Poor Sleep at Baseline Predicts Worse Mood Outcomes in Patients With Co-Occurring Bipolar Disorder and Substance Dependence

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ABSTRACT

Background: Sleep problems are common in patients with bipolar disorder and have been shown to predict subsequent mood symptoms. Sleep problems have also been shown to lead to worse substance use outcomes in individuals with substance use disorder. However, the relationship between sleep and clinical outcomes in a population with co-occurring bipolar disorder and substance use disorder is unclear.

Method: This secondary analysis included 60 outpatients (mean age = 38.1 years; recruited via advertisements, fliers, clinician referrals, and hospital treatment programs) who met *DSM-IV* criteria for both bipolar disorder and substance use disorder (assessed with the Structured Clinical Interview for *DSM-IV* Axis I Disorders) and who participated in a randomized clinical trial comparing integrated group therapy for bipolar disorder and substance use disorder to group drug counseling for substance use disorder alone. A 12-week treatment period preceded a 24-week follow-up. Poor sleep was assessed with the Pittsburgh Sleep Quality Index, which provides 7 component subscores and an overall sleep score. Data were collected from August 2003 through April 2007.

Results: When analyses were controlled for baseline mood, substance use, and treatment condition, baseline sleep score predicted mood over the course of the 12-week treatment ($\beta = 0.28$; $P < .05$) and 24-week follow-up ($\beta = 0.46$; $P < .01$): worse sleep was associated with worse mood outcomes. Sleep was not associated with substance use outcomes.

Conclusions: Impaired sleep is a prognostic factor for mood outcomes in patients with co-occurring bipolar and substance use disorders. Further investigation is warranted into the long-term clinical outcomes of poor sleep in this population with co-occurring bipolar disorder and substance use disorder so that appropriate interventions can be developed.

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Poor sleep, including problems with falling asleep and sleep duration, is common in patients with bipolar disorder[1–3] or substance use disorders,[4,5] with considerably higher rates of poor sleep in these patients than in the general population.[6–8] Indeed, hypersomnia, insomnia, and decreased need for sleep are included as diagnostic criteria for bipolar disorder.[9]

Sleep quality may play a role in the course of these disorders and affect treatment outcomes. For example, an increase in sleep duration has been correlated with next-day or same-day depressive symptoms,[10,11] and a decrease in sleep duration has been correlated with the onset or exacerbation of manic or hypomanic symptoms.[10–13] Furthermore, Perlman et al[14] reported that a shorter routine sleep duration predicted depression over 6 months. Sleep quality can also affect the course of substance use disorders: poor sleep at baseline[15,16] and during early abstinence[17] has been shown to predict relapse among alcohol-dependent patients.

Substance use disorders occur commonly in individuals with bipolar disorder (60.3% of those with bipolar I disorder and 40.4% of those with bipolar II disorder).[18] Patients with bipolar disorder and a co-occurring substance use disorder have a worse prognosis than those with bipolar disorder alone, including lower remission rates from manic episodes,[19] poorer medication adherence,[19,20] and a higher risk for suicide attempts.[21] Despite the high rates of co-occurrence, the worse prognosis, and the evidence that poor sleep in both bipolar disorder and substance use disorder predicts worse outcomes, no studies have examined the quality of sleep and its potential prognostic implications in patients with co-occurring bipolar disorder and substance use disorder.

In our present study, part of a larger overall study of a new integrated group therapy,[22] 60 patients with co-occurring bipolar disorder and substance dependence were asked about their sleep quality, substance use, and mood throughout 12 weeks of treatment and the 24 weeks following treatment completion. We hypothesized that most patients would have poor sleep and that poor sleep at baseline would predict worse mood outcomes and worse substance use outcomes.

METHOD

Patients

Sixty outpatients, all of whom had co-occurring *DSM-IV* bipolar disorder and substance use disorder, were participating in a larger study[22] (clinicaltrials.gov Identifier: NCT00227838) comparing 2 forms of group treatment given in 12 weekly hour-long sessions: integrated group therapy[23] and standard group drug counseling.[24] Integrated group therapy, designed specifically for patients with co-occurring bipolar disorder and substance use disorder, is based on a cognitive-behavioral model that integrates treatment for the 2 disorders by focusing on similarities in the recovery process in bipolar and substance use disorders. Group drug counseling is a standardized treatment designed to approximate the treatment patients receive in a substance use disorder community treatment program; group drug counseling has been empirically validated and is used routinely in clinical research.[25] Participants were randomized to either group drug counseling

Clinical Points

- Sleep disturbances in patients with co-occurring bipolar disorder and substance use disorder are extremely common.
- Evaluation of sleep disturbances at baseline may be useful as a prognostic factor for mood outcomes in patients with co-occurring bipolar and substance use disorders; patients with worse sleep problems have poorer outcomes.

or integrated group therapy. The 12-week treatment preceded a 24-week follow-up. Data were collected from August 2003 through April 2007. The results of that study[22] showed that integrated group therapy outperformed group drug counseling: patients in integrated group therapy reported significantly higher rates of total abstinence than patients in group drug counseling. Patients in integrated group therapy also reported fewer days of substance use during follow-up and were less likely to have mood episodes during treatment compared to patients in group drug counseling.

Participants were recruited via advertisements, fliers, clinician referrals, and McLean Hospital treatment programs in Belmont, Massachusetts. Inclusion criteria were (1) current diagnoses of bipolar disorder and substance dependence other than nicotine, (2) substance use within 60 days of intake, (3) a mood stabilizer regimen for ≥ 2 weeks, and (4) ≥ 18 years of age. Exclusion criteria were (1) current psychosis, (2) current danger to self or others, (3) concurrent group treatment, and (4) residential treatment restricting substance use. The McLean Hospital Institutional Review Board approved this research protocol. Written informed consent was obtained from each patient at the initial appointment after research staff reviewed the study procedures with the patient. Table 1 shows a description of the sample.

Table 1. Sample Description at Baseline (N = 60)

Baseline Characteristic	Baseline Data
Age, mean (SD) (range), y	38.1 (11.1) (18–65)
Sex, male, %	58.3
Race, white, %	91.7
Not currently married, %	71.7
Education < college, %	51.7
Not employed, %	55.0
Bipolar disorder type, %	
Bipolar I	78.3
Bipolar II	15.0
Bipolar not otherwise specified	6.7
Lifetime substance dependence, %	
Drugs and alcohol	65.0
Alcohol only	26.7
Drugs only	8.3
Duration of illness, mean (SD) (range), lifetime y[a]	
Bipolar disorder	18.0 (11.9) (1–45)
Alcohol dependence	16.7 (11.3) (< 1–43)
Drug dependence	15.8 (10.3) (1–33)
Mood stabilizer treatment, %	
Valproate	30.0
Lithium	28.3
Olanzapine	18.3
> 1 drug	34.4
Other current treatment, %	
Individual psychotherapy	65.0
12-Step group attendance	58.3

[a]The Structured Clinical Interview for *DSM-IV* Axis I Disorders duration data were incomplete for bipolar disorder (n = 13), alcohol dependence (n = 1), and drug dependence (n = 2).

Assessments

Sleep. Participants completed the Pittsburgh Sleep Quality Index (PSQI)[26] at baseline and every 12 weeks thereafter for 36 weeks. The PSQI, a self-report measure designed to characterize sleep in clinical populations, has 7 components, each with scores ranging from 0 (no dysfunction) to 3 (severe dysfunction); the total global score (0–21) is the sum. The components are (1) subjective sleep quality, (2) sleep latency, (3) sleep duration, (4) habitual sleep efficiency, (5) sleep disturbances, (6) use of sleeping medication, and (7) daytime dysfunction. The components and items demonstrate strong internal consistency, and global and component scores are stable over time.[26] Although there is limited evidence of correlation with polysomnography, the scale has been repeatedly validated to identify those with insomnia.[26–28] Defining *poor sleep* as a global PSQI score > 5, as recommended,[26] has discriminated those with good sleep (controls) from those with poor sleep among patients with primary insomnia or psychiatric disorders, although the optimal cutoff ranged from 5.5 to 6.0.[27,28] The PSQI has been used across diverse populations and was the only sleep measure of 22 assessed[29] that met the standards for reliability, validity, responsiveness, and interpretability. Sixty of the 61 participants in our study had valid PSQI data at baseline; therefore, the sample considered here comprises these 60 participants.

Mood. Participants completed the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID)[30] and the Longitudinal Interval Follow-Up Evaluation.[31] The SCID was administered at baseline, at the end of treatment, and 12 weeks after treatment; the Longitudinal Interval Follow-Up Evaluation was administered every 4 weeks, both during treatment and during the 24 weeks after treatment. Participants were considered to be ill during a particular week if they met criteria for a depressive, manic, hypomanic, or mixed mood syndrome.

Substance use. Participants were assessed monthly with the fifth edition of the Addiction Severity Index,[32] using the timeline follow-back method[33] to determine the number of days of substance use. Substance use disorder diagnoses were assigned using the SCID.

Data Analysis

In this secondary-analysis study, linear regression analysis was used to examine the effects of baseline sleep scores (ie, overall sleep scale) on mood and substance use outcomes, adjusted for treatment condition and baseline measures of mood (ie, number of weeks ill with bipolar disorder in the past 4 weeks) and substance use (ie, number of days of substance use in the past 30 days); these models considered the 12-week treatment period separately from the 24-week

follow-up period and pooled the repeated measures of the outcomes for each period into a single post-baseline summary outcome. Next, a complementary set of longitudinal analyses examined the effects of baseline sleep scores on mood and substance use over the combined 36 weeks postbaseline, using generalized estimating equations to account for the correlation among the repeated measures of the outcomes; these analyses were adjusted for the same covariates described above. Analysis adjusted for gender and age showed that neither variable was significantly related to either mood or substance use; hence, data are reported without adjustment for these variables. Data were analyzed with SPSS, version 17.0 (SPSS Inc, Chicago, Illinois).

Table 2. Distribution of Component Scores of the Pittsburgh Sleep Quality Index (N = 60[a])

Component	Score of 0, %	Score of 1, %	Score of 2, %	Score of 3, %	Mean Score (SD)
Use of sleep medication	28.8	8.5	8.5	54.2	1.9 (1.3)
Sleep latency	11.7	36.7	25.0	26.7	1.7 (1.0)
Daytime dysfunction	5.0	43.3	40.0	11.7	1.6 (0.8)
Sleep disturbances	5.0	38.3	46.7	10.0	1.6 (0.7)
Subjective sleep quality	20.3	40.7	35.6	3.4	1.2 (0.8)
Sleep duration	45.0	25.0	21.7	8.3	0.9 (1.0)
Habitual sleep efficiency	61.0	23.7	6.8	8.5	0.6 (0.9)

[a]Due to missing items, N = 59 for 3 components: use of sleep medication, subjective sleep quality, and habitual sleep efficiency.

Table 3. Number of Weeks With a Mood Episode and Days of Substance Use by Sleep Score and Other Baseline Characteristics[a]

	No. of Weeks With Mood Episode		Days of Substance Use	
Baseline Characteristic	Treatment, β (N = 58)	Follow-Up, β (N = 51)	Treatment, β (N = 60)	Follow-Up, β (N = 54)
Sleep score	0.28*	0.46**	0.15	−0.60
Number of weeks ill	0.40	−0.10	−2.41	−1.96
Days of substance use	0.03	−0.03	1.07**	0.97*
Treatment condition	0.98	−0.19	4.27	3.52
Adjusted R^2, %	14.4*	15.8*	15.8**	10.0

[a]Coefficients (βs) for follow-up (based on a 24-week interval) were divided by 2 for ease of comparison with βs for treatment (based on a 12-week interval).
*$P < .05$, **$P < .01$.

RESULTS

Do Patients With Co-Occurring Bipolar Disorder and Substance Dependence Have Poor Sleep?

The mean PSQI score at baseline was 9.6 (standard deviation [SD] = 4.1) on a scale from 0 to 21, with higher scores indicating worse sleep. When we used the clinical cutoff score of > 5 to distinguish those with poor sleep from those with good sleep,[26] 52 of the 60 patients (86.7%) had poor sleep (Table 2 shows the distribution of scores). For each component other than habitual sleep efficiency, most participants (55.0%–95.0%, n = 33–57) scored greater than 0, indicating some dysfunction. Almost all participants reported sleep disturbances, daytime dysfunction, and delayed sleep-onset latency in the past month, with most indicating moderate to severe difficulty (a score of 2 or 3). Most patients also indicated difficulty with subjective sleep quality and sleep duration. The use of sleep medications was bimodally distributed, with most patients reporting the use of sleep medications 3 or more times a week (a score of 3), about a quarter using no sleep medications in the past month (a score of 0), and a few using them once or twice a week (a score of 1 or 2).

Does Poor Sleep at Baseline Predict Worse Mood Outcomes?

Seventy percent (n = 42) of patients had at least 1 week of illness with bipolar disorder (depressed, hypomanic, manic, or mixed) in the 4 weeks before baseline, with a mean of 2.3 of 4 weeks of illness (SD = 1.7 weeks). We collected mood data for all 12 weeks of treatment from 59 patients (98.3%) and collected all 24 weeks of posttreatment follow-up data from 52 patients (86.7%). Patients reported a mean of 1.2 weeks of illness with bipolar disorder per 4 weeks (SD = 1.2 weeks) during treatment and 1.1 weeks of illness per 4 weeks (SD = 1.2 weeks) during the posttreatment follow-up period.

In analyses that considered the 12-week treatment period separately from the 24-week follow-up period, baseline sleep score predicted the number of weeks ill with bipolar disorder during treatment and during follow-up; these analyses also adjusted for number of weeks ill with bipolar disorder in the 4 weeks before baseline, number of days of substance use in the 30 days before baseline, and treatment condition. Specifically, those participants with worse sleep had more weeks of illness with bipolar disorder (Table 3); interestingly, the effect of baseline sleep scores was 1.6 times higher at follow-up compared to treatment (β = 0.28 for treatment and β = 0.46 for follow-up). For example, a 3-point increase in the baseline sleep score was associated with approximately 1 additional week of illness with bipolar disorder ($3 \times 0.28 = 0.84$) during treatment and approximately 1.5 weeks of illness with bipolar disorder ($3 \times 0.46 = 1.38$) during follow-up. (A 3-point change in sleep score, equivalent to the range of each of the 7 components, can be considered a clinically meaningful increase.) When this analysis was repeated for each PSQI component, 3 of the 7 components were found to significantly predict weeks of illness with bipolar disorder during treatment and follow-up: subjective sleep quality, sleep latency, and sleep duration at baseline; the remaining components were not significantly related to bipolar disorder.

Next, a complementary set of longitudinal analyses were used to predict the probability of a week of illness with bipolar disorder during the 36 weeks postbaseline. In logistic regression analysis of the probability of a week of illness with bipolar disorder, baseline sleep score, adjusted for the remaining baseline predictors, was significantly related to mood. Specifically, each 3-point increase in sleep score at baseline was associated with a 60% greater likelihood of a week of illness with bipolar disorder during the 36 weeks postbaseline (estimated odds ratio [OR] = 1.6, $P = .01$). That is, at any of the 36 weeks postbaseline, those with baseline

sleep scores 3 points higher were approximately 60% more likely to have an additional week of illness with bipolar disorder.

Does Poor Sleep at Baseline Predict Worse Substance Use Outcomes?

To predict substance use outcomes across the 36 weeks (12 weeks of treatment and 24 weeks of follow-up), we employed a procedure similar to that for mood: baseline sleep score, number of weeks of illness with bipolar disorder in the 4 weeks before baseline, number of days of substance use before baseline, and treatment condition were examined. In contrast to mood outcomes, baseline sleep score was not related to days of substance use during treatment or during the follow-up period; only the number of days of substance use at baseline was related to number of days of substance use during these 2 time periods, with greater use at baseline related to greater use postbaseline (see Table 3). This analysis was repeated for each PSQI component; as expected, none of the components were significant predictors of subsequent substance use. Complementary longitudinal analysis to predict days of substance use over the 36 weeks postbaseline also showed that baseline sleep score was not related to the number of days of substance use postbaseline. Finally, in additional analyses, number of days of alcohol use and number of days of drug use were considered separately, using the linear-regression and generalized-estimating-equation analyses; sleep scores at baseline were not related to either outcome (data not shown).

DISCUSSION

Overview

The aim of this study was to report the characteristics of sleep in patients with co-occurring bipolar and substance use disorders and to examine the association between poor sleep at baseline and subsequent mood and substance use outcomes. We found that poor sleep at baseline was quite prevalent in our sample and was associated with worse mood outcomes but not worse substance use outcomes during treatment and 24 weeks of posttreatment follow-up. Specifically, the sleep components of subjective sleep quality, sleep latency, and sleep duration at baseline were associated with subsequent weeks of illness with bipolar disorder.

Sleep Scores

More than twice as many patients with co-occurring bipolar and substance use disorders met the clinical cutoff for poor sleep compared to a general US population sample (87% vs 36%[8]). This finding indicates that individuals with co-occurring bipolar and substance use disorders appear to have more dysfunctional sleep than the general population. This association is not surprising; poor sleep is a criterion for a diagnosis of bipolar disorder,[9] and chronic substance abuse is associated with sleep disturbances.[15–17,34]

The rates of poor sleep in this sample are comparable to rates for patients with bipolar disorder only or substance use disorder only, in both the proportion of patients meeting criteria for poor sleep[1–3,34–37] and the mean PSQI score.[16,35,38] Since estimates for the proportion of those with poor sleep among patients with bipolar disorder or substance use disorder alone are already so high, a ceiling effect could limit the likelihood that co-occurrence of these disorders would elevate the rates of poor sleep much further.

The PSQI requires patients to fill out the average value for each question. For example, a participant is asked, "How long (in minutes) has it usually taken you to fall asleep each night?" A person who sometimes falls asleep right away but at other times takes hours to fall asleep might thus select an answer in the middle. This average answer can mask the variability of a patient's sleep difficulty. This variability is supported by previous research, which has shown that individuals with bipolar disorder have more variable sleep duration[3] and circadian rhythm than controls, even when the 2 groups do not differ on sleep measures.[39]

Treatment Outcomes

Our findings indicate that poorer subjective sleep quality, as measured by the PSQI, is associated with worse mood outcomes during and after treatment for co-occurring bipolar and substance use disorders, even while baseline mood was not; baseline sleep scores, however, were not associated with substance use outcomes.

Although several studies have shown that sleep duration in bipolar patients is correlated with next-day mood symptoms,[10–13] we are aware of only 1 study that examined how usual sleep patterns affect bipolar disorder outcomes: Perlman and colleagues[14] reported that shorter sleep duration was correlated with the severity of depressive symptoms in bipolar patients across a 6-month follow-up period after partial recovery when analyses were controlled for baseline depression. Our study found comparable results, in that poorer sleep, as measured by the PSQI, predicted worse mood outcomes during 12 weeks of treatment and 24 weeks of follow-up.

The lack of association between baseline sleep score and alcohol use, drug use, or total substance use during treatment and follow-up was surprising, as the effect of sleep quality on substance use disorder outcomes is more robust, particularly for alcohol use. Greater subjective sleep onset latency,[15,16,40] shorter subjective total sleep time,[40] and subjective estimates of awakening after sleep onset[40] have all predicted relapse either during or after treatment for alcohol dependence. One reason for the lack of correlation between sleep and substance use outcomes may be that our sample had a mix of alcohol and drug dependence, while most of the strongest evidence of a relationship between baseline sleep and substance use outcomes has been related to alcohol dependence. For other drugs, evidence that sleep affects outcomes is more limited and shows mixed results.[5,41] This explanation is limited, however, by the fact that we found no correlation between poor sleep at baseline and alcohol use, drug use, or overall substance use outcomes postbaseline. Our failure to find such an association may be related to the fact that the

current sample scored better on some PSQI components (eg, sleep quality, sleep duration, and sleep efficiency) than what was reported in previous studies among patients with alcohol dependence.[16,35]

Strengths and Limitations

One strength of this study is that sleep has not been previously examined in patients with co-occurring bipolar and substance use disorders. Given the high prevalence of co-occurrence and the rates of dysfunctional sleep in each disorder, understanding the effect that sleep has in this dual-diagnosis population is important.

The fact that the PSQI is a self-report and not an objective measure of sleep difficulty can be considered both a strength and a limitation of this research since its creators[26] found only a weak relationship between the PSQI and polysomnographic measures of sleep. Previous studies that utilized both subjective and objective measures found similar results. For example, both polysomnographic and subjective sleep latency predicted alcohol use outcomes in 1 study.[15] Even when there is a discrepancy between subjective and objective reports, subjective reports are sometimes better predictors of substance use outcomes than are objective measures.[40] Because subjective sleep measures are easier to administer than polysomnography or actigraphy and because they remain predictive of outcomes of interest (sometimes more so than objective measures), the PSQI appears to be a clinically useful tool to measure sleep in this population.

Another limitation of the PSQI is that it conflates answers across nights. For example, a patient who sleeps 4 hours per night half the time and 10 hours per night half the time can answer certain questions like someone who sleeps 7 hours every night. Since bipolar patients are distinguished from controls in part by the variability of their circadian rhythms[39] and sleep duration,[2] and because next-day mood in patients with bipolar disorder has been shown to be influenced by sleep duration the night before,[10–13] specific nights or clusters of nights may be responsible for some of the mood outcomes in our study. Perhaps variability of sleep across nights—not just the mean measures—may contribute to worse outcomes.

Future Research

Given the impact of poor sleep on long-term mood outcomes, future studies may examine the effect of sleep interventions on clinical outcomes in this population. It is unclear whether improved sleep would have a beneficial effect on mood outcomes.

Drug names: lithium (Lithobid and others), olanzapine (Zyprexa), valproate (Depacon and others).

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REFERENCES

1. Harvey AG, Schmidt DA, Scarnà A, et al. Sleep-related functioning in euthymic patients with bipolar disorder, patients with insomnia, and subjects without sleep problems. *Am J Psychiatry*. 2005;162(1):50–57.
2. Millar A, Espie CA, Scott J. The sleep of remitted bipolar outpatients: a controlled naturalistic study using actigraphy. *J Affect Disord*. 2004;80(2–3):145–153.
3. Saiz-Ruiz J, Cebollada A, Ibañez A. Sleep disorders in bipolar depression: hypnotics vs sedative antidepressants. *J Psychosom Res*. 1994;38(suppl 1): 55–60.
4. Burke CK, Peirce JM, Kidorf MS, et al. Sleep problems reported by patients entering opioid agonist treatment. *J Subst Abuse Treat*. 2008; 35(3):328–333.
5. Oyefeso A, Sedgwick P, Ghodse H. Subjective sleep-wake parameters in treatment-seeking opiate addicts. *Drug Alcohol Depend*. 1997;48(1):9–16.
6. LeBlanc M, Mérette C, Savard J, et al. Incidence and risk factors of insomnia in a population-based sample. *Sleep*. 2009;32(8):1027–1037.
7. Morphy H, Dunn KM, Lewis M, et al. Epidemiology of insomnia: a longitudinal study in a UK population. *Sleep*. 2007;30(3):274–280.
8. Roth T, Jaeger S, Jin R, et al. Sleep problems, comorbid mental disorders, and role functioning in the National Comorbidity Survey Replication. *Biol Psychiatry*. 2006;60(12):1364–1371.
9. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition. Washington, DC: American Psychiatric Association; 1994.
10. Bauer M, Glenn T, Whybrow PC, et al. Changes in self-reported sleep duration predict mood changes in bipolar disorder. *Psychol Med*. 2008;38(7):1069–1071.
11. Bauer M, Grof P, Rasgon N, et al. Temporal relation between sleep and mood in patients with bipolar disorder. *Bipolar Disord*. 2006;8(2): 160–167.
12. Barbini B, Bertelli S, Colombo C, et al. Sleep loss, a possible factor in augmenting manic episode. *Psychiatry Res*. 1996;65(2):121–125.
13. Leibenluft E, Albert PS, Rosenthal NE, et al. Relationship between sleep and mood in patients with rapid-cycling bipolar disorder. *Psychiatry Res*. 1996;63(2–3):161–168.
14. Perlman CA, Johnson SL, Mellman TA. The prospective impact of sleep duration on depression and mania. *Bipolar Disord*. 2006;8(3):271–274.
15. Brower KJ, Aldrich MS, Hall JM. Polysomnographic and subjective sleep predictors of alcoholic relapse. *Alcohol Clin Exp Res*. 1998;22(8): 1864–1871.
16. Foster JH, Peters TJ. Impaired sleep in alcohol misusers and dependent alcoholics and the impact upon outcome. *Alcohol Clin Exp Res*. 1999; 23(6):1044–1051.
17. Brower KJ, Aldrich MS, Robinson EA, et al. Insomnia, self-medication, and relapse to alcoholism. *Am J Psychiatry*. 2001;158(3):399–404.
18. Merikangas KR, Akiskal HS, Angst J, et al. Lifetime and 12-month prevalence of bipolar spectrum disorder in the National Comorbidity Survey Replication. *Arch Gen Psychiatry*. 2007;64(5):543–552.
19. Goldberg JF, Garno JL, Leon AC, et al. A history of substance abuse complicates remission from acute mania in bipolar disorder. *J Clin Psychiatry*. 1999;60(11):733–740.
20. Perlis RH, Ostacher MJ, Miklowitz DJ, et al. Clinical features associated with poor pharmacologic adherence in bipolar disorder: results from the STEP-BD study. *J Clin Psychiatry*. 2010;71(3):296–303.
21. Dalton EJ, Cate-Carter TD, Mundo E, et al. Suicide risk in bipolar patients: the role of co-morbid substance use disorders. *Bipolar Disord*. 2003;5(1):58–61.
22. Weiss RD, Griffin ML, Jaffee WB, et al. A "community-friendly" version of integrated group therapy for patients with bipolar disorder and substance dependence: a randomized controlled trial. *Drug Alcohol Depend*. 2009;104(3):212–219.
23. Weiss RD, Connery HC. *Integrated Group Therapy for Bipolar Disorder and Substance Abuse*. New York, NY: Guilford Press; 2011.
24. Daley DC, Mercer D, Carpenter G. *Group Drug Counseling for Cocaine Dependence*. Rockville, MD: US Department of Health and Human Services; 2002.
25. Crits-Christoph P, Siqueland L, Blaine J, et al. Psychosocial treatments for cocaine dependence: National Institute on Drug Abuse Collaborative Cocaine Treatment Study. *Arch Gen Psychiatry*. 1999;56(6):493–502.
26. Buysse DJ, Reynolds CF 3rd, Monk TH, et al. The Pittsburgh Sleep

Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;28(2):193–213.
27. Backhaus J, Junghanns K, Broocks A, et al. Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *J Psychosom Res*. 2002;53(3):737–740.
28. Doi Y, Minowa M, Uchiyama M, et al. Psychometric assessment of subjective sleep quality using the Japanese version of the Pittsburgh Sleep Quality Index (PSQI-J) in psychiatric disordered and control subjects. *Psychiatry Res*. 2000;97(2–3):165–172.
29. Devine EB, Hakim Z, Green J. A systematic review of patient-reported outcome instruments measuring sleep dysfunction in adults. *Pharmacoeconomics*. 2005;23(9):889–912.
30. First M, Spitzer M, Gibbon M, et al. *Structured Clinical Interview for* DSM-IV *Axis I Disorders, Research Version (Patient Edition)*. New York, NY: Biometrics Research, New York State Psychiatric Institute; 1996.
31. Keller MB, Lavori PW, Friedman B, et al. The Longitudinal Interval Follow-Up Evaluation: a comprehensive method for assessing outcome in prospective longitudinal studies. *Arch Gen Psychiatry*. 1987;44(6): 540–548.
32. McLellan AT, Kushner H, Metzger D, et al. The fifth edition of the Addiction Severity Index. *J Subst Abuse Treat*. 1992;9(3):199–213.
33. Sobell LC, Sobell MB. Timeline follow-back: a technique for assessing self-reported alcohol consumption. In: Litten RZ, Allen JP, eds. *Measuring Alcohol Consumption: Psychosocial and Biochemical Methods*. Totowa, NJ: Humana Press; 1992: 41–72.
34. Stein MD, Herman DS, Bishop S, et al. Sleep disturbances among methadone maintained patients. *J Subst Abuse Treat*. 2004;26(3): 175–180.
35. Cohn TJ, Foster JH, Peters TJ. Sequential studies of sleep disturbance and quality of life in abstaining alcoholics. *Addict Biol*. 2003;8(4): 455–462.
36. Mahfoud Y, Talih F, Streem D, et al. Sleep disorders in substance abusers: how common are they? *Psychiatry (Edgmont)*. 2009;6(9):38–42.
37. Peles E, Schreiber S, Adelson M. Variables associated with perceived sleep disorders in methadone maintenance treatment (MMT) patients. *Drug Alcohol Depend*. 2006;82(2):103–110.
38. Endicott J, Paulsson B, Gustafsson U, et al. Quetiapine monotherapy in the treatment of depressive episodes of bipolar I and II disorder: improvements in quality of life and quality of sleep. *J Affect Disord*. 2008;111(2–3):306–319.
39. Jones SH, Hare DJ, Evershed K. Actigraphic assessment of circadian activity and sleep patterns in bipolar disorder. *Bipolar Disord*. 2005;7(2): 176–186.
40. Conroy DA, Todd Arnedt J, Brower KJ, et al. Perception of sleep in recovering alcohol-dependent patients with insomnia: relationship with future drinking. *Alcohol Clin Exp Res*. 2006;30(12):1992–1999.
41. Coffey SF, Dansky BS, Carrigan MH, et al. Acute and protracted cocaine abstinence in an outpatient population: a prospective study of mood, sleep and withdrawal symptoms. *Drug Alcohol Depend*. 2000;59(3): 277–286.