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ORIGINAL ARTICLE

Sleep-Wake Patterns of Adolescents with Borderline Personality Disorder and Bipolar Disorder

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Abstract Sleep-wake patterns are rarely examined in adolescents with borderline personality disorder (BPD) or bipolar disorder (BD). Within a developmental perspective, this study explores the sleep-wake cycle of adolescents aged 12–17 years with BPD or BD and healthy controls (HC) during periods with and without entrainment by school/work schedules. Eighteen euthymic BPD, six euthymic BD, and 20 HC adolescents wore wrist actigraphy during nine consecutive days to assess sleep-wake patterns. During school/work days, BPD adolescents spent more time awake when they were in bed compared to HC and BD adolescents ($p = 0.039$). On schedule-free days, BPD and BD youths spent more time in bed compared to HC adolescents ($p = 0.015$). BPD adolescents woke up over 1 h later compared to HC ($p = 0.003$). Total sleep time was more variable between nights in BPD adolescents compared to the HC group ($p = 0.031$). Future research should explore if sleep-wake pattern disruptions are a cause or a consequence of BPD symptomatology in adolescents. Addressing sleep-wake pattern during clinical assessment and treatment of BPD adolescents may potentially reduce their symptoms; this therapeutic effect still needs to be evaluated.

Keywords Actigraphy · Sleep · Adolescent · Borderline personality disorder · Bipolar disorder · Circadian rhythm

Introduction

Context

The issue of sleep in psychiatric disorders characterised by emotional dysregulation, such as borderline personality disorder (BPD) and bipolar disorder (BD), has generated considerable attention in adult patients over the last decades. As such, questionnaire studies show that BPD patients have more sleep complaints, use more hypnotics, and report more nightmares and negative toned dreams [1–4]. Reviews of polysomnography studies highlight the presence of longer sleep latencies, a higher number of night awakenings, reduced rapid eye movement (REM) sleep latencies, longer REM sleep duration, and higher REM density in BPD adult patients compared to healthy controls (HCs) [5, 6]. Since BPD patients show comparable sleep disturbances to patients with major depressive disorder, such as delayed sleep onset and reduced total sleep time [7–10], studies must examine sleep parameters when patients are free of mood symptoms in order to control for depression as a confounding factor [5]. Assessing BPD patients without depressive disorders allows determining what sleep disturbances are more specific to this personality disorder, therefore improving knowledge about its aetiology. Although the rate of a cormobid DSM-IV mood disorder is high (55.3–58.7 %) in BPD adolescents [11, 12], they do not all have major depressive disorder or

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dysthymia. Even if BPD and major depressive disorder show overlap of symptoms, they are two distinct conditions which do not respond to antidepressant medication in a similar way [13]. Depressive symptoms in BPD are qualitatively different from major depression: they are more transient and stress-related, can disappear rapidly when the conflict is resolved, serve to express distress and are characterised by feelings of loneliness, emptiness and boredom, by clinging dependency, by unstable negative affect and by a deep sense of badness [13]. Affective instability in BPD usually involves fluctuating states including anger, anxiety and even euthymia, and not only depression [14]. Therefore, studies need to be conducted without the presence of its main confounder, i.e. depressive disorders, to better understand aetiology, course and treatment specific to BPD.

Sleep disturbances are also commonly observed in BD patients, particularly during acute mood episodes. Reduction of sleep need during mania and presence of insomnia/hypersomnia during depressive states are recognized as diagnostic features of BD according to DSM-IV and ICD-10 criteria [15, 16]. A systematic literature review has calculated the median prevalence of early symptoms in BD and reported that sleep disturbance is the most important prodrome for a maniac episode and it is the sixth most frequently observed prodrome for a depressive episode [17]. Furthermore, 23.6 % of adult BD patients still suffer from insomnia during euthymia (between mood episodes), and more than half are receiving sedating medication (sedating antipsychotics, benzodiazepines, sleeping aids) [18]. Actigraphy results show that euthymic BD adults also exhibit a longer sleep latency and higher interdaily variability of the sleep-wake rhythm than healthy adults [19, 20].

Many studies have highlighted the importance of adopting a developmental perspective when studying sleep, since adolescence is characterized by specific sleep features: sleep need during adolescence is about 10 h per night, slow wave sleep time decreases by 40 % during that age period, and level of daytime sleep tendency increases during midpuberty [21]. Furthermore, adolescents present a biological phase delay of their circadian rhythms (i.e. compared to children and adults, they have later bedtimes and rise times, melatonin secretion occurs later in the evening, etc.) [22–24]. In humans, this shift is strengthened by social obligations: part-time jobs, more homework, extracurricular activities, friendships and love, etc. These different biological, social and psychological factors create a common phenomenon which is being reported across the globe: adolescents are not getting enough sleep [25]. The deleterious effect of sleep disturbances on cognition and emotion in adolescents, especially those with psychiatric disorders, has been recognised over the past decades [26–30].

Sleep researches in adolescent psychiatry have focused on depression, attention deficit hyperactivity disorder (ADHD), autistic spectrum disorders, substance abuse and anxiety disorders. However, there are few studies on BD and BPD during adolescence, even though there is a solid scientific body of knowledge on the topic in adults. Studying specifically BD and BPD is relevant since both disorders are characterised by emotional dysregulation and impulsive behaviours which may increase and be increased by sleep disturbances [31, 32]. Also, both BD and BPD youths present a high-risk for suicidality [33, 34] as sleep disturbances, especially insomnia, aggravate suicide risk in BPD patients [35]. Sleep research in these two psychopathologies can shed a light on their respective aetiology. If BD and BPD symptoms are strongly linked to sleep disturbances, improving sleep in these patients may reducing symptomatology and lowering suicide risk.

Until recently, there was a lack of research on BPD conducted among adolescent populations, since there was a controversy surrounding the diagnosis of BPD in youths [36]. Mental health clinicians were reluctant to diagnose BPD in adolescents because it was believed that personality is in flux during that age period and that this diagnosis stigmatised the patients [37, 38]. Attitudes in research BPD have changed recently and this diagnosis is being recognised as a valid mental disorder in adolescence [38, 39]. To our knowledge, sleep in BPD adolescents has only been studied by our team. We have demonstrated the relevance of assessing sleep using actigraphy in a case report of a BPD adolescent girl [40] and shown an association between poor sleep and impulsive and aggressive behaviours [41]. Literature describing sleep in BD children and adolescents is more extensive and it has been produced mainly over the last decade. In BD youths, questionnaire studies have shown a decreased need for sleep during mania, and presence of insomnia during depressive states [42–47]. Euthymic and non-euthymic BD adolescents have more difficulties falling asleep and have more frequent and longer night awakenings than healthy adolescents [48]. Moreover, sleep disturbances often precede the diagnosis of BD in childhood and adolescence [49]. Reports of objective findings are rare and the few polysomnography studies show that depressed adolescents who later developed BD had initially more stage 1 and less stage 4, increased REM latency, lower REM density, and less REM sleep than the unipolar group who never developed BD. A recent comparative study observed longer sleep duration and less wakefulness after sleep onset in euthymic BD adolescents (11–17 years old) compared to ADHD and healthy youths using actigraphy. However, BD adolescents self-reported a higher number of awakenings than their ADHD counterparts [50]. All these results obtained from BD youths must be interpreted with caution since some

studies included both prepubertal children and postpubertal adolescents or did not apply all the diagnostic criteria for DSM-IV BD while others had not controlled for current mood state.

Aim of Current Study

This study explores the sleep-wake cycle of euthymic adolescents with BPD or BD. Diagnoses are defined by DSM-IV criteria as assessed by standardised interviews, and sleep-wake patterns are objectively measured using actigraphy during periods with and without entrainment by school/work schedules. Healthy adolescents are recruited as a comparison group to disentangle what is specific to this age span and what is associated with psychopathology.

Hypotheses

We hypothesise that both BPD and BD adolescents will present higher sleep disturbances, such as night awakenings and longer sleep latencies, compared to HC adolescents. We also expect that sleep-wake patterns disruptions will be more characteristic of the BD group. Hypotheses derive mainly from adult scientific literature and, therefore, predictions are limited considering that there are few studies concerning only BPD or BD adolescents. Developmental features specific to adolescence may explain differences found between data obtained among adolescents and those obtained among adults.

Methods

Participants

Eighteen BPD participants (16.0 ± 1.1 years old; 3 boys and 15 girls) and 6 BD (16.7 ± 1.0 years old; 2 boys and 4 girls) were recruited at Rivière-des-Prairies Hospital (Montréal, Canada), a university-affiliated psychiatric hospital for children and adolescents, and at Institute Philippe-Pinel de Montréal, a university-affiliated hospital specialised in forensic psychiatry. They were referred to this study by their psychiatrist. Among eligible participants, written consent was only obtained for 25 BPD and 10 BD adolescents. Actigraphy data of 2 BPD patients and 3 BD patients were not analyzable because of technical difficulties. Also, 6 BPD adolescents and 1 BD adolescent drop out of the study during data collection.

Concerning participants who completed the study, one BD and 3 BPD youths were not attending school or working during data collection. One BD and five BPD

youths dropped out of school, and 2 BD and 5 BPD adolescents were working part time. One BD girl and one BPD girl were living in a youth centre.

For the two clinical groups, psychiatric diagnoses were established using the best-estimate method: clinical diagnoses were made by the treating psychiatrist using all available sources of information. Results from the K-SADS-PL (Schedule of Affective Disorders and Schizophrenia for School-Age Children—Present and Longitudinal Version, parent informant) and from the DIB-R (Diagnostic Interview for Borderlines Revised, adolescent informant) were included.

In this research protocol, eligible patients were included if they: (1) were euthymic at the time of data collection (defined as no current depressive symptoms), (2) had a current clinical diagnosis of BPD confirmed by the treating psychiatrist and by a DIB-R score ≥8 for the BPD group; (3) had a current clinical diagnosis of BD confirmed by the treating psychiatrist and by the K-SADS-PL semi-structured interview for the BD group: 3) were currently in treatment. As explained in the introduction, euthymia was necessary to control current depressive symptoms as a potential cofounding factor. Being in treatment was considered as an inclusion criteria to facilitate mental health care in case of emergency. Exclusion criteria were: (1) mental retardation or pervasive development disorders, (2) sleep disorder of organic origin as reported in the patient's medical file by his/her treating physician, (3) current psychotic features, (4) current mania, (5) BD diagnosis (for the BPD group) or BPD diagnosis (for the BD group).

Clinical characteristics of the clinical groups are detailed in Table 1.

Twenty healthy adolescents (HC; 7 boys and 13 girls; 14.7 ± 1.0 years old) were recruited by public advertisement sent by email to all employees of the Rivière-des-Prairies Hospital and posted on message boards throughout the hospital. One of the authors (CH) also visited a regional high school during school hours to explain the project to three classes of about 35 students each. Parents of adolescents who were interested were contacted for the selection procedure. Exclusion criteria were psychopathological problems as assessed by the Child Behaviour Checklist (CBCL) filled by the parent and the Youth Self-Report (YSR) filled by the adolescent using a T-score ≤70 as the threshold for all scales and subscales of the CBCL and the YSR as recommended by the authors of these questionnaires [51]. BPD symptomatology was also ruled out in HC adolescents by using the Abbreviated Diagnostic Interview for Borderlines (Ab-DIB), with a threshold ≤12) to be included in the study.

Table 1 Clinical features of participants by clinical group

	Borderline personality disorder (n = 18)	Bipolar disorder (n = 6)
K-SADS-PL diagnoses	Last 12 months (lifetime)	Last 12 months (lifetime)
Mood disorders		
Major depressive disorder	6 (12)	0 (0)
Dysthymic disorder	2 (2)	0 (0)
Bipolar disorder	0 (0)	6 (6)
Anxiety disorders		
Separation anxiety disorder	0 (3)	0 (0)
Panic disorder	3 (5)	1 (2)
Specific phobia	2 (2)	3 (3)
Social phobia	2 (2)	0 (0)
Generalised anxiety disorder	2 (2)	1 (2)
Post-traumatic stress disorder	1 (1)	0 (2)
Obsessive–compulsive disorder	2 (2)	0 (0)
Disruptive behaviour disorders		
Attention deficit hyperactivity disorder	0 (1)	1 (1)
Oppositional defiant disorder-conduct disorder	8 (12)	2 (6)
Alcohol/substance use related disorders	1 (4)	2 (4)
Others	1 (5)	0 (4)
Current psychotropic use		
Mood stabilisers		
Lithium	0	3
Antidepressants		
Fluoxetine	2	0
Mirtazapine	2	0
Venlafaxine	1	0
Norepinephrine reuptake inhibitors		
Atomexetine	1	0
Atypical antipsychotics		
Olanzapine	1	1
Quetiapine	4	4
Risperidone	1	1
Psychostimulants		
Dextroamphetamine	1	0
Methylphenidate	2	0
Others		
Clonidine	0	1
Propranolol	1	0

Measures

Diagnoses

The K-SADS-PL comprises 35 Axis I psychiatric disorders according to DSM-IV criteria. It is one of the most commonly used instruments in child and adolescent psychiatry studies [52]. In this study, we used the K-SADS-PL to compile Axis I comorbidities. The original version in English showed high test–retest reliability kappa coefficients (0.77–1.00) for major depression, BD, generalised anxiety disorder, conduct disorder and oppositional defiant disorder [52]. We chose to use the parent as the only informant for the K-SADS-PL and not to obtain data from the adolescent in order to avoid overloading an already long data collection period for the patients with an additional 90 min of interview.

The CBCL and the YSR are questionnaires assessing general psychopathology in children and adolescents commonly used in epidemiological and clinical studies [53]. These questionnaires investigate the presence of externalised and internalised symptoms and syndromes in the last 6 months. Normative data exists in general populations from North America and others cultures [51].

The DIB-R is a semi-structured interview divided in four sections corresponding to key elements of BPD symptomatology (emotions, cognitions, impulsivity, and interpersonal relationships) and follows a dimensional approach [54, 55]. In this study, the clinical threshold was 8 out of a maximum of 10, which constitutes the best compromise between specificity (0.80), sensitivity (0.82), positive predictive value (0.74), and negative predictive value (0.87) [55]. We used a version we adapted for adolescents, with the timeframe reduced to 6 months instead of 2 years, and certain items adapted to behaviours and life situations specific to this age group.

The Ab-DIB allows the assessment of BPD symptoms through 26 yes–no items. The French adolescent version was validated in suicidal adolescents: internal consistency varies between 0.80 and 0.86, inter-rater agreement is high (0.77–0.95), and sensitivity and specificity of the instrument are good to excellent (0.88 and 0.73–0.82 respectively) [56].

Mood Assessment

The beck depression inventory (BDI-II) is the most frequently used depression scale in studies on adolescents. It evaluates cognitive, behavioural, affective and somatic aspects of depression. Many studies have shown its validity and reliability [57–59]. A score of 19 or lower on the BDI-II was required to be included in the study, since BPD

patients tend to overestimate their depressive symptoms [60].

The Young Mania Rating Scale (YMRS) is filled by a trained clinician after a short interview in order to assess mania [61] and can be used with children and adolescents [62, 63]. The French version shows a very good inter-rater reliability (ICC > 0.89) and a high concurrent validity (r_s > 0.96) [64]. A total score of 11 or lower was required to be included in this study.

Actigraphy

Actigraph (AW-64 Model, MiniMitter, Bend, OR) is a piezzo-electric device, worn as a wristwatch on the non-dominant hand that continuously monitors movements. More precisely, acceleration is measured and generates a voltage value [65]. This electric data is filtered and amplified inside the device, and then transferred offline to a computer. Activity counts are numerical values obtained from the digital integration, which takes into account the surrounding epochs through a weighting algorithm [65]. Actigraphic data was analysed by a commercially available software provided by the actigraph manufacturer (Actiware V. 5.0), using 1-min epochs. An activity count of 40 was used as a threshold to distinguish wake from sleep since it has a high sensitivity/specificity ratio [66]. Table 2 defines the variables and the terms used in the present paper.

We also calculated a Coefficient of Variation (CV = standard deviation (SD)/mean) to obtain a value for variability of total sleep time as previously described by BC Mullin, AG Harvey and SP Hinshaw [50]. Bed time and rise time variability was estimated by averaging individual SDs.

Protocol

This investigation was carried out in accordance with the Declaration of Helsinky (2008). The protocol and the consent form were approved by the research ethic board of Rivière-des-Prairies Hospital. Written consent was obtained from both the adolescent and one of his/her parents. All the participants were enrolled on a voluntary basis and were offered a $25 music store voucher for their participation.

After obtaining the written consent, parents of the clinical groups had to complete the K-SADS-PL if it was not already done in the past 12 months and parents of the HC group filled the CBCL. BPD symptomatology was assessed with the adolescent using the appropriate instrument (DIB-R for the clinical groups, Ab-DIB for the HC group). All adolescents in the three groups filled out the BDI-II and completed the YMRS with one of the researchers, making sure that all participants were currently euthymic.

All participants were asked to wear the wrist actigraph on their non-dominant hand for at least 9 days including 2 weekends, therefore allowing for recorded sleep-wake patterns under different schedule constraints. Participants were also given a sleep diary to be filled every morning upon waking up, recollecting characteristics of the previous night. Of note, the sleep diary did not examined nightmares. Since actigraphy only assessed sleep-wake patterns and could not determine presence of nightmares, exploration of this topic was considered beyond the scope of this current study. Participants identified the rest interval onset and offset (i.e. from lights out to final rise up) by using the event marker button. If the patient did not press the button, sleep diary data was used if time corresponded to a decline in activity as shown by actigraphy data. If sleep diary data was missing on this particular date, rest interval was determined by identifying periods of low activity levels on the graphs. Details concerning protocol feasibility are given elsewhere [67].

For each patient, actigraphy data was broken down into school/work days and schedule-free days. A school/work day was defined as having stable sleeping and rising hours because of school or work. A schedule-free day was defined as days where the patient did not have any time-related obligations. There was no contact with participants while they were wearing the actigraph.

Statistical Analyses

Statistical analyses were performed with Statistica 6.1 (StatSoft, Tulsa, OK). Means and SD were obtained for all descriptive data.

Since the sample sizes were small, non parametric Kruskal–Wallis ANOVA tests were used to compare actigraphy data between the three groups. Upon significant results, post hoc Mann–Whitney U-Tests were performed to detect which group was different from the others. Alpha level was set at 0.05.

Since this study was exploratory and the sample size was small, Bonferroni's corrections became too conservative, and they increased the risk of Type II error [68]. In this article, Benjamini-Hochberg's procedure was applied to reduce the false discovery rate, although it is possible that this correction may be too conservative considering the exploratory nature of our results. Therefore, both corrected and non-corrected results will be presented. Cohen's d for size effects will also be given.

Results

On school/work days (Table 3), BPD adolescents spent a higher percentage of their time awake during their time in bed compared to euthymic BD youths (BPD: 21.5 ± 6.6 %; BD:

Table 2 Definition of actigraphy variables

Variables	Definitions
AC/min	Average activity counts, per minute
Time in bed	Time spent in bed as determined by actiwatch markers, in minutes, and confirmed by sleep diary
Time awake in bed	Number of minutes scored as awake during the time in of bed
Sleep onset latency	Time elapsed between the start of the rest period and the onset of the first 10 min period of immobility (AC = 0)
Total sleep time	Number of minutes scored as sleep during the period starting with the sleep onset latency and ending with the final awakening
Sleep efficiency	(minutes scored as sleep/minutes of time in bed) × 100
Final awakening	Interval between the end of the sleep interval and the end of the rest interval, in minutes
Sleep time	Minutes scored as sleep (AC ≤ 40)
Awake time	Minutes scored as awake (AC > 40)

13.6 ± 7.2 %; $p = 0.04$), and consequently a lower percentage of time asleep (BPD: 78.5 ± 6.6 %; BD: 86.6 ± 7.3 %; $p = 0.04$). Comparing these results, BPD patients were awake about three quarters of an hour more than those with BD. Though these three results became not statistically significant after Benjamini–Hochberg's correction, their effect size was large. Time awake during the time in bed in the BPD and HC groups were not significantly different. Although the null hypothesis was not rejected between the three groups, BPD and HC adolescents seem to show more interdaily total sleep time variability than BD youths (large effect size).

On schedule-free days (Table 4), BD and BPD youths both spent more time in bed than HC adolescents (non significant after Benjamini–Hochberg's correction, but the effect size was large). All groups of patients went to bed at the same time, however the clinical groups rose up one hour later, which meant they spent more time in bed and slept longer. This result remained statistically significant after correction and the effect size was moderate (total sleep time: BPD vs. HC) to large (time in bed: BPD vs. HC; time in bed: BD vs. HC; total sleep time: BD vs. HC). Although the null hypothesis was not rejected between the three groups, BPD adolescents seem to show more interdaily total sleep time variability than BD and HC youths (large effect size). The BD group show less variability than the HC group (large effect size). Also, even though the result was not statistically significant, a large effect size was observed in the time that the clinical groups got out of bed compared to HC.

When data from school/work and schedule free days were combined, rise time in the two patient groups was about an hour later than HC adolescents (Table 5). Post hoc analyses showed that this difference was actually significant between the BPD and the HC group. The two clinical groups had more total sleep time than the HC group; the difference was found to be statistically

significant only between BPD and HC adolescents. Variability analyses showed that total sleep time was more variable from one night to the other in BPD adolescents compared to the HC group. There was more variability in rising time in BPD adolescents compared to HC youths. All the differences found when data from school/work and schedule free days were combined were no longer significant after Benjamini–Hochberg's correction, but their effect size remained large.

For school/work days, schedule-free days and both types of days combined, there were no significant differences for sleep onset latency, sleep efficiency and final awakening.

Discussion

The following trends emerged from this explorative study on sleep-wake patterns in BPD and BD adolescents: (1) stabilised euthymic BD adolescents sleep longer and better than BPD and HC adolescents on school/work days, (2) BD and BPD patients woke up later than the HC adolescents on schedule-free days and (3) BPD adolescents have a higher variability in total sleep time and in rise time compared to BD and HC youths. Of note, when differences were calculated, a number of results were significant when correction was not performed, but they were no longer statistically significant after applying Benjamini–Hochberg's procedure. However, the calculated effect size was large for all these results, suggesting that they were not due to chance. Considering the small sample size and the exploratory nature of this research, correction might be too conservative [68, 69]. To determine if this is the case, replication through confirmation studies is needed with a larger sample size to increase statistical power and to determine clinical significance. Meanwhile, this section will discuss our exploratory data before statistical correction. Observations reported in this study should be viewed as

Table 3 Sleep actigraphy comparisons between groups – school/work days

	BPD n = 14	BD n = 5	HC n = 20	p value[a]	BPD versus BD (p value[b]/Cohen's d)	BPD versus HC (p value[b]/Cohen's d)	BD versus HC (p value[b]/Cohen's d)
Number of recording days	5.7 (2.2)	7.4 (3.1)	7.6 (2.8)	n.s	n.c./−0.63	n.c./−0.75	n.c./−0.07
Bed time (h:min)	22:42 (01:09)	22:44 (01:01)	22:38 (00:51)	n.s.	n.c./−0.03	n.c./0.07	n.c./0.11
Time in bed (min)	508.36 (44.47)	494.16 (78.87)	506.31 (41.55)	n.s.	n.c./0.22	n.c./0.05	n.c./−0.19
Time awake in bed (min)	108.66 (32.00)	65.30 (28.02)	96.12 (30.52)	0.039	0.01/1.44	n.c./0.40	0.042/−1.14
Sleep latency (min)	27.54 (24.33)	19.99 (7.91)	20.00 (15.68)	n.s.	n.c./0.42	n.c./0.37	n.c./0.00
Total sleep time (min)	376.15 (55.57)	386.31 (102.52)	355.92 (75.12)	n.s.	n.c./−0.12	n.c./0.31	n.c./0.34
Sleep efficiency (%)	74.02 (9.05)	77.83 (12.68)	70.28 (14.09)	n.s.	n.c./−0.35	n.c./0.32	n.c./0.56
Final awakening (min)	19.13 (10.83)	15.36 (9.71)	20.63 (9.85)	n.s.	n.c./0.38	n.c./−0.14	n.c./−0.54
Rise time (h:min)	7:05 (00:58)	7:08 (00:40)	7:03 (00:39)	n.s.	n.c./−0.06	n.c./0.04	n.c./0.13
Variability							
Total sleep time (CV)	0.08 (0.02)	0.13 (0.03)	0.07 (0.01)	n.s.	n.c./−1.96	n.c./0.63	n.c./2.68
Bed time (SD-min)	42.74 (28.09)	49.06 (33.75)	29.74 (18.49)	n.s.	n.c./−0.20	n.c./0.54	n.c./0.71
Rise time (SD-min)	31.73 (36.48)	38.05 (35.69)	28.66 (23.76)	n.s.	n.c./−0.18	n.c./0.10	n.c./0.31

BPD borderline personality disorder, *BD* bipolar disorder, *HC* healthy controls, *CV* coefficient of variation, *n.s.* non significant

n.c. = no statistical calculation was performed

[a] Kruskal–Wallis ANOVA; no correction

[b] Post-hoc Mann–Whitney U-Test; no correction

interesting research questions that need further investigation, rather than definitive conclusive results.

Each of the two clinical groups showed selective differences compared to the HC group. We found that stabilised euthymic BD adolescents sleep longer and better than BPD and HC adolescents on school/work days. This result is in agreement with a previous report showing that euthymic BD youths (n = 13; all on mood stabilisers and/or antidepressants and none taking stimulants or hypnotics during data collection) slept longer than ADHD (n = 14; three patients with antidepressants and none with stimulants or hypnotics during data collection) and healthy youths (n = 21) according to actigraphic recordings [50]. Longer sleep duration has also been reported in previous actigraphy studies using euthymic BD adults [19, 20, 70, 71].

It could be argued that long sleep durations in our study could be due to the fact that more BD patients (67 %) than BPD adolescents (22 %) were being prescribed quetiapine, which has sedating qualities and which is effective to improve sleep quality and quantity [72]. However, other sedating medications were also prescribed to the BPD group (see Table 1). Pharmacotherapy cannot fully explain this finding. Another possible explanation could be that therapeutic interventions, including education about sleep, may be involved. In the present study, all BD adolescents, but none of the BPD youths, received psychoeducation about their disorder. Addressing the issue of sleep-wake disturbances during treatment seems to, therefore, be beneficial in euthymic BD adolescents. A similar approach may also help BPD youths to have better sleep hygiene. Presently, only Dialectical Behaviour Therapy addresses sleep problems in BPD [35]. Cognitive Behavioural Therapy can also be considered as a potential intervention to reduce dysfunctional sleep cognitions which maintain sleep disturbances [73] which are associated with non-recovery

Table 4 Sleep actigraphy comparisons between groups—schedule-free days

	BPD n = 18	BD n = 6	HC n = 20	p value[a]	BPD versus BD (p value[b]/Cohen's d)	BPD versus HC (p value[b]/Cohen's d)	BD versus HC (p value[b]/Cohen's d)
Number of recording days	6.0 (3.4)	5.5 (3.3)	6.1 (2.4)	n.s	n.c./0.15	n.c./−0.03	n.c./−0.21
Bed time (hr :min)	00:02 (01:23)	00:12 (01:34)	23:46 (00:59)	n.s.	n.c./−0.11	n.c./0.22	n.c./0.33
Time in bed (min)	602.01 (76.41)	612.60 (46.68)	549.00 (42.63)	0.015	n.c./−0.17	0.026/0.86	0.004/1.42
Time awake in bed (min)	128.11 (41.90)	105.97 (36.62)	106.26 (39.60)	n.s.	n.c./0.56	n.c./0.54	n.c./−0.01
Sleep latency (min)	20.78 (12.65)	23.28 (7.29)	14.42 (8.32)	n.s.	n.c./−0.24	n.c./0.59	n.c./1.13
Total sleep time (min)	438.21 (92.73)	473.49 (84.87)	383.16 (77.70)	0.019	n.s./0.40	0.030/0.64	0.016/1.11
Sleep efficiency (%)	72.61 (10.93)	77.19 (11.64)	70.02 (13.47)	n.s.	n.c./−0.41	n.c./0.21	n.c./0.57
Final awakening (min)	27.52 (21.35)	22.29 (17.11)	19.91 (10.01)	n.s.	n.c./0.27	n.c./0.46	n.c./0.17
Rise time (h:min)	10:03 (1:15)	10:24 (1:59)	8:53 (1:15)	0.003	n.s./−0.21	0.0004/0.93	n.s./0.91
Variability							
Total sleep time (CV)	0.17 (0.02)	0.11 (0.04)	0.14 (0.02)	n.s.	n.c./1.90	n.c./1.50	n.c./−0.95
Bed time (SD-min)	84.74 (47.63)	79.30 (51.53)	61.60 (45.76)	n.s.	n.c./0.11	n.c./0.50	n.c./0.36
Rise time (SD-min)	85.61 (41.95)	56.06 (41.53)	63.72 (29.18)	n.s.	n.c./0.71	n.c./0.61	n.c./−0.21

BPD borderline personality disorder, *BD* bipolar disorder, *HC* healthy controls, *CV* coefficient of variation, *n.s.* non significant

n.c. no statistical calculation was performed

[a] Kruskal–Wallis ANOVA; no correction

[b] Post-hoc Mann–Whitney U-Test; no correction

in BPD adults [74]. Luminotherapy constitutes another therapeutic strategy that can help improving certain aspects of BPD [75, 76].

On schedule-free days, we found that both BD and BPD patients woke up later than the HC group. Without any obligations during these schedule-free days, BD patients may have felt less pressured to comply with principles of sleep education, and optimal and stable sleep schedules. This may be interpreted as BD adolescents having a greater need to sleep during euthymic phases. We speculate that both clinical groups do not participate as much in activities requiring an early rising time during weekends (seeing friends for social activities, sport training, family gatherings) than the HC adolescents. Other studies report that BPD youths have more difficulties concerning peer relationships and family interactions compared to children and adolescents with other personality disorders or with other Axis I disorders [12, 77]. It can be hypothesized that this poor social functioning can lead to low participation in social activities. To the best of our knowledge, no study has specifically investigated social activities engagement in BD

and BPD adolescents. Research in this area will shed light on this hypothesis.

Our results also show that BPD adolescents have a higher variability in total sleep time and in rise time compared to BD and HC youths. This observation is in agreement with a previous study by Verkes et al. [78] who examined 24 h periodicity to assess circadian variability using actigraphy. In actigraphy analysis, a 24 h periodicity refers to the presence of a peak of activity counts which is repeated every 24 h. Presence of 24 h periodicity can be translated as having a regular schedule for everyday life activities such as eating, sleeping, and physical activity. Non-24 periodicity defines the presence of more than one peak of motor activity or the presence of a single peak occurring in a time period smaller or larger than 24 h. Verkes et al. [78] found that among suicidal adults, in whom 69.5 % had a diagnosis of BPD, patients with a non-24 h periodicity had significantly more BPD symptoms than those with 24 h periodicity. Furthermore, non-24 h periodicity was significantly correlated with depression and hopelessness and an improvement of mood was associated

Table 5 Sleep actigraphy comparisons between groups—both types of days combined

	BPD n = 18	BD n = 6	HC n = 20	p value[a]	BPD versus BD (p value[b]/Cohen's d)	BPD versus HC (p value[b]/Cohen's d)	BD versus HC (p value[b]/Cohen's d)
Number of recording days	10.4 (2.6)	11.7 (4.1)	13.6 (2.5)	0.007	n.s./−0.38	0.001/−1.25	n.s/−0.58
Bed time (h:min)	23:36 (01:33)	23:40 (01:33)	23:08 (00:55)	n.s.	n.c./−0.05	n.c./0.37	n.c./0.45
Time in bed (min)	558.10 (50.76)	564.55 (75.97)	525.00 (35.61)	n.s.	n.c./−0.10	n.c./0.75	n.c./0.67
Time awake in bed (min)	115.45 (32.07)	90.90 (37.10)	100.61 (35.21)	n.s.	n.c./0.71	n.c./0.44	n.c./−0.27
Sleep latency (min)	22.91 (14.42)	22.57 (4.78)	17.11 (11.25)	n.s.	n.c./0.03	n.c./0.45	n.c./0.63
Total sleep time (min)	413.11 (68.77)	438.13 (93.37)	368.14 (71.71)	0.038	n.s./−0.31	n.s./0.64	0.016/0.84
Sleep efficiency (%)	73.88 (8.57)	77.46 (11.23)	70.33 (13.31)	n.s.	n.c./−0.36	n.c./0.32	n.c./0.58
Final awakening (min)	24.05 (16.54)	18.70 (8.85)	20.57 (8.45)	n.s.	n.c./0.40	n.c./0.26	n.c./−0.22
Rise time (h:min)	8:53 (1:12)	9:09 (2:21)	7:52 (00:59)	0.028	n.s./−0.14	0.005/0.93	n.s./0.71
Variability							
Total sleep time (CV)	0.19 (0.01)	0.16 (0.03)	0.13 (0.02)	0.031	n.s./1.34	0.007/3.79	n.s./1.18
Bed time (SD-min)	79.31 (45.58)	87.47 (51.10)	56.58 (24.15)	n.s.	n.c./−0.17	n.c./0.62	n.c./0.77
Rise time (SD-min)	108.83 (35.48)	99.80 (39.69)	75.00 (33.25)	0.030	n.s./0.24	0.009/0.98	n.s./0.68

BPD borderline personality disorder, *BD* bipolar disorder, *HC* healthy controls, *CV* coefficient of variation, *n.s.* non significant

n.c. no statistical calculation was performed

[a] Kruskal–Wallis ANOVA; no correction

[b] Post-hoc Mann–Whitney U-Test; no correction

with more regular bed times within subjects. This non-24 h periodicity reflected a diminished entrainment of the activity rhythm to *zeitgebers* in suicidal BPD adults.

Thus, irregular sleep pattern is a phenomenon observed in both adult and adolescent BPD. What is the impact of this sleep disturbance on mood and behaviour in adolescents, especially those with BPD symptomatology? In young adolescents without psychopathology, longer sleep duration during weekends and larger differences in total sleep time between weekdays and weekends increased the association between perceptions of conflict between parents (reported by the mother and the adolescent) and the adolescent's level of aggressiveness [79]. It is proposed that irregular sleep duration and timing between weekdays and weekends cause a jetlag-like syndrome and increases a state of pre-existing vulnerability (emotional regulation difficulties, fatigue, irritability, and impulsiveness) weakening the adolescent's capacities to cope with psychosocial stress [80] and exacerbating BPD symptoms. This is

confirmed by our own observation according to which total sleep time and aggressiveness/impulsiveness are inversely related [41]. This supports the view that sleep disturbances actually BPD symptomatology.

Furthermore, better sleep quality seems to be associated with BPD recovery in adults [74]. This supports the view that sleep disturbances are actually associated with worse BPD symptomatology. The precise mechanisms underlying the association between sleep disturbances and symptoms in BD or BPD are currently unknown. There are a few hypotheses that have received preliminary support, however. For example, it was proposed that sleep loss may increase impulsivity and aggressiveness observed in BD and in BPD by decreasing serotonin activity [81, 82]. Low serotonin levels have been observed in both disorders [83, 84]. Sleep disturbances could also explain the reduced connectivity between the frontal cortex and the limbic system observed in these disorders [85–87], so the frontal cortex could not efficiently regulate the expression of

intense emotions. Conversely, emotional arousal caused by BD or BPD symptoms can disrupt sleep. In the BPD literature, it has been suggested that emotional cascades during the day, which are defined as self-amplifying loops between rumination and negative emotions leading to emotional dysregulation behaviours, increases arousal during sleep, which can lead to night awakenings and nightmares [88]. Moreover, maladaptive sleep-related cognitions can explain why sleep disturbances are more prevalent in non-recovered BPD patients compared to recovered BPD patients [73, 74]. Further research is needed to uncover the mechanisms that link sleep and emotions and behaviours in BD and BPD. This will hopefully allow for a better understanding of the nature and phenomenology of sleep disturbances in these two psychiatric disorders and eventually improve assessment and treatment.

This study has methodological limitations that need to be considered. First, the sample size was small, particularly in the BD group, so that the present results need to be confirmed with more participants. Power calculations were performed to estimate the sample size required (44 participants per group). Based on prevalence of BPD and of BD at Rivière-des-Prairies Hospital in the last 2 years (2004–2005) before the beginning of data collection, the calculated targeted number of participants seemed realistic. During the data collection period (August 2006–December 2011), 100 adolescents registered at Rivière-des-Prairies Hospital received an official diagnosis of BPD and 33 other adolescents were diagnosed with BD. However, only 58 BPD and 23 TB patients were eligible based on our inclusion and exclusion criteria. This low eligibility rate was explained mainly by the fact that many patients had a current mood episode or still had residual depressive symptoms during our recruitment period. In other words, euthymia was the limiting criterion. Also, consent rate was low and not all participants who consented completed the study. Therefore, the small sample size could be explained by a low number of eligible euthymic participants who consented and completed the study. Second, we could not control for factors which may influence sleep patterns, such as sex, socio-economical status, race, pubertal development, BMI, and drug abuse, by analyzing subgroups. Third, a number of patients used illicit drugs and/or were prescribed medication during data collection. Although these factors have an impact on sleep, they are extremely common in BPD and BD patients seen in clinical settings. To perform a pharmacological washout of many days with adolescents suffering from severe psychopathology would be unethical and would be unrepresentative of these clinical populations. Our results may possibly include the effects of medication and/or illicit drugs use. Because of the sample size, it was not possible to control for this important

confounding factor. A replication study taking this factor into account is thus needed. Finally, although this study attempted to control depression as a cofounding factor by excluding BPD patients with current depressive disorders, it is acknowledged that this sample is therefore selective and can be atypical from BPD adolescents seen in clinical settings [89].

Beyond these limitations, this study presents methodological strengths. Unlike previous studies, especially those concerning paediatric BD, our groups did not include prepubertal children. Moreover, we used strict diagnostic criteria. All patients had to present all DSM-IV criteria for their respective disorder, including the required duration and number of symptoms. In the case of BD, patients had to have a mania episode as defined by the DSM-IV, including cardinal symptoms such as euphoric mood and grandiosity for at least 4 days (or less if hospitalisation was needed). By doing so, patients who presented severe mood dysregulation and not BD [90] were not included and all patients were euthymic. Finally, using actigraphy as an ambulatory assessment, we were able to examine sleep-wake patterns as they occur in naturalistic environments.

Although this study is exploratory, it is among the first to examine sleep-wake patterns in BPD adolescents using actigraphy. This research also brings further evidence that sleep remains a crucial issue that needs to be addressed during adolescence, both in the presence and in the absence of psychopathology [26]. The presence of sleep disturbances in adults with BD is acknowledged in clinical practice and these are often assessed and addressed during therapy. For example, interventions targeting sleep disturbances, such as education about sleep and stabilisation of social rhythms, exist for BD adult patients to prevent relapse and maintain euthymia [91]. In the present research, we found that such interventions were also beneficial for euthymic BD adolescents: they actually sleep longer and better that the two other groups, probably because sleep disturbances were targeted during treatment. In BPD adolescents, on the contrary, sleep problems and sleep-wake patterns variability were not systematically addressed during assessment and therapy. Targeting sleep at the beginning of the disorder when the BPD diagnosis is made could contribute improving cognition and behaviour and introduce a positive asset in the course of their disorder.

Education about sleep should prove to be beneficial for youths with psychopathology. Healthy adolescents naturally experience a phase delay and many suffer from sleep restriction due to disturbing external factors. Unfortunately, they are not made aware about simple sleep hygiene rules that can improve sleep duration and quality [25]. More generally, we know that over 40 % of adolescents have sleep problems, with negative consequences on school performance and mood of students [25, 92]. As it is

the case for drug use and sexuality, education about sleep should thus also be implemented in schools so that teachers, school psychologists and nurses as well as administrators are informed and involved. Such a strategy could prevent the worsening of mood and behavior problems in adolescents, with or without psychopathological problems.

The present study also demonstrates that use of actigraphy in BD and BPD adolescents is feasible and useful in order to collect data concerning sleep-wake patterns in a natural environment: as of yet, few studies have used actigraphy in such patients [76]. Further research should combine actigraphy with additional ambulatory assessment tools (applications for tablets and smart phones) to examine more behavioral and emotional aspects of BD and BPD adolescents in real-time [93]. This should allow for a better understanding of the association between sleep-wake patterns and psychopathological manifestations.

Summary

In sum, this research suggests that sleep-wake disruptions are part of BD and BPD pathology, even in the absence of depression. We hypothesize that these problems can lead to impairment in daily functioning. This reinforces our overall key messages: (1) sleep disturbances are more than mere symptoms, they constitute factors contributing to the onset, maintenance or relapse of psychopathologies; (2) sleep should be systematically assessed and addressed while treating adolescents with BD or BPD due to the influence of sleep on quality of life, daily functioning, and symptoms [94, 95].

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