Behavioral Treatment of Insomnia in Bipolar Disorder

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Sleep disturbance is common in bipolar disorder. Stimulus control and sleep restriction are powerful, clinically useful behavioral interventions for insomnia, typically delivered as part of cognitive-behavioral therapy for insomnia (CBT-I). Both involve short-term sleep deprivation. The potential for manic or hypomanic symptoms to emerge after sleep deprivation in bipolar disorder raises questions about the appropriateness of these methods for treating insomnia. In a series of patients with bipolar disorder who underwent behavioral treatment for insomnia, the authors found that regularizing bedtimes and rise times was often sufficient to bring about improvements in sleep. Two patients in a total group of 15 patients reported mild increases in hypomanic symptoms the week following instruction on stimulus control. Total sleep time did not change for these individuals. Two of five patients who underwent sleep restriction reported mild hypomania that was unrelated to weekly sleep duration. Sleep restriction and stimulus control appear to be safe and efficacious procedures for treating insomnia in patients with bipolar disorder. Practitioners should encourage regularity in bedtimes and rise times as a first step in treatment, and carefully monitor changes in mood and daytime sleepiness throughout the intervention.

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Reduced need for sleep and insomnia or hypersomnia are common symptoms of the manic and depressive phases of bipolar disorder. This sleep disturbance persists into the period between episodes (the interepisode period) and may contribute to illness relapse. In one study, for example, 55% of an interepisode bipolar group met diagnostic criteria for insomnia (1). Compared with bipolar patients with longer sleep times, short sleepers exhibit more symptoms of mania, depression, anxiety, and irritability (2). A meta-analysis found sleep disturbance to be the most common prodrome of mania and the sixth most common prodrome of depression (3). Finally, experimentally induced sleep deprivation is associated with the onset of hypomania or mania (4), and sleep loss is highly correlated with daily manic symptoms (5).

Increasingly, nonpharmacological interventions, mostly cognitive-behavioral in content, are used to treat sleep disturbance. These are typically administered as a multicomponent treatment known as cognitive-behavioral therapy for insomnia (CBT-I). The efficacy of CBT-I has been summarized in systematic reviews (6), but the treatment techniques have not been evaluated in bipolar disorder.

Stimulus control (7) and sleep restriction (8) are the most carefully evaluated components of CBT-I. These interventions are designed to strengthen the association between sleep and the sleeping environment, to develop a consistent sleep-wake schedule, and to strengthen the homeostatic sleep drive. Stimulus control traditionally involves four components (1): using the bed and bedroom only for sleep and sex (2); going to bed only when sleepy (3); leaving the bedroom if unable to fall asleep or return to sleep within 15–20 minutes; and (4) arising at the same time each morning. Sleep restriction is derived from the proposal that excessive time in bed perpetuates insomnia. Time spent awake in bed leads to elevated levels of arousal and rumination, and the bed is associated with wakefulness rather than with sleep. To reverse this association, sleep restriction involves limiting time in bed to the actual amount of time slept. Based on sleep diary data, a specific amount of time in bed is prescribed, referred to as a "sleep window." For example, for a patient reporting a weekly average of 8 hours in bed and 6 hours of sleep, a 6-hour sleep window is prescribed at the start of treatment. The goal is to raise sleep efficiency, defined as total sleep time divided by time in bed, to 85%−90% (80% in older adults). The duration of the sleep window is reviewed weekly and increased or decreased on the basis of sleep efficiency from the previous week. Sleep opportunity is gradually increased to an optimal sleep time.

There are several reasons to suspect that stimulus control and sleep restriction are promising interventions for sleep disturbance in bipolar disorder (9). First, individuals with bipolar disorder exhibit night-to-night variability in total sleep time (2), along with reduced sleep efficiency and increased nighttime wakefulness when measured with actigraphy (1, 10) and with prospective self-report (11). As the goal of sleep restriction and stimulus control is to increase sleep efficiency and regularize the sleep-wake cycle (6), it may be particularly useful for this population. Moreover, the optimal intervention for insomnia is one that alleviates insomnia without causing adverse interactions with prescribed medication; therefore, a nonpharmacological intervention may be the best choice for the management of insomnia in patients with bipolar disorder (9). Finally, there is

This article is featured in this month's AJP Audio and is an article that provides Clinical Guidance (p. 720)

A 40-year-old woman with bipolar I disorder, currently euthymic, presents with persistent insomnia, worry about sleep duration, and concern about manic relapse.

"Ms. D" is a 40-year-old woman referred for treatment for persistent difficulty initiating sleep. She reports fatigue, irritability, and concern over her sleep, as she has heard that sleep loss can trigger manic episodes. She denies additional symptoms of depression or mania. Ms. D currently alternates between 50 mg of trazodone and 10 mg of zolpidem nightly. It takes her up to 2 hours to fall asleep at night, and she reports 8 hours in bed nightly and 6.5 hours per night of sleep on average.

Sleep restriction and stimulus control are two of the most effective nonpharmacological interventions for insomnia, yet both involve short-term sleep deprivation. Ms. D would be required to limit her time in bed to 6.5 hours per night, equivalent to her current total sleep time. Given the potential for manic or hypomanic symptoms to emerge after sleep deprivation, however, it is unclear whether it is safe to make these recommendations to treat her insomnia.

To institute standard sleep restriction and stimulus control, Ms. D was asked to limit her time in bed to 6.5 hours per night and to get out of bed if she was unable to sleep. Symptoms of mania and depression were monitored weekly. Another treatment goal was to regularize her bedtimes and rise times. Although she experienced a slight short-term decrease in total sleep time with her new behavioral regimen, she reported no increase in manic symptoms. Over the course of 8 weeks, Ms. D reported a decrease in her latency to fall asleep and reported feeling more sleepy at bedtime. Ms. D also noted feeling like she had more control over her illness and feeling grateful for the "tools" she had learned for managing her sleep.

evidence that nonpharmacological interventions for insomnia are more acceptable to patients (12) and produce more durable effects (13, 14) than hypnotic medications alone.

Even so, there are at least three reasons to question the applicability of stimulus control and sleep restriction for bipolar patients with insomnia. First, waiting to feel sleepy before getting into bed may be contraindicated. Numerous researchers have proposed that increased goal-oriented behavior is a hallmark of bipolar disorder and suggested that disengagement from arousing stimuli may be difficult even between episodes (15, 16). For example, Talbot et al. (17) found that increasing positive affect before bedtime through a positive mood induction in interepisode bipolar patients led to an increase in length of sleep onset. Individuals with bipolar disorder may need to get into bed, even though not yet sleepy, in order to begin the process of down-regulating sufficiently to achieve sleep onset. Second, the recommendation to get out of bed in the middle of the night may lead to further engagement in rewarding and arousing activities that reduce the potential for sleep. Third, and perhaps most fundamentally, both stimulus control and sleep restriction involve short-term sleep deprivation; this sleep deprivation helps to increase nighttime sleep pressure, consolidate sleep, and realign the circadian clock. In bipolar patients, however, experimental evidence suggests that in a subset of individuals, acute sleep deprivation can result in next-day hypomanic or manic symptoms (4). Therefore, the safety of implementing these interventions in individuals with bipolar disorder is unknown. We present treatment results for 15 patients who showed acceptable safety for the intervention.

Participants and Treatment

All patients met DSM-IV-TR criteria for bipolar I disorder and Research Diagnostic Criteria for insomnia (18). All participants were euthymic at the start of treatment, defined as the absence of diagnoses of current mania/hypomania and depression according to the Structured Clinical Interview for DSM-IV Axis I Disorders (19) and confirmed by a score ≤24 on the Inventory of Depressive Symptomatology–Clinician Version (IDS-C) (20) and a score <12 on the Young Mania Rating Scale (YMRS) (21), as research has suggested a YMRS score of 12 or greater would merit criteria for a DSM-IV-TR hypomanic episode (22). The first of eight therapy sessions involved devising a detailed case formulation for each patient (23), establishing treatment goals, educating patients on sleep in bipolar disorder, and creating wind-down and wake-up routines around a set bedtime and rise time of the patient's choosing. When indicated, sleep restriction was typically introduced in the second therapy session. Stimulus control was typically presented immediately after or at the beginning of the third session. Stimulus control and sleep restriction (when delivered) became rolling interventions that were revisited in subsequent sessions. Mood was assessed at the beginning of each session using the Quick Inventory of Depressive Symptomatology (24) and the YMRS (21). Participants' mean age was 38.1 years (SD=11.5), and at the start of treatment they had a mean score of 12.5 (SD=5.9) on the IDS-C and a mean score of 2.3 (SD=1.5) on the YMRS.

Results

Stimulus Control

All patients were introduced to stimulus control. For three individuals, stimulus control instructions were individualized because of poor tolerability. One patient reported watching television during periods of wakefulness, from which she found it difficult to disengage; in collaboration with the therapist, she decided to remain in bed practicing relaxation strategies (e.g., abdominal breathing) when awake at night. Another patient, who had extended awakenings in the middle of the night, reported that he monitored the time he spent out of bed by the passing

trains or the number of pages he read, which heightened his insomnia-related anxiety. After 2 weeks of complying with stimulus control, he decided in collaboration with the therapist to remain in bed. A third patient, who met criteria for comorbid generalized anxiety disorder and specific phobia at the start of treatment, reported using the bed and bedroom as an escape from overwhelming anxiety; he found it difficult to comply with the stimulus control recommendation to limit activities in bed to sleep and sex, and he continued to perform a majority of his activities (work, leisure, computer use) in bed throughout the day.

Sleep Restriction

Five of the 15 patients were introduced to sleep restriction. Sleep restriction was not introduced to the remaining 10 patients because setting regular bedtimes and rise times in session 1 led to marked improvement in sleep efficiency by session 2 for the vast majority of participants (N=8). Other reasons sleep restriction was not introduced included a baseline average total sleep time that was too low to safely suggest further restriction (total sleep time, ≤5.5 hours) and an inability to change bedtimes and rise times given rules set by the group home in which the patient was residing.

Mood

To evaluate the safety of each technique, weekly changes in mood were carefully assessed. Four of the 15 participants developed mild to moderate symptoms of mania during the 8 weeks of treatment. Two of these four individuals were introduced to sleep restriction; the first reported mood elevation lasting 2 days that coincided with the first week of sleep restriction, and the second reported mood elevation 4 weeks after sleep restriction was introduced. Examination of sleep diaries from these two patients revealed that total sleep time was unaffected during these periods of elevation, and both patients had mood elevations in the asymptomatic to mild range (YMRS scores ≤12).

All patients in the study, including the four who developed mood elevation, were introduced to stimulus control. Two of the four patients developed these hypomanic symptoms in the week immediately following stimulus control instruction; average total sleep time, however, did not decrease substantially for either patient. One patient developed hypomanic symptoms 1 week before stimulus control was introduced, and the other developed mood elevation 3 weeks after it was introduced.

Conclusions

Preliminary analysis of results suggested that the CBT-I intervention had a positive impact on sleep in this sample of 15 patients. On a self-report measure of insomnia severity (25), pre- and postintervention scores decreased from 17.9 to 6.0 ($t=8.3$, $df=14$, $p<0.001$). We also observed an improvement in sleep efficiency according to sleep diary, from 84.8% before intervention to 88.2% after intervention, although the difference fell short of significance ($t=-2.0$, $df=14$, $p=0.06$). We chose to focus on subjective outcome measures (self-reported symptom severity and sleep diary measures) following current guidelines, which recommend that insomnia diagnosis and assessment be made with subjective measures and not polysomnography (26–28).

Our objectives in this study were to evaluate the safety and tolerability of sleep restriction and stimulus control in individuals with insomnia and bipolar disorder. Considering sleep restriction first, we found limited evidence for induction of manic or hypomanic episodes after implementation. Two of five individuals who were introduced to sleep restriction developed brief and mild mood elevation in the weeks following. One patient reported that 2 days of mood elevation coincided with psychosocial household changes, and another patient attributed 1 day of mild mood elevation to a new job prospect. For both patients, total sleep time in the week of reported hypomanic symptoms was unchanged (i.e., unaffected by sleep restriction instruction). We offer three possible explanations as to why mood elevation was inconsistently observed after implementation of sleep restriction. First, studies examining a causal link between sleep deprivation and next-day hypomania have relied on total sleep deprivation paradigms (4), and prospective monitoring has shown that a decrease of more than 3 hours in time in bed is associated with elevated mood the following day (29). For our patients, sleep window decreases were generally less than 3 hours, and no patient underwent total sleep deprivation. It is possible, then, that the "dose" of sleep deprivation was insufficient to bring about a manic or hypomanic episode. Alternatively, as sleep restriction improves sleep continuity, it may be the case that any reduction in total sleep time would have been mitigated by the positive effects of more consolidated sleep. Third, setting a sleep window with regular bedtimes and rise times may have led to circadian rhythm entrainment, facilitating mood stabilization (9).

Behavioral intervention with sleep restriction and stimulus control thus appears to be an attractive option for addressing the high rate of sleep disturbance in bipolar disorder.

It is noteworthy that merely setting a sleep schedule in the first session was sufficient to resolve wakefulness at night in a majority of our patients. Accruing evidence suggests that bipolar disorder is characterized by significant schedule variability (10, 11), along with hypothesized sensitivity to schedule disruption and circadian changes (30), and schedule regularity is an established form of treatment in bipolar disorder (31). As a first step in treatment, the patient and practitioner should establish a consistent

bedtime and rise time to be adopted both weekdays and weekends. Compliance with this routine should be reinforced and revisited weekly.

Turning next to stimulus control, we found insufficient evidence that it contributed to the development of manic symptoms. Four of the 15 patients developed hypomanic symptoms during treatment. Although two of the four developed mild hypomania in the week following stimulus control instruction, total sleep time was unchanged for these individuals, and neither reported engaging in arousing or goal-oriented behaviors at night. Of the remaining two patients, one developed mild mood elevation the week before stimulus control was introduced, and the other developed moderate mood elevation 3 weeks after introduction of stimulus control.

Behavioral intervention with sleep restriction and stimulus control thus appears to be an attractive option for addressing the high rate of sleep disturbance in bipolar disorder. Although all participants in our study met diagnostic criteria for insomnia, average total sleep time at baseline was 7.16 hours. Sleep restriction below 6 hours was not warranted. Our strategies may not be as safe in bipolar patients who have insomnia with short sleep duration (32), as there may be a lower limit of sleep restriction before hypomanic symptoms begin to emerge. Even so, evidence suggests that individuals with bipolar disorder display longer sleep durations and more total sleep time than the general population (33, 34) but still experience insomnia at high rates (1). Finally, all our patients were taking psychotropic medications, and we did not exclude individuals who were currently taking hypnotic or other sedating medications for sleep. We acknowledge that changes in medication administration might have influenced mood instability and recommend that patients continue at a steady dosage with psychiatrist approval. We also note that we used a small sample size in this preliminary investigation and recommend that future investigations evaluate stimulus control and sleep restriction over a longer time frame than 8 weeks.

Treatment Considerations

We offer the following recommendations for addressing sleep problems in bipolar disorder without introducing pharmacological agents:

1. Monitor sleep regularly, including time to fall asleep, time awake in the middle of the night, early morning awakenings, and daytime naps. Encourage compliance with a simple sleep diary (35) to set a sleep window and evaluate progress between sessions.

2. Monitor symptoms of depression and mania regularly. Negotiate a safety plan with patient should mood grow unstable during treatment. If symptoms of mania emerge after sleep restriction or stimulus control, consider modifying or temporarily suspending the techniques.

3. Monitor sleepiness regularly using an instrument such as the Epworth Sleepiness Scale. When sleepiness levels reach clinical significance (a score of 10 on the Epworth scale), discourage patients from driving or other potentially unsafe behaviors during periods of drowsiness.

4. Begin by suggesting that the patient adopt a regular sleep schedule across both weekdays and weekends. After 1–2 weeks of this schedule, calculate weekly sleep efficiency with the patient; if sleep efficiency is below recommended guidelines, consider implementing sleep restriction (8).

5. Introduce stimulus control and explain the rationale to patient, underscoring the role of conditioning factors in maintaining insomnia (7, 8). Monitor compliance with stimulus control, along with adverse reaction to stimulus control, in subsequent sessions.

6. Encourage the use of friends, family, and technology to aid in adherence to regularizing bedtime and rise times, sleep restriction, and stimulus control. Setting an alarm as reminder to begin a wind-down period or to wake up at the same time each morning can be helpful for implementation. Likewise, recruiting the support of family and friends to call or visit in the morning so as to prevent oversleeping, or to respect a "no-call" period in the hour before bedtime to promote a relaxing wind-down, can be crucial to the success of these strategies.

7. Encourage a system of regular rewards and positive reinforcement to facilitate behavior change. Establish small daily rewards, like a morning trip to the coffee shop, for complying with treatment recommendations. Highlight successes in sessions rather than failures. For example, if a patient's weekly sleep diaries reveal that naps were taken on 4 of 7 days, underscore the 3 days on which naps were not taken, perhaps doing a functional analysis of how naps were avoided and pointing out positive nighttime sleep parameters (e.g., reduced sleep onset latency or nighttime wakefulness) on nap-free days.

8. Encourage patients to continue using sleep restriction and stimulus control after treatment has ended. Work with patients to review the main components of the tools and anticipate with patients any setbacks to sleep, along with how stimulus control and sleep restriction can be used to prevent the re-emergence of insomnia.

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Clinical Guidance: Behavioral Treatment of Insomnia in Bipolar Disorder

Adopting a consistent sleep schedule throughout the week is the first step in behavioral treatment of insomnia for bipolar patients. It has the added benefit of helping to regulate the patient's daily life, which in itself is an established treatment for bipolar disorder. Many patients' insomnia responds to simple stimulus control, i.e., limiting time in bed to sleep and sex. In treating 15 patients with bipolar disorder, Kaplan and Harvey found that increased manic symptoms did not result from either stimulus control or restriction of sleep time to the amount documented in sleep diaries.