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Lifetime Characteristics of Evening-Preference and Irregular Bed-Rise Time Are Associated With Lifetime Seasonal Variation of Mood and Behavior: Comparison Between Individuals With Bipolar Disorder and Healthy Controls

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Sleep–wake cycle disruption and seasonal variation in mood and behavior have been associated with mood disorders. This study aimed to investigate the lifetime characteristics of the sleep–wake cycle and its association with the lifetime characteristics of seasonality in individuals with bipolar disorder. Circadian preference, regularity of bed–rise time, and seasonality were evaluated on a lifetime basis using the Composite Scale of Morningness, the Sleep Timing Questionnaire, and the Seasonal Pattern Assessment Questionnaire in clinically stable individuals with bipolar I/II disorders ($n = 103/97$) and healthy controls ($n = 270$). Bipolar groups were more likely to have evening preference and irregular bed–rise time. These characteristics were interrelated and, particularly, more prevalent in bipolar II disorder. Seasonality, which was also more prevalent in the bipolar groups, was associated with evening preference and irregularity of the weekday bed–rise time.

Abnormalities in the sleep–wake cycle are among the most common features of bipolar disorder (BP), and these abnormalities could be associated with the core neurobiological mechanism of the illness (Harvey, 2008). Sleep difficulties, including insomnia or hypersomnia, are commonly observed at any state of bipolar disorder (Sylvia et al., 2012). In addition, sleep manipulation can improve or worsen mood symptoms (Colombo, Benedetti, Barbini, Campori, & Smeraldi, 1999; Wirz-Justice & Van den Hoofdakker, 1999).

In addition to problems related to total sleep duration, temporal shifting of sleep timing is also frequently observed in individuals with BP. Circadian preference or individual differences in the temporal organization of biological and behavioral rhythms (Natale, Adan, & Scapellato, 2005), which is typically classified as either morning or evening preference, has been widely studied. Individuals with BP often exhibit an evening preference, that is, they have a tendency to be more active or energetic during the evening time (Ahn et al., 2008; Mansour et al., 2005; Wood et al., 2009). Circadian preference is a physiological trait with a distinct biological basis, and individuals with evening preference show a phase delay in their peak body temperature and cortisol level compared to individuals with morning preference (Hilliker, Muehlbach, Schweitzer, & Walsh, 1992; Kerkhof & Van Dongen, 1996).

Another characteristic of the sleep–wake cycle that might be related to BP is an irregular bed–rise time. In studies using sleep diaries, individuals with BP reported frequent changes in their bed–rise time (Eidelman, Talbot, Gruber, & Harvey, 2010). A similar finding was also observed in studies using actigraphy (Jones, Hare, & Evershed, 2005; Millar, Espie, & Scott, 2004). Irregularities in bed–rise time and daily activities were also observed in individuals putatively at risk for BP (Meyer & Maier, 2006). According to the social zeitgeber theory (Reid, Towell, & Golding, 2000), physiological rhythm (circadian preference) and socio-behavioral rhythm (when to have meals, get exercise, get socialized, and take a rest within a day) were closely associated. A mismatch between circadian preference and daytime schedule could have a detrimental effect on sleep quality (Roepke & Duffy, 2010; Soehner, Kennedy, & Monk, 2011) and eventually worsen the clinical course of BP.

The sleep–wake cycle is related to an individual's biological response to light (Bass, 2012). Seasonality, which is defined as seasonal variations in mood, behavior, and vegetative symptoms, is also related to an individual's biological response to seasonal variations of the light and dark cycle. Higher seasonality was also reported in participants with BP compared to healthy participants (Choi et al., 2011; Goikolea et al., 2007; Shand, Scott, Anderson, & Eagles, 2011). Thus, increased seasonality and sleep–wake cycle disruptions (i.e., evening preference

and more irregular bed–rise time) could be interrelated and share a common neurobiological mechanism that might also be involved in the development of BP.

Previous studies have consistently reported that bipolar II disorder (BP-II) exhibits distinct clinical characteristics (Baek et al., 2011; Bega, Schaffer, Goldstein, & Levitt, 2012; Judd et al., 2003) and treatment response (Bond, Noronha, Kauer-Sant’Anna, Lam, & Yatham, 2008) from bipolar I disorder (BP-I). In several studies, BP-II also exhibited contrasting neuropsychological (Ha et al., 2012), genetic (Huang et al., 2012; S. Lee et al., 2012) and neuroimaging (Ha et al., 2011; Liu et al., 2010) findings, indicating that BP-I and BP-II might have different neurobiological underpinnings. In particular, individuals with BP-II also showed a more cycling nature, including increased episode frequency and rapid cycling (Judd et al., 2003). In our prior study, females with BP-II showed greater seasonality and more frequent premenstrual symptoms compared to BP-I (Choi et al., 2011).

This study aimed to determine the characteristics of the sleep–wake cycle (circadian preference and regularity of bed–rise time) and its association with seasonality in patients with BP. We hypothesized that individuals with BP would exhibit a higher rate of sleep–wake cycle dysregulation and seasonality, and that these characteristics would be more evident among individuals with BP-II relative to BP-I. We also hypothesized that these traits would correlate with each other.

METHODS

Participants

Two hundred patients who met the diagnostic criteria of the DSM-IV (APA, 1994) BP-I ($n = 103$) or BP-II ($n = 97$) were recruited from the outpatient and inpatient units of Samsung Medical Center and Seoul National University Bundang Hospital. Board-certified psychiatrists who had at least one year of research experience examined the participants’ psychiatric diagnoses using the DSM-IV criteria with either the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; Han & Hong, 2000) or the Korean version of the Diagnostic Interview for Genetic Studies (DIGS; Joo et al., 2004). SCID was used at Seoul National University Bundang Hospital and DIGS was used at the Samsung Medical Center. No significant difference in terms of demographic characteristics was detected between participants evaluated using SCID and DIGS. Considering the age-related changes in sleep architecture that become prominent since the patient’s mid-40s (Zhou, Liu, van Heerikhuizen, Hofman, & Swaab, 2003), we limited the participants’ ages to between 18 and 45 years. Participants who took part in our prior study (Choi et al., 2011; $n = 61$) on seasonality and premenstrual symptoms were also included in this study. Participants were required to be clinically stable, that is, they had to have a score of 3 (mildly ill) or lower on the Clinical Global Impression of Severity (CGI-S; Guy, 1976), and a validated (Beneke & Rasmus, 1992) seven-point one-item scale, at the time of assessment. The clinical severity was also determined by the same psychiatrists who performed the clinical diagnosis on the patients. Using comprehensive psychiatric evaluation, we only included individuals who could reliably report their lifetime trait of seasonality and lifetime habitual pattern in their sleep–wake cycle. We excluded participants if they could not clearly remember their lifetime traits due to illnesses. Moreover, to prevent the effect of current mood

state or medication in evaluating their lifetime characteristics, we included participants after their medication had been stabilized. All participants were under the standard pharmacological treatment, including mood stabilizers (lithium, valproate, lamotrigine, and carbamazepine) or atypical antipsychotics (quetiapine, olanzapine, risperidone, aripiprazole, and ziprasidone). As healthy controls, 270 age- and sex-matched participants without any history of psychiatric illness were recruited. Individuals with mental retardation, substance abuse, medical illness, or long-term use of hormonal agents known to affect mood were excluded from both patients and control groups. Because sustained social rhythm deeply affected biological rhythm (Grandin, Alloy, & Abramson, 2006) and recall on previous sleep–wake pattern, night-shift workers were also excluded as participants. The demographic characteristics of the participants are summarized in Table 1. Written informed consent was obtained from all participants after they were provided with a complete explanation of the study. This study was approved by the institutional review board of Samsung Medical Center and Seoul National University Bundang Hospital.

Measurements

The circadian preference was measured using the Composite Scale of Morningness (CSM) (Smith, Reilly, & Midkiff, 1989), and the regularity of bed–rise time was measured using the Sleep Timing Questionnaire (STQ; Monk et al., 2003). The CSM is a questionnaire evaluating circadian preference. The CSM contains 13 questions concerning what time the participants preferred to go to bed, wake up, and perform specific activities. The sum of the answers of these questions yields a single score, which represents the level of circadian preference (range, 13–55), with lower numbers indicating a stronger evening preference. The STQ is a “single-shot” questionnaire designed to measure the habitual bed and rise time separately for weekdays (from Sunday night through Friday morning) and weekends (from Friday night through Sunday morning). We asked participants to describe their usual bed and rise time, to determine the trait aspect of their bed–rise time pattern. The STQ also yields scores of variability of the subject’s habitual bed–rise time (on a 1–11 scale: 1, variable within 0–15 min; 2, variable within 16 to 30 min . . . 9, variable within 2–3 h; 10, variable within 3–4 h; 11, variable more than 4 h; with higher numbers indicating greater variability).

Seasonality was measured using the Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal et al., 1984), which contained six items measuring seasonal variation in sleep, social activity, mood, weight, appetite, and energy level. The sum of the individual item scores (each on a 5-point scale ranging from 0 = *no change* to 4 = *extremely marked change*) represents the global seasonality score (GSS). A GSS of higher than 11, in addition to a subjective rating of having at least moderate difficulty with seasonal changes (on a 6-point scale from 0 = *no difficulty* to 5 = *a disabling difficulty*; 2 = *moderate*) was regarded as seasonal affective disorder (SAD) (Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989). A GSS between 9 to 10 with a subjective rating of having more than moderate difficulty with seasonal change or a GSS of more than 11 with a subjective rating of having mild difficulty (= 1) was designated as subsyndromal SAD (SSAD).

We used the standardized Korean version of the CSM (Yoon, Shin, Kook, & Lee, 1997). SPAQ and STQ were translated into Korean by the authors. According to the preliminary

TABLE 1
Sociodemographic Characteristics of Subjects

	BP-I (n = 103)	BP-II (n = 97)	Healthy Controls (n = 270)	Statistics	p-value	Pairwise Comparison, p-value		
						BP-I vs. BP-II	BP-I vs. Controls	BP-II vs. Controls
Age, years, mean (SD)	30.3 (6.9)	32.2 (7.7)	30.8 (6.3)	$F = 2.26$	0.105			
Duration of illness, months, mean (SD)	89.9 (68.8)	91.0 (68.3)		$T = 0.36$	0.717			
Sex, M, n (%)	38 (36.9)	31 (32.0)	107 (39.6)	$\chi^2 = 1.81$	0.405			
Education: College graduate or more, n(%)	58 (56.3)	57 (58.8)	237 (87.8)	$\chi^2 = 56.18$	<0.001	1.000	<0.001	<0.001
Occupation, present, n (%)	79 (76.7)	83 (85.6)	267 (98.9)	$\chi^2 = 51.11$	<0.001	0.330	<0.001	<0.001
Currently married, n (%)	25 (24.3)	41 (42.3)	103 (38.1)	$\chi^2 = 8.35$	0.015	0.021	0.036	1.000

Note. BP-I, bipolar I disorder; BP-II, bipolar II disorder; SD, standard deviation. Post-hoc correction in pairwise comparison was done using Bonferroni correction.

evaluation of the validity and reliability of the SPAQ (with 20 participants, the mean test–retest interval of 60 weeks), the SPAQ exhibited internal consistency and test–retest reliability with acceptable levels, that is, Cronbach’s alpha of 0.93 and statistically significant intraclass correlation between the test and retest scores for all of the six items included in the SPAQ.

Procedures

We assessed the lifetime characteristics of the seasonality and sleep–wake cycle, that is, the circadian preference and the regularity of bed–rise time, using self-reporting rating scales. We gave the following instruction to all participants: “In this study, we want to evaluate your lifetime characteristics of sleep–wake pattern and seasonality. Please answer all questions based on your habitual patterns regardless of disease, medications, or any specific events happening in your life.” In the case of the patient group, all questionnaires were administered in an interview by board-certified psychiatrists or a research nurse with more than 2 years of clinical experience to clarify the time frame of all questions.

Statistical Analysis

In analyses of sociodemographic variables, parametric variables were compared using analysis of variance (ANOVA) followed by post-hoc pairwise comparisons using the least significant difference (LSD). Categorical data were analyzed using a chi-square test, and the Bonferroni correction was applied for post-hoc pairwise comparisons.

The mean GSS, CSM score, scores of variability of bed and rise times, and the prevalence of SAD were compared among the three participant groups using analyses of covariance (ANCOVA). Because all of these measures violated the assumption of the normality of distribution, we applied log-transformation when performing ANCOVA. Age, sex, and occupation were entered as covariates in the analyses. The prevalence of SAD was compared among the three groups using a chi-square test. Bonferroni correction was applied for the post-hoc pairwise comparisons.

We determined the interrelationship between the sleep–wake cycle characteristics and seasonality. Partial correlation analyses were performed between every pair of sleep cycle-related indices, that is, the CSM score and variability scores of bed and rise times. In evaluating the relationship between sleep–wake cycle disruption and seasonality, sleep cycle-related indices were compared between individuals with SAD or SSAD and those without SAD and SSAD using ANCOVA. Age, sex, occupation, and diagnosis (BP-I, BP-II, or controls) were entered as covariates in all analyses.

A probability (p) value of less than 0.05 was considered statistically significant. All of the statistical analyses were performed using Predictive Analytics Software (PASW) version 18.0 (2008).

RESULTS

Compared to the control group, participants with BP showed a lower level of education, occupation status, and current marriage rate (Table 1).

Participants with BP-II exhibited significantly lower CSM scores, which indicate more evening preferences, compared to healthy controls and those with BP-I. Patients with BP-I also had significantly lower CSM scores compared to healthy controls (Table 2). In terms of the variability of the bed and rise time, BP groups scored higher in the variability of weekday bed and rise time. Detailed statistics of group differences are presented in Table 2.

The mean GSS and the prevalence of SAD and SAD+SSAD were significantly different among the groups ($p < 0.001$; Table 3). In post-hoc analyses, both the BP-I and BP-II groups exhibited a significantly higher mean GSS compared to controls. They also showed significantly higher SAD and SAD+SSAD rates compared to the control group. No significant difference was observed between the BP-I and BP-II in terms of these seasonality indices.

In analyses of the association between sleep–wake cycle characteristics, a relatively high correlation was observed between the variability scores of the bed and rise times on weekdays/weekends ($\rho = 0.409\text{--}0.644$, $p < 0.001$; Table 4). These scores also showed a significant negative correlation with the CSM score ($\rho = -0.275$ to -0.220 , $p < 0.001$; Table 4). In the analysis of the association between the sleep–wake cycle indices and seasonality, individuals with SAD or SSAD ($n = 154$) showed significantly lower CSM scores and higher variability scores of weekday bed and rise time compared to those without SAD and SSAD ($n = 316$; Table 5). In addition, we separated all three groups (BP-I, BP-II, and control groups) and compared those with SAD or SSAD and those with no seasonality (GSS < 9 and no functional impairment due to season). In all groups, those with SAD or SSAD had lower CSM scores and higher variability scores of weekday bed and rise time, but the results were only statistically significant in the healthy controls (detailed data not shown).

DISCUSSION

In this study, we evaluated the association between sleep–wake cycle characteristics and seasonality in individuals with BP. As we hypothesized, individuals with BP exhibited greater evening preference, more variable bed and rise time on weekdays, and higher seasonality compared to healthy controls. The sleep–wake cycle-related characteristics were more prominent in individuals with BP-II. The sleep–wake cycle-related variables were interrelated and significantly associated with seasonality.

The CSM scores of the current participants were consistent with those generated from previous studies (CSM scores of healthy participants ranged from 32.6 to 34.2; CSM scores of participants with BP ranged from 30.5 to 33; Caci, Deschaux, Adan, & Natale, 2009; K. Y. Lee et al., 2010; Wood et al., 2009). As a heritable trait (Klei et al., 2005) that is stable over time in an individual during nonelderly ages (Greenwood, 1994; Wood et al., 2009), circadian preference seems to be determined by circadian body temperature regulation (Kerkhof & Van Dongen, 1996) and physiological sleep architecture (Hilliker et al., 1992). These two biological factors are also known to be involved in the pathophysiology of BP (Harvey, 2008).

Although irregular bed–rise time is commonly encountered in patients with BP in a clinical setting, only a small number of studies have focused on this issue. Using sleep diaries (Eidelman et al., 2010) and actigraphy (St-Amand, Provencher, Belanger, & Morin, 2013), previous studies reported more irregular bed–rise time in participants with BP. The STQ has shown similar results with sleep diaries in evaluating the irregular bed–rise time in healthy participants (Monk

TABLE 2
Comparison of Circadian Preference and Regularity of Sleep–Wake Time in Subjects With Bipolar I and Bipolar II Disorder, and Healthy Controls

	BP-I (n = 103)	BP-II (n = 97)	Healthy Controls (n = 270)	Statistics	p-value	Pairwise Test, p-value		
						BP-I vs. BP-II	BP-I vs. Control	BP-II vs. Control
Score of Composite Scale of Morningness, mean (SD)	31.2 (7.3)	30.2 (8.7)	34.6 (6.7)	$F = 15.98$	<0.001	0.040	0.003	<0.001
Variability of bed–rise time, mean (SD)								
Weekday bed time	4.9 (3.1)	5.4 (3.0)	4.3 (2.5)	$F = 4.34$	0.014	0.056	0.508	0.004
Weekday rise time	3.9 (2.9)	4.2 (3.0)	2.8 (2.3)	$F = 13.95$	<0.001	0.167	0.001	<0.001
Weekend bed time	5.9 (3.0)	6.0 (3.1)	5.8 (2.8)	$F = 0.29$	0.746			
Weekend rise time	5.3 (3.2)	5.4 (2.9)	5.5 (2.8)	$F = 1.30$	0.274			

Note. BP-I, bipolar I disorder; BP-II, bipolar II disorder; SD, standard deviation. Covariate: age, sex, occupation; bed–rise time variability was measured by a scale of 1–11, with higher numbers indicating greater irregularity.

TABLE 3
Comparison of Seasonality Among Subjects With Bipolar I and Bipolar II Disorder, and Healthy Controls

	<i>BP-I</i> (<i>n</i> = 103)	<i>BP-II</i> (<i>n</i> = 97)	<i>Healthy</i> <i>Controls</i> (<i>n</i> = 270)	<i>Statistics</i>	<i>p-value</i>	<i>Pairwise Test, p-value</i>		
						<i>BP-I vs.</i> <i>BP-II</i>	<i>BP-I vs.</i> <i>Control</i>	<i>BP-II vs.</i> <i>Control</i>
Seasonality, <i>n</i> (%) or mean (<i>SD</i>)								
GSS	7.9 (5.5)	8.6 (5.8)	6.0 (4.1)	$F = 7.63$	0.001	0.196	0.028	<0.001
SAD	23 (22.3)	23 (24.0)	15 (5.6)	$X^2 = 53.51$	$p < 0.001$	1.000	<0.001	<0.001
SAD+SSAD	45 (43.7)	54 (55.7)	55 (20.4)	$X^2 = 47.51$	$p < 0.001$	0.270	<0.001	<0.001

Note. BP-I, bipolar I disorder; BP-II, bipolar II disorder; *SD*, standard deviation; GSS, global seasonality score; SAD, seasonal affective disorder; SSAD, subsyndromal seasonal affective disorder. Post-hoc correction in pair-wise comparison was done using LSD for GSS, Bonferroni correction for SAD, SAD+SSAD. Covariate: age, sex, occupation. Seasonal affective disorder was defined as GSS > 11 with moderate/severe functional impairment; subsyndromal seasonal affective disorder was defined as GSS 9–10 with moderate/severe functional impairment or GSS > 11 with less than mild functional impairment

TABLE 4
Partial Correlation Between Sleep–Wake Rhythm Characteristics (ρ Value)

	<i>CSM Score</i>	<i>Variability</i> <i>of Weekday</i> <i>Bed Time</i>	<i>Variability</i> <i>of Weekday</i> <i>Rise Time</i>	<i>Variability</i> <i>of Weekend</i> <i>Bed Time</i>
Variability of weekday bed time	−0.237*			
Variability of weekday rise time	−0.266*	0.532*		
Variability of weekend bed time	−0.220*	0.644*	0.409*	
Variability of weekend rise time	−0.275*	0.494*	0.441*	0.630*

* < 0.001 .

Note. CSM, Composite Scale of Morningness. Age, sex, occupation and diagnosis (bipolar I disorder; bipolar II disorder; healthy controls) were entered as covariates.

TABLE 5
Comparison of Sleep–Wake Rhythm Characteristics According to the Seasonality

	<i>SAD/SSAD</i> (<i>n</i> = 154)	<i>Non-SAD/SSAD</i> (<i>n</i> = 316)	<i>Statistics</i>	<i>p-value</i>
CSM scores	30.72 (7.6)	33.99 (7.3)	$F = 6.85$	0.009
Variability of weekday bed time	5.38 (3.06)	4.29 (2.57)	$F = 6.71$	0.010
Variability of weekday rise time	4.03 (3.03)	2.98 (2.38)	$F = 4.97$	0.026
Variability of weekend bed time	6.09 (3.02)	5.77 (2.82)	$F = 0.47$	0.493
Variability of weekend rise time	5.71 (3.21)	5.34 (2.77)	$F = 0.31$	0.578

Note. CSM, Composite Scale of Morningness. Age, sex, occupation and diagnosis (bipolar I disorder, bipolar II disorder, healthy controls) were entered as covariates in all analyses.

et al., 2003). In previous studies using the STQ, increased variability in sleep timing was also reported in participants at risk of BP as defined by high scores in the Hypomanic Personality Scale (Ankers & Jones, 2009; Meyer & Maier, 2006) and in natural short sleepers who usually sleep from three to six hours (Monk et al., 2003).

In the current study, participants with BP-II showed lower CSM scores and more irregular weekday bed–rise time. These results suggest that cyclicity-related characteristics may be more associated with BP-II than with BP-I. In a broad sense, these results are consistent with previous findings indicating more frequent and more rapidly cycling mood episodes in BP-II than in BP-I (Judd et al., 2003; Vieta, Gasto, Otero, Nieto, & Vallejo, 1997). To the best of our knowledge, few studies have investigated the characteristics of sleep–wake cycles in individuals with BP-II. Only two previous studies have evaluated the CSM scores of those with BP-II separately from those with BP-I; one study found no difference between BP-I and BP-II (Wood et al., 2009) and the other study reported lower CSM scores in BP-II (K. Y. Lee et al., 2010). Evening preference was also correlated with characteristics including anxiety (Gaspar-Barba et al., 2009) and poorer social adjustment (Giannotti, Cortesi, Sebastiani, & Ottaviano, 2002), which were presented more often in BP-II compared to BP-I patients (Baek et al., 2011; Mantere et al., 2006; Mantere et al., 2008). There has been no prior study comparing sleep irregularity between BP-I and BP-II.

Consistent with a previous report (Soehner et al., 2011), evening preference and variability of bed–rise time were closely correlated in the current study, and the variability indices were also correlated with each other. The sleep pattern of individuals with evening preference could hardly be maintained due to its mismatch with daytime schedules. It is well known that an irregular sleep–wake cycle can worsen mood symptoms in BP (Sylvia et al., 2009). If patients with BP who are vulnerable to changes in the sleep–wake cycle, that is, having an evening preference, have also had an irregular life schedule, the vicious cycle that may develop could eventually worsen the clinical course of BP. Interpersonal social rhythm therapy (IPSRT) focuses on maintaining a stable daily routine, including sleep timing and demonstrated efficacy in decreasing depressive symptoms and preventing a new episode in BP (Frank et al., 2005).

In the current study, seasonality was associated with an evening preference and irregular bed–rise time. Previous studies reported similar findings in healthy controls (Murray, Allen, & Trinder, 2003; Natale et al., 2005). Lee et al. (2011) also reported that participants with delayed sleep phase syndrome showed higher seasonality. Lewy, Sack, Singer, White, and Hoban (1988) postulated the phase shift hypothesis of SAD, which indicates that winter depression might be caused by an abnormal delay of the circadian phase in winter. Furthermore, Natale et al. (2005) hypothesized that individuals with an evening preference, who normally have a delayed circadian phase, might be predisposed to SAD. The correlation between circadian preference and seasonality might derive from a common biological mechanism between these two traits, which might also be related to the core neurobiological underpinning of BP. On the basis of the findings obtained from genetic studies, the melatonin (Delavest et al., 2012; Etain et al., 2012) or serotonin (Willeit et al., 2003) system might be involved in the development of these traits. In addition, common personal and psychosocial factors could contribute to both the seasonality and sleep–wake cycle. For instance, lifestyles or habits related to the seasonality could change the sleep–wake cycle; vice versa, an irregular bed–rise time and evening preference could worsen the seasonality by decreasing the amount of time that the patient is exposed to light.

The present study has specific limitations that should be taken into consideration when interpreting the data. First, the measurement of seasonality and sleep–wake cycle was based on a self-reporting questionnaire. More direct physiological measures such as actigraphy or prospective evaluation of mood and behavior following seasonal changes can help to confirm these findings. Second, although we recruited clinically stable patients who were expected to reliably report on their premorbid histories of sleep–wake cycle and seasonality, recollections on these conditions might have been biased in some cases by their residual manic or depressive symptoms at the time of the study. Third, although night-shift workers were excluded as participants, we could not completely adjust for the effect of other social environments that could affect the individual’s lifestyle and sleep–wake cycle. Lastly, although we measured the patients’ sleep and seasonality parameters based on lifetime characteristics, the current study design could not evaluate the directionality of the association between the disease and the circadian and seasonal characteristics.

CONCLUSIONS

Within the above-mentioned limitations, this study revealed that participants with BP had more evening preference and a more irregular bed–rise time. These behavioral patterns were interrelated with each other and were more prominent in participants with BP-II compared to those with BP-I. The sleep–wake cycle characteristics were associated with seasonality (of mood and behavior), which was also more frequently observed in patients with BP compared to healthy controls. Circadian and seasonal rhythm-related traits appeared to have a common biopsychosocial basis that might also be related to the development of BP.

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