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DERLEME **REVIEW**

Social Rhythm in Bipolar Disorder and Interventions to Regulate Social Rhythm

Yeşim Ayar[1*], Tuğba Şahin Tokatlıoğlu[2]

[1]Istanbul University-Cerrahpasa, Institute of Graduate Studies, Psychiatric and Mental Health Nursing Doctoral Program, Istanbul, Türkiye
[2]Istanbul Aydın University, Faculty of Health Sciences, Department of Nursing, Istanbul, Türkiye

Abstract

This review aims to compile interventions targeting social rhythms to reduce symptoms, regulate sleep, and improve functioning in bipolar disorder. Factors influencing relapse in bipolar disorder include stressful life events, nonadherence to pharmacological treatment, and disruptions in social rhythms. Although the findings are inconsistent, bright light therapy, as a chronotherapeutic intervention, appears to be an effective and safe adjunctive treatment, particularly during depressive episodes of bipolar disorder. On the other hand, interventions implemented during manic episodes, such as dark therapy and the use of blue light-blocking glasses, have been found to reduce manic symptoms, improve sleep quality, and decrease the need for pharmacological treatment. In terms of psychotherapeutic interventions, CBT-I has been associated with higher depression remission rates, reduced use of anxiolytics, decreased insomnia severity, and fewer maladaptive sleep-related cognitions. Furthermore, CBT-I adapted for bipolar disorder (CBT-I-BD) has been shown to reduce insomnia severity, the frequency of manic/hypomanic episodes, and the use of psychotropic medication for sleep among individuals diagnosed with BD. Interpersonal and Social Rhythm Therapy (IPSRT), whether delivered individually or in group formats, has been found to reduce both depressive and manic symptoms, improve social rhythm regularity, and enhance overall functioning. In conclusion, approaches targeting circadian rhythms show promise, particularly in treatment-resistant cases of bipolar disorder, due to their rapid effects and neurobiological basis. However, further scientific data are needed, especially in areas such as standardized intervention protocols, larger sample sizes, and long-term follow-up of clinical outcomes.

Keywords: *Bipolar Disorder, Social Rhythm, Circadian Rhythm*

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Bipolar Bozuklukta Sosyal Ritim ve Sosyal Ritmi Düzenlemeye Yönelik Müdahaleler

Özet

Bu derlemede, bipolar bozuklukta semptomları azaltmak, uykuyu düzenlemek ve işlevselliği artırmak için sosyal ritimleri ele alan müdahaleleri derlemek amaçlanmıştır. Bipolar bozuklukta nüksü etkileyen faktörler; stresli yaşam olayları, ilaç tedavisine uyumsuzluk ve sosyal ritimlerdeki bozulmalardır. Kronoterapötik müdahalalerden olan parlak ışık terapisi ile ilgili tutarsız sonuçlar olsa da bipolar bozukluğun özellikle depresif dönemlerinde etkili ve güvenli bir tamamlayıcı müdahale yöntemi olabileceği görülmektedir. Öte yandan, manik dönemlerde uygulanan karanlık terapisi ve mavi ışık filtreleyici gözlük kullanımı gibi müdahalelerin, manik semptomlarda azalma sağladığı, uyku kalitesini artırdığı ve farmakolojik tedavi gereksinimini azalttığı bulunmuştur. Psikoterapötik müdahaleler incelendiğinde ise, BDT-I uygulandıktan sonra, depresyon remisyon oranının daha yüksek olduğu, anksiyolitik kullanımında, uykusuzluk şiddetinde ve uykuya ilişkin olumsuz düşüncelerde azalma olduğu belirlenmiştir. BDTI-BD ise bipolar bozukluk tanısı olan bireylerde uykusuzluk şiddeti, manik/hipomanik atakların sıklığını ve uyku için psikotrop ilaç kullanımını azaltmıştır. Kişilerarası ilişkiler ve Sosyal Ritim Terapisinin de (KİSRT) hem bireysel hem de grup formatında uygulanarak, depresif ve manik semptomlarda azalma, sosyal ritim düzenliliğinde iyileşme ve işlevsellikte artış sağlayabildiği görülmektedir. Sonuç olarak, sirkadiyen ritmi hedef alan yaklaşımlar, özellikle farmakolojik tedavilere direnç gösteren bipolar bozukluk olgularında, hızlı etki göstermesi ve nörobiyolojik temellere dayanması açısından umut vaat etmektedir. Ancak, daha geniş örneklemli, müdahale protokollerinin standardizasyonu ve klinik etkilerin uzun dönem takibi gibi alanlarda daha fazla bilimsel veri gereklidir.

Anahtar Kelimeler*: Bipolar Bozukluk, Sosyal Ritim, Sirkadyen Ritim*

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***Sorumlu Yazar / Corresponding Author:** E-mail (YA): yesimayr92@gmail.com. ORCID: 0000-0002-7248-5946
E-mail (TŞT): tsahintokatlioglu@aydin.edu.tr. ORCID: 0000-0003-2569-9906

INTRODUCTION

Bipolar disorder is a major mood disorder characterized by fluctuating manic and depressive symptoms over time. The emotional burden of the disease, along with cognitive, psychosocial, and occupational dysfunctions, can lead to increased suicide rates, comorbid medical conditions, and early mortality. Although genetics play an important role in the etiology of the disease, environmental factors are also known to significantly influence its course. The onset typically occurs between the ages of 15 and 25, with approximately 75% of the symptomatic course characterized by depressive episodes or symptomatology (1,2).

Factors influencing the recurrence of bipolar disorder include stressful life events, medication noncompliance, and disruptions in social rhythms (3,4). Psychosocial stress has been shown to have profound effects on the onset and course of the disease, with severe life events being associated with critical periods and the onset of bipolar disorder. While the role of psychosocial stress factors and medication noncompliance in triggering relapse is well established, the role of social rhythms remains less understood (5). Disruptions in social rhythms can trigger a relapse, which is further exacerbated by disturbances in biological rhythms, particularly sleep. Circadian and sleep/wake cycle processes can be controlled through chronotherapeutic and psychotherapeutic approaches. Psychological treatments and chronotherapeutic interventions applied to individuals diagnosed with bipolar disorder are known to reduce the risk of recurrence (6,7). This article aims to discuss interventions targeting social rhythms in individuals diagnosed with bipolar disorder to address mood regulation and highlight the importance of these interventions in the treatment and care process.

Social Rhythm Theory in Bipolar Disorder

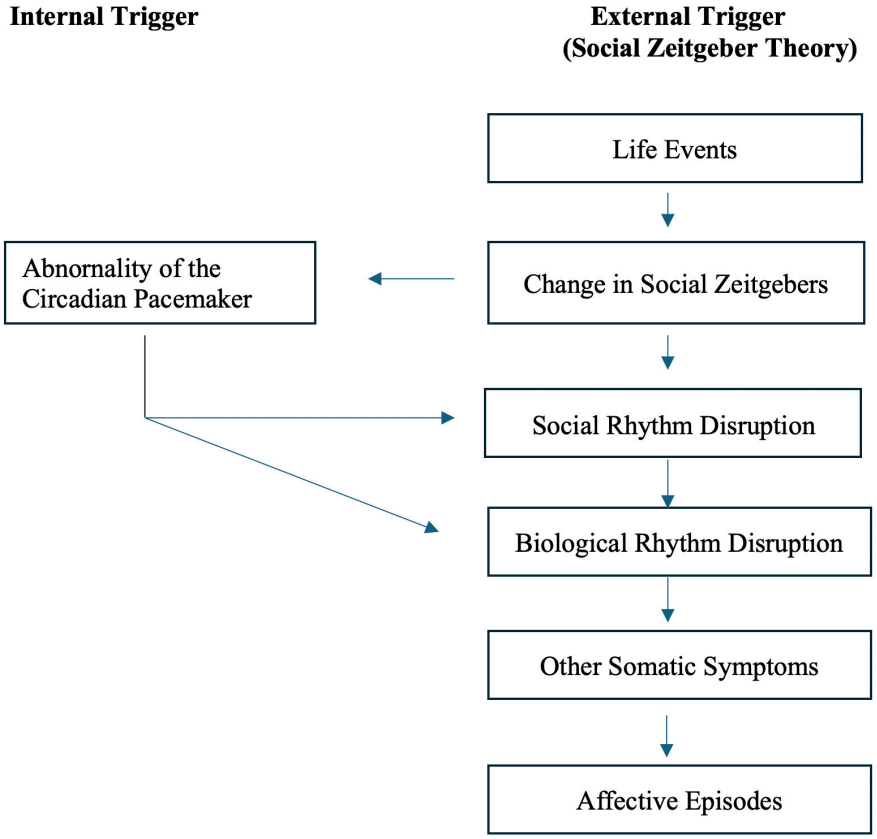
Ehlers (1988) defines social rhythms as social relationships, social demands, or tasks that influence the regulation of biological rhythms. The Social Zeitgeber Theory suggests that stressful life events, by disrupting the timing of "social zeitgebers" such as sleep time, wake time, and mealtimes, may lead to circadian rhythm disturbances, which can trigger mood episodes in individuals predisposed to the disorder. The term “zeitgeber,” derived from German, means "time giver," where "zeit" means time, and "geben" means to give. "Social timekeepers," or zeitgebers, are social conditions that influence circadian rhythms, such as sleep-wake cycles (8). Some external stimuli (zeitgebers) have the capacity to synchronize biological clocks and adjust bodily rhythms. The most potent physical zeitgeber is light, while social relationships and the environment also play a significant role in the regulation of circadian rhythms (9,7). Lifestyle activities, including sleep-wake hours, mealtimes, and social interactions, affect social rhythms. According to the social zeitgeber theory, changes in mood occur because of societal events and disrupt social rhythms. Disruptions in social rhythms, in turn, can cause significant disturbances in circadian rhythms. Social rhythms are involved in neurohormonal processes such as the regulation of cortisol and melatonin secretion, which are responsible for circadian rhythms (1). All biological processes work cyclically, but they require certain triggers to maintain their rhythms. These are synchronized with a 24-hour cycle driven by the light/dark cycle, mediated through the suprachiasmatic nucleus located in the hypothalamus. Individuals diagnosed with bipolar disorder are more sensitive to the cognitive and somatic effects of disrupted social rhythms (10,11).

It has been stated that disruptions in social rhythms following stressful life events can lead to disturbances in biological rhythms, as well as disrupt sleep, potentially contributing to depressive disorders. Furthermore, the Social Zeitgeber Theory has been shown to be effective in explaining manic and hypomanic episodes in individuals with bipolar disorder (12). Modern living conditions, including the 24-hour continuous fast-paced urban life, flexible working hours due to intense work schedules, shift work, and delayed meal and sleep times caused by the busyness of the day, are known to disrupt the rhythm regulated by the rising and setting of the sun (8).

The circadian clock maintains 24-hour rhythms

even in the absence of external stimuli. The circadian pacemaker is in the suprachiasmatic nucleus of the brain. It regulates the activity of peripheral clocks through the neuroendocrine and autonomic nervous systems. Circadian rhythm disturbances can occur due to external stimuli or because of dysfunction in the hypothalamus (suprachiasmatic nucleus). Figure 1 shows the impact of internal and external factors on affective episodes (12).

Figure 1. A model of the social zeitgeber theory



It is taken from the article titled "The Social Zeitgeber Theory, Circadian Rhythms, and Mood Disorders: Review and Evaluation.

Intervention for Regulating Social Rhythm in Bipolar Disorder

Bright light and dark therapy

Bright light therapy regulates circadian rhythm timing by suppressing melatonin production. Seasonal affective disorder is defined as a recurring major depression that begins every fall or winter and spontaneously improves in the spring. It has been observed that bright light therapy produces a strong antidepressant response within 3-4 days, and this response quickly dissipates after withdrawal of bright light or switching to a non-bright light device (13,7). Bright light therapy is known to be effective in the treatment of both seasonal and non-seasonal major depressive disorder, either as a monotherapy or as an adjunct to antidepressants (14). The daily exposure duration typically ranges from 15-30 minutes in the morning and midday. Light devices should generally be applied at eye level, at 12-13 inches (30 to 33 cm), with an intensity of 7,000 to 10,000 lux (a measure of light intensity dependent on the distance from the light source). Hypomanic symptoms that arise during the treatment period can be prevented by reducing the daily exposure duration (15,7).

Bright light therapy has been employed as a complementary intervention during the depressive episodes of bipolar disorder. In a randomized controlled trial conducted by Zhou et al. (2018), bright light therapy was administered to individuals diagnosed with bipolar disorder for one hour each morning over a two-week period. The findings indicated that this intervention was an effective and safe adjunctive treatment during depressive episodes (16). Similarly, in a randomized controlled study by Sit et al. (2018), individuals with bipolar I and II disorder experiencing depressive episodes received white bright light therapy (7000 lux). The results demonstrated significantly lower depression scores and higher remission rates at weeks 4 to 6, supporting the efficacy of bright light therapy in bipolar depression (17). Kupeli et al. (2018) also assessed the effectiveness of bright light therapy in individuals with bipolar disorder during depressive episodes. In their study, participants received bright light therapy (10000 lux) for 30 minutes each morning over a two-week period, resulting in a significant reduction in depression scores. Headaches were reported as a side effect (18). These findings suggest that bright light therapy may exert beneficial effects at varying intensities and durations. In a study conducted by Benedetti et al. (2007), an intervention protocol combining sleep deprivation and low-intensity light therapy was applied to individuals in the depressive phase of bipolar disorder. The intervention consisted of three 36-hour periods of continuous wakefulness followed by three nights of normal sleep. Additionally, participants were exposed to light at an intensity of 400 lux for 30 minutes. Approximately two-thirds of the intervention group exhibited clinically significant reductions in depression levels from day one to day seven (19). On the other hand, the literature also reveals that bright light therapy does not always yield positive outcomes. In a study involving hospitalized adolescents diagnosed with major depressive disorder, bright light therapy (10000 lux) was administered in addition to routine treatment over a four-week period. No significant superiority was found for bright light therapy compared to placebo red light therapy (20). Similarly, in a study by Chojnacka et al. (2016) involving individuals diagnosed with bipolar disorder in the depressive phase, bright light therapy was applied for two weeks. While improvements were observed, no statistically significant differences emerged between the intervention and control groups (21).

In the literature, it has been reported that the application of darkness therapy to patients in rapid cycling or manic episodes leads to a reduction in manic symptoms, a decreased need for antipsychotic medication, and shorter hospitalization durations. Additionally, the use of glasses that filter blue wavelengths can create an "artificial darkness," which facilitates melatonin secretion and promotes the onset of sleep (22,6). Laboratory studies have provided significant insights into the neural pathways responsible for light input. Intrinsically photosensitive retinal ganglion cells (ipRGCs) naturally contain light-sensitive pigments. These ipRGCs can interact with melatonin and influence mood in bipolar disorder. Research has shown that ipRGCs are particularly sensitive to blue light (8,23,24). In individuals diagnosed with bipolar disorder during manic episodes, the use of blue light-blocking glasses over a seven-day period has been associated with a reduction in manic symptoms. These individuals also demonstrated higher sleep efficiency and quality, as well as a decreased need for pharmacological intervention (25,26). The researchers suggest that blue light-blocking glasses may serve as an effective and beneficial intervention to support sleep regulation in hospitalized patients experiencing manic episodes (26). Similarly, in a study conducted by Barbini et al. (2005), darkness therapy was administered as an adjunct to routine treatment for individuals in a manic episode. Participants receiving darkness therapy showed a more rapid reduction in manic symptoms compared to those receiving medication alone, and they were discharged earlier. These findings indicate that darkness therapy may hold potential as an adjunctive intervention to alleviate manic symptoms during manic episodes (27). In a systematic review, blue light-blocking glasses were reported to be an effective intervention for facilitating sleep onset in individuals with sleep disorders, jet lag, and shift work schedules (24).

A review by Swanson et al. (2025) on chronotherapeutic interventions (e.g., bright light and dark therapy) in psychiatric disorders indicated that while these treatments show promise, current research is constrained by small sample sizes and a limited focus on depression. The authors underscored the necessity of conducting larger studies to improve the generalizability of findings and to refine treatment protocols for the development of individualized treatment approaches (28).

Sleep deprivation

Sleep deprivation can be a rapid, effective, and short-term option for individuals with depressive symptoms. After sleep deprivation, a rapid improvement in mood can be observed, which highlights the importance of the mechanisms within the circadian rhythm that regulate sleep in the onset of depression. The antidepressant effect of sleep deprivation on depressive symptoms is closely related to serotonergic, noradrenergic, and dopaminergic mechanisms within the circadian system. Mood improvement achieved through sleep deprivation may also result in a reduction in depression. The reduction of REM sleep and the application of sleep deprivation have antidepressant properties (29, 30).

Sleep deprivation therapy is implemented in various forms as an adjunctive intervention aimed at the rapid reduction of depressive symptoms. The types of application are classified as total sleep deprivation, partial sleep deprivation, sleep phase advancement, and sleep phase delay. In total sleep deprivation, individuals are required to remain awake for approximately 36 hours, typically from the morning of one day until the evening of the following day. In partial sleep deprivation, sleep duration is restricted to either the first or second half of the night, generally limited to 4–5 hours. Sleep phase advancement is an intervention following total sleep deprivation, in which sleep times are systematically shifted earlier by one hour each day, with the goal of achieving a normal nocturnal sleep schedule (23:00–06:00). This method is frequently used as a relapse prevention strategy. In sleep phase delay, time spent in bed initially occurs between 02:00 and 07:00 and is then advanced by 30 minutes daily until the target sleep phase is reached. Although sleep deprivation therapy has been shown to produce rapid antidepressant effects within the first 24–48 hours following the intervention, it has also been noted that the subsequent return to regular sleep may diminish these clinical effects. According to the literature, total sleep deprivation is reported to be the most effective method in terms of antidepressant efficacy (31). Circadian rhythm-targeted interventions in the treatment of the depressive phase of bipolar disorder have attracted attention, particularly due to their rapid onset of action. In this context, the study conducted by Barbini et al. (1998) demonstrated that three sessions of total sleep deprivation administered to individuals experiencing a major depressive episode produced different clinical effects depending on the diagnostic subtype of mood disorder. The three sessions of total sleep deprivation yielded more favorable clinical outcomes in individuals with bipolar disorder and those experiencing a single episode. Notably, unipolar patients showed worse depression scores following the three total sleep deprivation sessions compared to the other groups (32). In a separate study, Benedetti et al. (2001) aimed to examine the role of dopaminergic enhancement in sleep deprivation therapy. They combined three consecutive sessions of total sleep deprivation with amineptine, a dopamine reuptake inhibitor, in individuals diagnosed with bipolar depression. Although significant improvements in mood scores were observed following the first two deprivation sessions, no significant difference was found between the medication and placebo groups by the end of the intervention (33). In contrast, Colombo et al. (2000) highlighted the importance of combination therapies by demonstrating that the efficacy of total sleep deprivation was enhanced when combined with lithium and bright light therapy (34).

Studies evaluating the long-term effects of combined circadian interventions have also drawn attention. Wu et al. (2009), in a study combining total sleep deprivation, bright light therapy, and sleep phase advance as an adjunct to antidepressant treatment, reported that the antidepressant effect—initiated within 48

hours—was maintained over a 7-week period (35). Similarly, in a study conducted by Sikkens et al. (2019) involving individuals with both unipolar and bipolar depression, combined chronotherapeutic interventions yielded rapid and significant clinical responses, even in treatment-resistant depression (36). A systematic review conducted by D'Agostino et al. (2020), which analyzed 25 studies involving Triple Chronotherapy (Total Sleep Deprivation + Sleep Phase Advance + Bright Light Therapy) protocols, concluded that this approach represents an effective and safe adjunctive treatment option, with reported improvements in depression scores ranging from 50% to 84% and remission rates between 33% and 77%. However, the authors emphasized the need for further randomized controlled trials to assess the sustainability and generalizability of these effects (37).

Pharmacological treatments affecting circadian rhythms and the role of melatonin

It has been suggested that psychotropic drugs may affect sleep through mechanisms such as regulating the activity of the suprachiasmatic nucleus, aligning the sleep/wake cycle, and stabilizing biological rhythms. Specifically, lithium has been shown to affect the suprachiasmatic nucleus through the inhibition of Glycogen Synthase Kinase-3 Beta (GSK-3β) (6). In addition, lithium promotes the rapid proteasomal degradation of the nuclear receptor Rev-erbα, leading to the activation of the core clock gene Bmal1. A lithium-insensitive form of Rev-erbα interferes with circadian gene expression, highlighting the importance of Rev-erbα stability as a biological target of lithium therapy (38).

Selective serotonin reuptake inhibitors (SSRIs) have also been shown to influence circadian rhythms by regulating the suprachiasmatic nucleus. Moreover, the two-week latency typically observed before SSRIs exert their antidepressant effects aligns with the time required to re-regulate the disrupted circadian clock and rhythm. Fluoxetine has been shown to modulate the circadian system in depression by advancing the phase activation of suprachiasmatic nucleus neurons (6). N-Acetyl-5-methoxytryptamine (Melatonin) is a natural hormone first identified in 1958, secreted by the pineal gland during the night. Its secretion level is regulated through the suprachiasmatic nucleus, reaching a peak during the night, while it is typically undetectable during the day. By transmitting photoperiodic information to the body's organ systems and peripheral clocks, it supports the phase adjustment of the body's circadian rhythms (39). Melatonin levels are most measured in blood or saliva. In addition to its role as a significant chronobiotic agent, it also possesses sleep-promoting, antioxidant, anti-apoptotic, immune-enhancing, and oncostatic properties (40). Melatonin, which plays a role in the initiation of sleep, begins approximately 2 hours before the usual bedtime, marking the onset of evening sleep. It peaks around 02:00-03:00 AM. With the onset of sleep, melatonin levels rise at night and decrease in the morning. Melatonin is known to help initiate sleep by activating gamma-aminobutyric acid (GABAergic) mechanisms in the suprachiasmatic nucleus. While melatonin use has been observed to increase sleep duration and quality, drowsiness and sedation are the most common side effects. Depressive symptoms and suicidal thoughts may also be observed as side effects (Gottlieb et al., 2019). In this context, agomelatine—a novel antidepressant with proven efficacy in major depressive disorder—acts both as an agonist at melatonergic MT1 and MT2 receptors and as an antagonist at 5-HT2C receptors. Animal studies have shown that agomelatine increases noradrenaline and dopamine in the frontal cortex, restores disrupted circadian rhythms, and exerts antidepressant effects. Its therapeutic action is believed to stem from the synergy between melatonergic and serotonergic modulation in the suprachiasmatic nucleus, highlighting its dual chronobiotic and antidepressant role (41).

Disruptions in the melatonin secretion rhythm have been observed in individuals with bipolar disorder, with melatonin levels particularly reduced during depressive episodes. In line with this, a study conducted by Li et al. (2024) measured melatonin levels using saliva samples collected over two days from individuals diagnosed with depression and bipolar disorder

and examined their association with anhedonia. The findings indicated that the peak phase of melatonin secretion was delayed in the depression group, that the rhythm differed from that observed in bipolar disorder, and that this difference was associated with anhedonia (42). The therapeutic potential of melatonin and similar agents has been investigated, particularly as adjunctive treatment options in bipolar disorder. In a study by Mahableshwarkar et al. (2017), individuals with a diagnosis of bipolar I disorder were administered sublingual ramelteon (0.1 mg/day) in addition to their ongoing treatment. The results showed no significant difference between the ramelteon and placebo groups (43). Similarly, a meta-analysis by Kishi et al. (2019) reviewed studies involving the use of ramelteon and melatonin. The analysis found that ramelteon did not significantly differ from placebo in terms of recurrence of manic/hypomanic or mixed episodes, sleep quality, depressive symptoms, quality of life, or side effects such as headache, insomnia, somnolence, anxiety, and dizziness. However, ramelteon was found to significantly reduce recurrence related to depressive episodes compared to placebo (44). Studies focusing on the effects of melatonin on manic symptoms have yielded mixed results. In a placebo-controlled study conducted by Moghaddam et al. (2020), the efficacy of adjunctive melatonin treatment was evaluated in individuals with bipolar disorder experiencing acute manic episodes, in combination with lithium and risperidone. The study found that melatonin led to significant improvements in manic symptoms and overall clinical status, suggesting that it may be an effective adjunct in the treatment of acute mania (45). However, a study by Quested et al. (2021) evaluated the safety of melatonin in the treatment of manic and hypomanic episodes in bipolar disorder and reported that melatonin did not produce a significant therapeutic effect on these episodes over a three-week treatment period. The authors also noted the small sample size as a limiting factor (46). In another study by Bersani et al. (2000), patients diagnosed with bipolar I disorder who were undergoing antimanic treatment were administered 3 mg of melatonin for four weeks to address treatment-resistant insomnia. A significant reduction in manic symptoms was observed following the intervention (47). Conversely, an eight-week study by Romo-Nava et al. (2014) involving individuals with bipolar I disorder found no significant difference between the intervention and control groups following the administration of 5 mg melatonin (48).

Overall, while melatonin and similar agents appear to offer potential benefits in managing depressive symptoms and sleep-related disturbances in bipolar disorder, findings regarding their effects on manic and hypomanic symptoms remain inconsistent.

Cognitive behavioral therapy for insomnia protocol (CBT-I)

Cognitive Behavioral Therapy for Insomnia (CBT-I) has been observed to be used in regulating sleep in various psychiatric disorders such as bipolar disorder, depression, psychotic disorders, alcohol use disorder, and post-traumatic stress disorder (49). Benzodiazepines and antidepressant medications are commonly used in the treatment of sleep disorders (50). Although these pharmacological treatments are frequently used, they are known to have many disadvantages (51). Considering the long-term effectiveness, treatment approaches that do not rely on pharmacological methods are gaining prominence. Among these psychosocial interventions, Cognitive Behavioral Therapy for Insomnia Protocol is frequently used in the treatment of insomnia disorder (52,53).

In CBT-I interventions, the treatment process typically ranges from 4 to 8 sessions, with each session lasting between 60 and 120 minutes. The number and duration of sessions may vary depending on individual factors such as the client's educational level and the severity of the sleep disorder. Within the framework of the CBT-I protocol, a comprehensive approach is adopted, incorporating various techniques such as cognitive interventions, stimulus control, sleep restriction, and relaxation techniques. An examination of session content reveals that the first session primarily focuses on the client's reason for seeking treatment, involving a comprehensive assessment that addresses the potential medical and psychological origins of sleep problems. At this stage, the treatment

method is explained to the client, and the practice of maintaining a sleep diary is initiated. The graphical analysis of the sleep diary aims to enhance the client's awareness of their sleep patterns. In the second session, dysfunctional sleep behaviors are identified based on the data is collected from the client's sleep diary, and an individualized intervention plan is developed accordingly. During this session, stimulus control and sleep restriction techniques are also introduced, and clients are instructed to implement these techniques as homework. The third session addresses the concept of sleep hygiene, and lifestyle adjustments tailored to the client's specific circumstances are developed. In the fourth session, previous interventions are reviewed, difficulties encountered are evaluated, and the treatment is adjusted accordingly. The fifth session focuses on cognitive components, aiming to identify and restructure dysfunctional beliefs and thoughts related to sleep. Throughout the process, interventions such as stimulus control techniques, sleep restriction, relaxation exercises, and cognitive restructuring are employed as core components (54).

In recent years, the effects of CBT-I on insomnia comorbid with psychiatric disorders have been increasingly investigated. This growing interest stems from the fact that the coexistence of insomnia with psychiatric conditions has been associated with more severe psychiatric symptoms, and some studies have suggested that CBT-I may have beneficial effects not only on insomnia but also on accompanying psychiatric symptoms (55). For instance, in individuals newly diagnosed with depression or bipolar disorder who also experience insomnia, a four-session CBT-I intervention has been associated with higher depression remission rates and reduced use of anxiolytics. Furthermore, a decrease in negative cognitions related to sleep has also been observed (56).

In a meta-analysis conducted by Hertenstein and colleagues (2022), the effectiveness of CBT-I was examined in individuals with co-occurring psychiatric disorders and insomnia. The findings indicated that CBT-I significantly reduced the severity of insomnia and also led to improvements in depressive symptoms following treatment. Moreover, in individuals diagnosed with bipolar disorder, CBT-I was found to significantly decrease insomnia severity (49). Another meta-analysis conducted by Uğurlu et al. (2025) evaluated the effectiveness of CBT-I in individuals with psychotic disorders. This study included research involving individuals diagnosed with bipolar disorder or unipolar depression with psychotic features, who had been experiencing sleep problems for at least one month and were receiving treatment. The findings revealed that CBT-I significantly reduced insomnia symptoms and improved sleep quality both in the short and long term among individuals with psychotic disorders. Additionally, it was reported that CBT-I contributed to the reduction of psychotic symptoms and enhancement of overall psychological well-being in the short term (57).

CBT-I has been modified for individuals diagnosed with bipolar disorder. By incorporating techniques from interpersonal and social rhythm therapy, chronotherapy, and motivational interviewing, CBT-I has been adapted to create CBTI-BD, aimed at regulating mood and sleep, as well as improving functionality in individuals with bipolar disorder (7). The core components of this method include functional assessment/case formulation, goal setting, motivational interviewing, and education on sleep and circadian rhythms (6). In a study conducted by Harvey et al. (2015), a bipolar disorder-specific adaptation of CBT-I, known as CBTI-BD, was implemented in individuals diagnosed with Bipolar I Disorder who were also experiencing insomnia. The group that received eight sessions of CBTI-BD demonstrated lower rates of hypomanic/manic episode recurrence compared to those in the psychoeducation group. Additionally, participants in the CBTI-BD group showed a significant reduction in insomnia severity and improvements in sleep quality (23). Similarly, Swagemakers and colleagues (2021) applied a seven-session group-based CBTI-BD intervention to individuals diagnosed with Bipolar I or II Disorder. Their findings indicated a significant decrease in insomnia severity from pre- to post-treatment. Moreover, throughout the group CBTI-BD program, mood episode recurrence was prevented, and

the total number of nights during which psychotropic medications were used for sleep was reduced (58).

Interpersonal and social rhythm therapy (IPSRT)

Interpersonal and Social Rhythm Therapy (IPSRT) is an intervention designed for individuals diagnosed with bipolar disorder, whose mood regulation processes are marked by variability (59). The approach integrates principles from both interpersonal psychotherapy and social rhythm therapy. Grounded in the social zeitgeber theory, IPSRT emphasizes the complex relationship between psychological symptoms and stress arising from interpersonal relationships (60).

The primary objective of IPSRT is to regulate daily rhythms and enhance mood stability by establishing consistent sleep hygiene (59). Therapeutic sessions typically last approximately 45 minutes and are conducted on a weekly basis. The duration of the intervention may vary from several weeks to several months, depending on the individual's clinical needs. The therapy is delivered across four distinct phases: the initial phase, the intermediate phase, the maintenance phase, and the termination phase (61,62,63). In the initial phase of IPSRT, a comprehensive clinical history is first obtained, which includes an assessment of interpersonal problem areas and the provision of psychoeducation about the patient's disorder. A timeline of significant life events is constructed to analyze the characteristics of past mood episodes, including their duration, triggering factors, and treatment modalities. To contextualize the patient's experience, an analysis of key individuals in their life—both past and present—is conducted to better understand their interpersonal dynamics. The relationship between mood symptoms and interpersonal disruptions is also thoroughly assessed (61,62,63). During the intermediate phase, therapeutic efforts focus on stabilizing key variables such as sleep-wake cycles, mealtime regularity, and work schedules. For patients with significant variability in sleep duration or timing, interventions are aimed at standardizing these rhythms. Similarly, irregular eating patterns are restructured, and social interactions are adjusted to a balanced level, avoiding both excessive engagement and social withdrawal. The reduction of overstimulating activities is also a key objective. Simultaneously, structured strategies are developed to address ongoing interpersonal difficulties (61,62,63). The maintenance phase is centered on the application of acquired skills to sustain stable social and biological rhythms. Patients are encouraged to preserve this stability, particularly during transitional periods like job changes, holidays, or unexpected stressors. The primary focus is to maintain consistency in daily routines and to enhance the patient's self-awareness of their internal and external rhythms (61,62,63). Finally, the termination phase involves a comprehensive review of the patient's progress. Interpersonal functioning is re-evaluated, and relapse prevention strategies are developed to help patients identify and respond to early warning signs of a potential mood episode. This final stage is typically carried out gradually over four to six months through structured follow-up sessions, marking the conclusion of the therapeutic process (61,62,63).

Several randomized controlled trials have been conducted in the literature regarding Interpersonal and Social Rhythm Therapy (IPSRT). In a study by Swartz et al. (2012), individuals with bipolar II depression who had not previously used medication were assigned to receive either IPSRT or quetiapine. No significant differences in outcomes were observed between IPSRT monotherapy and quetiapine treatment over a 12-week follow-up period. The authors suggested that future studies should investigate the characteristics of individuals who respond differentially to psychotherapy versus pharmacotherapy (64). In another study involving adolescents diagnosed with bipolar disorder, the addition of IPSRT or supportive care to pharmacological treatment resulted in reductions in depressive and manic symptoms and improvements in social functioning (65). However, in a subsequent 78-week long-term follow-up study conducted by the same research team, the addition of IPSRT to pharmacological treatment did not yield significant improvements in depression or mania scores (66). This

finding highlights the need for further research into the long-term efficacy of IPSRT. Nonetheless, a group-based IPSRT study by Bouwkamp et al. (2013) reported positive outcomes at a one-year follow-up, including reductions in depressive symptoms, increased regularity of social rhythms, and fewer hospital admissions (67). Similarly, in another group-format IPSRT study investigating its effects on bipolar depression, two individual and six group sessions were administered. The results indicated symptom reduction, which persisted over a 12-week period (68). A study conducted by Crowe et al. (2020) found that the combination of IPSRT and medication management over an 18-month period did not significantly affect relapse rates but did lead to improvements in functional outcomes (69). In a separate study by the same authors, individuals with major depressive disorder who received IPSRT over a 12-month period demonstrated improvements in depressive symptoms and functioning compared to baseline (70). In another study by Moot et al. (2021), approximately 24 sessions of IPSRT were administered to individuals with bipolar disorder and comorbid substance use disorder. The results suggested that lifetime substance use disorders may negatively influence psychotherapy outcomes (71). Finally, in a study by Steardo et al. (2020), IPSRT administered over a 12-week period was found to have beneficial effects on manic and depressive symptoms, responsiveness to mood stabilizers, and overall functioning. Notably, improvements in manic and depressive symptoms were sustained during a 6-month follow-up period (72).

CONCLUSION

The social zeitgeber theory postulates that perturbations in the regularity of social zeitgebers—such as sleep-wake cycles and meal timing—induced by stressful life events may result in disturbances of circadian rhythms. These disruptions, in turn, have the potential to precipitate mood episodes in individuals with a predisposition to bipolar disorder. Furthermore, alterations in both social and biological rhythms detrimentally impact sleep quality, thereby contributing to the emergence of depressive, manic, and hypomanic episodes.

The literature describes various interventions aimed at regulating social rhythms, including bright light therapy, dark therapy, sleep deprivation, pharmacological treatments, CBT-I, and IPSRT. Findings indicate that bright light therapy may serve as an effective and safe adjunctive treatment, particularly during depressive episodes of bipolar disorder. The regulatory effect of bright light on circadian rhythms is attributed to its suppression of melatonin secretion, which supports mood stabilization. However, some studies have reported inconsistent efficacy. Conversely, interventions such as dark therapy and the use of blue light-blocking glasses during manic episodes have been shown to reduce manic symptoms, improve sleep quality, and decrease the need for pharmacological treatment. Overall, light-based interventions—including bright light therapy, dark therapy, and blue light-blocking glasses—appear to be effective adjuncts to pharmacotherapy in the treatment of bipolar disorder. More recently, protocols combining sleep deprivation with bright light therapy and sleep phase advance have demonstrated rapid therapeutic effects as well as prolonged maintenance of these effects. Notably, Triple Chronotherapy protocols, which integrate total sleep deprivation, sleep phase advance, and bright light therapy, have yielded over 50% improvements in depression scores and high remission rates, demonstrating both efficacy and safety. Nevertheless, further randomized controlled trials are needed to confirm the generalizability and long-term durability of these outcomes. Although melatonin and related agents show potential benefits in alleviating depressive symptoms and sleep disturbances in bipolar disorder, findings regarding their effects on manic and hypomanic symptoms remain inconclusive.

Regarding psychotherapeutic interventions, recent studies have demonstrated that CBT-I is an effective treatment for insomnia comorbid with psychiatric disorders. Following CBT-I, increased rates of depression remission, reductions in anxiolytic use, decreased insomnia severity, and diminished dysfunctional beliefs about sleep have been reported. The CBT-I protocol has been restructured under the name CBTI-BD by integrating it with interpersonal and social rhythm therapy, chronotherapy, and

motivational interviewing. CBTI-BD has been shown to reduce both insomnia severity and the frequency of manic/hypomanic episodes, as well as decrease psychotropic medication use for sleep in individuals diagnosed with bipolar disorder. IPSRT, administered in both individual and group formats, has demonstrated reductions in depressive and manic symptoms, improvements in social rhythm regularity, and enhancements in functional outcomes. However, some longitudinal studies report that symptomatic improvements are not always sustained over the long term.

In conclusion, chronotherapeutic interventions (including bright light and dark therapy, sleep deprivation, and melatonin treatment) alongside psychotherapeutic approaches (such as CBTI-BD and IPSRT) have been shown to reduce relapse rates and alleviate symptoms, while positively influencing sleep processes and overall functioning in individuals with bipolar disorder. Circadian rhythm-targeted treatments hold promise, especially for patients with pharmacoresistant bipolar disorder, due to their rapid onset of action and neurobiological basis. However, more research is needed to provide scientific data on a broader range of topics, including studies with larger sample sizes, the standardization of intervention protocols, and the long-term follow-up of clinical outcomes. The limited number of studies conducted in Turkey underscores the need for expanded research into circadian and social rhythm-based interventions and their integration into clinical practice.

Author's contribution

Conceptualization: YA, TŞT; Analysis and/or Interpretation: YA, TŞT; writing: YA, TŞT; review: YA, TŞT; editing: YA, TŞT. All authors have read and agreed to the published version of the manuscript.

Conflict of Interest

There is no financial or other conflict of interest related to the study.

REFERENCES

1. Frank E, Swartz HA, Boland E Interpersonal and social rhythm therapy: an intervention addressing rhythm dysregulation in bipolar disorder. Dialogues in Clinical Neuroscience 2007;9,325–332.
2. Nierenberg, A, Agustini B, Köhler-Forsberg O, Cusin C, Katz, D, Sylvia LG., ... & Berk M. Diagnosis and treatment of bipolar disorder: a review. Jama 2023;330(14), 1370-1380.
3. Post RM, Leverich GS. The role of psychosocial stress in the onset and progression of bipolar disorder and its comorbidities: The need for earlier and alternative modes of therapeutic intervention. Dev Psychopathol 2006;18, 1181–1211.
4. Malkoff-Schwartz S, Frank E, Anderson BP, Hlastala SA, Luther JF, Sherrill JT, Houck PR, Kupfer DJ. Social rhythm disruption and stressful life events in the onset of bipolar and unipolar episodes. Psychol Med 2000;30(5):1005–16.
5. Crowe M, Whitehead L, Wilson L, et al. Disorder-specific psychosocial interventions for bipolar disorder – a systematic review of the evidence for mental health nursing practice. International Journal of Nursing Studies 2010;47, 896–908.
6. Gıca Ş, Selvi Y. Şizofreni ve Bipolar Bozukluğun Tedavisinde Uyku Alanında Müdahaleler. Archives of Neuropsychiatry/Noropsikiatri Arsivi 2021;58,53-60.
7. Gottlieb JF, Benedetti F, Geofroy P, et al.The chronotherapeutic treatment of bipolar disorders:A systematic review and practice recommendations from the ISBD task force on chronotherapy and chronobiology. Bipolar Disorders 2019; 21:741–773.
8. Sylvia LG, Alloy LB, Hafner JA, Gauger MC, Verdon K, Abramson LY. Life events and social rhythms in bipolar spectrum disorders. J Affect Disord 2009;113(1-2):117–25.
9. Ehlers C.L, Frank E, Kupfer D.J. Social zeitgebers and biological rhythms. A unified approach to understanding the etiology of depression. Archives of General Psychiatry 1988;45, 948–952.

10. Boland E.M, Bender R.E, Alloy L.B, et al. Life events and social rhythms in bipolar spec- trum disorders: an examination of social rhythm sensitivity. Journal of Affective Disorders 2012;139, 264–272.

11. Zou H, Zhou H, Yan R, Yao Z, Lu Q. Chronotype, circadian rhythm, and psychiatric disorders: Recent evidence and potential mechanisms. Frontiers in neuroscience 2022; 16, 811771.

12. Grandin, LD, Alloy LB, Abramson LY. The social zeitgeber theory, circadianrhythms, and mood disorders: Review and evaluation. Clinical Psychology Review 2006;26 (6), 679–694. https://doi.org/10.1016/j.cpr.2006.07.001.

13. Rosenthal NE, Sack DA, Gillin JC, et al. Seasonal affective disorder: a description of the syndrome and preliminary findings with light therapy. Arch Gen Psychiatry 1984;41:72-80.

14. Al-Karawi D, Jubair L. Bright light therapy for nonseasonal depression: Meta-analysis of clinicaltrials.JAffectDisord 2016;198:65-71.

15. Terman M, Terman JS. Light therapy for seasonal and nonseasonal depression: efficacy, protocol, safety, and side effects. CNS Spectr 2005;10:647-663.

16. Zhou TH, Dang WM, Ma YT, et al. Clinical efficacy, onset time and safety of bright light therapy in acute bipolar depression as an adjunctive therapy: a randomized controlled trial. Journal of affective disorders 2018;227, 90-96.

17. Sit DK, McGowan J, Wiltrout C, et al. Adjunctive bright light therapy for bipolar depression: a randomized double-blind placebo-controlled trial. American Journal of Psychiatry 2018;175(2), 131-139.

18. Kupeli, NY, Bulut NS, Bulut GC, et al. Efficacy of bright light therapy in bipolar depression. Psychiatry research 2018;260, 432-438.

19. Benedetti F, Dallaspezia S, Fulgosi MC, et al. Phase advance is an actimetric correlate of antidepressant response to sleep deprivation and light therapy in bipolar depression. Chronobiology International 2007;24, 921–937.

20. Legenbauer T, Kirschbaum-Lesch I, Jörke C, et al. Bright light therapy as add-on to inpatient treatment in youth with moderate to severe depression: a randomized clinical trial. JAMA psychiatry 2024;81(7), 655-662.

21. Chojnacka M, Antosik-Wójcińska AZ, Dominiak M, et al. A sham controlled randomized trial of adjunctive light therapy for non- seasonal depression. J Affect Disord 2016;203:1-8.

22. Phelps J. Dark therapy for bipolar disorder using amber lenses for blue light blockade. Med Hypotheses, 2008;70:224–229.

23. Harvey AG, Soehner AM, Kaplan KA, et al. Treating insomnia improves mood state, sleep, and functioning in bipolar disorder: A pilot randomized controlled trial. J Consult Clin Psychol 2015;83:564–577.

24. Hester L, Dang D, Barker CJ, Heath M, Mesiya S, Tienabeso T, Watson K. Evening wear of blue-blocking glasses for sleep and mood disorders: a systematic review. Chronobiology International 2021;38(10), 1375-1383.

25. Henriksen TE, Skrede S, Fasmer OB, et al. Blue-blocking glasses as additive treatment for mania: a randomized placebo-controlled trial. Bipolar Disord 2016;18(3):221–32.

26. Henriksen TE, Grønli J, Assmus J. et al. Blue-blocking glasses as additive treatment for mania: effects on actigraphy-derived sleep parameters. Journal of sleep research 2020;29(5), e12984.

27. Barbini B, Bendetti F, Colombo A, et al. Dark therapy for mania: a pilot study. Bipolar Disorders 2005;7:98–101.

28. Swanson LM, Schubert JR, Raglan GB, Conroy DA. Chronotherapeutic treatments for psychiatric disorders: a narrative review of recent literature. Current Psychiatry Reports 2025;1-15.

29. Wirz-Justice A. Biological rhythms and depression: treatment opportunities. WPA Bulletin on Depression 2008;12:5-8.

30. Gottlieb JF, Goel N, Chen S, et al. Meta-analysis of sleep deprivation in the acute treatment of bipolar depression. Acta Psychiatrica Scandinavica 2021;143(4), 319-327.

31. Ramirez-Mahaluf JP, Rozas-Serri E, Ivanovic-Zuvic F, et al. Effectiveness of sleep deprivation in treating acute bipolar depression as augmentation strategy: a systematic review and meta-analysis. Frontiers in Psychiatry 2020;11, 70.

32. Barbini B, Colombo C, Benedetti F, et al.The unipolar–bipolar dichotomy and the response to sleep deprivation. Psychiatry Research 1998;79(1), 43-50.

33. Benedetti F, Campori E, Barbini B, et al. Dopaminergic augmentation of sleep deprivation effects in bipolar depression. Psychiatry Research 2001;104(3), 239-246.

34. Colombo C, Lucca A, Benedetti F, et al. Total sleep deprivation combined with lithium and light therapy in the treatment of bipolar depression: replication of main effects and interaction. Psychiatry research 2000;95(1), 43-53.

35. Wu JC, Kelsoe JR, Schachat C, et al. Rapid and sustained antidepressant response with sleep deprivation and chronotherapy in bipolar disorder. Biological psychiatry, 2009;66(3), 298-301.

36. Sikkens D, Riemersma-Van der Lek RF, Meesters Y, et al. Combined sleep deprivation and light therapy: Clinical treatment outcomes in patients with complex unipolar and bipolar depression. Journal of Affective Disorders 2019;246, 727-730.

37. D'Agostino A, Ferrara P, Terzoni S, et al. Efficacy of triple chronotherapy in unipolar and bipolar depression: a systematic review of the available evidence. Journal of Affective Disorders 2020;276, 297-304.

38. Yin L, Wang J, Klein PS, et al. Nuclear receptor Rev-erbα is a critical lithium-sensitive component of the circadian clock. Science 2006;311(5763):1002-5.

39. Claustrat B, Brun J, Chazot G. The basic physiology and pathophysiology of melatonin. Sleep Med Rev 2005;9(1):11-24.

40. Perumal S, Srinivasan V, Maestroni G, et al. Melatonin: nature's most versatile biological signal? The FEBS J 2006;273(13):2813-2838.

41. Racagni G, Riva MA, Popoli M. The interaction between the internal clock and antidepressant efficacy. Int Clin Psychopharmacol 2007;22 Suppl 2:S9–S14.

42. Li X, Yu J, Jiang S, et al. Circadian rhythms of melatonin and its relationship with anhedonia in patients with mood disorders: a cross-sectional study. BMC psychiatry 2024;24(1), 165.

43. Mahableshwarkar AR, Calabrese JR, Macek TA., et al. Efficacy and safety of sublingual ramelteon as an adjunctive therapy in the maintenance treatment of bipolar I disorder in adults: a phase 3, randomized controlled trial. Journal of Affective Disorders 2017;221, 275-282.

44. Kishi T, Nomura I, Sakuma K, et al. Melatonin receptor agonists—ramelteon and melatonin—for bipolar disorder: a systematic review and meta-analysis of double-blind, randomized, placebo-controlled trials. Neuropsychiatric disease and treatment 2019;1479-1486.

45. Moghaddam HS, Bahmani S, Bayanati S, et al. Efficacy of melatonin as an adjunct in the treatment of acute mania: a double-blind and placebo-controlledtrial. International Clinical Psychopharmacology 2020;35(2), 81-88.

46. Quested DJ, Gibson JC, Sharpley AL, et al. Melatonin In Acute Mania Investigation (MIAMI-UK). A randomized controlled trial of add-on melatonin in bipolar disorder. Bipolar Disorders 2021;23(2), 176-185.

47. Bersani G, Garavini A. Melatonin add-on in manic patients with treatment resistant

insomnia. Prog Neuropsychopharmacol Biol Psychiatry 2000;24(2):185-191.

48. Romo-Nava F, Gonzlez D, Orellana AF, et al. Melatonin attenuates antipsychotic metabolic effects: an eight-week randomized, doubleblind, parallel-group, placebo-controlled clinical trial. Bipolar Disord 2014;16(4):410-421.
49. Hertenstein E, Trinca E, Wunderlin M, et al. Cognitive behavioral therapy for insomnia in patients with mental disorders and comorbid insomnia: A systematic review and meta-analysis. Sleep medicine reviews 2022; 62, 101597.
50. Neubauer DN, Pandi-Perumal SR, Spence DW, et al. Pharmacotherapy of insomnia. J CentNervSystDis, 2018 10:1179573518770672.
51. Smith MT, Perlis ML, Park A, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. Am J Psychiatry 2002;159:5–11.
52. Morse CD, Klingman KJ, Jacob BL, et al. Exercise and ıinsomnia risk in middle-aged women. J Nurse Pract 2019;15:236-240.
53. Randall C, Nowakowski S, Ellis JG. Managing acute insomnia in prison: evaluation of a "one-shot" cognitive behavioral therapy for insomnia (CBT-I) intervention. Behav Sleep Med 2019;17:827-836.
54. Çıvgın U, Dirik G. Bilişsel Davranışçı Terapi-Uykusuzluk Protokolü ve Etkililik Çalışmalarının Gözden Geçirilmesi. Psikiyatride Güncel Yaklaşımlar 2020;12(2), 274-286.
55. Jansson-Fröjmark M, Norell-Clarke A. Cognitive behavioural therapy for insomnia in psychiatric disorders. Current sleep medicine reports 2016;2(4), 233-240.
56. Chung KF, Yeung WF, Ho FY, et al. Cognitive behavioural therapy for insomnia as an early intervention of mood disorders with comorbid insomnia: a randomized controlled trial. Early Interv Psychiatry 2024;18(2):82–93.
57. Ugurlu M, Karakas Ugurlu G, Kabadayi Sahin E, et al. Short and long-term effects of cognitive behavioral therapy on sleep problems and psychotic symptoms in patients with psychotic disorders: a meta-analysis. Brazilian Journal of Psychiatry 2025;47, e20243623.
58. Swagemakers O, Nugter A, Engelsbel F, et al. Feasibility of group cognitive behavioural therapy for insomnia (CBT-I) in bipolar disorder. European Psychiatry 2021;64(S1), S195-S196.
59. Frank E, Kupfer DJ, Thase ME, et al. Two-year outcomes for interpersonal and social rhythm therapy in individuals with bipolar I disorder. Archives of General Psychiatry 2005;62(9), 996-1004.
60. Goldstein TR, Merranko J, Krantz M, et al.Early intervention for adolescents at-risk for bipolar disorder: A pilot randomized trial of Interpersonal and Social Rhythm Therapy (IPSRT). Journal of affective disorders 2018;235,348-356.
61. Hızlı Sayar G, Özten E, Ünsalver B. Bipolar bozuklukta kişilerarası ilişkiler ve sosyal ritim terapisinin temel ilkeleri. Psikiyatride Güncel Yaklaşımlar 2014;6(4),438-446.
62. Frank E, Swartz H. A, Kupfer DJ. Interpersonal and social rhythm therapy: managing the chaos of bipolar disorder. Biol Psychiatry 2000;48, 593–604.
63. Frank E. Interpersonal and social rhythm therapy: a means of improving depression and preventing relapse in bipolar disorder. Journal of Clinical Psychology 2007;463-473. https://doi.org/10.1002/jclp.20371.
64. Swartz HA, Frank E, Cheng YA. Randomized pilot study of psychotherapy and quetiapine for the acute treatment of bipolar II depression. Bipolar Disorders 2012;14, 211–216.
65. Inder M, Crowe MT, Luty SE. Randomized, controlled trial of ınterpersonal and social rhythm therapy for young people with bipolar disorder. Bipolar Disorders 2015;17(2), (s.128-138). doi: 10.1111/bdi.12273.

66. Inder ML, Crowe MT, Moor S, et al. Three-year follow-up after psychotherapy for young people with bipolar disorder. Bipolar Disord 2017;22. doi: 10.1111/bdi.12582.

67. Bouwkamp CG, de Kruiff ME, van Troost TM, et al. Interpersonal and social rhythm group therapy for patients with bipolar disorder. Int J Group Psychother 2013;63(1):97–115.

68. Hoberg AA, Ponto J, Nelson PJ, et al. Group interpersonal and social rhythm therapy for bipolar depression. Perspect Psychiatr Care 2013;49(4):226–34.

69. Crowe M, Porter R, Inder M, et al. Clinical effectiveness trial of adjunctive ınterpersonal and social rhythm therapy for patients with bipolar disorder. Am J Psychother 2020;1:73(3),107-114.

70. Crowe M, Inder M, Douglas K, et al. Interpersonal and social rhythm therapy for patients with major depressive disorder. American journal of psychotherapy 2020;73(1), 29-34.

71. Moot W, Crowe M, Inder M, et al. Functional and mood outcomes in bipolar disorder patients with and without substance use disorders undergoing psychotherapy. Frontiers in psychiatry 2021;12, 661458.

72. Steardo L, Luciano M, Sampogna G, et al.Efficacy of the interpersonal and social rhythm therapy (IPSRT) in patients with bipolar disorder: results from a real-world, controlled trial. Annals of general psychiatry 2020;19, 1-7.