced serotonin synthesis due to circadian phase shifts has been documented in depressed individuals, while bipolar mania is often accompanied by altered dopaminergic activity [11]. These neurochemical changes reinforce the role of temporal hormonal dynamics in shaping emotional outcomes. In addition, light acts as the dominant zeitgeber for aligning internal circadian rhythms with the external environment. Seasonal fluctuations in light availability—especially during the darker months of winter—have been strongly associated with the emergence of seasonal affective disorder (SAD), a condition marked by oversleeping, fatigue, and loss of interest in daily activities. Mechanistically, this is linked to diminished serotonergic activity and excessive melatonin secretion, both of which interfere with emotional regulation [3]. At the other end of the spectrum, exposure to

artificial light at night (ALAN)—particularly short-wavelength blue light from screens and LEDs—can interfere with melatonin synthesis and shift circadian phase timing. Studies have linked ALAN to later sleep onset, reduced sleep quality, and heightened vulnerability to depression and anxiety [10]. These findings underscore the ways in which contemporary lighting environments can act as chronodisruptors, altering internal biological timing and increasing affective risk. Interventionally, bright light therapy (BLT) has emerged as one of the most effective non-pharmacological treatments for SAD. It functions by phase-advancing circadian rhythms and reducing depressive symptoms, particularly in those with strong seasonal patterns [11]. The therapeutic efficacy of BLT lies in its ability to re-establish synchronized rhythmicity, counteracting the biological disruptions caused by insufficient daylight or excessive nighttime illumination. Taken together, the effects of environmental light—both natural and artificial—highlight its bidirectional role in mood regulation. Circadian misalignment induced by light dysregulation interacts with sleep patterns, endocrine signaling, and genetic vulnerabilities, forming a complex pathway that underlies the onset and maintenance of various mood disorders, including MDD, BD, anxiety, and SAD.

3. Mood disorders and circadian rhythm across different conditions

3.1. Major depressive disorder (MDD)

Disruptions in circadian rhythms are widely observed in individuals diagnosed with major depressive disorder (MDD), affecting core physiological domains such as sleep timing, hormone regulation, and thermoregulatory processes. One consistent finding is the attenuation of circadian amplitude—patients frequently exhibit delayed sleep onset, irregular waking patterns, and diminished rhythmic fluctuations in endocrine activity [13]. Melatonin secretion, in particular, often becomes phase-delayed or flattened, contributing to poor sleep continuity and persistent fatigue during daytime hours [5]. In addition to melatonin, cortisol rhythms are frequently disturbed. Many individuals with MDD demonstrate an exaggerated cortisol awakening response (CAR), which may reflect an overactive stress axis and impaired feedback regulation [8]. These disruptions suggest that misaligned circadian outputs are not merely byproducts of depression but may participate in symptom maintenance. Accordingly, phase-resetting interventions—including bright light exposure and controlled sleep deprivation—have shown promising effects in alleviating depressive symptoms by targeting circadian regulation directly [11].

3.2. Bipolar disorder (BD)

Bipolar disorder is strongly associated with internal rhythm instability, manifesting in erratic sleep-wake timing and recurrent mood fluctuations. A growing body of evidence highlights circadian phase variability as a distinguishing feature of BD, particularly in those with rapid cycling forms [4]. Genetic studies have revealed associations between manic episodes and polymorphisms in core circadian genes, notably within the CLOCK family [13], pointing to a heritable basis for rhythm vulnerability in bipolarity. Sleep disruptions further characterize BD, with marked reductions in slow-wave sleep and shorter REM sleep latency observed across both poles of the illness [12]. Compounding these physiological irregularities is the diminished regularity of social rhythms, such as activity timing and interpersonal routines, which has been linked to symptom exacerbation [14]. Interventions like social rhythm therapy (SRT) aim to restore temporal regularity in daily structure and have demonstrated efficacy in improving mood stability among BD patients [15].

3.3. Anxiety disorders

In anxiety-related disorders, particularly generalized anxiety disorder (GAD), disruptions in circadian physiology have been noted across both hormonal and autonomic systems. One hallmark feature is the dysregulation of HPA axis signaling—patients frequently exhibit a hyperactive CAR, indicating a heightened anticipatory stress response [8]. This is often accompanied by increased sympathetic tone and reduced parasympathetic activity during nocturnal periods, leading to delayed sleep onset and greater night-time arousal [4]. Circadian misalignment in anxiety does not appear limited to endocrine rhythms. Disruptions in the autonomic nervous system's circadian modulation may underlie persistent hyperarousal and fragmented sleep architecture [14]. Moreover, irregularities in sleep-wake timing, often stemming from inconsistent routines, have been linked to worsening anxiety symptoms over time. Interventions that target circadian alignment—such as timed melatonin administration or cognitive-behavioral strategies like CBT-I—are increasingly recognized as valuable adjuncts in anxiety treatment [10].

3.4. Seasonal affective disorder (SAD)

Seasonal affective disorder (SAD) represents one of the clearest clinical manifestations of circadian rhythm disruption linked to environmental light variability. The condition tends to emerge during periods of reduced photoperiod, particularly in winter months, when decreased daylight triggers a delay in circadian phase and prolongs melatonin secretion during the early morning hours [5]. This temporal misalignment is thought to underlie key depressive symptoms, including hypersomnia, diminished energy, and loss of pleasure [11]. Accumulating evidence supports the role of circadian dysregulation in winter-pattern depression. Patients with SAD frequently exhibit delayed sleep-wake cycles, as well as reduced serotonin turnover, which together may compromise mood stability. Among available treatments, bright light therapy (BLT) remains the most widely used and evidence-based approach. By delivering high-intensity light during the early morning, BLT advances circadian timing, enhances serotonergic transmission, and regulates nocturnal melatonin suppression, thereby alleviating mood symptoms and improving circadian coherence [15]. Circadian rhythm dysfunction manifests uniquely across different mood disorders, influencing sleep, hormonal regulation, and neurobiological processes. MDD is associated with blunted circadian patterns and cortisol dysregulation, BD with phase instability and rapid mood cycling, anxiety disorders with heightened HPA axis reactivity, and SAD with seasonal shifts in melatonin and serotonin balance. Understanding these distinct circadian patterns provides a foundation for developing targeted chronotherapeutic interventions, such as light therapy, melatonin regulation, and social rhythm therapy, to restore circadian stability and enhance treatment outcomes for individuals with mood disorders.

4. Therapeutic strategies targeting circadian rhythms in mood disorders

4.1. Chronotherapy and light-based interventions

Chronotherapeutic interventions are designed to realign disrupted biological rhythms by leveraging environmental time cues—particularly light exposure. Among these, bright light therapy (BLT) remains the most extensively validated, especially in the treatment of seasonal affective disorder (SAD). A meta-analysis demonstrated that BLT led to a moderate but clinically meaningful reduction in depressive symptoms, with a standardized mean difference of -0.37 and a response risk ratio of 1.42 relative to placebo [9]. At the physiological level, BLT is believed to exert its effects via melanopsin-containing retinal ganglion cells, which transmit photic input to the suprachiasmatic nucleus (SCN). This pathway facilitates circadian phase advancement while downregulating excessive melatonin synthesis, thereby mitigating mood-related circadian misalignment [5].

Additional chronotherapy modalities include wake therapy (as therapeutic sleep deprivation) and phase-advance strategies. Although these techniques can produce rapid antidepressant responses, their effects are often short-lived when applied in isolation. Emerging evidence suggests that combining BLT with sleep schedule stabilization and behavioral sleep hygiene protocols may yield more durable improvements in mood and circadian alignment.

4.2. Pharmacological modulation of circadian function

A range of pharmacological compounds have been investigated for their potential to influence circadian timing systems or modulate downstream physiological processes. Among the most direct are melatonin and its synthetic analogs, such as agomelatine, which act on MT1 and MT2 receptors to promote sleep onset and adjust circadian phase. Beyond their chronobiotic properties, these agents also exhibit antioxidant and anti-inflammatory effects in both animal and human models, reinforcing their neuroprotective potential in the context of mood regulation [16]. In addition to melatonin-based interventions, other compounds exert more indirect circadian effects. Lithium, for instance, has been shown to enhance CLOCK/BMAL1 transcriptional stability, thereby supporting circadian rhythm regularity at the molecular level. Some antidepressants also appear to influence circadian pathways through their effects on serotonergic and dopaminergic signaling. However, the specificity and consistency of these chronomodulatory effects remain areas of ongoing research.

4.3. Behavioral and lifestyle-based interventions

Non-pharmacological strategies aimed at reinforcing circadian stability include behavioral interventions such as Cognitive Behavioral Therapy for Insomnia (CBT-I), structured physical activity, and the strategic timing of food intake. Recent work highlights the influence of meal timing on metabolic rhythms, suggesting that aligning eating behaviors with endogenous circadian signals may support improved mood regulation and energy balance [17]. Moreover, maintaining consistent daily routines—particularly in sleep-wake cycles and exposure to natural light—serves to strengthen the synchronizing effect of environmental cues (zeitgebers) on the circadian system. These lifestyle-based approaches can augment pharmacological and light-based treatments by enhancing circadian entrainment and reducing rhythm fragmentation.

4.4. Social rhythm and psychosocial interventions

Social rhythm interventions are designed to promote stability in daily behavioral routines, such as consistent sleep-wake schedules, regular social engagement, and predictable mealtimes, with the goal of supporting internal circadian coherence. In bipolar disorder, mood fluctuations frequently follow periods of routine disruption, lending empirical support to the idea that temporal structure can serve as a protective factor against relapse. Therapeutic frameworks that combine rhythm stabilization with psychoeducation, interpersonal skill development, and behavioral monitoring have demonstrated potential for improving treatment adherence and long-term functional outcomes. These psychosocial strategies aim not only to regulate biological timing but also to reinforce environmental regularity, thereby complementing pharmacological and light-based therapies in a multidimensional treatment model.

5. Conclusion

Circadian rhythm disruption plays a fundamental role in the pathophysiology of mood disorders, including major depressive disorder (MDD), bipolar disorder (BD), seasonal affective disorder (SAD), and anxiety disorders. This review synthesizes current evidence showing that disturbances in sleep

architecture, hormonal rhythms (particularly cortisol and melatonin), clock gene expression (e.g., CLOCK, PER2, BMAL1), and light-dependent neuroendocrine signaling are intimately linked with emotional dysregulation. The bidirectional relationship between circadian misalignment and psychiatric symptoms reinforces the centrality of chronobiological processes in both the etiology and treatment of mood disorders. Mechanistic studies increasingly point to the interplay of genetic vulnerability and environmental modulation. Alterations in circadian genes, such as polymorphisms in CLOCK or epigenetic downregulation of PER3, appear to sensitize individuals to mood fluctuations, especially in BD and MDD populations. At the systems level, HPA axis hyperactivity and maladaptive cortisol rhythms exacerbate stress sensitivity, a hallmark of depressive and anxiety presentations. In the case of SAD, decreased natural light exposure and elevated artificial illumination (ALAN) contribute to disrupted melatonin–serotonin balance, further misaligning internal timing systems. In terms of intervention, circadian-focused treatments have gained empirical traction. Modalities such as bright light therapy (BLT), melatonin-based pharmacotherapies (e.g., agomelatine), and social rhythm therapy (SRT) have demonstrated potential in re-synchronizing biological rhythms and alleviating mood symptoms. Combinatory approaches, integrating BLT with cognitive behavioral therapy for insomnia (CBT-I), may offer enhanced durability by addressing both physiological and behavioral misalignments. Bringing circadian science into psychiatric practice represents more than a mechanistic insight; it provides a framework for rethinking the temporal dimension of mental illness. Bridging molecular chronobiology with applied therapeutic innovation offers not only more refined interventions but also the possibility of sustainable, rhythm-informed mental healthcare.

References

- [1] W. H. Walker, J. C. Walton, A. C. DeVries, and R. J. Nelson, 'Circadian rhythm disruption and mental health', *Transl. Psychiatry*, vol. 10, no. 1, p. 28, Jan. 2020, doi: 10.1038/s41398-020-0694-0.
- [2] H. K. Dollish, M. Tsyglakova, and C. A. McClung, 'Circadian rhythms and mood disorders: Time to see the light', *Neuron*, vol. 112, no. 1, pp. 25–40, Jan. 2024, doi: 10.1016/j.neuron.2023.09.023.
- [3] A. K. Gold and G. Kinrys, 'Treating Circadian Rhythm Disruption in Bipolar Disorder', *Curr. Psychiatry Rep.*, vol. 21, no. 3, p. 14, Mar. 2019, doi: 10.1007/s11920-019-1001-8.
- [4] W. Bechtel, 'Circadian Rhythms and Mood Disorders: Are the Phenomena and Mechanisms Causally Related?', *Front. Psychiatry*, vol. 6, Aug. 2015, doi: 10.3389/fpsy.2015.00118.
- [5] D. Gubin et al., 'Melatonin mitigates disrupted circadian rhythms, lowers intraocular pressure, and improves retinal ganglion cells function in glaucoma', *J. Pineal Res.*, vol. 70, no. 4, p. e12730, May 2021, doi: 10.1111/jpi.12730.
- [6] V. Ansu Baidoo and K. L. Knutson, 'Associations between circadian disruption and cardiometabolic disease risk: A review', *Obesity*, vol. 31, no. 3, pp. 615–624, Mar. 2023, doi: 10.1002/oby.23666.
- [7] M. J. McCarthy et al., 'Neurobiological and behavioral mechanisms of circadian rhythm disruption in bipolar disorder: A critical multi-disciplinary literature review and agenda for future research from the ISBD task force on chronobiology', *Bipolar Disord.*, vol. 24, no. 3, pp. 232–263, May 2022, doi: 10.1111/bdi.13165.
- [8] N. C. Nicolaides, E. Charmandari, G. P. Chrousos, and T. Kino, 'Circadian endocrine rhythms: the hypothalamic–pituitary–adrenal axis and its actions', *Ann. N. Y. Acad. Sci.*, vol. 1318, no. 1, pp. 71–80, May 2014, doi: 10.1111/nyas.12464.
- [9] E. Pjrek et al., 'The Efficacy of Light Therapy in the Treatment of Seasonal Affective Disorder: A Meta-Analysis of Randomized Controlled Trials', *Psychother. Psychosom.*, vol. 89, no. 1, pp. 17–24, 2020, doi: 10.1159/000502891.
- [10] T. L. Sletten et al., 'The role of circadian phase in sleep and performance during Antarctic winter expeditions', *J. Pineal Res.*, vol. 73, no. 2, p. e12817, Sep. 2022, doi: 10.1111/jpi.12817.
- [11] L. Basquin et al., 'Study of the different sleep disturbances during the prodromal phase of depression and mania in bipolar disorders', *Bipolar Disord.*, vol. 26, no. 5, pp. 454–467, Aug. 2024, doi: 10.1111/bdi.13429.
- [12] L. Gonzalez-Blanco and E. Romero, 'A multi-scale insight into gas transport in a deep Cenozoic clay', *Géotechnique*, vol. 74, no. 4, pp. 337–354, Apr. 2024, doi: 10.1680/jgeot.21.00208.
- [13] J. Scott et al., 'A systematic review and meta-analysis of sleep and circadian rhythms disturbances in individuals at high-risk of developing or with early onset of bipolar disorders', *Neurosci. Biobehav. Rev.*, vol. 135, p. 104585, Apr. 2022, doi: 10.1016/j.neubiorev.2022.104585.

- [14] T. H. Ng, K.-F. Chung, F. Y.-Y. Ho, W.-F. Yeung, K.-P. Yung, and T.-H. Lam, 'Sleep-wake disturbance in interepisode bipolar disorder and high-risk individuals: A systematic review and meta-analysis', *Sleep Med. Rev.*, vol. 20, pp. 46–58, Apr. 2015, doi: 10.1016/j.smrv.2014.06.006.
- [15] L. M. Lyall et al., 'Association of disrupted circadian rhythmicity with mood disorders, subjective wellbeing, and cognitive function: a cross-sectional study of 91 105 participants from the UK Biobank', *Lancet Psychiatry*, vol. 5, no. 6, pp. 507–514, Jun. 2018, doi: 10.1016/s2215-0366(18)30139-1.
- [16] A. K. Verma, S. Singh, and S. I. Rizvi, 'Age-dependent effect of continuous "artificial light at night" on circadian rhythm in male rats: neuroprotective role of melatonin', *Biogerontology*, vol. 22, no. 5, pp. 531–545, Oct. 2021, doi: 10.1007/s10522-021-09933-y.
- [17] R. Chamorro, C. Jouffe, H. Oster, N. H. Uhlenhaut, and S. M. Meyhöfer, 'When should I eat: A circadian view on food intake and metabolic regulation', *Acta Physiol.*, vol. 237, no. 3, p. e13936, Mar. 2023, doi: 10.1111/apha.13936.