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Journal

Bipolar Disorders, 21(2)

ISSN

1398-5647

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Publication Date

2019-03-01

DOI

10.1111/bdi.12658

Peer reviewed

HHS Public Access

Author manuscript

Bipolar Disord. Author manuscript; available in PMC 2020 March 01.

Published in final edited form as:

Bipolar Disord. 2019 March ; 21(2): 132–141. doi:10.1111/bdi.12658.

Double Trouble: Weekend Sleep Changes are Associated with Increased Impulsivity among Adolescents with Bipolar I Disorder

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Abstract

Objectives—Both sleep disruption and impulsivity are important predictors of the course of bipolar disorder (BD). Although sleep disruption has been shown to intensify impulsivity, little research has considered how these two important domains interact within BD. Adolescence is a critical period for the onset of BD, and often associated with increases in impulsivity and substantial changes in sleep. We tested the hypothesis that disruptions in sleep would increase impulsivity among adolescents, and that this effect would be more pronounced among those with BD.

Methods—Thirteen to nineteen-year olds diagnosed with BD I (n = 33, 16.2 years old ±1.66 years, 54.5 % female) and psychiatrically healthy controls (n = 26, 15.5 years old ±1.45 years, 55.6 % female) reported their past-week bedtime, rise time, and sleep duration, separately for school days and weekends, and completed a self-report questionnaire on impulsivity. Stepwise regression was used to examine the effects of sleep on impulsivity, and the moderation of this effect by BD status.

Results—Adolescents with BD reported significantly higher impulsivity, later and more variable rise time, and more variable time in bed and sleep duration on school days, than did controls. Greater change in sleep duration between school days and weekends was associated with significantly more impulsivity among adolescents with BD as compared to controls.

Conclusions—These findings highlight the importance of sleep on impulsivity among adolescents with BD and add to the growing evidence that establishing sleep routines may be an important therapeutic target for youth with BD.

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Financial Disclosures

Dr. Gershon receives research support from the National Institute of Mental Health. Dr. Johnson receives research support from the National Institute of Mental Health, the HF Guggenheim foundation, the Kauffman Foundation, and the Sean Costello Memorial Fund for Bipolar Research. Leigh Thomas has no financial disclosures to report. Dr. Singh receives research support from Stanford University's Child Health Research Institute, the National Institute of Mental Health, the Office of Research on Women's Health, the National Institute of Aging, Neuronetics, Johnson and Johnson, and the Brain and Behavior Foundation. She has been on the advisory board for Sunovion.

Keywords

Bipolar Disorder; Adolescents; Sleep; Impulsivity

Bipolar disorder (BD) is a serious and chronic disorder that affects approximately 2 to 4% of the U.S. population (1). Onset occurs by adolescence in at least half of cases (2). Onset during childhood or adolescence is related to particularly devastating long-term outcomes, including higher rates of rapid cycling, recurrences, suicidality, and comorbidities compared to adult onset (2–4). Such findings highlight the critical need for targeted early interventions for youth with BD. Identification of modifiable risk factors is required if progress is to be made in developing effective interventions to improve long-term outcomes for bipolar youth.

Among youth and adult samples, sleep and circadian disturbances are common and persistent features of BD (5, 6). A growing body of research has documented sleep differences in young people with BD relative to controls persist outside of acute periods of (7–9). A large body of work indicates that sleep disturbance predicts functioning and illness course among adults with BD (10). In parallel, the few available studies of youth with BD indicate that sleep disturbances, such as sleep loss and irregular sleep rhythms, predict symptom worsening over time (7, 9). ..

One particular BD correlate that may relate to sleep disturbances is heightened impulsivity. Indeed, one of the criteria for mania involves impulsive engagement in pleasurable activities without regard to potential danger (11). Elevated impulsivity is observed not only during mood episodes (12). but also during remission in BD (13–16) and has been found to predict the onset of BD in young adulthood among those with mild sub-threshold manic symptoms in two studies (17, 18). Elevated impulsivity has been linked to a variety of negative outcomes in individuals with BD, including poorer cognitive function, poorer quality of life, lower medication adherence, greater substance abuse, higher suicide risk, greater risk of non-suicidal self-injury, suicidal ideation, suicide attempts, and more sustained aggression after remission (12, 19–23). Although there are very few available studies of impulsivity in youth with BD, one study of bipolar spectrum youth found that impulsivity was significantly associated with parent-reported suicidal threats (21).

While impulsivity involves a number of components, several studies have now highlighted the importance of one specific facet of impulsivity, the pursuit of rewarding experiences without regard for the potential consequences of these actions, which is measured by the Fun-Seeking subscale of the Behavioral Activation Scale (BAS) (24). Specifically, in a 4.5 year longitudinal study assessing rates of conversion to BD-I or II among 18–24 year olds diagnosed with cyclothymia and BD not otherwise specified (BD-NOS), the Fun-Seeking scale emerged as the primary significant predictor of conversion to BD-II among the personality variables tested, after controlling for family history of BD, baseline mood symptoms, and treatment seeking status (18). In this study, the BAS-Fun Seeking scale also predicted progression to BD I among those diagnosed with BD II, cyclothymia, or BD-NOS (18). Another longitudinal study found that higher BAS-Fun Seeking scores uniquely fluctuated with manic symptom increases among 63 youth (17 to 20 year old) (25). Given

that the Fun Seeking component of impulsivity is a unique risk factor for mood episodes, it is critical to understand which etiological factors contribute to increased impulsivity.

Multiple sources of nonclinical data suggest that sleep may be a critical mechanism that guides fluctuations in impulsivity. Sleep disturbances have been consistently linked to impairment in a range of cognitive functions including decision-making (26). Of more direct relevance, sleep loss has been shown to impair performance on behavioral measures of impulsivity, such as response inhibition (27, 28). Consistent with the idea that even a modest loss of sleep can adversely impact impulsivity, one nonclinical study showed that eight year-old children were rated by their teachers as displaying more restless and impulsive behavior after a sleep restriction protocol of less than one hour per night for five nights (29). More general assessment of sleep disturbances, measured with items such as “sleeps too much” or “overtired”, also related to greater impulsivity in healthy 10–16 year old youth (30). In sum, considerable research suggests that sleep disruptions predict increases in impulsivity. Despite these important links, research in BD has considered sleep and impulsivity separately.

With the growing recognition that sleep and impulsivity are core facets of mood vulnerability, and that these two variables have important interactions in nonclinical samples, understanding the relations between them may lead to important discoveries about how BD develops. To our knowledge, no studies have examined the effects of sleep on impulsivity in BD. This is the primary aim of the current study. Our goal was to understand whether youth with BD would be differentially sensitive to the effects of sleep disturbances on impulsivity. Given that adolescence is a vulnerable period for BD onset and is a period of increased impulsivity and sleep change (31), examining the effects of sleep on impulsivity in youth with BD is important.

Thus, in this study, we consider the interface among sleep, impulsivity, and BD in an adolescent sample. Adolescents tend to show later bedtimes compared to children, possibly due to a natural biological shift in the intrinsic circadian timing system, and/or due to a psychosocial shift towards increased autonomy and increased social activities (32). On school days, early rise times combined with delayed bedtimes often curtail sleep duration, whereas on weekends, adolescents may have more of an opportunity to “catch up” on sleep. However, it is unclear whether such attempts at weekend sleep recovery are sufficient (33). We hypothesized that both BD and healthy control adolescents would report significantly delayed bedtimes and rise times, and longer sleep duration, on weekends relative to school days. We further hypothesized that shifts between school day and weekend sleep duration may represent a marker of sleep disturbance that predicts increased impulsivity. However, we hypothesized that greater changes in sleep duration between weekends and school days would be associated with increased impulsivity, and that those diagnosed with BD would be particularly susceptible to these effects. The current study is unique in considering individuals within one year of their first episode of mania, to avoid the confounds of prolonged illness such as excessive medication exposure, co-occurring substance use or other psychiatric conditions.

Method

Participants

Participants were 59 post-pubertal adolescents, 13–19 years old and fluent in English, recruited from the Stanford University Pediatric Mood Disorders Program or the surrounding San Francisco Bay Area community. Thirty-three were adolescents diagnosed with bipolar I disorder (16.2 years old ±1.66 years, 54.5 % female) and 26 were healthy adolescents without a personal or family history of any psychiatric disorders (15.5 years old ±1.43 years, 57.7 % female). The University research ethics board approved all study procedures. Those age 18 and older provided written informed consent. If under the age of 18, youth provided assent and parents provided written informed consent.

Participants were included in the BD group if they met diagnostic criteria for bipolar I disorder based on the Diagnostic and Statistical Manual (DSM IV-TR) (34), as measured by the Washington University in St. Louis Kiddie-Schedule for Affective Disorders and Schizophrenia (WASH-U KSADS) (35), and had experienced their first manic episode in the past year. Control group individuals were eligible if they did not meet diagnostic criteria for any DSM-IV Axis I disorder and had no first- or second-degree relative with a history of mood disorders or psychosis. Exclusion criteria included a history of neurological or developmental disorders, head injury with loss of consciousness for over 5 minutes, IQ of less than 80, or seizures. Exposure to psychotropic medications was an exclusion criterion for control group participants. Adolescents in the BD group were not excluded for current use psychotropic medication treatment because cessation of medication posed a potential risk of mood destabilization. In addition, because BD disorder often co-occurs with other psychiatric disorders (36), youth in the BD group were not excluded on the basis of current comorbid psychiatric diagnoses, with the exception of alcohol or substance abuse or dependence in the previous six months.

Measures

Demographic information—Parents and adolescents were asked to report age, gender, and ethnicity. The Hollingshead Four Factor Index (37) was used to assess family socioeconomic status.

Pubertal development—The Pubertal Development Scale (38) is a 5-item self-report scale that assessed pubertal status. Items assess for changes in body hair, changes in voice (for boys) or breast development (for girls), skin change, growth spurts, and facial hair (for boys) and menarche (for girls). In a large sample of sixth- and seventh-grade boys and girls (N=253), the measure was shown to have good reliability (α = 0.68 to 0.87) and high correlations between a questionnaire version and physician ratings (r = 0.61 to 0.67) (38).

Psychiatric diagnosis and current symptoms—To assess for current and lifetime psychiatric disorders, the Washington University in St. Louis Kiddie-Schedule for Affective Disorders and Schizophrenia (WASH-U KSADS) (35) was administered separately to participants and their parents. The WASH-U KSADS has been shown to have high inter-rater reliability for both BD diagnosis and individual mania items (39). A board-certified

child and adolescent psychiatrist (MKS) and Master's level researchers reviewed diagnoses by consensus for the present study (K > 0.90 for all diagnoses). Youth in the control group were required to have no personal or family history of mood disorders or psychosis in first or second-degree relatives as verified by the Family History-Research Diagnostic Criteria (FH-RDC) (40).

Children's Global Assessment Scale (C-GAS). Current global overall functioning was assessed by interviewers using the Children's Global Assessment Scale (C-GAS). The reliability and validity of the C-GAS have been established (41).

The Children's Depression Rating Scale-Revised (CDRS-R) (42), a 17-item clinician-administered scale, was administered to assess depression symptom severity in participants. Total scores range from 17 to 113. Higher scores indicate greater depression severity. The CDRS-R has demonstrated good reliability and validity in adolescents 7–18 years old (43).

The Young Mania Rating Scale (YMRS) (44), an 11-item clinician-administered scale, was used to assess mania symptom severity. Total scores range from 0 to 60. Higher scores indicate greater symptom severity. The measure is widely used and has exhibited good psychometric properties (44), including validation for use with children as young as 5 years old, with demonstrated reliability for measuring mania symptoms and excellent ability to separate bipolar from non-bipolar cases (45, 46).

Sleep—Adolescents were asked to complete self-report questionnaires to measure the average bedtime, rise time, and total sleep time for the past week, assessing these variables separately for weekdays versus weekends.

Impulsivity—Adolescents were administered the Fun-Seeking subscale of the Behavioral Activation Scale (BAS) (24). The BAS Fun-seeking subscale is at trait measure of the tendency to pursue new and potentially rewarding experiences (e.g., "I will often do things for no other reason than that they might be fun") and to act reflexively during goal pursuit (e.g., "I often act on the spur of the moment") without regard for the potential consequences of these actions. Impulsivity is often guided by poor decision-making. Scores range from 4 to 16. Higher scores indicate greater impulsivity. The BAS scales have been validated against psychiatric diagnoses (47), neural response to reward (48), and more specific to the current study, have been found to predict the onset and course of BD (18, 49).

Statistical Analysis

To evaluate for potential confounds, preliminary analyses examined group differences on demographic variables (age, gender, ethnicity, socioeconomic status, and pubertal development) using t-tests for continuous variables and chi-square tests for categorical variables. For chi-square analyses, Fisher's exact test was used when the expected cell size was < 5. We also considered group differences on clinician-rated symptom severity scales (CDRS-R and YMRS) and function (C-GAS) using t-tests. Then, to consider whether impulsivity and sleep related to diagnostic status, t-tests were used to examine group differences in self-reported bedtimes, rise times, time in bed, and total sleep time for school days and weekends separately, and the BAS Fun-Seeking scale. Pearson product correlations

were used to examine inter-correlations of sleep and impulsivity measures. To examine the primary hypothesis that those with BD would be particularly sensitive to the effects of sleep disruption on impulsivity, we conducted a regression model with BAS Fun-seeking scores as the outcome index. Diagnostic group, the effects of bedtime on school days, time in bed on school days, and change in total sleep time between school days and weekends, were entered using forced entry, and the three interactions of Diagnostic group x Sleep variables were entered using forward selection. Variables were z-transformed before entry in regression models. Within-group correlations were used to describe the form of the interaction between sleep and impulsivity. All tests were completed using SPSS for Windows (Version 23.0; SPSS v22.0, IBM Corporation, Armonk, New York).

Results

Kolmogorov-Smirnov tests for normality indicated non-normal distributions for bedtime on school days ($p = 0.036$), rise time on school days ($p < 0.001$), time in bed on school days ($p = 0.013$), bedtime on weekend days ($p < 0.001$), rise time on weekend days ($p = 0.001$), total sleep time on weekend days ($p < 0.001$), and BAS-Fun Seeking ($p = 0.017$). Rise time on school days and total sleep time on weekend days both had kurtosis values > 1.5. Log transformations were used to normalize the non-normal distributions; the transformed variables were used in analysis.

Demographic and clinical characteristics are presented in Table 1. The BD and control groups were well matched, in that they did not differ significantly in age, gender, ethnicity, socioeconomic status, or pubertal development status. As expected, BD group participants scored significantly lower on the C-GAS, and significantly higher on the YMRS and CDRS-R than did control group participants. Twenty-seven of 33 BD group participants (81.8%) and 0% of the control participants were taking psychotropic medications. These included antipsychotics, anticonvulsants, selective serotonin reuptake inhibitors (SSRIs), stimulants, lithium, bupropion, serotonin antagonist reuptake inhibitors (SARIs), tricyclic antidepressants (TCAs), tetracyclic antidepressant (TeCAs), or two or more of these. Seventeen of 33 BD group participants (51.5%) and 0% of the control group had at least one comorbid psychiatric diagnosis, including attention deficit and hyperactivity disorder ($n = 9$), past substance abuse or dependence ($n = 8$), anxiety disorders ($n = 5$), oppositional defiant disorder ($n = 5$), dysthymia ($n = 4$), conduct disorder ($n = 1$), and bulimia nervosa ($n = 1$).

Diagnostic group differences in sleep and impulsivity

As shown in Table 2, on school days, BD group participants reported a significantly later rise time than did controls. Psychotropic medication use (antipsychotics, anticonvulsants, SSRIs, stimulants, lithium, bupropion, SARIs, TCAs, TeCAs, or two or more of these) was not significantly related to rise time on school days (rs range from −0.15 to 0.29, $n = 33$). The groups did not differ in the mean values of any other sleep variables. However, relative to control group, the BD group showed significantly more variable rise time, time in bed, and total sleep time on school days. Although the BD group was more varied on the BAS Fun Seeking scale, their mean score was not significantly different than the control group.

Effects of sleep on impulsivity

Before testing for the effects of sleep on impulsivity we examined the inter-correlations of our measures. As shown in Table 3, sleep variables showed expected correlations. That is, on school days, both bedtime and rise time were significantly positively correlated with time in bed. School day rise time and time in bed were also significantly positively correlated with school day sleep duration. School day bedtime was significantly negatively correlated with school day sleep duration and with weekend time in bed. Weekend rise time was positively correlated with weekend time in bed. Weekend sleep duration was negatively correlated with school day rise time, school day time in bed, and school day sleep duration, and positively correlated with weekend rise time. As predicted, impulsivity was significantly related to change in sleep duration between school days and weekend days for both groups.

Regression models were constructed to test whether sleep effects differed by diagnostic group. As noted above, we found no group differences in demographic variables and therefore did not include them in our regression models. As shown in Table 4, the change in sleep duration between school days and weekends was significantly related to Fun Seeking, and the significant interaction of Group indicated that this effect was moderated by diagnostic group. Follow-up correlations were conducted to examine the role of change in sleep duration on impulsivity in each diagnostic group. Change in sleep duration was significantly related to greater Fun Seeking in adolescents with BD ($r = .503$, $p = .005$, $n = 26$), but not in controls ($r = -.018$, $p = .93$, $n = 25$).

Discussion

As predicted, adolescents with BD did not differ from control youth on most of the sleep indices measured. However, a greater change in sleep duration between school days and weekends was associated with greater impulsivity in adolescents with BD but not in controls. Our study is the first to show that an otherwise normative developmental change in sleep, whereby adolescents tend to sleep longer or “catch up” on sleep during the weekends relative to school days, may be associated with negative outcomes for BD relative to control youth. Specifically, our finding suggests that for youth with BD, the change in sleep duration between school days and weekends is associated with increased impulsivity and, as such, may require closer monitoring to address sleep schedule patterns that may be detrimental to impulse control among BD youth.

In this study, adolescents with BD showed a very specific profile of sleep disturbance compared to control youth. That is, they did not differ in their report of most of the sleep variables assessed with the exception that adolescents with BD reported significantly later rise times (nearly one hour later) on school days relative to controls. Significantly later rise times on school days may reflect a more pronounced circadian phase delay among BD youth relative to controls. Although circadian phase was not systematically assessed in the current study, studies have consistently suggested an association between BD and delayed circadian phase, that is, a preference for later rather than earlier timing of sleep and daily activities (50). Although adolescents with BD had near significantly longer time in bed on school days ($p = .052$), there was no group difference in sleep duration, consistent with previous reports of adequate sleep duration in youth with BD (6, 51). Importantly, however, our findings

show that adolescents with BD exhibit significantly greater variability in rise time, time in bed, and total sleep time on school nights than do controls. Variability in sleep rhythms has been documented in previous studies of youth with BD (9) and may represent an important marker of individual differences to consider in future research.

Interestingly, we did not find an association between sleep disturbance and impulsivity in the control group. It may be the case that sleep disturbance in the control group is associated with other functional outcomes that are related to impulsivity. Previous reports have found that sleep disturbances in healthy youth is associated with emotion regulation deficits (52), poorer academic performance (53), and increased delinquency (54). At the same time, researchers have found that in psychiatrically healthy populations, it takes greater neurocognitive disturbance to lead to impulsive behavior than it does in psychiatric populations (55). That is, we might have observed effects of sleep disturbances on impulsivity had we captured more severe sleep disruptions in the healthy controls.

Several limitations should be noted. First, sleep indices were assessed via self-report and, as such, susceptible to memory bias. We assessed sleep over the past week to reduce the potential for this bias. Nevertheless, our assessment would have benefitted from the addition of objective measures of sleep (e.g., actigraphy). Second, our small sample size limited power, and could have contributed to some of the null effects observed in comparing sleep and impulsivity between those with and without BD. Third, given the cross-sectional design, we cannot delineate the direction of effects. It is possible that impulsivity may be driving sleep disruption, rather than sleep disruption driving impulsivity. Fourth, we measured impulsivity using a single self-report scale. Although the BAS-Fun Seeking scale was chosen due to its demonstrated unique relation to BD vulnerability, future studies should incorporate a more comprehensive assessment of impulsivity, including behavioral tasks such as the Balloon Analogue Risk Task (56). Fifth, chronotype (preference for timing of sleep and daily activities) could play a role in the effects of sleep dysregulation on impulsivity. Future research should examine chronotype in relation to impulsivity in BD samples. Finally, due to the absence of a psychiatric comparison group, we cannot ascertain whether the identified between-group sleep differences are due to general psychopathology rather than BD, per se (57). It is possible that sleep problems contribute to the impulsivity that is increasingly documented transdiagnostically (58).

Despite these limitations, the findings underscore that the change in sleep duration that occurs between school days and weekends may be an important target for treatment in youth with BD. Several techniques have been developed to address to irregular or delayed sleep schedules. These include administration of melatonin to address later bed times and rise times (59) and behavioral modifications, such as maintenance of a consistent sleep schedule across the days of the week (60). Current findings suggest that these types of interventions may be particularly important to consider in BD. Indeed, recent findings in adults such that CBT for insomnia is promising in reducing mood symptoms in BD (61), but effects of such interventions on reducing impulsivity remain unknown. Future interventions may need to consider the important relations between sleep and impulsivity in order to yield tractable outcomes for adolescent patients with BD.

Acknowledgments

This research was supported by the National Institute of Mental Health (NIMH) Research Scientist Development Award K01MH100433 to AG and an NIMH R01MH105469, K23MH085919, the Klingenstein Third Generation Foundation, and American Psychiatric Association to MKS.

References

1. Merikangas KR, Akiskal HS, Angst J, Greenberg PE, Hirschfeld RM, Petukhova M, et al. Lifetime and 12-month prevalence of bipolar spectrum disorder in the National Comorbidity Survey replication. Archives of General Psychiatry. 2007; 64:543–52. [PubMed: 17485606]
2. Perlis RH, Miyahara S, Marangell LB, Wisniewski SR, Ostacher M, DelBello MP, et al. Long-term implications of early onset in bipolar disorder: data from the first 1000 participants in the systematic treatment enhancement program for bipolar disorder (STEP-BD). Biological Psychiatry. 2004; 55:875–81. [PubMed: 15110730]
3. Birmaher B, Axelson D, Goldstein B, Strober M, Gill MK, Hunt J, et al. Four-year longitudinal course of children and adolescents with bipolar spectrum disorders: the Course and Outcome of Bipolar Youth (COBY) study. Americal Journal of Psychiatry. 2009; 166:795–804.
4. Lewinsohn PM, Seeley JR, Buckley ME, Klein DN. Bipolar disorder in adolescence and young adulthood. Child and Adolescent Psychiatric Clinics of North America. 2002; 11:461–75. vii. [PubMed: 12222078]
5. Harvey AG, Schmidt DA, Scarna A, Semler CN, Goodwin GM. Sleep-related functioning in euthymic patients with bipolar disorder, patients with insomnia, and subjects without sleep problems. American Journal of Psychiatry. 2005; 162:50–7. [PubMed: 15625201]
6. Roybal DJ, Chang KD, Chen MC, Howe ME, Gotlib IH, Singh MK. Characterization and factors associated with sleep quality in adolescents with bipolar I disorder. Child Psychiatry and Human Development. 2011; 42:724–40. [PubMed: 21701911]
7. Gershon A, Singh MK. Sleep in Adolescents With Bipolar I Disorder: Stability and Relation to Symptom Change. Journal of Clinical Child & Adolescent Psychology. 2017; 46:247–57. [PubMed: 27472039]
8. Levenson JC, Axelson DA, Merranko J, Angulo M, Goldstein TR, Mullin BC, et al. Differences in sleep disturbances among offspring of parents with and without bipolar disorder: association with conversion to bipolar disorder. Bipolar Disorders. 2015; 17:836–48. [PubMed: 26547512]
9. Lunsford-Avery JR, Judd CM, Axelson DA, Miklowitz DJ. Sleep impairment, mood symptoms, and psychosocial functioning in adolescent bipolar disorder. Psychiatry Research. 2012; 200:265–71. [PubMed: 22884306]
10. Harvey AG. Sleep and circadian functioning: critical mechanisms in the mood disorders? Annual Review of Clinical Psychology. 2011; 7:297–319.
11. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5. Washington, DC: American Psychiatric Press; 2013.
12. Strakowski SM, Fleck DE, DelBello MP, Adler CM, Shear PK, McElroy SL, et al. Characterizing impulsivity in mania. Bipolar Disorders. 2009; 11:41–51. [PubMed: 19133965]
13. Gilbert KE, Kalmar JH, Womer FY, Markovich PJ, Pittman B, Nolen-Hoeksema S, et al. Impulsivity in Adolescent Bipolar Disorder. Acta Neuropsychiatrica. 2011; 23:57–61. [PubMed: 21483649]
14. Nandagopal JJ, Fleck DE, Adler CM, Mills NP, Strakowski SM, DelBello MP. Impulsivity in adolescents with bipolar disorder and/or attention-deficit/hyperactivity disorder and healthy controls as measured by the Barratt Impulsiveness Scale. Journal of Child and Adolescent Psychopharmacology. 2011; 21:465–8. [PubMed: 22040191]
15. Swann AC, Moeller FG, Steinberg JL, Schneider L, Barratt ES, Dougherty DM. Manic symptoms and impulsivity during bipolar depressive episodes. Bipolar Disorders. 2007; 9:206–12. [PubMed: 17430294]
16. Swann AC, Pazzaglia P, Nicholls A, Dougherty DM, Moeller FG. Impulsivity and phase of illness in bipolar disorder. Journal of Affective Disorders. 2003; 73:105–11. [PubMed: 12507743]

17. Kwapil TR, Miller MB, Zinser MC, Chapman LJ, Chapman J, Eckblad M. A longitudinal study of high scorers on the hypomanic personality scale. Journal of Abnormal Psychology. 2000; 109:222–6. [PubMed: 10895560]
18. Alloy LB, Urosevic S, Abramson LY, Jager-Hyman S, Nusslock R, Whitehouse WG, et al. Progression along the bipolar spectrum: a longitudinal study of predictors of conversion from bipolar spectrum conditions to bipolar I and II disorders. Journal of Abnormal Psychology. 2012; 121:16–27. [PubMed: 21668080]
19. Johnson SL, Carver CS, Tharp JA. Suicidality in bipolar disorder: The role of emotion-triggered impulsivity. Suicide and Life-Threatening Behavior. 2016
20. Muhtadie L, Johnson SL, Carver CS, Gotlib IH, Ketter TA. A profile approach to impulsivity in bipolar disorder: the key role of strong emotions. Acta Psychiatrica Scandinavica. 2014; 129:100–8. [PubMed: 23600731]
21. Papolos D, Hennen J, Cockerham MS. Factors associated with parent-reported suicide threats by children and adolescents with community-diagnosed bipolar disorder. Journal of Affective Disorders. 2005; 86:267–75. [PubMed: 15935246]
22. Swann AC, Dougherty DM, Pazzaglia PJ, Pham M, Moeller FG. Impulsivity: a link between bipolar disorder and substance abuse. Bipolar Disorders. 2004; 6:204–12. [PubMed: 15117399]
23. Victor SE, Johnson SL, Gotlib IH. Quality of life and impulsivity in bipolar disorder. Bipolar Disorders. 2011; 13:303–9. [PubMed: 21676133]
24. Carver CS, White TL. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. Journal of Personality and Social Psychology. 1994; 67:319–33.
25. Meyer B, Johnson SL, Carver CS. Exploring behavioral activation and inhibition sensitivities among college students at risk for bipolar spectrum symptomatology. Journal of Psychopathology and Behavior Assessment. 1999; 21:275–92.
26. Killgore WD, Balkin TJ, Wesensten NJ. Impaired decision making following 49 h of sleep deprivation. Journal of Sleep Research. 2006; 15:7–13. [PubMed: 16489997]
27. Anderson C, Platten CR. Sleep deprivation lowers inhibition and enhances impulsivity to negative stimuli. Behavioral Brain Research. 2011; 217:463–6.
28. Drummond SP, Paulus MP, Tapert SF. Effects of two nights sleep deprivation and two nights recovery sleep on response inhibition. Journal of Sleep Research. 2006; 15:261–5. [PubMed: 16911028]
29. Gruber R, Cassoff J, Frenette S, Wiebe S, Carrier J. Impact of sleep extension and restriction on children's emotional lability and impulsivity. Pediatrics. 2012; 130:e1155–61. [PubMed: 23071214]
30. Moore M, Slane J, Mindell JA, Burt SA, Klump KL. Sleep problems and temperament in adolescents. Child: Care, Health, and Development. 2011; 37:559–62.
31. Carskadon MA, Acebo C, Jenni OG. Regulation of adolescent sleep: implications for behavior. Annals of the New York Academy of Sciences. 2004; 1021:276–91. [PubMed: 15251897]
32. Carskadon, MA. Factors influencing sleep patterns of adolescents. In: Carskadon, MA, editor. Adolescent sleep patterns: Biological, social, and psychological influences. New York: Cambridge University Press; 2002. 4–26.
33. Agostini A, Carskadon MA, Dorrian J, Coussens S, Short MA. An experimental study of adolescent sleep restriction during a simulated school week: changes in phase, sleep staging, performance and sleepiness. Journal of Sleep Research. 2017; 26:227–35. [PubMed: 27868260]
34. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4. Washington, DC: American Psychiatric Press; 2000. Text-Revision ed
35. Geller, B, Williams, M, Zimerman, B, Frazier, J. Washington University in St Louis Kiddie Schedule for affective disorders and schizophrenia (WASH-U-KSADS). St. Louis: Washington University; 1996.
36. Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry. 2005; 62:617–27. [PubMed: 15939839]
37. Hollingshead, AA. Four-Factor Index of Social Status. New Haven, CT: Yale University; 1975.

38. Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. Journal of Youth and Adolescence. 1988; 17:117–33. [PubMed: 24277579]
39. Geller B, Zimerman B, Williams M, Bolhofner K, Craney JL, DelBello MP, et al. Reliability of the Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS) mania and rapid cycling sections. J Am Acad Child Adolesc Psychiatry. 2001; 40:450–5. [PubMed: 11314571]
40. Andreasen NC, Endicott J, Spitzer RL, Winokur G. The family history method using diagnostic criteria. Reliability and validity. Archives of General Psychiatry. 1977; 34:1229–35. [PubMed: 911222]
41. Shaffer D, Gould MS, Brasic J, Ambrosini P, Fisher P, Bird H, et al. A children's global assessment scale (CGAS). Archives of General Psychiatry. 1983; 40:1228–31. [PubMed: 6639293]
42. Poznanski, EO, Mokros, HB. Children's Depression Rating Scale, Revised (CDRS-R) Manual. Los Angeles: California Western Psychological Services; 1995.
43. Mayes TL, Bernstein IH, Haley CL, Kennard BD, Emslie GJ. Psychometric properties of the Children's Depression Rating Scale-Revised in adolescents. Journal of Child and Adolescent Psychopharmacology. 2010; 20:513–6. [PubMed: 21186970]
44. Young RC, Biggs JT, Ziegler VE, Meyer DA. A rating scale for mania: reliability, validity and sensitivity. The British journal of psychiatry : the journal of mental science. 1978; 133:429–35. [PubMed: 728692]
45. Frazier TW, Demeter CA, Youngstrom EA, Calabrese JR, Stansbrey RJ, McNamara NK, et al. Evaluation and comparison of psychometric instruments for pediatric bipolar spectrum disorders in four age groups. Journal of Child & Adolescent Psychopharmacology. 2007; 17:853–66. [PubMed: 18315456]
46. Youngstrom EA, Danielson CK, Findling RL, Gracious BL, Calabrese JR. Factor structure of the Young Mania Rating Scale for use with youths ages 5 to 17 years. Journal of Clinical Child & Adolescent Psychology. 2002; 31:567–72. [PubMed: 12402575]
47. Johnson SL, Turner RJ, Iwata N. BIS/BAS levels and psychiatric disorder: An epidemiological study. Journal of Psychopathology and Behavioral Assessment. 2003; 25:25–36.
48. Kim SH, Yoon H, Kim H, Hamann S. Individual differences in sensitivity to reward and punishment and neural activity during reward and avoidance learning. Social Cognitive and Affective Neuroscience. 2015; 10:1219–27. [PubMed: 25680989]
49. Meyer B, Johnson SL, Winters R. Responsiveness to threat and incentive in bipolar disorder: Relations of the BIS/BAS scales with symptoms. Journal of Psychopathology & Behavioral Assessment. 2001; 23:133–43. [PubMed: 21765592]
50. Melo MC, Abreu RL, Linhares Neto VB, de Bruin PF, de Bruin VM. Chronotype and circadian rhythm in bipolar disorder: A systematic review. Sleep Medicine Reviews. 2016
51. Mullin BC, Harvey AG, Hinshaw SP. A preliminary study of sleep in adolescents with bipolar disorder, ADHD, and non-patient controls. Bipolar Disorders. 2011; 13:425–32. [PubMed: 21843282]
52. Baum KT, Desai A, Field J, Miller LE, Rausch J, Beebe DW. Sleep restriction worsens mood and emotion regulation in adolescents. Journal of Child Psychology and Psychiatry, and Allied Disciplines. 2014; 55:180–90.
53. Lee YJ, Park J, Kim S, Cho SJ, Kim SJ. Academic performance among adolescents with behaviorally induced insufficient sleep syndrome. Journal of Clinical Sleep Medicine. 2015; 11:61–8. [PubMed: 25515277]
54. Clinkinbeard SS, Simi P, Evans MK, Anderson AL. Sleep and delinquency: does the amount of sleep matter? J Youth Adolesc. 2011; 40:916–30. [PubMed: 20936500]
55. Johnson SL, Tharp JA, Peckham AD, Sanchez AH, Carver CS. Positive urgency is related to difficulty inhibiting prepotent responses. Emotion. 2016; 16:750–9. [PubMed: 27064288]
56. Lejuez CW, Read JP, Kahler CW, Richards JB, Ramsey SE, Stuart GL, et al. Evaluation of a behavioral measure of risk taking: the Balloon Analogue Risk Task (BART). Journal of Experimental Psychology. 2002; 8:75–84. [PubMed: 12075692]

57. Robillard R, Hermens DF, Naismith SL, White D, Rogers NL, Ip TK, et al. Ambulatory sleep-wake patterns and variability in young people with emerging mental disorders. Journal of Psychiatry & Neuroscience. 2015; 40:28–37. [PubMed: 25203899]

58. Berg JM, Latzman RD, Bliwise NG, Lilienfeld SO. Parsing the heterogeneity of impulsivity: A meta-analytic review of the behavioral implications of the UPPS for psychopathology. Psychological Assessment. 2015; 27:1129–46. [PubMed: 25822833]

59. Eckerberg B, Lowden A, Nagai R, Akerstedt T. Melatonin treatment effects on adolescent students' sleep timing and sleepiness in a placebo-controlled crossover study. Chronobiology International. 2012; 29:1239–48. [PubMed: 23005039]

60. Frank, E. Treating bipolar disorder: A clinician's guide to interpersonal and social rhythm therapy. New York: Guilford Press; 2005.

61. Harvey AG, Soehner AM, Kaplan KA, Hein K, Lee J, Kanady J, et al. Treating insomnia improves mood state, sleep, and functioning in bipolar disorder: a pilot randomized controlled trial. Journal of Consulting and Clinical Psychology. 2015; 83:564–77. [PubMed: 25622197]

Table 1

Demographic and Clinical Characteristics of Study Participants by Group

	BD (n=33)	Control (n=26)	χ^2 or *t* value	df	*p value*
Age in years	16.2±1.66	15.5±1.43	1.63	57	0.11
Gender, n (%) female	18 (54.5)	15 (57.7)	0.01	1	0.94
Caucasian, n (%)	22 (66.7)	15 (57.7)	8.61	5	0.13
Hollingshead Index	4.73±0.52	4.64±0.64	0.58	56	0.57
PDS Score	3.33±0.45	3.16±0.57	1.28	57	0.21
Current mood state					
Inter-episode, n (%)	11 (33)	-	-	-	-
Depressed, n (%)	10 (30)	-	-	-	-
Manic or hypomanic, n (%)	7 (21)	-	-	-	-
Mixed, n (%)	5 (15)	-	-	-	-
C-GAS score	53.7±13.6	86.5±25.2	6.25	57	<0.001
YMRS score	18.4±7.41	0.25±0.53	14.0	55	<0.001
CDRS-R score	45.2±14.0	18.1±1.54	11.03	55	<0.001
Current medications					
Antipsychotics, n (%)	23 (70)	-	-	-	-
Anticonvulsants, n (%)	17 (52)	-	-	-	-
SSRIs, n (%)	15 (45)	-	-	-	-
Stimulants, n (%)	11 (33)	-	-	-	-
Lithium, n (%)	8 (24)	-	-	-	-
Bupropion, n (%)	5 (15)	-	-	-	-
SARIs, n (%)	2 (6)	-	-	-	-
TCAs, n (%)	1 (3)	-	-	-	-
TeCAs, n (%)	1 (3)	-	-	-	-
Two or more classes, n (%)	23 (70)	-	-	-	-

Note. SD, standard deviation; PDS, Pubertal Development Scale; C-GAS, Children's Global Assessment Scale; YMRS, Young Mania Rating Scale. CDRS-R, Child Depression Rating Scale - Revised. SSRIs, selective serotonin reuptake inhibitors; SARIs, serotonin antagonist reuptake inhibitors; TCAs, tricyclic antidepressants; TeCAs, tetracyclic antidepressants. One control group participant was missing Hollingshead Index score and two control group participants were missing YMRS and CDRS-R scores. Underlines indicate *t* (rather than χ^2) values.

Table 2

Means, Standard Deviations, and Levene's Test for Homogeneity of Variances of Sleep and Impulsivity Variables by Group, and T-tests for Mean Differences Between Groups.

	BD (n=33)	Control (n=26)	Levene's F Test	df	*p value*	*t*-test for difference between means	df	*p value*
Sleep on School Days								
Bed time	22:19 (1:09)	22:23 (0:55)	2.14	57	0.149	−0.28	57	0.78
Rise time	7:45 (1:31)	6:53 (0:31)	19.1	57	<0.001	2.55	57	0.008
Time in bed (minutes)	566.2 (118.6)	510.2 (59.4)	6.23	57	0.016	1.98	57	0.052
Total sleep time (minutes)	465.6 (152.1)	475.0 (64.7)	6.94	56	0.011	−0.33	55	0.74
Sleep on Weekend Days								
Bed time	23:08 (1:35)	23:21 (1:25)	3.52	57	0.07	1.06	57	0.30
Rise time	9:36 (1:20)	9:23 (1:24)	0.25	55	0.62	0.64	55	0.52
Time in bed (minutes)	615.2 (102.5)	601.7 (107.9)	0.97	55	0.48	0.63	55	0.58
Total sleep time (minutes)	527.8 (100.1)	567.7 (103.9)	0.22	52	0.65	−1.18	52	0.25
Change in total sleep time between school days and weekend days	98.8 (86.0)	114.6 (80.7)	0.001	52	0.98	−0.70	52	0.49
Impulsivity								
BAS Fun seeking	12.1 (2.5)	11.7 (1.5)	4.124	57	0.05	0.70	57	0.49

Note. SD, standard deviation; BAS, Behavioral Activation Scale. Bold font indicates statistical significance. One bipolar group participant and one control group participant were missing school day total sleep time. Two bipolar group participants were missing weekend rise time, time in bed and total sleep time. Two additional bipolar group participants and one control group participant were missing weekend total sleep time.

Table 3

Pearson's Product–Moment Correlations of Sleep and Impulsivity Variables

	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
(1) School Bed time	−0.046	0.684***	−0.321*	−0.005	0.211	−0.408**	−0.188	0.134	0.001
(2) School Rise time	--	0.748***	0.408**	0.034	0.085	0.087	0.049	−0.319*	−0.159
(3) School Time in bed	--	--	0.536***	0.022	−0.110	0.319*	0.158	−0.348**	−0.128
(4) School Total sleep time	--	--	--	0.076	−0.139	0.165	0.316*	−0.379**	−0.125
(5) Weekend Bed time	--	--	--	--	−0.188	−0.186	−0.153	−0.188	0.079
(6) Weekend Rise time	--	--	--	--	--	0.449***	0.372**	0.441**	0.166
(7) Weekend Time in bed	--	--	--	--	--	--	0.718***	0.082	−0.007
(8) Weekend Total sleep time	--	--	--	--	--	--	--	−0.076	−0.108
(9) Change in Total sleep time	--	--	--	--	--	--	--	--	0.318*
(10) BAS Fun Seeking	--	--	--	--	--	--	--	--	--

Note. BAS, Behavioral Activation Scale.

*$p < .05$.

**$p < .01$.

***$p < .001$.

Table 4

Linear Regression Using Group, Sleep Variables, and the Interaction of Group by Sleep Variables to Predict BAS Fun Seeking Scores

Predictors	B (95% CI)	SE	β	*t value*	*p value*	R^2	df1, df2	*F*	*p value*	ΔR^2	*F Change*	Sig. *F* Change
Model 1												
(Constant)	−0.003 (−0.279,0.272)	0.137		−0.024	0.981							
Group	−0.158 (−0.457,0.141)	0.149	−0.153	−1.060	0.294							
School Bed time	−0.145 (−0.518,0.228)	0.186	−0.139	−0.780	0.439	0.126	4,49	1.769	0.150	0.126	1.769	0.150
School Time in bed	−0.124 (−0.531,0.284)	0.203	−0.118	−0.610	0.544							
Change in Total sleep time	0.326 (0.033,0.620)	0.146	0.315	2.235	0.030							
Model 2												
(Constant)	−0.015 (−0.280,0.250)	0.132		−0.111	0.912							
Group	0.257 (−0.211, −0.724)	0.232	0.249	1.104	0.275							
School Bed time	−0.122 (−0.481, −0.237)	0.179	−0.117	−0.683	0.498	0.211	5,48	2.560	0.039	0.084	5.127	0.028
School Time in bed	−0.069 (−0.464,0.326)	0.196	−0.066	−0.352	0.726							
Change in Total sleep time	1.219 (0.378,2.060)	0.418	1.176	2.913	0.005							
Group x Change in Total sleep time	−1.047 (−1.976, −0.117)	0.462	−1.010	−2.264	0.028							

Note. BAS, Behavioral Activation Scale.