Brief Report

A preliminary study of sleep in adolescents with bipolar disorder, ADHD, and non-patient controls


Objectives: To compare the sleep of adolescents with bipolar disorder (BD) to groups of adolescents with attention-deficit hyperactivity disorder–combined type (ADHD-C) and those without psychopathology.

Methods: A sample of 13 adolescents diagnosed with BD who were not in the midst of a mood episode, 14 adolescents with ADHD-C, and 21 healthy controls, all between the ages of 11 and 17 years served as participants. They were psychiatrically evaluated using a structured diagnostic interview and completed four nights of in-home sleep monitoring using actigraphy and sleep diaries.

Results: Sleep diary estimates of sleep indicated that participants with BD experienced more awakenings than their peers with ADHD, whereas actigraphic estimates revealed that participants with BD slept longer and with less wakefulness than their peers.

Conclusions: In between mood episodes, adolescents with BD experience their sleep as more fragmented than that of their peers but do not exhibit more disturbed sleep as estimated by actigraphy. The possible influence of psychotropic medication is an important consideration when assessing sleep in the context of BD.

Sleep and circadian abnormalities represent core features of bipolar disorder (BD) in adulthood (1). Among adults with BD, compared to healthy controls, investigations document insomnia and reduced need for sleep during mania, both insomnia and hypersomnia during depression, and greater insomnia and more variable sleep and activity patterns during interepisode periods (2). Two studies have provided evidence that BD is associated with delayed circadian phase (3, 4). Moreover, theories of BD have emphasized the possible role of sleep and circadian rhythm instability in the onset and maintenance of mood episodes (2, 5, 6).

Despite increasing interest in early-onset BD, few studies have investigated sleep and circadian functioning in this population. Nonetheless, the literature indicates that sleep problems are significant (7). Questionnaire- and interview-based studies report sleep problems among 96–97% of youth with BD (8). Using one night of polysomnography (PSG), Mehl et al. (9) found that children with symptoms of BD (but not official diagnoses) exhibited impaired sleep efficiency, increased sleep-onset latency, and increased awakenings and nightmares compared to healthy peers. The only published study using actigraphy features case reports of two youths with BD who completed 5 to 7 days of actigraphic monitoring (10). Both individuals exhibited symptoms of insomnia, as well as marked diurnal and nocturnal...

The authors of this paper do not have any commercial associations that might pose a conflict of interest in connection with this manuscript.
hyperactivity. The use of actigraphy was a strength, but the case study design limits the generalizability of the findings.

Sleep functioning in early-onset BD is an essential area of investigation. Sleep is critically involved in mood regulation (11), particularly for individuals with BD, as they may be especially vulnerable to the destabilizing effects of sleep loss (2). Moreover, particular sleep and circadian profiles may serve as endophenotypic markers for BD (12). A current diagnostic challenge involves distinguishing between BD and attention-deficit hyperactivity disorder (ADHD), a much more common childhood-onset disorder (13, 14). However, one large longitudinal study found that the symptom of decreased need for sleep effectively discriminated between youth with BD (39.8%), ADHD (6.2%), and healthy participants (1.1%) (15). Whereas sleep abnormalities have been alleged in youth with ADHD (16), a recent meta-analysis revealed few significant differences between samples with ADHD and healthy groups (17).

The current study aimed to extend previous research by examining both self-report and objective estimates of sleep over a four-night period using carefully characterized samples of youth with BD who were between mood episodes, youth with ADHD–combined type (ADHD-C), and healthy adolescents. We chose ADHD-C as our clinical comparator because it is the most common form of ADHD (18), with symptoms of both inattention and impulsivity/hyperactivity, and therefore perhaps most representative of this condition. Based on previous research (19–21), we predicted that participants with BD would exhibit greater total sleep time, but also greater wakefulness during the night, lower sleep efficiency, and lower satisfaction with sleep than their peers. We also hypothesized that the participants with BD would exhibit later sleep onset and morning wake times than their peers (3, 4). These variables were conceptualized as proxies for a tendency toward a delayed phase. Finally, we predicted that the participants with BD would show more variable sleep onset times, morning wake times, and total sleep times than their peers in the comparison groups (20, 22).

Materials and methods

Participants

This study included 48 participants between the ages of 11 and 17 years. The sample was composed of three groups: individuals with BD (n = 13), individuals with ADHD-C (n = 14), and healthy controls (n = 21). Participants were recruited from parent-support groups, mental health support listservs, postings at mental health clinics, and an online community message board in the San Francisco Bay Area, CA, USA.

The BD group included participants who met DSM-IV-TR criteria (13) for bipolar I disorder (BD-I) (n = 5), bipolar II disorder (BD-II) (n = 4), or bipolar disorder not otherwise specified (BD-NOS) (n = 4). BD-NOS was assigned to individuals who met the stringent criteria implemented in the large Course and Outcome of Bipolar Illness in Youth study (23). None of the participants with BD were in the midst of manic or depressive episodes at the time of participation, based on the results of the clinical interview and the Parent–Young Mania Rating Scale (P-YMRS) (24), a 10-item parent-report scale measuring manic symptoms over the past week. A total score of ≥ 27 was used as a cutoff for determining mania status (25); none of the potential participants exceeded this threshold, although many were fairly symptomatic. All participants with BD were in treatment and receiving psychiatric medications (described below).

Participants in the ADHD-C group met DSM-IV-TR criteria for this condition. Healthy controls had not received any clinical diagnoses or received treatment for psychological problems. (See Table 1 for demographic characteristics.) Exclusion criteria included current or lifetime diagnoses of sleep disorders, schizophrenia, mental retardation, or

### Table 1. Participant characteristics

<table>
<thead>
<tr>
<th></th>
<th>Bipolar disorder (n = 13)</th>
<th>ADHD-C (n = 14)</th>
<th>Controls (n = 21)</th>
<th>F (2,46) p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex ratio</td>
<td>6/7</td>
<td>11/3</td>
<td>11/10</td>
<td>3.46</td>
</tr>
<tr>
<td>(male/female)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Age, years, mean (SD)</td>
<td>14.4 (2.1)</td>
<td>15.1 (2.1)</td>
<td>14.1 (2.0)</td>
<td>1.00</td>
</tr>
<tr>
<td>Race</td>
<td>69.2</td>
<td>57.1</td>
<td>61.9</td>
<td>0.86</td>
</tr>
<tr>
<td>(% Caucasian)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PDS score, mean (SD)</td>
<td>3.1 (0.8)</td>
<td>2.8 (0.7)</td>
<td>2.7 (0.9)</td>
<td>0.95</td>
</tr>
<tr>
<td>P-YMRS score, mean (SD)</td>
<td>10.2 (8.6)</td>
<td>5.1 (6.1)b</td>
<td>1.6 (2.8)b</td>
<td>8.73</td>
</tr>
<tr>
<td>P-YMRS sleep severity score, mean (SD)a</td>
<td>0.9 (1.2)</td>
<td>0.2 (0.6)</td>
<td>0.1 (0.3)</td>
<td>5.53</td>
</tr>
</tbody>
</table>

Between-group comparisons performed with ANOVA, $\chi^2$ (sex, race), or Kruskal–Wallis Test (P-YMRS Sleep Severity). ADHD-C = attention-deficit hyperactivity disorder–combined type; PDS = Pubertal Development Scale; P-YMRS = Parent–Young Mania Rating Scale.

*Has your child’s sleep decreased lately?: 1 = no; 4 = denies need for sleep, has stayed up one night or more.

*p < 0.05 versus bipolar disorder.
Sleep in youth with bipolar disorder

autism spectrum disorders. These conditions were excluded because each has been associated with various sleep disturbances, potentially obscuring understanding of sleep specifically within BD or ADHD-C. Out of 52 participants, four participants were excluded from analyses: two because they did not meet diagnostic criteria for BD, despite having received this diagnosis by their treating physicians; one from the ADHD-C group because of a comorbid autism spectrum diagnosis, and another with ADHD because of meeting criteria for ADHD-inattentive type rather than ADHD-C.

Table 2 documents the psychiatric medications in our sample. Stimulant and hypnotic medications were not permitted during the course of this study given their potential influence on sleep (26); discontinuation at least one day prior to, and throughout the four days of study participation, was required. One-day washout periods for stimulants are commonly used in studies of ADHD (e.g., 27). This stipulation affected seven participants from the ADHD-C group (all taking stimulants), and one participant in the BD group who was taking a benzodiazepine sporadically as needed at bedtime. Participants temporarily discontinuing these medications were required to obtain permission from their prescribing physician before doing so. When possible, we scheduled participation for times when the individual might not be taking these medications (e.g., long weekend). Participants in the BD group were each taking an average of 2.2 psychiatric medications; in the ADHD-C group, 0.2 (excluding stimulants); and no psychiatric medications in the control group. Although antidepressant, antipsychotic, and mood-stabilizing medications are likely to influence sleep, temporary discontinuation would have been both impractical (given long washout/titration periods) and unethical (given health-related risks of severely ill adolescents being unmedicated).

Table 2. Medication usage by diagnostic group

<table>
<thead>
<tr>
<th></th>
<th>Bipolar disorder (n = 13)</th>
<th>ADHD-C (n = 14)</th>
<th>Controls (n = 21)</th>
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</thead>
<tbody>
<tr>
<td><strong>Anticonvulsants/mood stabilizers</strong></td>
<td></td>
<td></td>
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<tr>
<td>Divalproex</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lamotrigine</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lithium</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Oxcarbazepine</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Topiramate</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Antidepressants (SSRIs and miscellaneous)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bupropion</td>
<td>2</td>
<td>1</td>
<td>0</td>
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<tr>
<td>Duloxetine</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Escitalopram</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Sertraline</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Trazodone</td>
<td>1</td>
<td>0</td>
<td>0</td>
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<tr>
<td><strong>Anxiolytics</strong></td>
<td></td>
<td></td>
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<tr>
<td>Clonazepam</td>
<td>1</td>
<td>0</td>
<td>0</td>
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<tr>
<td><strong>Atypical antipsychotics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aripiprazole</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Clozapine</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ziprasidone</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Norepinephrine reuptake inhibitors</strong></td>
<td></td>
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<tr>
<td>Atomoxetine</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td><strong>Psychostimulants</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dextroamphetamine</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Methylphenidate</td>
<td>0</td>
<td>5</td>
<td>0</td>
</tr>
</tbody>
</table>

ADHD-C = attention-deficit hyperactivity disorder–combined type; SSRI = selective serotonin reuptake inhibitor.

aThese medications were not taken during study participation.
were summed into 60-sec epochs, which were downloaded to a PC and analyzed using the Actiware 3.4 software package (32). This software classifies epochs as sleep or wake using a validated algorithm. It also calculates variables such as total sleep time, sleep onset latency, minutes of wake after sleep onset, and sleep efficiency (i.e., minutes of actual sleep/minutes in bed, multiplied by 100). Participants were instructed to press the ‘event marker’ buttons on the actigraphs when they got into bed at night and again when they got out of bed in the morning. The time of these markers was checked against the sleep diary; in cases where the participant forgot to press the event marker, bedtime was imputed from the sleep diary.

Sleep diary
Each morning, participants completed a sleep diary over the phone with a research assistant. They were asked about bedtime, sleep onset time, number of awakenings, total time awake, and overall quality of sleep on a 4-point scale (1 = very poor, 4 = excellent). Daytime naps were not recorded.

Overview of analyses
Preliminary analyses were conducted to examine whether pubertal development and sex were related to sleep. We tested associations between the pubertal score and actigraphically measured total sleep time and sleep efficiency using bivariate correlations. Independent samples t-tests were used to investigate sex differences across these sleep variables. Group differences across sleep measures were evaluated using multiple one-way analysis of variances (ANOVA) with Tukey post-hoc comparisons. Effect sizes were measured using Cohen’s d. When examining sleep onset time and morning wake time, we converted standard clock times into variables representing the number of minutes from midnight. This transformation allowed us to perform accurate calculations of means and standard deviations (we have translated these values back to clock time in Table 3 for ease of viewing). Variability in actigraphy-measured total sleep time was estimated using the coefficient of variation (CV), with CV = (SD/mean). This statistic has been used to quantify within-individual variability in sleep across multiple recording periods (33). Because CV can only be computed with true ratio data, variability in sleep onset time and wake time were instead estimated using each individual’s standard deviation, calculated using the number of minutes from midnight at which each night’s sleep onset and wake time occurred.

<table>
<thead>
<tr>
<th></th>
<th>BD (n=13)</th>
<th>ADHD-C (n=14)</th>
<th>Controls (n=21)</th>
<th>Pairwise-comparison p-values (Effect size d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>F(2,46)  p-value BD vs. ADHD-C</td>
</tr>
<tr>
<td>Total sleep time (min)</td>
<td>492.3 (47.7)</td>
<td>424.2 (67.3)</td>
<td>436.9 (58.3)</td>
<td>5.26 0.009</td>
</tr>
<tr>
<td>Wake after sleep onset (min)</td>
<td>36.6 (19.7)</td>
<td>56.3 (17.4)</td>
<td>55.5 (19.2)</td>
<td>4.95 0.011</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>85.4 (6.9)</td>
<td>80.2 (6.1)</td>
<td>82.8 (4.7)</td>
<td>2.73 0.076</td>
</tr>
<tr>
<td>Sleep onset latency (min)</td>
<td>28.8 (19.2)</td>
<td>28.7 (18.0)</td>
<td>20.8 (12.1)</td>
<td>1.45 0.245</td>
</tr>
<tr>
<td>Mean no. of weeknights</td>
<td>3.3 (0.8)</td>
<td>2.6 (0.8)</td>
<td>3.0 (0.9)</td>
<td>2.96 0.062</td>
</tr>
<tr>
<td>in recording period (out of 4)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep onset time (hr:min)</td>
<td>23:15 (01:46)</td>
<td>23:35 (01:36)</td>
<td>23:27 (01:15)</td>
<td>0.16 0.851</td>
</tr>
<tr>
<td>Morning wake time (hr:min)</td>
<td>08:05 (01:40)</td>
<td>07:40 (01:41)</td>
<td>07:35 (00:52)</td>
<td>0.53 0.591</td>
</tr>
<tr>
<td>Variability</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Total sleep time (CV)</td>
<td>0.1 (0.1)</td>
<td>0.1 (0.1)</td>
<td>0.1 (0.1)</td>
<td>0.03 0.969</td>
</tr>
<tr>
<td>Sleep onset time (SD-min)</td>
<td>39.8 (22.9)</td>
<td>46.9 (20.8)</td>
<td>53.9 (35.8)</td>
<td>0.97 0.388</td>
</tr>
<tr>
<td>Morning wake time (SD-min)</td>
<td>52.0 (38.7)</td>
<td>62.1 (47.2)</td>
<td>56.9 (46.2)</td>
<td>0.17 0.843</td>
</tr>
</tbody>
</table>

Between-group comparisons made using one-way ANOVAs with Tukey post-hoc comparisons. Effect sizes were measured using Cohen’s d. BD = bipolar disorder; ADHD-C = attention-deficit hyperactivity disorder–combined type; CV = coefficient of variation.
Results

Pubertal development and sex

Pubertal development was not statistically related to any sleep variable. No sex differences were found for sleep onset latency, wakefulness after sleep onset, or sleep efficiency. However, girls exhibited significantly greater mean total sleep time (mean = 470.2 min) than boys (mean = 432.5 min), \( t(46) = 2.10, p = 0.042 \). Because sex was associated with a core sleep variable, we elected to examine this relationship in post-hoc analyses described later.

Actigraphy

Total sleep time was significantly different across groups (see Table 3). The BD participants slept longer than both the healthy control (\( d = 1.03 \)) and ADHD-C participants (\( d = 1.16 \)). The groups also differed on wakefulness after sleep onset. The BD group exhibited nearly 20 fewer minutes of wakefulness per night than both the healthy control (\( d = 0.98 \)) and ADHD-C groups (\( d = 1.06 \)). There was a marginally significant effect of diagnostic group on actigraphy-measured sleep efficiency, with the BD group exhibiting the highest mean value. Post-hoc analyses revealed a marginal difference between the BD and ADHD-C groups (\( d = 0.80 \)), but the BD and control groups did not differ significantly.

Sleep onset times did not differ between groups. No group differences were found in terms of variability (CV) in total sleep time, sleep onset times, or wake times.

Sleep diary

No group differences emerged for total sleep time on the sleep diary (Table 4). There was greater wakefulness after sleep onset among BD participants than healthy controls (\( d = 0.71 \)) at a marginal level of significance, though comparisons between the BD and ADHD-C groups, and the ADHD-C and control groups, were nonsignificant. Participants with BD reported more awakenings per night than both the ADHD-C (\( d = 0.95 \)) and healthy groups (\( d = 0.49 \)), though the latter comparison attained only marginal significance. The groups did not differ across sleep diary-derived sleep efficiency, with the mean values for all groups above 90%. However, group differences emerged for subjective satisfaction with sleep. The BD group reported lower satisfaction than the control group (\( d = 0.76 \)), at a level of marginal significance, but not relative to the ADHD-C group. The ADHD-C and healthy groups did not differ significantly.

Covariate analyses

Because there was a sex difference in actigraphy-derived total sleep time, we repeated all group sleep comparisons that had originally achieved statistical significance. Analysis of covariance (ANCOVA) was used to include sex as a covariate. For the actigraphy analyses, diagnostic group remained significantly related to mean total sleep time, \( p = 0.010 \), and mean wakefulness after sleep onset, \( p = 0.018 \), after controlling for sex (which became nonsignificantly related to either outcome). However, when sex was included as a covariate, neither

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Table 4. Sleep diary comparison between groups

<table>
<thead>
<tr>
<th></th>
<th>BD (n = 13)</th>
<th>ADHD-C (n = 21)</th>
<th>Controls (n = 21)</th>
<th>Pair-wise comparison p-values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>(Effect size ( d ))</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>BD vs. ADHD-C</td>
</tr>
<tr>
<td>Total sleep time (min)</td>
<td>532.1 (62.3)</td>
<td>504.2 (74.0)</td>
<td>511.7 (78.0)</td>
<td>0.51 0.605</td>
</tr>
<tr>
<td>Wake after sleep onset (min)</td>
<td>15.6 (28.0)</td>
<td>4.3 (7.7)</td>
<td>3.5 (3.7)</td>
<td>2.85 0.069 0.138 (0.58) 0.073 (0.71) 0.988 (0.11)</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>93.4 (8.0)</td>
<td>95.0 (3.2)</td>
<td>96.3 (2.3)</td>
<td>1.50 0.235</td>
</tr>
<tr>
<td>Awakenings (n)</td>
<td>1.5 (1.2)</td>
<td>0.7 (0.5)</td>
<td>0.9 (0.8)</td>
<td>3.48 0.040 0.040 (0.95) 0.099 (0.49) 0.790 (0.29)</td>
</tr>
<tr>
<td>Subjective satisfaction*</td>
<td>2.7 (0.6)</td>
<td>2.9 (0.5)</td>
<td>3.1 (0.5)</td>
<td>2.86 0.068 0.511 (0.38) 0.057 (0.76) 0.451 (0.41)</td>
</tr>
<tr>
<td>Sleep onset latency (min)</td>
<td>20.1 (14.1)</td>
<td>22.6 (16.0)</td>
<td>15.8 (11.3)</td>
<td>1.13 0.332</td>
</tr>
</tbody>
</table>

Between-group comparisons made using one-way ANOVAs with Tukey post-hoc comparisons. Effect sizes were measured using Cohen’s \( d \). BD = bipolar disorder; ADHD-C = attention-deficit hyperactivity disorder–combined type.

*1= very poor, 4 = excellent.
it nor diagnostic group was significantly associated with mean sleep efficiency. For the sleep diary analyses with sleep satisfaction as the dependent variable, diagnostic group remained a marginally significant predictor (p = 0.072), but sex was not. With mean number of awakenings as the dependent variable, sex was a nonsignificant predictor, while diagnostic group remained associated with this outcome at a marginal level of significance (p = 0.066). Similarly, with sex as a covariate, diagnostic group remained a marginally significant predictor of mean wakefulness after sleep onset (p = 0.083).

Discussion
To our knowledge, this is the first comparison of sleep among adolescents with BD, ADHD, and healthy controls, utilizing both actigraphy and sleep diaries. Consistent with our first hypothesis, actigraphy results revealed that participants with BD exhibited longer average sleep times than their comparison peers. This finding is consistent with previous actigraphy studies which reported extended sleep duration among remitted adults with BD (19–21), as well as the offspring of parents with BD (34). Furthermore, our sleep diary results indicated that participants with BD experienced more awakenings during the night relative to ADHD-C and control participants, and marginally significant findings of more wakefulness after sleep onset and lower satisfaction with sleep than healthy controls. These findings echo previous research documenting sleep impairments in youth with BD (8–10). However, contrary to our predictions, via actigraphy our participants with BD exhibited less wakefulness throughout the night than either of the other groups.

There are multiple reasons why youth with BD may sleep longer and with less wakefulness than their peers. First, the participants in our BD group were all taking at least one mood stabilizer or antipsychotic medication, with atypical antipsychotics being the most commonly used. These medications vary in their sedating properties, but they are generally less sedating than at least some of the traditional antipsychotics (35). Unfortunately, controlling for medications in between-group analyses was not practical, given the strong collinearity between medication usage and diagnostic group. For further discussion of the influence of psychiatric medications on sleep, see DeMartinis and Winokur (36).

A second possibility is that the extended sleep reflects a tendency toward hypersomnia, a phenomenon found in the depressive episodes of those with BD (37) and in about 25% of individuals who are inter-episode (38). Using the actigraphy estimate, the participants with BD slept 68 minutes more per night on average than the participants with ADHD-C, and 56 minutes per night more than the control group. Yet their mean total sleep time (8.2 hours) would not be considered outside of the normative range for this age group (39), and only one member of the BD group averaged more than nine hours of sleep per night.

Intriguingly, we observed some inconsistency between objective and subjective measures. Whereas sleep diaries indicated greater sleep problems in the BD sample than controls, actigraphy did not. This desynchrony is the topic of much interest and debate (40) and has been observed in other studies of youth with mood disorders. For example, Bertocci et al. (41) reported that children with major depressive disorder (MDD) rated their sleep as being of lower quality and with more wakefulness than controls; yet via laboratory PSG, the children with MDD appeared to sleep better than controls. One possibility is that PSG and actigraphy fail to detect subtle sleep disturbances in these groups (42). Alternatively, it is possible that sleep is measured accurately with actigraphy but that negative mood-congruent cognitive biases (particularly relevant to mood disorders) lead participants to evaluate their sleep in a globally negative way. That is, if an adolescent awakens in a negative mood, he or she may be more likely to appraise sleep negatively, independent of the objectively measured quality of that sleep.

It is notable that our participants with BD, although somewhat symptomatic, were not in the midst of full-blown mood episodes. Descriptions of dramatic sleep deficits and excesses in BD tend to originate from studies of patients during manic or depressive episodes, respectively. Whereas increasing sleep disturbance represents a reliable pro-drome for mania and depression in adults (2), perhaps when patients are experiencing relatively stable mood, sleep patterns tend to remain in the normative range (i.e., sleep problems may be a state marker, rather than a trait marker, of individuals with this disorder).

We garnered no evidence to support our hypotheses that participants with BD would exhibit more delayed sleep schedules or more variable sleep than their peers. This finding contrasts with studies in which adults with this diagnosis show more variable activity and sleep patterns than healthy controls in between affective episodes (22). Anecdotally, several parents of participants with BD reported that they attempted to enforce consistent sleep-wake schedules for their children to help
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prevent mood destabilization. Thus, the sleep patterns of participants with BD may have been more externally regulated than those of individuals in the comparison groups.

Moreover, most participants with BD were recruited from parent-support groups, whereas our participants with ADHD-C controls were typically referred from physicians’ offices and community postings. The families who attended support groups may have been particularly focused on their child’s functioning and care, and thus exerted more control over their child’s sleep schedule. It will be critical for future studies to capture the degree to which participants’ sleep schedules are regulated by external factors (e.g., parents, school schedules, etc.).

Several limitations should be considered. Foremost is the potentially confounding influence of medication on sleep. If research is conducted on only medication-free participants, samples are likely to be nonrepresentative and the results may not be generalizable to the majority of patients with BD. We recommend that investigators obtain more detailed information on medications, notably their dosages, duration of usage, and participants’ reports of sedating/alerting side effects. This is important because such side effects are often only present early in treatment (43), some medications can be either alerting or sedating, and sleep-related side effects are far from inevitable, present in an average of 4–37% of patients (26).

Given the pilot nature of this study, and the correspondingly small sample size, the findings should be interpreted with caution. However, a post-hoc calculation using our achieved t-test effect sizes (\(d \sim 1.0\)) suggests a power of 0.70, a reasonable balance of risk for type I and type II error. This study also represents one of the first attempts to use either actigraphy or sleep diaries with this population. Both methods possess limitations, with actigraphy possibly overestimating sleep in those who remain still while awake (44), and sleep diaries being subject to mood-congruent memory and cognitive biases. Standardized methods for acquiring and analyzing actigraphy data in children and adolescents are not well established.

In this preliminary study, we did not control whether sleep recording took place over weekends or weekday nights, or whether individuals participated over holidays or only during school weeks. As can be seen in Table 3, the ADHD-C group had the fewest average number of weeknights in their recording periods (i.e., a slightly higher proportion of weekend nights), although group differences were not significant. For most adolescents, the weekend is a period of longer sleep relative to weekdays (45), yet the ADHD-C group exhibited the shortest mean sleep periods using both sleep diaries and actigraphy. Therefore, it seems likely that if we had controlled the proportion of weeknights and weekend nights, our ADHD-C group would have exhibited even more reduced total sleep time relative to the other groups. Sleep-wake schedules are likely to be more regulated during the school week, but we have no reason to think that our groups differed in terms of participating during school weeks or over holiday breaks.

In summary, we are increasingly aware of the complexity of sleep disturbance in BD. In this study, we found evidence that adolescents with BD, who were between mood episodes, experience their sleep as more fragmented and less satisfying than their peers. However, when assessed using actigraphy, they appeared to sleep longer and with fewer interruptions than their peers. It is unclear whether sleep problems in adolescents with BD would be more objectively detectable during mood episodes or with more sophisticated methods. Although dramatic sleep problems were not consistently manifest in our sample, we emphasize that promoting good sleep practices in adolescents with BD should be a clinical priority, given the established importance of sleep to mood regulation in both healthy and adult BD samples.

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