Variability in Sleep Is Associated with Trait-Based and Daily Measures of Bipolar Spectrum Psychopathology

By: Sarah H. Sperry, Thomas R. Kwapil

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Abstract:

Introduction: Although sleep disturbances are well documented in bipolar spectrum disorders (BSDs), significantly less research has examined whether these disturbances are present in those at risk for developing BSDs or with subsyndromal symptoms. The present study examined associations between risk for BSDs, as measured by the Hypomanic Personality Scale (HPS), and sleep assessed using experience sampling. We assessed whether intraindividual variability in sleep was associated with affect, cognition, and behavior in daily life and potential directionality of these relationships.

Methods: 233 young adults oversampled for high scores on the HPS completed 14 days of experience sampling assessing total sleep time (TST), bed/rise time, sleep quality, affect (negative and positive affect), cognition (difficulty concentrating, racing thoughts), and behavior (impulsivity) in daily life. We used Dynamic Structural Equation Modeling (DSEM) to assess within person links between sleep and bipolar spectrum psychopathology.

Results: HPS scores were associated with less TST, later bedtime, and more variable TST and bedtime. Variability in TST was associated with negative affect, difficulty concentrating/racing thoughts, and impulsivity. Within person decreases in sleep were associated with next day increases in negative affect, stress, difficulty concentrating, and racing thoughts.

Limitations: Measurement of sleep was limited. Future studies should examine both objective measures of sleep (e.g., actigraphy) and fragmentation in sleep.

Conclusions: Risk for BSD was associated with similar patterns of sleep disruptions as seen in BSDs. Important dynamic links between sleep and bipolar spectrum psychopathology emerged indicating that sleep is an important target for improving symptoms of BSDs in daily life.

Keywords: sleep | sleep health | bipolar spectrum | hypomanic personality scale | dynamic structural equation modeling

Article:

Introduction

Although sleep disturbances are well documented in bipolar spectrum disorders (BSDs), significantly less research has examined whether these disturbances are present in those at risk for developing BSDs or with subsyndromal symptoms. The present study examined associations between risk for BSDs, as measured by the Hypomanic Personality Scale (HPS), and sleep assessed using experience sampling. We assessed whether intraindividual variability in sleep was associated with affect, cognition, and behavior in daily life and potential directionality of these relationships. 233 young adults oversampled for high scores on the HPS completed 14 days of experience sampling assessing total sleep time (TST), bed/rise time, sleep quality, affect (negative and positive affect), cognition (difficulty concentrating, racing thoughts), and behavior (impulsivity) in daily life. We used Dynamic Structural Equation Modeling (DSEM) to assess within-person links between sleep and bipolar spectrum psychopathology. HPS scores were associated with less TST, later bedtime, and more variable TST and bedtime. Variability in TST was associated with negative affect, difficulty concentrating/racing thoughts, and impulsivity. Within-person decreases in sleep were associated with next day increases in negative affect, stress, difficulty concentrating, and racing thoughts. Measurement of sleep was limited. Future studies should examine both objective measures of sleep (e.g., actigraphy) and fragmentation in sleep. Risk for BSD was associated with similar patterns of sleep disruptions as seen in BSDs. Important dynamic links between sleep and bipolar spectrum psychopathology emerged indicating that sleep is an important target for improving symptoms of BSDs in daily life.

Bipolar spectrum disorders (BSDs) are characterized by sleep disturbances both before episode onset and during manic/hypomanic and depressive episodes (e.g., Cassidy et al., [8]). Disturbances include more variable total sleep time (TST), longer or shorter TST, and disrupted sleep quality (see Ng et al., [26] for review). Sleep disturbances are related to a more severe course of BSDs including increased severity and frequency of depressive, manic, and mixed episodes (e.g., Boland & Alloy, [5]). However, overall instability of sleep and circadian rhythms is not limited to acute episodes and may exist independently of symptoms and course of the disorder. Indeed, those at risk for the development of BSDs or those who do not meet diagnostic criteria for the disorder experience less TST, later bedtimes, and worse sleep efficiency, as well as more variable TST and bed/rise times compared to healthy controls (Ankers & Jones, [1]; Hensch et al., [18]; Meyer & Maier, [23]; Shen et al., [28]).

Consistent with the social zeitgeber theory of mood disorders (Ehlers et al., [10]), disruptions in TST, bed/rise times, or sleep quality may be associated with the development and expression of bipolar symptoms, especially mood dysregulation. Bauer et al. ([3]) found that decreased TST was associated with symptoms of hypomania, whereas increased TST predicted depressive symptoms. This is consistent with findings from the STEP-BD study that found longer sleepers (>9hrs) had greater depressive symptoms, whereas short sleepers (<6hrs) showed greater mood elevation (Gruber et al., [13]). When examining the coupling between affect and sleep in interepisodic BSDs, Gershon et al. ([12]) reported that sleep was linked with negative affect (NA) but was generally unassociated with positive affect (PA). Lastly, greater variability in bed/rise time was associated with greater levels of depressive symptoms (Eidelman et al., [11]). Although the link between bipolar spectrum psychopathology (the full spectrum of symptoms and impairment underlying BSDs) and sleep is established in clinical BSDs, only one study to our knowledge has examined this link in those at risk for BSDs. Shen et al. ([28]) reported that social rhythm dysregulation (including bed/rise times) was associated with depressive symptoms and across-day

mood instability in those at risk for BSDs (Shen et al., [28]). Thus, an important next step in understanding the role of sleep and circadian disturbances in those at risk for BSDs is to examine the within-person associations between sleep, affect, cognition, and behavior in daily life.

The present study was the first to our knowledge to use experience sampling methodology (ESM), an ecologically valid daily diary method, to assess the within-person links between sleep and symptoms of BSDs in a large sample of young adults oversampled for high-risk scores on the Hypomanic Personality Scale (HPS; Eckblad & Chapman, [9]). Specifically, we examined (1) relationships between HPS scores and daily sleep, (2) the within-day and carry-over (day-to-day) associations between sleep duration (TST), affect (NA, PA, stress), cognition (difficulty concentrating, racing thoughts), and behavior (impulsivity), and (3) whether links between TST, affect, cognition, and behavior were associated with severity of risk as assessed by scores on the HPS. We hypothesized that greater variability in TST would be associated with greater expression of BSD symptoms in daily life.

Method

Participants

Participants were (n = 352) enrolled via general psychology courses at a large midwestern university or were recruited from departmental pre-screening if they scored 1.5 SD above the mean on the HPS. This recruitment strategy ensured a broad range of scorers on the HPS. College students with elevated scores on the HPS report bipolar symptoms and have heightened risk for BSDs (e.g., Walsh et al., [30]). The final sample included 233 participants (AgeM = 18.81, AgeSD = 1.04; 71% female, 52% White, 22% Asian, 13% Black) after excluding participants who withdrew from the study (n = 5), had technical difficulties with the ESM applications that prevented their participation (n = 47), completed too few ESM questionnaires (a priori criterion based on power analysis <20 per week; n = 71), or for invalid responding at the initial assessment (n = 4). Participants received course credit and those with ≥ 70% adherence on ESM questionnaires were entered into a drawing for a 100 USD gift card.

Materials & procedures

All procedures were approved by the Institutional Review Board. Participants completed informed consent, the HPS, and training on ESM procedures using their personal smartphones. The HPS is a well-validated 48-item self-report measure that assesses risk for BSDs. HPS scores are uniquely associated with BSDs in college students (Kwapil et al., [20]; Walsh et al., [30]) with up to 78% of high scorers on the measure meeting criteria for a past hypomanic episode (Eckblad & Chapman, [9]). In two different studies, approximately 30% of individuals in the upper quartile on the HPS met criteria for a DSM-IV-TR Bipolar Diagnosis (Kwapil et al., [20]; Walsh et al., [30]). The ESM questionnaire (supplemental Table 1) assessed NA (angry, irritable, nervous, afraid, sad, bored, sluggish) and PA (enthusiastic, excited, determined, confident), as well as cognitive (difficulty concentrating, racing thoughts) and behavioral (impulsivity) characteristics of bipolar spectrum psychopathology (Sperry & Kwapil, [29]). Each item was assessed on a 7-point Likert scale from 1: Not at all to 7: Very Much. Participants completed the ESM questionnaire eight times daily for 14 days between 10am and 10pm. ESM questionnaires were sent at random times within 90-minute stratified intervals and were required to be completed

within 10 minutes of the notification. Beginning the morning after their first day of participation, participants completed a three-item sleep diary that assessed bedtime, risetime, and sleep quality using ESM items adapted from the Pittsburgh Sleep Quality Index (Buysse et al., [7]). Sleep quality was rated: 0: Very good, 1: Fairly good, 2: Fairly Bad, 3: Very bad. Sleep diaries were sent to participants at 5:00am and were available for completion until the end of that day. TST was calculated as duration between the time participants reported falling asleep (bedtime) and the time they reported waking up (risetime).[1]

Statistical approach

Given that affect, cognition, and behavior were sampled at a higher rate that sleep (8 times per day for 14 days vs. once per day), we averaged affect, behavior, and cognition responses into a day-level measure in order to be able to examine cross-lag relationships. Next, we used Dynamic Structural Equation Modeling (DSEM; Asparaouhov et al., [2]; Hamaker et al., [14]) to fit several bivariate multilevel first-order vector autoregressive (VAR[1]) models. Specifying 10,000 iterations, each model was estimated using Bayesian Markov chain Monte Carlo estimation which imputes missing data. DSEM decomposes within- and between-person variance using latent mean centering and allows for multiple random effects and their covariances to be modeled simultaneously. Unconditional models (see Figure 1) were fit for each within-person variable of interest (NA, PA, stress, difficulty concentrating, racing thoughts, impulsivity). These models resulted in eight random effects: mean intensity, autoregressive coefficients, cross-lagged regressions, and variability (as indicated by random logs of unique innovation variance). Following estimation of each unconditional model, we fit models which regressed all random effects at the between-person level on HPS scores (grand mean centered).

Results

Recruitment of high scorers on the HPS was successful (M = 21.02, SD = 7.84, Range = 4–45, possible Range = 0–48, $\propto$ =.85). Descriptive statistics and within- and between-person zero-order correlations for all relevant study variables are presented in supplementary Table 2. Participants completed an average of 14 sleep diaries (SD =.47, Range = 10–15).[2] Participants slept on average 7hrs and 21 min (SD = 1:55[H:MM]) with an average bedtime of 1:14AM (SD = 2:38) and risetime of 8:37AM (SD = 2:17); however, there were significant individual differences and variability in these sleep parameters. At the between-person level, higher HPS scores were associated with less TST and later bedtimes but were not significantly associated with sleep quality or risetimes (see Table 1). As hypothesized, HPS scores were associated with greater night-to-night variability in TST, bedtime, sleep quality, but not risetime.

Next, we examined associations between average levels and variability of TST with affect, behavior, and cognition in daily life. Full correlation matrices between random effects at the between person level and unstandardized point estimates are available in supplemental Tables 3–9. First, a person's average duration of sleep (μ_{TST}) was largely unrelated to their average level of NA, PA, stress, difficulty concentrating, racing thoughts, or impulsivity. However, greater night-to-night variability in sleep duration ($\log(\pi_{TST})$) was associated with greater mean intensity of NA (μ_{NA}) and greater day to-day variability in NA ($\log(\pi_{NA})$). The same pattern emerged for difficulty concentrating, racing thoughts, and impulsivity. In addition, variability in TST ($\log(\pi_{TST})$) was associated with greater average levels of stress (μ_{Stress}).

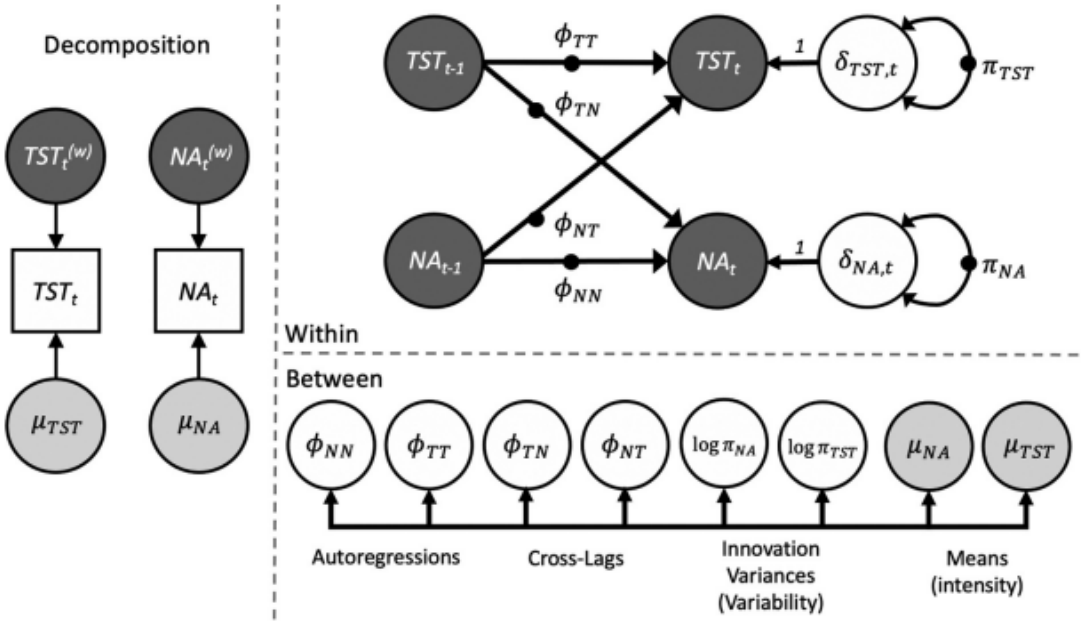

Figure 1. Example dynamic structural equation model (DSEM) with total sleep time and negative affect. (w) = within-individual variables. TST = Total Sleep Time. NA = Negative Affect. Example path diagram for unconditional bivariate multilevel VAR(1) model. Eight random effects were specified: two individual differences each (TST and HAN) for mean intensity, autoregressions, cross-lagged regressions, innovation variances (variability). This figure was adapted with permission from Hamaker et al. (2018). Full model outputs are available in supplemental material.

Table 1. Conditional DSEM model exploring associations between sleep parameters and HPS scores.

		Between Subjects Predictor of random effects (HPS)
Variable	Estimated Mean [95% Credibility Interval]	Standardized Point Biserial Estimate [95% Credibility Interval]
TST		
Mean intensity(μ_{TST})	7.39 [7.27, 7.50]	−.11 [−.21, −.01]
Variability (log(π_{TST}))	.91 [.81, 1.02]	.15 [.05, .26]
Bedtime		
Mean intensity (μ_{bed})	13.27 [13.12, 13.42]	.10 [−.00, .20]
Variability (log(π_{bed}))	.95 [.77, 1.12]	.13 [.04, .22]
Risetime		
Mean intensity (μ_{rise})	20.65 [20.52, 20.79]	.03 [−.08, .12]
Variability (log(π_{rise}))	.86 [.71, 1.01]	.09 [−.00, .18]
Sleep Quality		
Mean intensity ($\mu_{quality}$)	2.12 [2.00, 2.24]	−.06 [−.21, .08]
Variability (log($\pi_{quality}$))	−.98 [−1.11, −.86]	.13 [.03, .23]

For the purposes of analyses, bed and rise time are transformed into reverse military time and coded as HH.MM/60. For interpretation, add 12 to bedtime and subtract 12 from risetime (13.27 = 1:16AM; 20.65 = 8:39AM). In Bayesian estimation, 95% Credibility Intervals that do not contain 0 are viewed as significant (bolded).

Night-to-night variability in TST was unassociated with mean levels or day-to-day variability in PA. Next, we examined models where HPS was entered as a between subjects predictor of the eight random effects specified in each model (see Table 2). HPS scores were unassociated with average levels of TST in all models; however, the higher one's HPS score was, the greater night-tonight variability in sleep they had (log(π_{TST})).

Table 2. Conditional DSEM models with HPS as a predictor of random effects.

		Between Subjects Predictor of random effects (HPS)
Variable	Estimated Mean [95% CI]	Estimate [95% CI]
Negative Affect		
NA mean intensity (μ_{NA})	2.32 [2.21, 2.43]	.13 [.03, .23]
TST mean intensity (μ_{TST})	7.39 [7.27, 7.50]	−.12 [−.23, −.00]
NA Autoregression (ϕ_{NN})	.38 [.33, .43]	.03 [−.15, .23]
TST Autoregression (ϕ_{TT})	−.03 [−.07, .02]	.13 [−.25, .54]
$TST_{t-1} \rightarrow NA_t$ cross-lag (ϕ_{TN})	−.02 [−.03, −.01]	.04 [−.15, .27]
$NA_{t-1} \rightarrow TST_t$ cross-lag (ϕ_{NT})	−.05 [−.17, .06]	.22 [−.20, .85]
TST variability (log(π_{TST}))	.94 [.84, 1.04]	.16 [.04, .26]
NA variability (log(π_{NA}))	−1.50 [−1.62, −1.38]	.17 [.06, .26]
Positive Affect		
PA mean intensity (μ_{PA})	3.16 [3.03, 3.28]	.22 [.13, 32]
PA Autoregression (ϕ_{PP})	.38 [.33, 43]	−.07 [−.30, .12]
$TST_{t-1} \rightarrow PA_t$ cross-lag (ϕ_{TP})	−.00 [−.02, .01]	.02 [−.22, .27]
$PA_{t-1} \rightarrow TST_t$ cross-lag (ϕ_{PT})	−.01 [−.10, .07]	−.06 [−.46, .32]
PA variability (log(π_{PA}))	−.95 [−1.07, −.84]	.03 [−.08, .12]
Stress		
Stress mean intensity (μ_S)	2.75 [2.63, 2.88]	.10 [.01, .20]
Stress Autoregression (ϕ_{SS})	.29 [.25, .34]	.00 [−.24, .25]
TSTt-1 → $Stress_t$ cross-lag (ϕ_{TS})	−.03 [−.05, −.01]	−.07 [−.38, .28]
Stresst-1 → TST_t cross-lag (ϕ_{ST})	−.10 [−.16, −.04]	.19 [−.25, .65]
Stress variability (log(π_S))	−.21 [−.31, −.10]	.06 [−.05, .16]
Concentration		
Concentration mean intensity (μ_C)	2.71 [2.56, 2.87]	.17 [.07, .26]
Concentration Autoregression (ϕ_{CC})	.31 [.26, .36]	.10 [−.07, .30]
$TST_{t-1} \rightarrow Concentration_t$ cross-lag (ϕ_{TC})	−.03 [−.04, −.01]	.06 [−.18, .29]
$NA_{t-1} \rightarrow Concentration_t$ cross-lag (ϕ_{CT})	−.05 [−.11, .02]	.22 [−.37, .82]
Concentration variability (log(π_C))	−.56 [−.73, −.39]	.14 [.04, .23]
Racing Thoughts		
Racing thoughts mean intensity (μ_{RT})	2.27 [2.13, 2.42]	.25 [.15, .34]
Racing thoughts Autoregression (ϕ_{RR})	.31 [.25, .36]	.03 [−.12, .20]
$TST_{t-1} \rightarrow Racing\ thoughts_t$ cross-lag (ϕ_{TR})	−.01 [−.02, .00]	.02 [−.21, .27]
Racing thoughtst-1 → TST_t cross-lag (ϕ_{RT})	−.04 [−.11, .03]	.45 [−.03, .91]
Racing thoughts variability (log(π_{RT}))	−.94 [−1.14, −.73]	.25 [.16, .33]
Impulsivity		
Impulsivity mean intensity (μ_{IMP})	1.81 [1.70, 1.91]	.18 [.09, .27]
Impulsivity Autoregression (ϕ_{II})	.30 [.25, .35]	.04 [−.12, .22]
$TST_{t-1} \rightarrow Impulsivity_t$ cross-lag (ϕ_{TI})	−.00 [−.01, .01]	−.05 [−.21, .17]
$Impulsivity_{t-1} \rightarrow TST_t$ cross-lag (ϕ_{IT})	.00 [−.10, .09]	.58 [.21, .98]
Impulsivity variability (log(π_{IMP}))	−1.86 [−2.10, −1.62]	.16 [.07, .25]

Due to redundancy amongst models we do not present TST mean intensity (μ_{TST}), TST Autoregression (ϕ_{TT}), and TST variability (log(π_{TST})) after the first model. In Bayesian estimation, 95% Credibility Intervals that do not contain 0 are viewed as significant (bolded). Estimates for conditional models (with HPS) represent standardized posterior estimates.

Lastly, we examined autoregressive and cross-lagged relationships between TST and affect, cognition, and behavior (see Table 2; Figure 2). Significant autoregressive relationships emerged for all BSP items in daily life (ϕ_{NN}, ϕ_{PP}; ϕ_{SS}, ϕ_{CC}, ϕ_{RR}, ϕ_{II}) such that the more an individual deviated from their own baseline on one day, they more likely they were to continue to deviate

away from their personal baseline the next day – this was true regardless of HPS score. In terms of cross-lagged effects, if a person had less TST than their average, they were more likely to experience heightened NA (ϕ_{TN}), difficulty concentrating (ϕ_{TC}), and racing thoughts (ϕ_{TR}) the next day, but not vice versa. Interestingly, a different pattern emerged for stress – less TST predicted greater stress , and, the greater stress one experienced, the less TST they had that night. TST had no cross-lagged effect with PA or impulsivity. HPS scores did not predict cross-lagged effects with the exception of one model. The within-person link between impulsivity and TST (ϕ_{IT}) was stronger for those at greater risk for bipolar spectrum disorders.

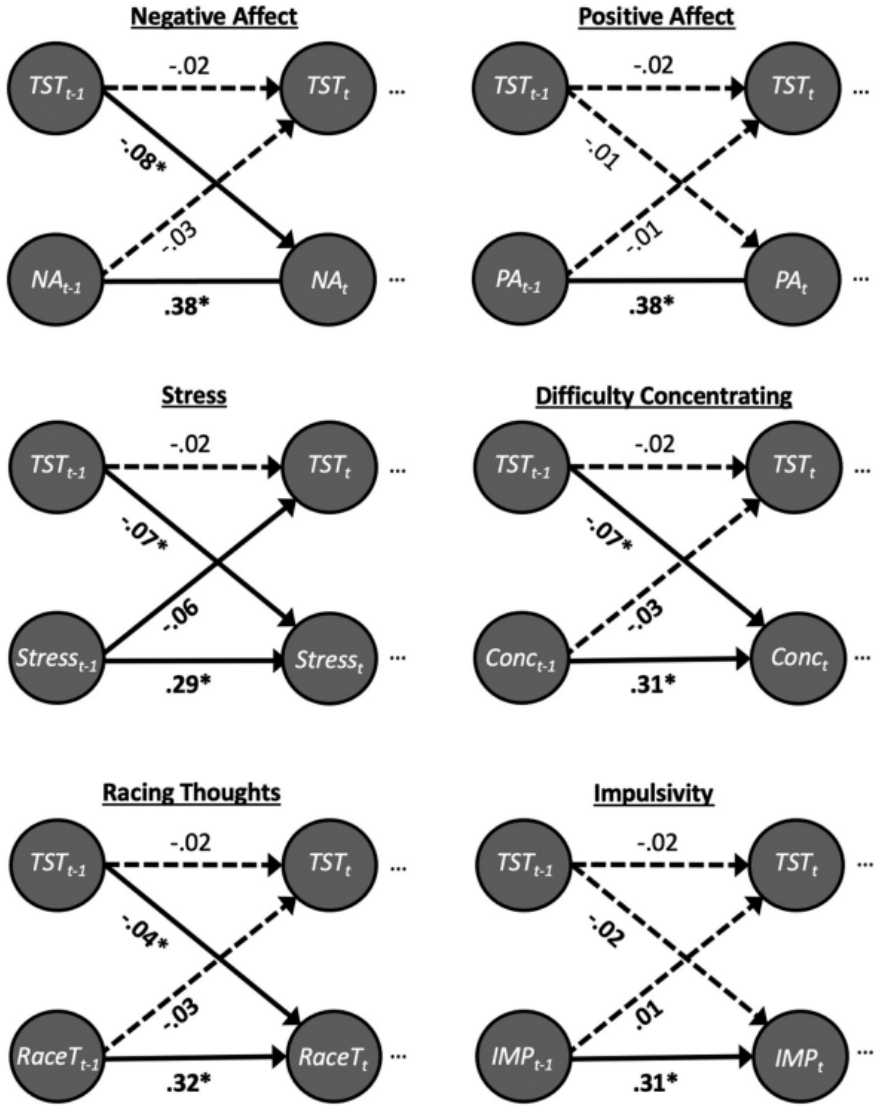

Figure 2. Within-level standardized posterior estimates for autoregressive and cross-lag random effects. Significant pathways (bolded solid lines with *) represent estimates that have a 95% Credibility Interval that does not contain 0. Paths that included 0 in the Credibility Interval are indicated by a dashed line. t-1 represents the lagged variable.

Discussion

Consistent with prior studies (e.g., Hensch et al., 2019; Meyer & Maier, 2006), HPS scores were associated with less TST and more variable TST, bedtime, and sleep quality. We did not find that HPS scores were associated with risetime. This was the first study to our knowledge to extend these findings to highlight within-person associations between TST and affective, cognitive, and behavioral characteristics of bipolar spectrum psychopathology using ESM. Two novel findings emerged: 1) intraindividual variability in TST was robustly associated with bipolar spectrum psychopathology in daily life, and 2) within-person changes in TST drove within-person changes in affect and cognition.

Sleep and affect

Prior work suggests that more variability in sleep (a proxy for less entrained 24 hr sleep-wake or circadian rhythm) is associated with greater affective dysregulation (e.g., Murray & Harvey, [24]) but does not seem to have as much of an effect on positive mood (Eidelman et al., [11]). Our findings replicated this pattern in a non-clinical sample oversampled for risk for BSDs and extended them in several important ways. On average, TST was unassociated with average levels of NA; however, when exploring the dynamic association between TST and NA critical patterns emerged. First, individuals with greater intraindividual variability in TST reported greater intensity of NA as well as greater mood lability (as evidenced by the innovation variances of the within-person residual for NA). Importantly, this relationship appeared to be largely driven by within-person changes in TST. When an individual got less sleep than was typical for them, they reported higher levels of NA the following day. The opposite was not true – when an individual experienced greater NA compared to their norm, they did not seem to have significantly reduced sleep that night. A slightly different pattern emerged for stress. When an individual got less sleep than was typical for them, they reported higher stress the following day. In addition, when individuals reported greater stress than was typical for them, they got less sleep that night. This indicates that while sleep seems to have a primary effect on NA, there is a vicious cycle between stress and sleep such that they both exacerbate one another.

Sleep and cognition

Theory suggests that the relationship between sleep and cognition may be bidirectional. Specifically, partial sleep deprivation and TST $\leq$ 6 hrs per night can result in adverse consequences for cognitive functioning, such as cognitive slowing, decreased processing speed, and a decline in working memory (see Boland & Alloy, [5] for review). In contrast, excessive cognitive arousal may be one facet of BSDs that precipitates sleep disturbances (Harvey et al., [15]). Specifically, patients with BD are much more likely to report "my mind keeps turning things over" and "I am unable to empty my mind" compared with good sleepers. Our findings suggested that, in non-clinical samples displaying a wide range of bipolar spectrum psychopathology, changes in sleep exerted primary effects on cognition but not vice versa. Specifically, when an individual slept less than was normal for them, they reported more difficulty concentrating and racing thoughts the next day. The opposite was not true – individuals who reported more racing thoughts than normal did not report significant changes in TST that night. Consistent with negative mood, greater intraindividual variability in sleep was associated with greater intensity of difficulty with

concentration and racing thoughts over the 14-day sampling period. It is important to note though that the relationship between sleep and cognition may look different in a clinical sample who is actively displaying symptoms of mania when individuals are at significantly higher rates of cognitive arousal. Thus, future studies should aim to assess the bidirectionality of this relationship in differing clinical states in patients with BSDs.

Sleep and impulsivity

Previous findings suggested that sleep deprivation may lead to more impulsive or risky decision making whereas improvements in TST may lead to reductions in impulsive thoughts (see Harvey et al., [16] for review). Although greater intraindividual variability in TST was associated with greater momentary reports of impulsive behaviors, we did not find evidence of cross-lagged relationships between TST and impulsivity on the whole. However, HPS scores were a significant predictor of the cross-lag relationship between impulsivity and TST but in the opposite direction than hypothesized – for individuals with higher severity of bipolar spectrum psychopathology, more daily impulsivity led to less sleep that night. This was surprising given that primary research on the topic has highlight the influence of sleep duration on impulsive or risky decision making, not the other way around.

Implications & future directions

Taken together, the present study reveals that circadian disruptions, in the form of more variable sleep duration, may leave individuals particularly vulnerable to negative affect, affective lability, difficulty concentrating, racing thoughts, and impulsivity, even in a non-clinical high-risk sample. Likewise, these findings provide initial evidence that changes in sleep seem to drive changes in the affective and cognitive symptoms of BSDs. Consistent with the social zeitgeber theory of mood disorders, it is clear that keeping a regular sleep routine may be especially important for those at risk for the development of BSDs and for reducing affective dysregulation, cognitive difficulties, and impulsivity in those displaying subthreshold symptoms.

Although these findings improve our understanding of the day-to-day influence of sleep in a high-risk sample, they do not speak to why individuals on the bipolar spectrum have greater interindividual variability in sleep to begin with. Several neurobiological indicators of disrupted circadian rhythms show promise as future candidates of study. Regardless of mood state, patients with BSDs have shown chronic suppression of melatonin in response to light (Lewy et al., [22]; Nurnberger et al., [27]). This hypersensitivity to light was reported in non-clinical samples with elevated scores on the General Behavior Inventory, a risk measure akin to the HPS (Bullock et al., [6]). Patients with BSDs also showed reduced cortisol reactivity to negative daily events, a higher amplitude of cortisol fluctuations, and flatter diurnal cortisol slopes (Havermans et al., [17]). This is especially relevant to our findings regarding the bidirectional relationship between sleep and stress – stress exerted its influence through the hypothalamic-pituitary axis (HPA) which has been directly linked to circadian systems (Nader et al., [25]). Given clear pathophysiological links between BSDs and sleep, future work should examine whether these biomarkers are present in those with high scores on risk measures such as the HPS or GBI and whether interventions targeted to regulate melatonin or cortisol result in decreased affective dysregulation, cognitive deficits, and behavioral impulsivity.

Limitations

The present study had several limitations. First, the sleep diary administered only assessed bedtime, risetime, and sleep quality. Thus, we were unable to estimate sleep onset latency (how long it takes to fall asleep), wake after sleep onset (an indication of fragmented sleep), or sleep efficiency (TST/time in bed). Future studies should examine whether other indicators of sleep disturbance (SOL, WASO, SE) are associated with bipolar spectrum psychopathology in daily life. Second, the present study did not assess interpersonal or global functioning. This is an important area of future study as sleep disturbances are associated with poorer social functioning (e.g., Kyle et al., [21]) and a systematic review highlighted the importance of understanding the interrelationships between sleep, emotion, and interpersonal factors (Beattie et al., [4]). Lastly, several studies have noted that there is some discordance between objectively assessed sleep and self-reported sleep (e.g., Kaplan et al., [19]). Future studies should simultaneously assess both self-reported and objectively measured sleep to assess whether sleep parameters established via wrist-worn actigraphy show similar dynamic associations with bipolar spectrum psychopathology.

Nevertheless, the present study used ESM to measure sleep, affect, cognition, and behavior related to bipolar spectrum psychopathology in a large sample oversampled for high HPS scorers. In general, trait bipolar spectrum psychopathology was associated with less regular sleep which in turn was associated with symptoms of BSD in daily life.

Data and model scripts and outputs will be made publicly available via Open Science Framework and can be accessed via the following URL:
https://osf.io/tgnc2/?view_only=a291870b8c1c42cf9d4d86bd2e699b0f

Disclosure statement

No potential conflict of interest was reported by the authors.

Supplementary material

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Notes

[1] Note that this measure of TST does not account for wake after sleep onset (WASO) as participants were not asked about their nighttime awakenings. However, it is calculated based on the time the participant fell asleep and woke up, not the times they got into bed or got out of bed.

[2] Sleep surveys were automatically set to be available each morning. One participant completed an extra sleep survey the day after the ESM study was completed.