



Research paper

Abnormal sleep duration associated with hastened depressive recurrence in bipolar disorder



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ABSTRACT

Background: Abnormal sleep duration (ASD, < 6 or ≥ 9 h) is common in bipolar disorder (BD), and often persists beyond acute mood episodes. Few longitudinal studies have examined the ASD's impact upon BD illness course. The current study examined the longitudinal impact of ASD upon bipolar depressive recurrence/recovery.

Methods: Outpatients referred to the Stanford BD Clinic during 2000–2011 were assessed with the Systematic Treatment Enhancement Program for BD (STEP-BD) Affective Disorders Evaluation at baseline, and with the Clinical Monitoring Form at monthly follow-ups for up to two years of naturalistic treatment. Prevalence and clinical correlates of ASD in 93 recovered (euthymic ≥ 8 weeks) and 153 depressed BD patients were assessed. Kaplan-Meier analyses (Log-Rank tests) assessed relationships between baseline ASD and longitudinal depressive severity, with Cox Proportional Hazard analyses assessing potential mediators.

Results: ASD was only half as common among recovered versus depressed BD outpatients, but was significantly associated with hastened depressive recurrence (Log-Rank $p = 0.007$), mediated by lifetime anxiety disorder and attenuated by lifetime history of psychosis, and had only a non-significant tendency towards association with delayed depressive recovery (Log-Rank $p = 0.07$). In both recovered and depressed BD outpatients, baseline ASD did not have significant association with any baseline BD illness characteristic.

Limitations: Self-reported sleep duration. Limited generalizability beyond our predominately white, female, educated, insured American BD specialty clinic sample.

Conclusions: Baseline ASD among recovered BD patients may be a risk marker for hastened depressive recurrence, suggesting it could be an important therapeutic target between mood episodes.

1. Introduction

Bipolar disorder (BD) is a severe and recurrent disorder that affects approximately 2.4% of the U.S. population, and entails a significant public health burden (Merikangas et al., 2007, 2011). Although mood elevation (mania or hypomania) is the defining aspect of bipolar disorder, depression is more pervasive (Judd et al., 2003, 2002). Despite advances in the treatment of BD, the risk of recurrence remains high (Gitlin et al., 1995; Goldberg et al., 1995; Perlis et al., 2006). Findings from both naturalistic studies and randomized clinical trials, indicate that one third to one half of recovered BD patients will experience a new mood episode within two years, even while receiving pharmacotherapy (Vazquez et al., 2015). Furthermore, BD is associated with substantive residual symptoms and persistent functional impairment between episodes (Altshuler et al., 2006; Coryell et al., 1993; Goldberg and Harrow, 2011; Joffe et al., 2004; MacQueen et al., 2003;

Tohen et al., 2003; Vieta et al., 2008). These findings underscore the importance of identifying factors associated with recurrence risk and interepisode functional impairment. Identification of such factors could help lead to more effective treatments that lower or prevent vulnerability to recurrence and illness burden.

A considerable body of literature demonstrates the important role of disturbed sleep in BD (Harvey, 2008). Sleep disturbance is common in both depression and mood elevation. During depression individuals often experience hypersomnia or insomnia, whereas during mood elevation, individuals commonly experience insomnia or reduced need for sleep (DSM-5; American Psychiatric Association, 2013). Furthermore, research has shown that sleep disturbances persist between mood episodes in BD (Cretu et al., 2016; Gershon et al., 2012; Gruber et al., 2009; Harvey et al., 2005), contributing to both residual symptoms and functional impairment (Cretu et al., 2016; Gruber et al., 2009).

It has long been proposed that disturbed sleep may trigger episode

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recurrence in BD (Goodwin and Jamison, 1990; Wehr et al., 1987). Although there has been an extensive body of cross-sectional studies on sleep in bipolar disorder (Asaad et al., 2016; Banks et al., 2016; Baroni et al., 2012; Brill et al., 2011; Karthick et al., 2015; Rocha et al., 2013; Roybal et al., 2011; Russo et al., 2015; St-Amand et al., 2013; Steinar et al., 2016a; Walz et al., 2013), only a few longitudinal studies have examined the impact of sleep disturbances on BD illness course. In one such study, shorter sleep duration predicted worsening of depressive, but not manic symptoms, across a 6-month follow-up period in 54 adults with bipolar I disorder (BD-I) (Perlman et al., 2006). In another longitudinal study, 59 adults with BD provided daily reports of their mood, sleep duration and time in bed over 5 months. This study found shorter sleep duration or time in bed predicted increased next-day self-reported mania and longer sleep duration or time in bed predicted increased next-day self-reported depression (Bauer et al., 2006).

In addition, three longitudinal studies examined relationships between sleep and illness course among patients in the National Institute of Mental Health-funded Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). First, Gruber et al. (2011) found that among adults with bipolar spectrum disorders (N=196), who had recovered clinical status at study entry, reduced sleep duration at study entry was associated with increased manic symptoms, and more variable sleep duration was associated with increased manic and depressive symptoms, over a 12-month assessment period (Gruber et al., 2011). Second, Sylvia et al. (2012) found that reduced sleep duration, as measured using the sleep item from the Montgomery-Asberg Depression Rating Scale (MADRS) (Montgomery and Asberg, 1979), was associated with increased risk of mood episode recurrence among 483 recovered adults with BD-I and bipolar II disorder (BD-II) (Sylvia et al., 2012). Third, Cretu et al. (2016) found that among 89 recovered patients with bipolar spectrum disorders, assessed when recovered using the Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989), those classified as “poor sleepers” according to PSQI global score (i.e., with score > 5) had earlier mood episode recurrence over a one-year follow-up period (Cretu et al., 2016). Furthermore, the authors found that the relationship between poor sleep and episode recurrence was independent of residual mood symptoms, suggesting that poor sleep could represent an independent recurrence risk factor in BD. Taken together, these investigations suggested reduced sleep duration or quality could be important independent predictors of mood symptoms and episodes in BD.

The aim of the present study was to investigate the impact of abnormal sleep duration (ASD) on episode recurrence and recovery in a large, well-characterized sample of BD-I and BD-II outpatients. Although a handful of prior longitudinal studies have examined the impact of sleep duration on illness course in BD, all but one (Sylvia et al., 2012) followed patients for relatively short time periods (5–12 months). Sylvia et al. (2012) assessed sleep using an item drawn from a depression scale, which assessed specifically for reduced sleep duration. The present study examined impact of ASD upon BD illness course, assessing the effects of both long and short sleep duration.

2. Method

We included outpatients with BD-I or BD-II referred by community practitioners (primarily psychiatrists) to the Stanford University BD Clinic between 2000 and 2011. Patients were assessed with the Systematic Treatment Enhancement Program for BD (STEP-BD) Affective Disorders Evaluation (Sachs et al., 2003), which included the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (First et al., 1996) mood disorders module and Clinical Global Impression-Bipolar Version-Overall Severity (CGI-BP-OS) score (Spearing et al., 1997). The Mini International Neuropsychiatric Interview (MINI) (Sheehan et al., 1998) was used to confirm bipolar and comorbid psychiatric disorder diagnoses. Clinical status at each follow-up visit was determined by

symptom ratings on the STEP-BD Clinical Monitoring Form (CMF) (Sachs et al., 2002) while patients received measurement- and guideline-based naturalistic treatment (with monthly modal visit frequency) for up to two years.

Baseline ASD (mean of maximum and minimum duration in week prior to enrollment < 6 h or ≥ 9 h) was determined from available medical records and patient report, as assessed by the STEP-BD Affective Disorders Evaluation. Current mood symptoms were determined from patient report, as assessed by the STEP-BD Affective Disorders Evaluation at the time of enrollment, and clinician observation and reflected any mood symptoms in the ten days prior to enrollment for the primary analysis, and mood symptoms thresholded for occurring on at least four or seven of the ten days prior to enrollment for the secondary analysis.

As described below, clinical characteristics of participants were evaluated and prospective clinical course of participants meeting diagnostic criteria for either current recovery (euthymic ≥ 8 weeks) or depression (a current major depressive episode) at enrollment were assessed. The STEP-BD protocol and the subsequent similar Stanford-specific Assessment, Monitoring, and Centralized Database protocol were approved by the Stanford University Administrative Panel on Human Subjects, and patients provided verbal and written informed consent prior to participation. Trained medical and research staff collected data on 6 demographic parameters and 19 illness characteristics/current mood symptoms. The demographic parameters assessed were: (A) Age (in years); (B) Gender; (C) Race/Ethnicity; (D) Education; (E) Marital Status; and (F) Employment status. The illness characteristics/current mood symptoms/current psychotropic medications assessed were: (1) Lifetime anxiety disorder; (2) Lifetime alcohol/substance use disorder; (3) Lifetime eating disorder; (4) Lifetime personality disorder; (5) Bipolar II Disorder; (5 A) Lifetime psychosis (which is very commonly associated with Bipolar I Disorder); (5B) Lifetime prior psychiatric hospitalization (which is also very commonly associated with Bipolar I Disorder); (6) ≥ One first-degree relative with mood disorder; (7) Onset age (in years); (8) Childhood (age < 13 years) onset; (9) Illness duration (in years); (10) Long illness duration (≥ 15 years); (11) Episode accumulation (≥ 10 prior mood episodes); (12) Lifetime suicide attempt; (13) Rapid cycling (≥ 4 episodes) in prior year; (14) CGI-BP-OS; current (i.e., any in the prior 10 days); and (15) Sadness; (16) Anhedonia; (17) Euphoria; (18) Irritability; and (19) Anxiety.

Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) Version 23.0 software (IBM Corp.; Armonk, NY) on an Apple MacBook Air computer (Apple Corporation, Cupertino, CA). Prevalence and clinical correlates of baseline ASD were examined in currently recovered (i.e., euthymic ≥ 8 weeks) and currently depressed (i.e., with a current major depressive episode) patients. Analytical baseline statistics included Fisher's Exact Test comparisons of categorical data and independent-sample *t*-test comparisons of continuous variables. Primary longitudinal analyses consisted of Kaplan-Meier survival analyses (Log-Rank tests), which compared times to recurrence and recovery in patients with versus without baseline ASD. We used the standard approaches of censoring patients with mood elevation prior to depressive recurrence in assessing time to depressive recurrence, and censoring patients with depressive prior to mood elevation recurrence in assessing time to mood elevation recurrence (Tohen et al., 1990). Secondary metrics included for patients with versus without baseline ASD were Kaplan-Meier estimated recurrence/recovery rates for significant longitudinal depressive associations. Additional secondary analyses included Cox proportional hazard analyses (hazard ratios (HRs) and 95% confidence intervals (CIs)) for recurrence and recovery, as well as for potential mediators of statistically significant longitudinal illness findings. To select parameters for entry into mediator models, univariate Cox proportional hazard analyses were performed for all 25 (6 demographic and 19 clinical) assessed parameters. Parameters with $p < 0.05$ were entered into a

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