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Poor subjective sleep quality and trait impulsivity in patients with bipolar disorder

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Abstract

Background. Sleep disturbance and impulsivity are key components of mood vulnerability in bipolar disorder (BD), but few studies have assessed the association between these two symptoms among patients with BD.

Methods. Forty-seven euthymic patients with bipolar I disorder (BDI) or bipolar II disorder (BDII) and 58 age- and sex-matched healthy controls were enrolled in this cross-sectional study. Trait impulsivity was measured using the Barratt Impulsiveness Scale Version 11 (BIS-11), which yielded 3 second-order factors: attention, motor, and non-planning. Subjective sleep quality was assessed using the self-reported Pittsburgh Sleep Quality Index (PSQI). General linear models (GLMs) were used to assess the associations between subjective poor sleep and trait impulsivity with multiple testing corrections.

Results. Patients with BD scored higher in BIS-11 and PSQI than healthy controls. PSQI total scores positively correlated with BIS-11 total scores, while sleep disturbance and daytime dysfunction were associated with attentional impulsiveness after controlling for covariates. Participants with higher PSQI total scores (>10) had higher scores in BIS-11 total, attention, and non-planning than those with low PSQI scores (≤5).

Conclusion. These findings support the hypothesis that poor sleep quality might lead to impulsivity and add to the growing evidence that improving sleep quality may be a therapeutic target for patients with BD.

Introduction

Impulsivity, defined as "a predisposition toward rapid, unplanned responses to internal or external stimuli with disregard for the negative consequences of these reactions," is a core feature of bipolar disorder (BD).[1] Individuals with BD exhibit greater impulsivity than those with other affective disorders.[2] This heightened impulsivity persists not only during manic episodes but also during remission in BD, suggesting it is both a state feature and a trait feature of BD.[3] Emerging research indicates that BD may be more accurately conceptualized as a multidimensional condition rather than a categorical diagnosis, characterized by distinct neurobiological underpinnings and a range of comorbidities.[4,5] Elevated impulsivity in patients with BD is linked to a spectrum of adverse outcomes, including cognitive dysfunction, increased risk of substance abuse and suicidal behaviors, reduced medication adherence, and sustained aggression post-remission.[6,7] Furthermore, our recent research has identified higher trait impulsivity, associated with altered prefrontal cortex (PFC) structure, in patients with BD with a history of suicide attempts compared to those without such history and healthy individuals.[8] Sleep disturbance, a prevalent feature of BD, may be related to heightened impulsivity.[9] Both impulsivity and sleep disturbance are associated with symptomatic relapse in BD and can predict the trajectory of BD.[10,11] Poor sleep has been connected to several maladaptive neurocognitive processes that regulate mood and impulsive behavior,[12,13] underscoring the importance of sleep as a potential mediator and therapeutic target for impulsivity and its detrimental effects. Nonetheless, research into the interplay between these two domains in patients with BD remains scarce.

Impulsivity consists of state impulsivity and trait impulsivity. State impulsivity refers to momentary responses to contextual triggers and is generally assessed with behavioral tasks, while trait impulsivity denotes an individual's personal characteristics. Studies investigating impulsivity and sleep have mostly enrolled healthy individuals or patients with attentional deficits, with the majority focusing on state impulsivity.[14] Experimental studies have found that sleep deprivation is associated with more impulsive responses in behavioral tasks related to decision-making or response inhibition.[14] McGowan and Coogan observed inverse correlations between sleep duration/efficiency and trait impulsivity, as measured by the Barratt Impulsiveness Scale

Version 11 (BIS-11), within an undergraduate student population.[15] In summary, extensive research suggests that sleep duration or efficiency negatively correlates with impulsivity in healthy individuals.[14] Despite these significant connections, the study of BD has largely examined sleep and impulsivity as separate entities, and the association between the two features in BD has been scarcely examined. Russo et al. demonstrated associations between disturbed sleep/daytime dysfunction and trait impulsivity among euthymic patients with BD.[16] However, the data were limited by the exploratory design, which described only bivariate associations between variables without adjusting for important covariates such as age, sex, educational level, and psychotropic medication use.

Recognizing that sleep and impulsivity are critical elements of mood vulnerability in BD, and considering their significant interactions in nonclinical subjects, a deeper comprehension of trait impulsivity and its correlation with sleep disturbance could provide valuable insights into the prognosis and potential therapeutic target of BD. Our objective was to explore the relationship between subjective sleep quality and trait impulsivity in patients with BD. We hypothesized that poor sleep quality would be associated with increased trait impulsivity in euthymic patients with BD.

Methods

Forty-seven consecutive outpatients, aged 20–50 years, diagnosed with BD based on the Mini-International Neuropsychiatric Interview (MINI) and the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), were enrolled from the psychiatric outpatient department of Taipei Veterans General Hospital. Fifty-two patients were screened; 5 were excluded for being over 50 years old. According to DSM-5, bipolar I disorder (BDI) requires at least 1 manic episode for diagnosis, while bipolar II disorder (BDII) requires at least 1 hypomanic episode and 1 major depressive episode. All participants were in a stable mood state, indicated by scores below 7 on both the Montgomery–Asberg Depression Rating Scale (MADRS) and the Young Mania Rating Scale (YMRS).[17,18] Exclusion criteria included major physical diseases (e.g., head injury with sustained loss of consciousness or cognitive aftermath, neurological disorders) and a lifetime history of schizophrenia, other psychoses, or intellectual disability as defined by the DSM-5. Patients were receiving treatment with various antipsychotics, mood stabilizers, and hypnotics according to the Taiwan consensus on the biological treatment of BD.[19] A cohort of 58 healthy participants without any DSM-5 diagnoses, matched for age and sex, was recruited through posters within the clinic and community. These participants underwent thorough clinical evaluations by a psychiatrist to confirm the absence of psychiatric illness. The Institutional Review Board of Taipei Veterans General Hospital approved the study, which adhered to the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before study inclusion.

Clinical assessment

Demographic characteristics, including age, sex, education, duration of illness, history of substance or alcohol use disorder (according to DSM-5 criteria), and use of psychotropic medications, were collected from all participants. The severity of depression and manic symptoms was evaluated using the MADRS and the YMRS, respectively. The BIS-11 was used to gather self-reported assessments of trait impulsivity through 30 queries, graded on a four-point Likert scale from 1 (rarely/never) to 4 (almost always/always),[20] assessing three impulsivity categories: attentional, motor, and non-planning. Higher scores indicate increased trait impulsivity. Subjective sleep quality over the previous month was assessed using the Pittsburgh Sleep Quality Index (PSQI).[21] This index includes 19 items covering seven components: subjective sleep quality (1 item), sleep latency (2 items), sleep duration (1 item), habitual sleep efficiency (3 items), sleep disturbance (9 items), use of sleep medication (1 item), and daytime dysfunction (2 items). The sleep disturbance component is derived from the sum of eight item scores reflecting the frequency of sleep-related difficulties. Each component is scored from 0 to 3, indicating the degree of difficulty, with a total score ranging from 0 to 21 for overall sleep quality; higher scores suggest poorer sleep quality. A PSQI score > 5 was used to identify sleep disorders, and a score > 10 was used to determine clinically significant insomnia.[21]

Statistical analyses

Continuous variables were compared using analysis of variance (ANOVA), and categorical data were evaluated with the chi-squared test. The Levene test assessed variance homogeneity among groups. When the variance distribution was uneven, Welch's 1-way ANOVA was used, followed by Games–Howell post hoc pairwise comparisons. Participants were stratified by their PSQI ratings (≤5, 5–10, > 10), and general linear models (GLMs) assessed impulsivity levels across groups, adjusting for age, sex, education, mood symptoms, substance/alcohol use disorder history, psychotropic medication use, and disease group. Linear regression models examined the association between BIS-11 and PSQI scores, controlling for the same covariates. The main effects of demographic data and clinical symptoms on BIS-11 and PSQI scores were tested, as well as interactions between diagnosis and PSQI scores in relation to BIS-11 scores. The Benjamini–Hochberg false discovery method managed multiple comparisons. A p-value of <0.05 was considered significant. Statistical analyses were conducted using Statistical Package for the Social Sciences (SPSS) 11.5 (SPSS Inc., Chicago, IL, USA).

Results

Compared with controls, patients with BDI or BDII exhibited higher rates of substance or alcohol use disorder history and were more frequently receiving treatment with antipsychotics, mood stabilizers, and hypnotics (all $p < 0.005$; Table 1). Patients with BD had significantly higher BIS-11 total scores and exhibited greater attention and non-planning impulsiveness than healthy subjects. Additionally, patients with BD experienced poorer sleep quality across global PSQI measures, including sleep latency, sleep disturbance, use of sleeping medications, and daytime dysfunction, relative to healthy controls (all $p < 0.005$; Table 1). The percentage of patients dissatisfied with their sleep quality (PSQI total score > 5) was 95.5% for BDI, 72% for BDII, and 17.2% for controls.

Patients with BD who took hypnotics ($n = 18$) had higher PSQI total scores and greater daytime dysfunction than those who did not use hypnotics ($n = 29$) (PSQI total: 11.39 versus 8.72, $p = 0.04$; daytime dysfunction: 2.00 versus 1.24, $p = 0.11$). Those who experienced interrupted sleep (question 5b in PSQI) more than once per week ($n = 31$) exhibited higher attentional impulsiveness compared to patients with less frequent interrupted sleep (less

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Table 1. Demographic Data and Clinical Variables between Patients with BD and Healthy Controls

	BD (n = 47)					
Demographic variables	BDI (n = 22)	BDII (n = 25)	Healthy control (n = 58)	p^*	p^+	Post hoc
Age (year, SD)	33.8 (8.1)	34.7 (9.2)	34.6 (7.3)	0.860	0.920	
Female (n, %)	9 (40.9)	13 (52.0)	30 (51.7)	0.380	0.661	
Education (years, SD)	15.5 (2.0)	14.6 (2.0)	16.3 (1.5)	<0.001	0.001	HC > BDII
Age at onset (years, SD)	21.0 (6.8)	22.5 (6.0)			0.404	
Duration of illness (years, SD)	11.4 (7.4)	12.0 (6.9)			0.780	
History of substance or alcohol use disorder (n, %)	4 (18.2)	5 (20)	0	0.001	0.002	BDI, BDII > HC
MADRS total score	1.3 (1.8)	2.2 (2.0)		<0.001		
YMRS total score	1.6 (1.9)	1.7 (1.9)		<0.001		
Medications						
Antipsychotics	17 (77.3)	11 (44.0)	0	<0.001	<0.001	BDI, BDII > HC
Mood stabilizers	16 (72.7)	11 (44.0)	0	<0.001	<0.001	BDI, BDII > HC
Hypnotics	10 (45.5)	8 (32.0)	0	<0.001	<0.001	BDI, BDII > HC
Impulsivity scale						
BIS total	62.0 (17.6)	66.6 (10.0)	54.8 (14.3)	0.001	0.002	BDI, BDII > HC
Attention	17.4 (3.5)	18.4 (3.2)	14.6 (2.3)	<0.001	<0.001	BDI, BDII > HC
Motor	22.1 (4.3)	23.7 (4.5)	20.8 (2.7)	0.003	0.014	BDII > HC
Non–planning	26.7 (4.3)	26.2 (4.1)	22.9 (3.2)	<0.001	<0.001	BDI, BDII > HC
Sleep scale						
PSQI total	10.4 (3.7)	9.2 (4.9)	3.6 (2.1)	<0.001	<0.001	BDI, BDII > HC
Sleep quality	1.7 (0.7)	1.3 (0.8)	1.1 (0.7)	0.013	0.005	BDI > BDII, HC
Sleep latency	1.8 (1.0)	1.5 (1.0)	0.6 (0.8)	<0.001	<0.001	BDI, BDII > HC
Sleep duration	0.6 (1.1)	0.8 (1.1)	0.3 (0.5)	0.027	0.050	
Sleep efficiency	0.7 (1.1)	0.6 (1.0)	0.1 (0.5)	0.001	0.017	
Sleep disturbances	1.5 (0.6)	1.5 (0.7)	1.1 (0.5)	<0.001	0.001	BDI, BDII > HC
Use of sleeping medications	2.6 (1.1)	2.0 (1.3)	0.0 (0.2)	<0.001	<0.001	BDI, BDII > HC
Daytime dysfunction	1.6 (1.0)	1.5 (1.1)	0.4 (0.6)	<0.001	<0.001	BDI, BDII > HC

*Independent t-test comparing the BD and HC groups.
+ANOVA comparing the BDI, BDII, and HC groups.
BDI, bipolar I disorder; BDII, bipolar II disorder; BIS, Barratt Impulsiveness Scale; MADRS = Montgomery–Asberg Depression Rating Scale; PSQI, Pittsburgh Sleep Quality Index; YMRS, Young Mania Rating Scale.

than once per week, n = 16) (18.77 versus 16.20; p = 0.013). Subgroup analyses revealed this association only in patients with BDI (18.73 versus 14.00; p = 0.002), not in those with BDII. Among the eight items assessing sleep disturbance (5b–5i), only interrupted sleep (5b) was linked to attentional impulsiveness.

Linear regression analyses revealed a positive association between the PSQI total score and the BIS-11 total score after adjusting for covariates (Table 2). The second-order attention factor in the BIS-11 was positively correlated with the PSQI total score, sleep disturbances, and daytime dysfunction. However, subanalyses stratified by disease groups did not demonstrate the aforementioned associations between the PSQI and BIS-11 scores. In the total sample, tests for an interaction effect between the PSQI and disease groups revealed no significant interactions (Table 3).

The GLM, adjusted for covariates, revealed that participants with higher PSQI total scores (>10) exhibited elevated BIS-11 total and non-planning scores compared to those with low and medium PSQI scores (≤5, 5–10) (all $p < 0.05$; Figure 1). The GLM detected no differences in the second factor (motor) between groups.

Discussion

The current study found that patients with BD scored higher on the BIS-11 and the PSQI, and subjective sleep quality was negatively associated with trait impulsivity. Given the multifaceted nature of impulsivity, we further explored the contribution of the three BIS-11 subscales to subjective sleep quality. Our findings demonstrated significant correlations between sleep disturbance and daytime dysfunction with attentional impulsiveness, highlighting that the connections between sleep problems and impulsivity primarily stemmed from a specific relationship with the subscale covering difficulties concentrating. We also found that a higher frequency of interrupted sleep (difficulty maintaining sleep) was the only

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Table 2. Associations between the BIS and PSQI, with the Adjustment of Age, Sex, Education, Substance/Alcohol Use Disorder History, Mood Symptom Severity, Psychotropic Medication Use, and Disease Group

	BIS total			Second-order factor Attention			Second-order factor Motor			Second-order factor Non-planning		
	B (95% CI)	t	*p*	B (95% CI)	t	*P*	B (95% CI)	t	*p*	B (95% CI)	t	*p*
PSQI total	**1.173 (.237–2.109)**	**2.490**	**0.015**	**0.275 (.085–.465)**	**2.895**	**0.005**	0.213 (−.027–.454)	1.760	0.081	0.243 (.003–.484)	2.008	0.047
Sleep quality	1.428 (−2.868–5.724)	0.660	0.511	0.396 (−.494–1.286)	0.884	0.379	0.643 (−.442–1.728)	1.178	0.242	0.734 (−.352–1.819)	1.344	0.183
Sleep latency	3.287 (−.145–6.718)	1.902	0.060	0.364 (−.346–1.075)	1.020	0.311	0.041 (−.854–.936)	0.091	0.927	−0.408 (−1.312–.497)	−0.897	0.373
Sleep duration	1.831 (−1.984–5.646)	0.953	0.343	0.571 (−.183–1.324)	1.507	0.136	0.880 (−.047–1.807)	1.888	0.063	0.559 (−.431–1.549)	1.122	0.265
Sleep efficiency	3.391 (−.600–7.382)	1.687	0.095	0.356 (−.446–1.158)	0.881	0.380	0.631 (−.355–1.618)	1.272	0.207	1.125 (.139–2.112)	2.264	0.026
Sleep disturbances	5.571 (.126–11.016)	2.032	0.045	**1.406 (.313–2.499)**	**2.556**	**0.012**	0.520 (−.897–1.937)	0.729	0.467	0.263 (−1.163–1.689)	0.366	0.715
Use of sleeping medications	3.253 (−1.034–7.541)	1.507	0.135	0.884 (.027–1.741)	2.051	0.043	0.584 (−.470–1.637)	1.102	0.274	1.232 (.186–2.277)	2.342	0.022
Daytime dysfunction	3.405 (−.416–7.225)	1.770	0.080	**1.230 (.491–1.969)**	**3.306**	**0.001**	0.556 (−.411–1.523)	1.144	0.256	1.019 (.067–1.971)	2.127	0.036

BIS, Barratt Impulsiveness Scale; CI, confidence interval; PSQI, Pittsburgh Sleep Quality Index.
Bold font indicates statistical significance.

Table 3. Correlation of the BIS and PSQI from GLMs with the Adjustment of Age, Sex, Education, Substance/Alcohol Use Disorder History, Mood Symptom Severity, and Psychotropic Medication Use

	B (95% CI)	t	*p*		B (95% CI)	t	*p*		B (95% CI)	t	*p*
					BIS total score						
PSQI total	1.173 (.237–2.109)	2.490	0.015	Sleep disturbances	5.571 (.126–11.016)	2.032	0.045	Daytime dysfunction	3.405 (−.416–7.225)	1.770	0.080
Group			0.392	Group			0.226	Group			0.342
HC	0			HC	0			HC	0		
BDI	−3.464 (−17.764–10.836)	−.481	0.632	BDI	1.916 (−10.459–14.292)	.308	0.759	BDI	0.748 (−12.408–13.904)	.113	0.910
BDII	3.483 (−8.588–15.555)	.573	0.568	BDII	7.328 (−3.204–17.860)	1.382	0.170	BDII	6.873 (−4.353–18.100)	1.216	0.227
p for interaction			0.667	*p* for interaction			0.426	*p* for interaction			0.609
Second–order factor: attention											
PSQI total	0.275 (.085–.465)	2.895	0.005	Sleep disturbances	1.406 (.313–2.499)	2.556	0.012	Daytime dysfunction	1.230	0.372	0.001
Group			0.487	Group			0.848	Group			0.103
HC	0			HC	0			HC	0		
BDI	0.882 (−1.876–3.641)	.636	0.527	BDI	1.976 (−.395–4.347)	1.656	0.101	BDI	1.193 (−1.280–3.666)	.959	0.340
BDII	2.299 (−.028–4.627)	1.964	0.053	BDII	3.015 (.974–5.056)	2.936	0.004	BDII	2.414 (.288–4.540)	2.257	0.027
p for interaction			0.517	*p* for interaction			0.695	*p* for interaction			0.288

BDI, bipolar I disorder; BDII, bipolar II disorder; BIS, Barratt Impulsiveness Scale; PSQI, Pittsburgh Sleep Quality Index.

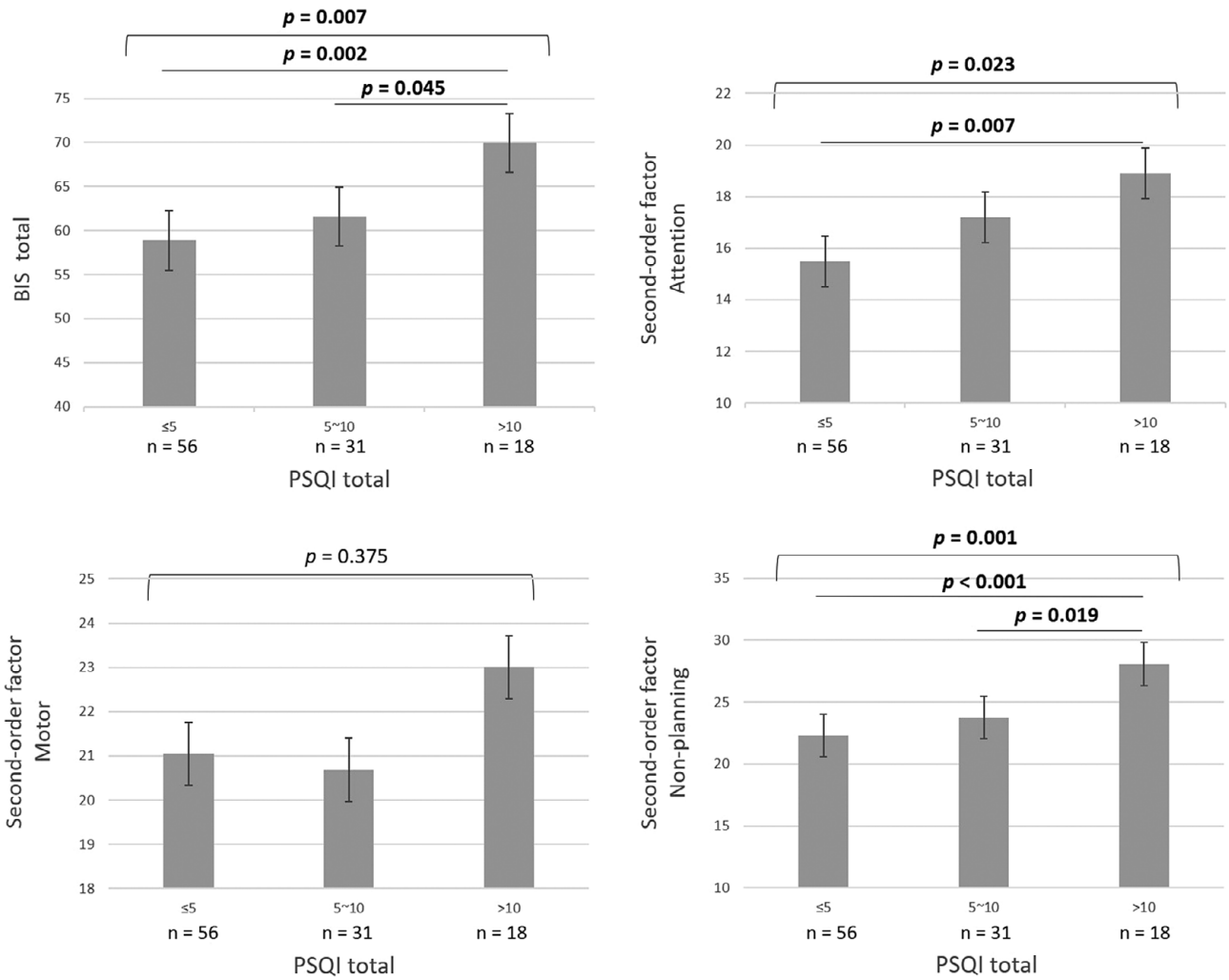

Figure 1. Comparison of BIS-11 scores among participants of different PSQI score ranges, adjusting for age, sex, education, psychotropic medication use, substance use disorder, mood symptoms, and disease group.

component of the 8 sleep disturbance items to be associated with higher attentional impulsiveness, which includes impaired task focus, intrusive thoughts, and racing thoughts. Despite the non-significant linear association between PSQI scores and the non-planning factor of the BIS-11 after multiple testing corrections, participants with PSQI total scores higher than 10 exhibited greater non-planning impulsiveness than those with low or medium PSQI scores. As patients with BD also scored high on non-planning impulsiveness items, it could be that other aspects of trait impulsivity, as opposed to attentional impulsiveness, are less likely to be directly related to sleep. Additionally, we found that total scores of the BIS-11 and PSQI were positively associated, while subgroup analyses based on different disease categories showed no such association. Our findings suggest the possibility of a shared and transdiagnostic role of sleep-related mechanisms in influencing inhibitory control, which could be independent of the specific pathological underpinnings of distinct mood disorders. The absence of statistical significance after employing multiple testing corrections or in the subgroup analyses might have resulted from the limited sample size within each subgroup. Future studies with larger cohorts are warranted to confirm our findings.

The relationship between sleep and impulsivity continues to be a subject of debate, with the bidirectional hypothesis gaining traction. Experimental studies have demonstrated that sleep deprivation significantly increases neural and autonomic responses to negative emotional stimuli,[12] and disturbed sleep may lead to increased impulsive action.[22] Conversely, evidence suggests that poor impulse control or a propensity for risk-taking may contribute to the development of sleep problems.[23] Individuals with deficits in self-regulation or higher levels of trait impulsivity and reward-seeking behavior are thought to be inclined to delay bedtime by engaging in activities that provide immediate gratification, such as media consumption or playing video games.[24,25] An alternative view suggests that sleep and impulsivity may not have a causal relationship but may instead share a common psychopathological basis. Future research should aim to clarify the relationship between poor sleep quality and impulsivity. Prospective studies could investigate whether improving sleep quality effectively reduces impulsivity. There have been a few longitudinal studies: Serena V. Bauducco found bidirectional links between sleep duration/insomnia and trait impulsivity in a cohort of 2767 young adolescents.[26] In contrast, Van Veen *et al.* found no longitudinal correlations between sleep quality and impulsivity, whether self-reported or clinician-rated, in 83 male forensic psychiatric patients.[27] The interaction between sleep and impulsivity may be particularly significant in individuals with abnormal prefrontal

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function, which could explain the inconsistencies in previous research findings. Sleep deprivation disrupts the functional connectivity of the PFC, a critical region for inhibitory control, with this effect being more pronounced in those with prefrontal dysfunction.[28] Poor sleep quality is correlated with higher impulsivity specifically in individuals with reduced PFC connectivity.[29] From an evolutionary standpoint, the reduction in inhibitory control during sleep loss may represent an adaptation for increased responsiveness to negative stimuli.[30]

A potential neural explanation exists for the impact of poor sleep on trait impulsivity. The top-down control exerted by the PFC, which mediates inhibitory capabilities, is vulnerable to sleep loss,[31] and functional connectivities between prefrontal networks, the insula, and the limbic system undergo alterations during inadequate sleep.[12,13] Sleep deprivation-induced impairments in attention and vigilance coincide with reduced metabolic activity in the prefrontal and posterior parietal cortices. These brain regions are also pivotal in the regulation of trait impulsivity.[32–34]

Our results have clinical significance for psychiatric cohorts, as concurrent insomnia may exacerbate trait impulsivity. This is noteworthy because impulsivity is linked to adverse outcomes in BD, such as suicidality and risky behavior.[35] Therefore, reducing impulsivity is a critical therapeutic objective, and improving sleep quality could serve as a potential intervention. Chronotherapeutic strategies aimed at increasing the regularity of an individual's routines, including interpersonal and social rhythm therapy and cognitive behavioral therapy for insomnia, have proven effective in treating BD-related insomnia. These therapies have been shown to delay relapse and decrease suicidal ideation.[36,37] However, it remains unclear whether these behavioral interventions can also reduce impulsivity, necessitating further research. Additionally, the potential pathophysiological mechanisms connecting sleep and impulsivity warrant investigation. Specifically, poor sleep quality is associated with maladaptive changes in the hypothalamus–pituitary–adrenal (HPA) axis, which has been implicated in impulsivity.[38,39] A better understanding of HPA axis responsiveness and its moderating effect on the sleep–impulsivity relationship could inform psychiatric classification and the development of targeted interventions for impulsivity-related undesirable behaviors.[40]

This study had several limitations. First, sleep and impulsivity indices were evaluated using self-report measures, which are susceptible to memory bias. Incorporating objective measures of sleep and a more comprehensive assessment of behavioral impulsivity would have been beneficial. Actigraphy, with its extended sampling intervals, could have provided estimates of sleep phase, duration, interdaily stability, and amplitude of the circadian rest–activity rhythm. Second, the cross-sectional design limits our ability to determine directionality. Future studies should identify which actigraphic parameters most strongly correlate with psychopathologies in affective disorders, and longitudinal designs could clarify potential intraindividual relationships between actigraphic metrics and trait impulsivity. Additionally, this study only examined the sleep–impulsivity relationship in euthymic patients with BD. The mediating role of affective disturbance in this relationship warrants further investigation. Third, the patients were on psychotropic medications during clinical assessments, which could have influenced the results, as antipsychotics and mood stabilizers can have sedative effects and modify impulsive behaviors. Nevertheless, allowing patients to continue their medications was ethically sound and likely prevented relapse and recurrence; we have accounted for the influence of psychotropic medications in our statistical analyses. Fourth, we used the PSQI, a comprehensive measure of subjective sleep quality that assesses various facets of sleep and associated symptoms, including insomnia, snoring, breathing difficulties, and daytime fatigue. However, we could not determine the specific impact of sleep disorders such as sleep apnea on impulsivity trajectories. Symptoms of particular sleep disorders could inflate cumulative PSQI scores and act as a predisposing factor for increased impulsivity. Finally, chronotype may influence the effects of sleep dysregulation on impulsivity. Previous research has linked later chronotype and eveningness preference with multiple aspects of impulsivity[41]; eveningness may adversely affect PFC and striatal function, which are implicated in impulsivity.[42,43] Future research should explore the relationship between impulsivity and chronotype in BD.

The findings corroborate the hypothesis that poor sleep quality correlates with increased trait impulsivity in individuals with BD, bolstering the growing body of evidence that enhancing sleep quality could be beneficial for those suffering from BD.

Author contribution. Validation: Y.E.C.; Writing – review & editing: Y.E.C., W-C.M., T-P.S.; Data curation: Y-H.K., T-P.S., M-H.H.; Project administration: Y-H.K., T-P.S.; Resources: Y-H.K., T-P.S.; Methodology: W-C.M., M-H.H.; Supervision: W-C.M., T-P.S.; Funding acquisition: T-P.S.; Conceptualization: M-H.H.; Formal analysis: M-H.H.; Investigation: M-H.H.; Writing – original draft: M-H.H.

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Credit authorship contribution statement. Mao-Hsuan Huang conceptualized the study, curated the data, involved in formal analysis, investigated the data, designed the methodology, and wrote the original draft. Yi-Hsuan Kuan curated the data, administered the project, and provided resources. Yee-Lam E Chan wrote, reviewed, and edited the manuscript and validated the data. Wei-Chung Mao designed the methodology; supervised the data; and wrote, reviewed, and edited the manuscript. Tung-Ping Su curated the data; acquired funding; administered the project; provided resources; supervised the data; and wrote, reviewed, and edited the manuscript.

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