Published in final edited form as:

Psychiatry Res. 2012 December 30; 200(2-3): 265–271. doi:10.1016/j.psychres.2012.07.037.

Sleep impairment, mood symptoms, and psychosocial functioning in adolescent bipolar disorder

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Abstract

Few empirical studies have investigated the role of sleep impairment in the course of adolescent bipolar spectrum disorders (BSD). The present study examined the longitudinal associations between sleep disruption, mood symptom severity, and psychosocial functioning in a 2-year follow-up of patients with adolescent BSD. Fifty-three adolescents with BSD (mean [SD] age: 14.6 [1.6]) participated in a two-site randomized trial of family focused treatment for adolescents (FFT-A) or enhanced care, a briefer psychoeducational treatment; both treatments were administered with pharmacotherapy. Sleep disturbance was assessed with the Adolescent Sleep Habits Questionnaire (ASHQ) filled out by patients every 6 weeks in the first study year and every 3 months in the second year. Main outcomes included clinician-rated measures of mania, depression and psychosocial impairment over 2 years. Sleep impairment was significantly associated with mania and depression severity scores and psychosocial impairment ratings across the 2-year follow-up. Despite its efficaciousness in reducing mood symptoms, FFT-A was not more effective than enhanced care in improving sleep habits. Sleep impairment may play a substantial role in the course of adolescent BSD. Youth with BSDs may benefit from targeted psychosocial interventions that emphasize sleep regularity.

Keywords

Depression; mania; psychosocial treatment; sleep disturbance; family therapy; familyfocused treatment

1. Introduction

For up to 65% percent of individuals with bipolar spectrum disorders (BSD), onset occurs during or prior to the adolescent years (Perlis et al., 2004), with earlier onset associated with

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a poorer prognosis in adulthood (Suppes et al., 2001; Coryell et al., 2003; Schneck et al., 2004). In adult BSD, sleep disturbance represents a core symptom associated with poorer quality of life and impaired psychosocial functioning (Wehr et al., 1987; Barbini et al., 1996; Leibenluft et al., 1996; Lenox et al., 2002; Jackson et al., 2003; Hasler et al., 2006; Gruber et al., 2010). Sleep disruption has been described as the “final common pathway” in explaining the genesis of mania among adults (Wehr et al., 1987).

Comparatively little is known regarding the impact of sleep changes on the course of BSD in adolescence. Although sleep disturbance is common in healthy adolescents (Carskadon, 2002), it has been described as a particularly impairing feature of early-onset BSD (Harvey, 2008; Lofthouse et al., 2010). Elucidating the prognostic role of sleep impairment among adolescents with BSD may contribute to the development of early interventions focused on sleep regularity, which may translate into improved symptomatic outcomes in adulthood.

Cross-sectional investigations of sleep dysfunction in adolescent BSD indicate that a majority of affected youth report significant sleep disruptions, including problems awakening in the morning (Staton et al., 2008, as reported in Staton, 2008), insomnia or parasomnia (Faedda et al., 2004), and decreased need for sleep during (hypo)manic episodes (Findling et al., 2001). As expected, the majority of adolescents report significant insomnia during their worst depressive episode and decreased need for sleep during their worst manic episode (Lofthouse et al., 2007). In addition, increased fatigue, insomnia, and decreased need for sleep distinguish adolescents with BSD from those with unipolar affective disorder, disruptive behaviors, ADHD, anxiety, and adjustment disorders (Meyers and Youngstrom, 2007; Luckenbaugh et al., 2009). Impairments of sleep appear to be particularly distressing to families of patients with adolescent BSD. In a web-based survey, 85% percent of parents reported consulting medical, mental health, or school professionals regarding their child's mood-related sleep problems (Lofthouse et al., 2010).

The few existing studies on sleep and adolescent BSD did not examine the covariation of sleep and manic or depressive symptoms over time. In addition, most of these cross-sectional studies relied solely on parent report, even though children and adolescents may be more accurate reporters of sleep problems than their parents (Tillman et al., 2004; Lofthouse et al., 2007; Stoléru et al., 2007). Prospective research incorporating adolescent reports of sleep habits is necessary to clarify the role of sleep impairment in the symptomatic course of adolescent BSD.

Secondarily, sleep disruption may result in a diminished capacity of adolescent BSD patients to function in social domains. Patients with adolescent BSD exhibit pervasive impairment in family relationships, peer interactions, and academic performance (Geller et al., 2000; Wolf and Wagner, 2003; Rucklidge, 2006). In adolescent community samples, sleep irregularities are associated with detrimental psychosocial outcomes (Carskadon, 2002). Less is known regarding the impact of sleep impairment on the psychosocial functioning of adolescents with BSD. In one cross-sectional study, 97% of parents of patients with pediatric BSD reported sleep-related psychosocial impairment at home, in school, or with peers, with sleep disturbance most commonly occurring during mixed episodes (Lofthouse et al., 2008). No studies, however, have examined the longitudinal covariation of sleep dysfunction and psychosocial outcomes in adolescent BSD.

The primary objectives of the current study were to examine the longitudinal associations between sleep disruption and (a) mood symptom severity and (b) psychosocial functioning in adolescent patients with DSM-IV bipolar spectrum disorders (bipolar I, bipolar II, or bipolar, not otherwise specified [NOS] disorder). Data were collected in the context of a 2-site randomized trial of family-focused treatment for adolescents (FFT-A), a 9-month, 21

session psychoeducational treatment for adolescents with BSD following a mood episode, versus enhanced care (EC), a briefer psychoeducational treatment; both treatments were administered with pharmacotherapy (Miklowitz et al., 2008). Assessments of mood symptoms, sleep regularity, and psychosocial functioning were conducted prospectively over a 2-year period. We hypothesized that the level of self-reported sleep impairment would correlate with (a) the severity of mania and depression symptoms over the follow-up period, and (b) greater psychosocial impairment at follow-up, even after covarying concurrent symptoms.

Patients in both FFT-A and EC were acquainted with the importance of keeping regular sleep/wake cycles to manage mood (Frank et al., 2005). Further, patients in the FFT-A condition were encouraged to keep track of their sleep and daily routines through a daily mood chart. Thus, a secondary aim was to explore the effects of FFT-A on sleep dysfunction, to determine whether adolescents with BSD who received FFT-A and pharmacotherapy demonstrated less sleep impairment than those receiving EC and pharmacotherapy. We reasoned that if sleep disturbance is linked to better clinical or functional outcomes in adolescent BSD, then psychoeducational treatments that include a focus on sleep stabilization should lead to better short-term outcomes of the disorder.

2. Methods

2.1 Subjects

Adolescents aged 12 years 0 months through 17 years 11 months were recruited from areas surrounding the University of Colorado at Boulder and the University of Pittsburgh School of Medicine. Participants met DSM-IV-TR criteria for bipolar I or bipolar II disorder, or bipolar NOS, defined as a distinct period of abnormally elevated, expansive, or irritable mood plus two (three if irritable mood only) DSM-IV-TR symptoms of mania that resulted in a change in functioning, persisted for at least one day, and was present for a total of at least four days during the adolescent's lifetime (Axelson et al., 2006; Birmaher et al., 2006). These operational criteria are similar to the broad clinical spectrum criteria proposed for bipolar disorder among adults (e.g., Akiskal et al., 2000). Further, the adolescent had to have experienced a mood episode, defined as 1 week of mania, hypomania, or mixed state or 2 weeks of depression, in the 3 months prior to study intake. For inclusion, all participants indicated a willingness to engage in pharmacological treatment with a study psychiatrist and had at least one parent who agreed to participate with their child in family therapy. Potential participants were excluded from the study if they met criteria for substance or alcohol disorder in the 3 months prior to intake, pervasive developmental disorder, an eating disorder or medical disorder necessitating hospitalization, or current psychosis lasting at least 3 months.

This study was approved by Institutional Review Boards at the University of Colorado at Boulder and the University of Pittsburgh School of Medicine. All adolescents and parents signed an informed assent or consent form prior to participating in the study.

2.2 Procedures

2.2.1 Initial assessment—To determine initial eligibility, project coordinators screened prospective participants by telephone. If the participant was deemed likely to fit inclusion criteria, an appointment was set during which trained interviewers administered the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997) and the Child's Global Assessment Scale (CGAS) to each adolescent and parent separately. To establish current episodes, the adolescent and parent were queried regarding the worst week of mania and the worst week

of depression in the 3 months prior to this intake assessment. To more adequately assess severity and impairment associated with mood symptoms, the K-SADS Mania Rating Scale (MRS; Axelson et al., 2003b) and Depression Rating Scale (DRS; Chambers et al., 1985) were substituted for the mood sections of the K7 SADS-PL. Interrater reliability for K-SADS MRS and DRS scores (51 ratings) was 0.97 and 0.89 (intraclass r), and 0.70 (Kappa statistic) for distinguishing among comorbid disorders.

Following the conclusion of the K-SADS-PL, consensus scores from the parent and adolescent interviews were created to determine lifetime and current (present in the last 3 months) diagnoses and ratings of global functioning for each participant. If the K-SADS evaluators agreed with the study psychiatrist's diagnosis of bipolar I, bipolar II, or bipolar NOS (based on a separate medical evaluation), the adolescent was invited to participate in the study.

2.2.2 Treatment—Once eligibility was established, each participant was randomly assigned to receive FFT-A or EC. Families assigned to FFT-A received 21 fifty-minute sessions of psychoeducation, communication enhancement training, and problem-solving skills training, administered weekly, then biweekly, then monthly up to 9 months. Those assigned to the EC condition received weekly fifty-minute sessions of psychoeducation in the first month after randomization, followed by case management as needed for the next 9 months (Miklowitz, 2008; Miklowitz et al., 2008). Randomizations were stratified by study site.

Patients in the FFT-A condition were encouraged to keep track of their sleep and daily routines through a daily mood chart. There were no other sleep interventions in either condition; however, clinicians implementing FFT-A had more opportunities than clinicians implementing EC to examine the adolescents' sleep/wake habits and suggest strategies to improve regularity.

All participants received pharmacological treatment from a study psychiatrist for the duration of the study. Study psychiatrists consulted current pharmacological guidelines in treating adolescents with BSD (Kowatch et al., 2005), which consisted primarily of lithium, atypical antipsychotics, and anticonvulsants alone or in combination. Pharmacological treatment of comorbid psychiatric disorders, including antidepressants and anxiolytics, was permitted as needed.

2.3 Measures

2.3.1 Assessment of sleep outcomes—Participants completed the Adolescent Sleep Habits Questionnaire (ASHQ) every 3 months in year one and every 6 months in year two. The 23-item ASHQ is a shortened, self-report version of the Children's Sleep Habits Questionnaire (CSHQ; Owens et al., 2000). The CSHQ is comprised of subscales that include bedtime behavior and sleep onset, sleep duration, anxiety about sleep, night waking, morning waking, and daytime sleepiness over the prior week. Participants rated each item as occurring rarely (0–1 time a week), sometimes (2–4 times a week), or usually (5–7 times a week). The internal consistency of the total ASHQ score in this study was 0.76 (Cronbach's α) at baseline, and remained reliable across follow-up assessments (mean α = 0.75).

Exploratory factor analysis using data from this sample revealed that ratings on the ASHQ loaded primarily on a single "sleep disturbance" factor (Factor 1 eigenvalue = 5.51), which accounted for 24% of the variance in ASHQ item ratings. The items loading highly on this factor included: "You sleep too little", "You sleep the right amount" (reverse scored), "You awake more than once during the night", "You wake up in a negative mood", "You have difficulty getting out of bed in the morning", and "You take a long time to become alert in

the morning.” Thus, a total ASHQ sleep disturbance score was calculated for each patient at each available time point. Secondary analyses explored the relationship of specific ASHQ items to adolescents’ mania and depression scores.

2.3.2 Assessment of mood and psychosocial functioning outcomes—To assess changes in mood over time, participants were evaluated every 3 months in year one and every 6 months in year two by an independent evaluator who was “blind” to treatment condition. Follow-up evaluations were most frequent during the active psychosocial treatment period (first 12 months). Patients were followed less frequently in the second year, during which no active psychosocial interventions were administered (Miklowitz et al., 2008). At each follow-up, independent evaluators administered the K-SADS MRS and DRS to the child and parent separately, creating consensus scores for the presence and severity of mania, hypomania, and depressive symptoms for the worst week of depression and the worst week of mania in the preceding month.

In addition, evaluators administered the Adolescent Longitudinal Interval Follow-up Evaluation (A-LIFE) Psychiatric Status Rating Scales (Keller et al., 1987; Birmaher et al., 2006) and Psychosocial Scales (Leon et al., 2000) to the child and parent separately, assigning consensus ratings for the presence and severity of manic, hypomanic, and depressive symptoms for each week since the previous assessment (prior 12 weeks in year one, and prior 26 weeks in year two). Weekly Psychiatric Status Ratings for the depression and hypomania scales ranged from 1 to 6, with ratings of 1 or 2 signifying asymptomatic or minimally symptomatic states, ratings of 3 or 4, subthreshold symptoms and impairment, and ratings of 5 or 6, fully syndromal conditions. For ratings of mania, the Psychiatric Status Rating Scale could be rated a 1 (absent) or a 5 or 6 (severe or extremely severe), with no intermediate points. Following conventions, a total mania score was computed for each week. This score was equivalent to the hypomania score if the patient did not meet the DSM-IV syndromal threshold for mania during the week, or to the mania score if the patient met syndromal criteria. Interrater reliability for distinguishing syndromal episodes of depression or mania/hypomania using the A-LIFE ranged from 0.74 – 0.91 (Kappa statistics).

Based on separate parent and adolescent reports, global psychosocial scores on the ALIFE were determined for each month since the last follow-up. In making these ratings, clinicians took into consideration educational attainment and functioning at work and in interpersonal relationships. In addition, separate ratings were made for academic, peer, and family functioning domains at each point of follow-up. Ratings ranged from 1 to 5, with a score of 1 indicating optimal functioning and a score of 5 signifying severe impairment. The A-LIFE Psychosocial Scales have been validated on samples of individuals with bipolar spectrum disorders, demonstrating sound interrater reliability (ICC = 0.94) and internal consistency (coefficient alpha ranging from 0.78 to 0.84) (Leon et al., 1999, Leon et al., 2000), and were found to have discriminative validity in a prior study of psychosocial functioning in adolescent BSD conducted at the Pittsburgh site (Goldstein et al., 2009).

2.4 Data analyses

Linear mixed effects regression models covarying for treatment condition, time, and their interaction were used to assess the relationship of ASHQ sleep disruption scores to the severity of mood symptoms (MRS and DRS scores) over time. To assess the relationship of changes in ASHQ scores to changes in mood symptoms on the weekly A-LIFE Psychiatric Status Ratings at follow-up, standardized betas were calculated for the ASHQ, the Psychiatric Status Rating for depressed mood, and the Psychiatric Status Rating for manic/hypomanic mood. Then, general linear models controlling for treatment condition used the

calculated regression coefficients for the A-LIFE Psychiatric Status Ratings to predict changes in sleep (also represented by standardized betas).

Linear mixed effects regression models were used to investigate the relationship between ASHQ total scores and A-LIFE psychosocial functioning scores (impairment in academic, peer, familial, and global functioning) over two years, using concurrent non-sleep-related mood symptoms as covariates (Lavori et al., 1994). These analyses used MRS and DRS scores that were recalculated without the sleep items.

To assess the efficacy of FFT-A versus EC in reducing sleep problems, multilevel mixed models covarying for baseline levels of sleep disturbance assessed the effects of treatment, time, and a treatment by time interaction on ASHQ scores. With 53 participants in two treatment conditions, we had 99% power to detect a medium (Cohen's $f = 0.25$) or large effect ($f = 0.40$) of treatments on sleep scores measured over 6 points of follow-up ($P < 0.05$). However, we did not have adequate power (48%) to detect a small effect (Cohen's $f = 0.10$). Thus, we considered the treatment/outcome analyses to be secondary and exploratory.

3. Results

3.1 Participants

Of the 58 youth participating in the treatment study, data from 53 adolescents (29 females [54.7%] and 24 males [45.3%]) were used in the current investigation; 5 could not be included because they did not complete an ASHQ during the follow-up period. Thirty-four participants were from the Colorado site (64.2%) and 19 (35.8%) from the Pittsburgh site. The mean ($\pm$ SD) age at intake was 14.6 ± 1.6 ; 35 participants were diagnosed with bipolar I disorder, 5 with bipolar II, and 13 with bipolar NOS. Eleven participants [21%] had comorbid ADHD. The mean ($\pm$ SD) CGAS score for the 2 weeks prior to random assignment was 57.8 ± 11.1 (range 35–95) and 44.9 ± 12.9 (range 35–95) for the most severe past episode.

Levels of sleep, depression, and mania were variable at baseline. The mean MRS score at intake exceeded a cutoff score of 12, indicating clinically significant symptoms of mania ($M = 28.19 \pm 10.49$; range = 2–50). A cutoff score of 12 has been previously shown to distinguish those with clinically significant levels of mania from those with non-clinically significant levels of mania with a sensitivity of 87% and a specificity of 81% (Axelson et al., 2003b). The mean DRS score was $22.46 (\pm 10.0)$; range = 2–43, indicating moderate levels of depression (on average) but with significant range. ASHQ scores covering the prior week averaged 43.76 ± 7.83 (range = 29–60), indicating that the average patient had a moderate level of sleep impairment. However, there are no established norms for the ASHQ.

Participants were followed for an average of 83.9 ± 31.6 weeks, and completed a mean of 4.2 ± 2.0 (range = 1–7) ASHQ questionnaires over the 24-month period (see Table 1).

3.2 Cross-sectional comparisons at baseline

At the baseline pretreatment assessment, sleep ratings on the ASHQ (covering the prior week) did not differ significantly by gender ($F[1, 40] = 0.13, P = 0.72$), bipolar subtype ($F[2, 39] = 0.32, P = 0.73$), treatment condition ($F[1, 40] = 0.08, P = 0.78$), comorbid ADHD ($n = 11; F[1, 40] = 0.19, P = 0.67$), or site (Colorado vs. Pittsburgh: $F[1, 40] = 0.16, P = 0.69$). Because comorbid ADHD was not associated with sleep score on the ASHQ, it was not included as a covariate in subsequent analyses. Baseline sleep disturbance scores on the ASHQ were positively correlated with baseline DRS scores ($F[1, 40] = 11.63, P < 0.01, r = 0.48$), suggesting that patients reporting more sleep impairment also had higher depression ratings upon direct interview. Baseline ASHQ scores were not associated with baseline MRS scores ($F[1, 40] = 0.10, P = 0.76, r = 0.05$).

3.3 Relationship of sleep scores and mood symptom scores over time

Mixed effects modeling controlling for time, treatment condition, and their interaction revealed that ASHQ sleep disturbance scores significantly predicted MRS scores over time ($F[1, 160] = 15.98, P < 0.0001, d = 0.63$), such that as the level of sleep disturbance increased, ratings of mania also increased (See Fig. 1). In contrast, changes in sleep scores on the ASHQ were not significantly related to changes in mania scores (as represented by standardized betas) on the ALIFE Psychiatric Status Rating Scale over 2 years ($F[1, 42] = 1.37, P = 0.25, d = 0.36$).

Linear mixed effects models revealed that sleep scores on the ASHQ significantly predicted depression scores on the DRS across the 2-year period, independent of treatment condition, time, and their interaction ($F[1, 161] = 12.12, P < 0.001, d = 0.55$). Greater levels of sleep impairment were associated with more severe depressive symptoms (see Fig. 2). Similarly, general linear modeling demonstrated that changes in the level of sleep impairment on the ASHQ were significantly related to changes in depression symptoms on the A-LIFE Psychiatric Status Rating Scale (as represented by standardized betas) across the follow-up period ($F[1, 41] = 9.63, P = 0.004, d = 0.97$).

3.4 Relationship of specific items on the ASHQ to mood symptom scores over time

To identify the specific components of sleep dysfunction that best explained these results, we used linear mixed effects regression models to examine the relationship of specific items on the ASHQ to depression and mania scores over time. An item related to stability of sleep rhythms, “You sleep the same amount each day,” was significantly associated with longitudinal MRS scores across time and treatment condition ($F[1, 159] = 5.57, P = 0.02, d = 0.37$), such that greater endorsement of the item was correlated with decreased ratings of mania. In addition, items related to unstable morning routines and difficulty awakening were significantly related to increased mania scores over the 2-year follow-up period. The items “Someone else has to wake you up” ($F[1, 160] = 5.95, P = 0.02, d = 0.39$) and “You have difficulty getting out of bed in the morning” ($F[1, 160] = 8.01, P = 0.005, d = 0.45$) were positively correlated with increased MRS scores over time.

Linear mixed effects regression models revealed that the ASHQ items that best predicted longitudinal depression (DRS) scores were related to difficulties with middle insomnia, such as “You awake more than once during the night” ($F[1, 148] = 6.81, P = 0.01, d = 0.43$) and “You are awake more than 20 minutes a night” ($F[1, 160] = 8.12, P = 0.005, d = 0.45$), with greater endorsement being associated with higher depression scores over the 24-month period. The item “You go to bed at the same time each night” was inversely associated with depression scores over time, such that greater endorsement was associated with lower DRS scores ($F[1, 161] = 4.54, P = 0.03, d = 0.34$).

To assess whether the role of sleep impairment in mood symptoms differed by diagnosis (narrowly defined versus broadly defined BSD), we conducted the same mixed models with diagnostic subtype as an additional independent variable. There were no main effects of diagnostic subtype or interactions between subtype and treatment condition on sleep impairment at follow-up (for all, $P > .10$).

3.5 Relationship of sleep scores to psychosocial functioning over time

A linear mixed effects regression model covarying for concurrent DRS scores, MRS scores, treatment condition, and time revealed that sleep scores on the ASHQ significantly predicted parent-reported global psychosocial functioning scores ($F[1, 110] = 4.01, P < 0.05, d = 0.38$), such that higher levels of sleep impairment were associated with more psychosocial impairment. Results were similar when adolescents reported their level of global

psychosocial functioning. ASHQ scores significantly predicted global functioning scores ($F[1, 140] = 5.87, P = 0.02, d = 0.41$), such that as sleep disturbances increased, self-reported ratings of global impairment also increased (See Fig. 3), over and above mania severity on the MRS, depression severity on the DRS, treatment condition, and time.

Covarying for time, treatment condition, and level of depression on the DRS, ASHQ scores significantly predicted academic functioning on the A-LIFE ($F[1, 34.4] = 4.59, P = 0.04, d = 0.73$), such that higher levels of sleep impairment were associated with more severe academic impairment. Controlling for levels of mania on the MRS, sleep problems on the ASHQ did not predict academic functioning on the A-LIFE ($F[1, 54.2] = 0.95, P = 0.33, d = 0.27$). ASHQ scores were not significantly associated with peer or familial functioning.

3.6 Effect of FFT-A or Enhanced Care on sleep dysfunction

As reported in Miklowitz et al. (2008), FFT-A was associated with a more favorable trajectory of A-LIFE Psychiatric Status Rating Scale depression scores than EC (treatment by time interaction, $P = 0.002$) but not of mania or hypomania scores ($P = 0.96$). A linear mixed effects regression model controlling for baseline sleep disruption scores revealed that the main effect of treatment condition ($F[1, 45] = 0.01, P = 0.92, d = 0.03$), time ($F[1, 125] = 0.16, P = 0.69, d = .07$), and the interaction effect of treatment condition by time ($F[1, 125] = 0.45, P = 0.50, d = 0.12$) on longitudinal ASHQ scores were not significant. To assess whether this pattern was similar among narrowly defined versus broadly defined BSD, we expanded the model to include diagnosis, the diagnosis by condition interaction, and the diagnosis by condition by time interaction. None of these main effects or interactions was significant. Thus, despite the effects of FFT-A on depressive symptoms, patients in FFT-A did not demonstrate significantly different sleep scores over time than those receiving EC.

4. Discussion

This was the first prospective investigation of self-reported sleep impairment in a young population with BSD. The primary objectives were to examine the longitudinal associations between sleep disruptions and (a) severity of mood symptoms, and (b) psychosocial impairment. A secondary aim was to investigate the efficacy of FFT-A in reducing sleep difficulties in this population. In accordance with our hypotheses, results indicated that sleep impairment and severity of manic and depressive symptoms were significantly intercorrelated over time among adolescents with BSD. These results are similar to emerging evidence from the adult bipolar literature, which has indicated that sleep duration and variability are prospectively associated with severity of mania and depression symptoms, constituting candidate targets for treatment (Malkoff-Schwartz et al., 2000; Gruber et al., 2010).

Specific sleep disturbances differed across manic and depressive states, although irregularities in sleep rhythms were associated with both mood states. The ASHQ items most closely associated with mania symptoms measured sleep rhythm irregularity (e.g., sleeping different amounts each night or having unstable morning routines). The ASHQ items most closely associated with depression scores included sleep rhythm irregularity and frequent nighttime awakenings. These results underscore the importance of maintaining stable sleep/wake rhythms as a protective factor against mood episodes. The results are also consistent with findings in the adult literature concerning relationships between unstable social rhythms and mood episodes, particularly phase delays in the timing of morning routines (Leibenluft et al., 1996; Ashman et al., 1999; Shen et al., 2008).

Sleep irregularities were also associated with academic and social impairment among adolescents with BSD, even when mood severity was controlled. In normal adolescents, insufficient or disturbed sleep is associated with psychosocial impairment (Wolfson and Carskadon, 1998), as well as with learning problems and poor academic performance (Wolfson and Carskadon, 2003). Specifically, reduced sleep time results in difficulties with new memory acquisition and retrieval, both of which are essential processes for maintaining effective social functioning and academic success (Carskadon, 2011). Although sleep changes are part of bipolar illness and generally more severe than the normal variability observed in adolescence, it is unclear whether the present results have specific relevance to adolescent BSD. Future studies prospectively following adolescents with BSD and normal controls (perhaps using real-time, repeated measures of sleep and mood severity) may be necessary to distinguish processes that are specific to BSD.

Contrary to our hypothesis, youth receiving FFT-A did not demonstrate greater sleep improvements over time than those receiving EC. The absence of a significant effect of treatment condition on sleep impairment may be attributed to the specific goals emphasized in family treatment. Although FFT-A educates youth and family members about the importance of regular daily rhythms and sleep/wake cycles, the central tenets of FFT-A focus on reducing familial conflict and mastering a wide-range of self-management strategies, of which sleep regulation is only one. Given the observed relationships between sleep rhythm instability and mania and depression symptoms in the present study, targeted psychosocial interventions emphasizing sleep regularity as a primary objective, such as Interpersonal and Social Rhythm Therapy (Frank et al., 2005; Hlastala and Frank, 2006), may be essential for reducing the severity of mood symptoms in adolescents with BSD. In addition, although not yet fully developed for adolescent BSD patients, cognitive-behavioral therapy focused on relieving insomnia (CBT-I) has also shown promise in the treatment of mood disorders in adults (Manber et al., 2008), and may prove beneficial for relieving mood symptoms in adolescent BSD (Harvey, 2008).

The conclusions of this study should be interpreted in the context of several limitations. First, sleep disturbances were assessed via a self-report questionnaire. On one hand, self-report should be preferred to parent-report, as adolescents are generally more accurate reporters of sleep impairment (Tillman et al., 2004; Lofthouse et al., 2007; Stoléru et al., 2007). However, it is also possible that when youth are in a mood episode, they are more likely to recall instances of sleep disturbance than during periods of euthymia. In addition, norms for the ASHQ have not been reported in the literature. Thus, it is not known what score on the ASHQ distinguishes clinically significant from non-clinically significant levels of sleep impairment.

Because weekly ratings of sleep were not obtained, the direction of causation between sleep abnormalities and mood variation, and between sleep problems and psychosocial impairment, are unknown. Whereas the findings of this study could indicate that improved sleep leads to better clinical and functional outcomes, it is possible that mood instability and social difficulties (and the resulting generation of stress; Hammen, 2006) result in poorer sleep. Experience sampling methods (e.g., Trull and Ebner-Priemer, 2009) may help determine whether changes in sleep precede or are a consequence of changes in mood in this population. In addition, the frequency of ASHQ administration may not have been frequent enough to identify subtle changes in sleep that portend greater changes in mood. Finally, the sample size was too small to examine the impact on sleep impairment of disorders that are frequently comorbid with BSD among youth (i.e., ADHD, anxiety disorders, and conduct disorder).

Future research should utilize objective measures of sleep impairment to prospectively examine sleep disturbances in youth with adolescent BSD. Longitudinal studies using polysomnographic or actigraphic methods (e.g., Axelson et al., 2003a; Faedda and Teicher, 2005; Mehl et al., 2006) would clarify the specific types of sleep impairment experienced in BSD over the course of the illness, and possibly, aid in the development or enhancement of psychosocial and pharmacological treatments for the early-onset form of the disorder. In addition, studies could be further enhanced by the incorporation of real-time assessments of mood or functioning, such as weekly text-messaged mood ratings (Bopp et al., 2010). Real-time mood measures may clarify whether shifts in mood state represent triggers or consequences of objectively assessed disturbances in sleep.

In conclusion, the present study provides evidence for a robust link between sleep problems and mood symptoms and functioning in adolescents with BSD followed for up to 2 years. Psychosocial treatments focusing on sleep stabilization may prove effective in improving the prognosis of adolescent BSD-related sleep irregularities. Furthermore, given that an association between sleep disturbance, mood, and daytime functioning has been shown in most adolescents, including those recruited from community high school samples (Wolfson and Carskadon, 1998), the development and empirical evaluation of therapies targeting sleep may benefit not only adolescents with BSD, but also the larger pediatric population.

Acknowledgments

Financial support was provided by National Institute of Mental Health (NIMH) grants MH62555 and MH073871 (Dr. Miklowitz), the National Alliance for Research on Schizophrenia and Depression (Dr. Miklowitz), and NIMH grant MH01878 (Dr. Axelson). The authors thank Elizabeth George, Dawn Taylor, Adrine Biuckians, Tina Goldstein, Eunice Kim, Kimberley Mullen, Amy Schlonski, and Tim Winbush for serving as study therapists; Christopher Schneck for serving as a study physician; Susan Wassick, Amy Mechels, Chad Morris, Victoria Cosgrove, Laura Wagenknecht, and Lindsey Gagnon for serving as independent evaluators; and Mary Beth Hickey and Chris Hawkey for serving as data managers.

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Figure 1.

Relationship of ASHQ and MRS scores over time

Abbreviations: ASHQ, Adolescent Sleep Habits Questionnaire; MRS, Mania Rating Scale.

For ease of illustration, the graphs present the unadjusted ASHQ scores.

Figure 2.

Relationship of ASHQ and DRS Scores over Time

Abbreviations: ASHQ, Adolescent Sleep Habits Questionnaire; DRS, Depression Rating Scale. For ease of illustration, the graphs present the unadjusted ASHQ scores.

Figure 3.

Relationship of ASHQ and A-LIFE global psychosocial functioning over time

Abbreviations: ASHQ, Adolescent Sleep Habits Questionnaire; A-LIFE, Adolescent Longitudinal Interval Follow-up Evaluation. For ease of illustration, the graphs present the unadjusted ASHQ scores.

Table 1

Demographic Characteristics of Sample at Baseline

Variable	Values
Mean (SD)	
Age in Years	14.6 (1.60)
DRS Score	22.46 (10.00)
MRS Score	28.19 (10.49)
ASHQ Score	43.76 (7.83)
Number of Weeks Followed	83.9 (31.6)
Number of ASHQs Completed	4.2 (2.0)
Number (%)	
Sex (female)	29 (54.70)
Race	
White	48 (90.57)
Native American	1 (1.89)
Asian	1 (1.89)
Biracial	3 (5.66)
Ethnicity	
Hispanic	2 (3.77)
Non-Hispanic	51 (96.23)
Diagnosis	
Bipolar I	35 (66.04)
Bipolar II	5 (9.43)
Bipolar NOS	13 (24.53)
Comorbid ADHD Diagnosis	11 (20.75)
Comorbid Anxiety Diagnosis	1 (1.89)