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Publication Date

2022-06-01

DOI

10.1016/j.jpsychires.2022.03.055

Peer reviewed

HHS Public Access

Author manuscript

J Psychiatr Res. Author manuscript; available in PMC 2022 December 01.

Published in final edited form as:

J Psychiatr Res. 2022 June ; 150: 257–263. doi:10.1016/j.jpsychires.2022.03.055.

Temporal relationships of ecological momentary mood and actigraphy-based sleep measures in bipolar disorder

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Abstract

Sleep disturbances are a key feature of bipolar disorder (BD), and poor sleep has been linked to mood symptoms. Recent use of ecological momentary assessment (EMA) has allowed for nuanced exploration of the sleep-mood link; though, the scale and directionality of this relationship is still unclear. Using EMA, actigraphy, and self-reported sleep measures, this study examines the concurrent and predictive relationships between sleep and mood. Participants with BD (n = 56) wore actigraphy devices for up to 14 days and completed validated scales and daily EMA surveys about mood and sleep quality. Linear mixed models were used to examine overall and time-lagged relationships between sleep and mood variables. EMA mood ratings were correlated with validated rating scales for depression, mania, anxiety, and impulsivity. Poor self-reported sleep quality was associated with worse overall ratings of sadness and anger. Worse self-reported sleep

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Declaration of competing interest

The authors have no relevant conflicts of interests to report. Dr. Malhotra reports income related to medical education from Corvus, Equillium and Livanova. ResMed provided a philanthropic donation to UC San Diego.

Appendix A.: Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2022.03.055.

quality was associated with greater sadness the following day. Higher daytime impulsivity was associated with worse sleep quality the following night. Exploratory analyses found relationships between worse and more variable mood (sadness, anger, and impulsivity) with worse and more variable sleep that evening (efficiency, WASO, and sleep onset time). The sample size was modest, fairly homogenous, and included mainly euthymic persons with BD. EMA-based assessments of mood and sleep are correlated with validated scale scores and provide novel insight into intra-individual variability. Further work on the complex two-way interactions between sleep and mood is needed to better understand how to improve outcomes in BD.

Keywords

Bipolar disorder; Sleep; Variability; Ecological momentary assessment

1. Introduction

As the sixth leading cause of disability among young and middle-aged adults, bipolar disorder (BD) is associated with depressive and manic episodes that lead to hospitalizations, risky and suicidal behaviors, and impaired functioning (Cloutier et al., 2018). While mood-stabilizing pharmacotherapies are a key linchpin in the treatment of BD, these treatments are associated with major side effects and often have poor adherence (Bai et al., 2019; MacDonald et al., 2016; Velligan et al., 2017). Novel psychosocial approaches to improving mental health outcomes of BD are needed, especially in early identification of mood instability. Recent studies have shown that daily mood ratings through ecological momentary assessment (EMA) can provide longitudinal mood profiles that are linked to clinical outcomes (Faurholt-Jepsen et al., 2019; Faurholt-Jepsen et al., 2015; O'Donnell et al., 2018). Linking daily mood changes to other behavioral assessments, such as sleep and activity, may facilitate prediction of worsening mood symptoms and enable early-stage interventions, perhaps including those focused on regularizing sleep and circadian rhythms.

Sleep disturbances and worse sleep quality are key features of BD. Adults with BD report lower sleep efficiency and poorer overall sleep quality (i.e., higher total scores on the Pittsburgh Sleep Quality Index (PSQI) than controls (Cretu et al., 2016; Geoffrey et al., 2014; Ng et al., 2015). Actigraphy-based studies show that individuals with BD have longer total sleep time (TST) and wake after sleep onset (WASO or total duration of overnight awakenings) than controls (Geoffroy et al., 2015; Ng et al., 2015; Robillard et al., 2015).

Prior studies have supported a relationship between sleep and mood symptoms in BD. Higher PSQI scores have been associated with worse residual depression and earlier mood episode relapse in BD (Cretu et al., 2016) as well as more hypomanic symptoms among people vulnerable to BD (Hensch et al., 2019). Various objective sleep studies have reported relationships between mean sleep measures and mood symptoms, though consensus about the most influential aspects of sleep are lacking. One study found that longer TST was positively correlated with depressive symptom severity and negatively correlated with manic symptom severity (Mukherjee et al., 2018), and another reported that lower sleep efficiency predicted worse longitudinal manic symptoms (Robillard et al., 2016).

Furthermore, the intra-individual variability (IIV) of subjective and objective sleep features may be more sensitive measures of optimal sleep, compared to mean sleep measures. We and other researchers have previously reported that while persons with BD have similar mean TST and WASO, they have higher variability in TST, WASO, and sleep onset latency compared to non-psychiatric comparison cohorts when measured both by actigraphy-based and self-report (Lee et al., 2021; Ng et al., 2015). Hensch et al. (2019) reported that hypomanic symptoms were more related to night-by-night variability of sleep duration rather than mean measures from actigraphy and PSQI. Recent EMA research has allowed for more nuanced assessment of this phenomenon. One EMA study of self-reported sleep duration in adults with BD found that participants with more variable sleep duration had higher mean daily ratings of anger, anxiety, stress, and impulsivity as well as greater variability in daily ratings of energy, sadness, and impulsivity (Kaufmann et al., 2016). On a nightly basis, Kaufmann et al. (2016) found that sleep duration of less than 7–8 h was associated with higher same-day negative affect and lower positive affect. Additional EMA studies have found that mood may be more predictive of sleep than vice versa (Li et al., 2019; Merikangas et al., 2019). Passive tracking of sleep may have key clinical applications in monitoring early mood changes that reflect onset of manic or depressive episodes and provide just-in-time clinical interventions to prevent hospitalization, emergent care, or negative outcomes.

Moreover, recent studies have found that people with BD often inaccurately report their sleep (Kaufmann et al., 2019; Krishnamurthy et al., 2018). Krishnamurthy et al. (2018) found that greater inaccuracy between subjective and objectively assessed TST was associated with more severe depression in persons with BD. More refined exploration of the different relationships between mood and objective versus subjective sleep may provide useful insight for future behavioral interventions targeting sleep.

Using a combination of subjective and objective measures, our study aimed to examine the relationships between sleep variability and mood variability, to identify which mood states are most strongly associated with subjective and objective sleep measures, and to understand the intricacies of how sleep disturbances affect the next day's mood (and vice versa). We focused on how subjective sleep quality and sleep duration is related to mood and mood changes. We hypothesized that poorer and more variable self-reported sleep quality and sleep duration would be concurrently associated with worse and more variable mood ratings among adults with BD. We also hypothesized that poorer quality and more variable sleep (TST) would predict worse and more variable mood the following day and vice versa. Additionally, we explored the relationships of other subjective sleep measures (efficiency, WASO, sleep onset time) to overall mood and day-to-day mood changes.

2. Methods

2.1. Subjects

As part of a longitudinal study of aging in BD, English-speaking participants were recruited from the greater San Diego area. Data collection was performed in batches from November 2016 to October 2019. All participants had a BD diagnosis, as determined by Structured Clinical Interview for the DSM-IV-TR (SCID) (First et al., 2002). Participants were

excluded if they had: 1) another current DSM-IV-TR Axis I diagnoses; 2) acute illness or pregnancy; 3) recent vaccination; 4) history of neurodegenerative or a major neurological illness; 5) history of cancer treatment; and 6) uncontrolled medical illness affecting a subject's ability to complete study procedures. DSM-IV-TR criteria were used due to harmonization of the study protocol with other accelerated aging studies in schizophrenia and post-traumatic stress disorder populations which started prior to 2013, the publication date of the DSM-5 criteria. The protocol was approved by the University of California, San Diego (UCSD) Human Research Protections Program, and prior to participation, all participants gave informed written consent.

The participants completed annual assessments that included wearing a wrist-worn actigraphy device for a fourteen-day period, completing three in-clinic assessments, and responding to short daily surveys on a mobile device. Some participants had data from multiple (2–4) years.

2.2. Mood assessments

The Young Mania Rating Scale (YMRS) is a self-report assessment of manic symptoms over the last 48 h (Young et al., 1978). Of the 11-items, seven items are rated from 0 to 4. The remaining four items (irritability, speech, thought content, and disruptive/aggressive behavior) are rated on Likert scale from 0 to 8 to compensate for the poor cooperation of some patients. Higher scores indicate more manic symptoms.

The Hamilton Depression Rating Scale (HAM-D) is a 17-item clinician-administered assessment of depression over the past week (Hamilton, 1967). Nine of those items are scored from 0 (absent) to 4 (incapacitating). The remaining eight items are scored from 0 (absent) to 2 (present) because they may be more difficult for the patient to classify. Higher scores overall indicate higher levels of depression.

The Brief Psychiatric Rating Scale (BPRS) is an 18-item clinicianrated assessment of current psychiatric symptoms (Ventura et al., 1993). Each item is scored on a Likert scale ranging from 0 (not assessed) to 7 (extremely severe). Higher scores indicate worse psychiatric symptoms.

The 9-item Patient Health Questionnaire (PHQ-9) measures self-reported depression over the past 2 weeks (Kroenke et al., 2001). Each item is rated on a Likert scale of 0 (not at all) to 3 (nearly every day) with higher scores indicating higher levels of depression.

The Brief Symptom Inventory—Anxiety Subscale (BSIA) is a 6-item subscale of the original 53 item assessment (Derogatis and Melisaratos, 1983). Each item is rated on a Likert scale of 0 (not at all) to 4 (extremely). Higher scores suggest higher levels of anxiety.

The Barratt Impulsiveness Scale (BIS-11) is 30-item self-reported questionnaire assessing the personality and behavioral construct of impulsivity (Patton et al., 1995). Each item is rated on a Likert scale of 1 (rarely/never) to 4 (almost always/always). Higher scores indicate higher levels of impulsivity.

The HAM-D, YMRS, and PHQ-9 scales contain questions about sleep disturbance and insomnia.

2.3. EMA mood survey

During the two-week study burst, participants received ecological momentary assessments (EMA surveys) about mood via their mobile device three times each day. The assessments were sent sporadically within three intervals specified by the participant: one in the morning, one in the afternoon, and one in the evening. Each survey asked the participant to rate the intensity of different moods (sadness, anger, anxiety, and impulsivity) on a Likert scale of 1 (not at all) to 7 (very much).

2.4. Sleep assessments

Subjective sleep was measured using the Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989). The Total PSQI score is based on the sum of the seven components: subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbance, use of sleep medication, and daytime dysfunction. Possible scores range from 0 to 21 with higher scores indicating worse sleep quality. Subjective sleep quality was also measured daily as part of the EMA survey ("How would you rate the quality of your sleep last night?"). Participants rated their sleep quality on a 4-point Likert scale (−2 = very bad, −1 = fairly bad, 1 = fairly good, 2 = very good).

Objective sleep was measured using the wrist-worn actigraph device, the Actisleep-BT (Actigraph, Pensacola, FL), in combination with a sleep diary in which participants recorded the previous night's "go to bed" and "get up" times. For more accurate measurement, Cole-Kripke algorithms were used to determine total sleep time (TST), sleep efficiency, wake after sleep onset (WASO), and sleep onset time (Ancoli-Israel et al., 2003; Cole et al., 1992). Sleep efficiency is the percent of total time in bed that is spent sleeping. Sleep efficiency was calculated using the ratio of total sleep time to time in bed. WASO is the total amount of time awake after the initial bout of sleep. Sleep onset time is the time at which sleep began.

2.5. Variability

For the general analysis of sleep and mood over the two-week burst, we used root mean squared successive difference (RMSSD) as an assessment of intra-individual variability due to its ability to detect night-to-night changes (Kanady et al., 2017; Straus et al., 2015). RMSSD was calculated for both sleep (TST, sleep efficiency, WASO, sleep onset time, and EMA Sleep Quality) and mood (sadness, anger, anxiety, and impulsivity.

For the time-lagged analyses, we used atypicality, a measure of how different an individual's sleep or mean mood rating on a given day is from that individual's average sleep or mood over the entire burst period. This metric was calculated by squaring the difference between the individual's mean value and their specific day's value (Kaufmann et al., 2016). Atypicality was calculated for each of the sleep and mood measures. Higher atypicality values represent a more abnormal sleep or mood value for that day for that person.

2.6. Statistical analysis

For this study sample, we included individuals with both actigraphy and EMA data for 2–11 days to maximize the sample and allow for comparable variability calculations. Variables were assessed for violation of distribution assumptions. Mann-Whitney *U* test, independent sample t-tests and chi-square tests were used to assess differences in sociodemographic, psychopathology, physical health, mood, and sleep variables between the BD groups with one vs. with two or more assessments.

Linear mixed models (LMMs) were constructed to examine the relationship between mean sleep variables and mean mood variables. To study the specific utility of examining variability in both sleep and mood, we used time-lagged analyses (LMMs) to study how poor sleep or changes in sleep are associated with mood the following day and vice versa. In models examining how sleep influenced the following day's mood, the dependent variables were mood states and independent variables were sleep measurements from the previous night. Additional models examined the relationship between mood and the subsequent night's sleep, with the dependent variables as sleep measures and independent variables as mean and atypicality of mood ratings.

Significance level (α) was set at .05 (two-tailed) for all analyses. False Discovery Rate (FDR) was used to account for multiple comparisons in the hypothesis-driven regression models (sleep quality and TST) to ensure overall Type 1 error at α 0.05. Exploratory analyses on additional sleep measures (efficiency, WASO, and sleep onset time) were not FDR-corrected. Because effect sizes cannot be obtained from LMM's, mood and sleep variables were standardized before analyses to make them more comparable to one another and produce more meaningful B-estimates. Analyses were conducted in SPSS Statistics for Macintosh, Version 26 and 27 (IBM Corp., Armonk, N.Y., USA).

2.7. Covariates

All LMMs controlled for age, gender, education, and race. Random mixed effects controlled for participant and year. The role, if any, of sleep medications as a proxy of hyperarousal or in confounding the sleep-mood relationships is not clear. Results were similar when conducting the analyses with and without adjusting for sleep medications, so we presented the analyses without sleep medications for simplicity. LMMs analyzing mean mood and sleep included the RMSSD of the sleep measure as a covariate. Time-lagged models included both the mean sleep measure and atypicality measure to examine the individual impact of both factors. Prior day's mood was also a covariate in the time-lagged models to control for its influence on mood the following day. For models assessing relationships between mood atypicality and the following night's sleep, average same-day mood was included as a covariate to ensure the association of atypicality was independent from mood overall.

3. Results

The study sample included 56 individuals with BD, 22 with a single assessment and 34 with two or more annual assessments (Table 1). On average, we analyzed 68 surveys

per participant, making up 78% of overall deployed surveys. Overall, the mean age of participants at their first visit was about 47 years old. Most participants were female and Caucasian. Participants had 15 years of education and most reported use of sleep medication. The two groups (one vs. two or more assessments) differed by years of education, such that participants with two or more assessments were significantly more educated.

Mean EMA ratings of anxiety were significantly correlated (r_s = 0.29–0.56) with the scores on all validated mood rating scales (BPRS, YMRS, HAM-D, PHQ-9, BSIA, and BIS). EMA ratings of sadness were significantly correlated with all mood scales except the BIS (r_s = 0.26–0.48). Mean EMA ratings for anger were correlated with all mood scales except the PHQ-9 and BIS (r_s = 0.31–0.49). Mean EMA ratings for impulsivity were correlated with YMRS, BPRS, and BSIA scores (r_s = 0.23–0.34), but not with the BIS.

3.1. Linear mixed models of concurrent mood and sleep measures

As summarized in Fig. 1, linear mixed models show that mean mood ratings are related to subjective sleep quality measures. After FDR correction, two relationships remained significant. Worse self-reported sleep quality (via EMA) was associated with greater overall sadness (B = −0.48, SE = 0.10, FDR-corrected p = <0.001) and anger (B = −0.38, SE = 0.11, FDR-corrected p = 0.01).

Exploratory analyses found that more variable sleep onset time was associated with worse mean mood ratings (anger, anxiety, impulsivity) and more variable ratings of anger. Greater WASO was associated with more variable ratings of anxiety, and more variable WASO was associated with greater mean impulsivity.

3.2. Time-lagged analysis of sleep and mood

One relationship between sleep and the following day's mood remained significant after FDR correction. Poorer EMA sleep quality was associated with greater sadness the next day (independent of average sadness the day prior) (B = −0.14, SE = 0.03, FDR-corrected p = <0.001). Models analyzing the relationship of mood to sleep the next night found higher daytime impulsivity predicted worse EMA sleep quality that evening (B = −0.14, SE = 0.04, FDR-corrected p = 0.03). No other relationships between sleep and mood or mood atypicality the next day or vice versa remained significant.

Exploratory time-lagged models on additional objective sleep measures (efficiency, WASO, sleep onset) found several relationships between sleep and mood, independent of mean mood ratings the day prior (Supplemental Figs, 1a and 1b). These included relationships between atypicality of sleep efficiency and atypicality of anxiety, later sleep onset time with worse sadness the following day. Atypicality of mood was also associated with sleep the following day. Greater atypicality of sadness and anger predicted later sleep onset time and greater atypicality of sleep onset time that evening. Greater mean daytime impulsivity was associated with more atypical sleep efficiency that night. Greater atypicality of impulsivity predicted greater WASO the subsequent night

4. Discussion

These findings support the hypotheses of strong associations between mood and subjective sleep quality among adults with bipolar disorder. Mean ratings of sadness and anger were associated with mean self-reported sleep quality, but not with objective sleep measures. Time-lagged analyses found that worse EMA sleep quality was associated with greater sadness the next day. Further, greater daytime impulsivity predicted worse sleep quality that night. Exploratory analyses (not FDR-adjusted) on objective sleep measures revealed several associations of mood and mood atypicality with the previous and following night's sleep.

The current study participants were similar by gender and race to previous studies, though the mean age was older (Kanady et al., 2017; Kaufmann et al., 2016; Ng et al., 2015; Robillard et al., 2015). Our study sample had worse mean subjective sleep quality assessed by PSQI than three studies (Gershon et al., 2012; Harvey et al., 2005; Kanady et al., 2017), and similar objective sleep measures to four studies (Kanady et al., 2017; Kaufmann et al., 2016; Ng et al., 2015; Robillard et al., 2015).

The present study supports the utility of EMA ratings to assess unique mood states (i.e., anger and impulsivity) in BD, separate from commonly used clinical rating scales for psychopathology, depression, and mania. Mean EMA ratings were significantly correlated with most clinical mood scales, supporting the validity of EMA surveys as a measure of mood and mood symptoms. Moreover, our results suggest that EMA may provide additional insight into changing mood states that are not always captured via traditional clinical measurement. For example, EMA impulsivity was not associated with BIS scores, suggesting that moment-to-moment ratings of impulsivity may be distinct from trait-like impulsivity. EMA-based mood ratings capture a diversity of mood states that provide valid, unique, and non-overlapping information about the BD participants.

The current study found that mean mood ratings were associated with poor subjective sleep quality, but not worse objectively measured sleep averaged over the burst period. This is consistent with two previous studies that reported links between poor sleep quality, hypomania, mood volatility, and depression in high-risk for BD and BD samples (Cretu et al., 2016; Hensch et al., 2019). However, the current study's findings differed from studies that reported links between sleep duration and mood ratings (Gruber et al., 2011; Kaufmann et al., 2016; Mukherjee et al., 2018), which could be attributable to our use of actigraphy-assessed TST or the longer 14-day monitoring period.

Our findings suggest that mood and mood changes may be more closely related to subjective sleep experiences rather than objective measurements. It is possible that inaccuracies in self-reported sleep may have contributed to these findings. Krishnamurthy et al. (2018) found that individuals with BD report their sleep more inaccurately than their healthy counterparts. Further, the authors found depressive symptoms in people with BD to be associated with greater inaccuracy between self-reported and actigraphy-based TST rather than lower TST overall. Future sleep interventions should account for both subjective and objective sleep to personalize and better target health outcomes.

The current study found that higher mean impulsivity was associated with worse EMA sleep quality the subsequent night. Previous studies have demonstrated similar associations between poor/disturbed sleep and increased impulsivity in BD (Gershon et al., 2019; Russo et al., 2015), that may be mediated by brain connectivity (Tashjian et al., 2017) and the ability of certain brain areas (e.g., PFC) to inhibit and modulate emotional input (Anderson and Platten, 2011; McKenna and Eyler, 2012). The ability to inhibit impulsive responses may decrease as sleep quality deteriorates, resulting in a cycle of poorer sleep and gradually more impulsive behavior over time. Our impulsivity-related findings have important clinical implications, given that emotion-triggered impulsivity is associated with negative sequelae in BD, including risky behavior, suicidality, and self-harm (Johnson et al., 2017). Consequently, reducing impulsivity is a critical clinical target, and improving sleep quality may provide an important means of intervention.

Exploratory time-lagged analyses found that worse and more variable mood predicted objective and subjective sleep measures the following night, while sleep did not predict subsequent mood as consistently. These findings were analogous to Li et al. (2019), which examined 14-day self-report sleep measures with positive/negative affect, energy, impulsivity, and speed of thoughts in 20 participants (10 BD and 10 controls). Among the BD group, positive affect, energy, impulsivity, and speed of thoughts predicted lower total sleep time. Alternatively, Merikangas et al. (2019) examined objective sleep measures and EMA ratings of sadness, energy, and activity among 242 participants (including participants with major depressive disorder and BD). Greater activity predicted shorter total sleep time in all participants. Among participants with bipolar disorder type I, increased activity was associated with changes in sad mood and vice versa. Thus, physical activity appears to mediate the links between mood and sleep. The complex interrelationships between mood, sleep, and physical activity warrant further investigation.

Future studies should consider sleep- and activity-focused interventions for BD. For example, Interpersonal and Social Rhythms Therapy (IPSRT) is a form of psychotherapy that aims to stabilize circadian rhythm through stabilizing daily social rhythms (Frank et al., 2000). An important part of IPSRT is to regulate sleep and wake times. BD participants who undergo IPSRT have improved depressive and manic symptomatology as well as clinical psychological functioning in some (Crowe et al., 2020; Frank et al., 2000; Inder et al., 2015; Steardo et al., 2020) but not all studies (Frank et al., 2000). Effective interventions for improving sleep quality (e.g., CBT for Insomnia) may also have a role in improving mood, preventing the cyclical pattern of worsening mood and sleep. Furthermore, measuring sleep disturbance could allow clinicians to track mood among patients with BD, assessing individual-level changes over time.

Our study has a few limitations. Our sample size is modest (n = 56) and has moderately heterogeneous sleep. Future studies should examine these relationships in larger and more diverse samples with longer follow-up and more variable sleep. Our sample of people with BD are community-dwelling outpatients with relatively stable symptoms, so results may not be generalizable to inpatients or those in acute manic or depressive episodes. The current sample is also demographically homogenous, so our findings may not be generalizable to persons with BD across age and cultural groups as they do not account for social

determinants of health (Dietch et al., 2017; Smagula et al., 2016). Additionally, the sample did not include a healthy comparison group, so it is unclear if these findings are unique to bipolar patients specifically. Although they are valuable research tools, actigraphy watches and EMA surveys are not infallible. Actigraphy watches may mis-categorize periods of inactivity as sleep and vice versa. Our study of the EMA survey data was limited to four mood states, which may not fully capture all possible relationships between mood and sleep. The analyses did not account for concurrent mood states (e.g., high anxiety plus high anger). Despite these limitations, this study sheds new light on links between subjective and objective sleep with mood in BD.

Future EMA studies of the sleep-mood relationship in BD should include subjective and objective sleep assessments for a more comprehensive understanding of this connection. Further, additional research is required to confirm which aspects of mood are associated with sleep the previous night and vice versa. A larger sample size and extended actigraphy monitoring period may reveal more robust relationships. The prediction of mood changes through sleep assessment may allow for novel preventative measures in bipolar disorder through sleep management as well as short term adjustments to improve quality of life in adults with BD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Role of funding source

This study was supported, in part, by the National Institutes of Health [NIMH R01MH103318 (PI: Lisa T. Eyler), NIMH K23MH119375-01 (PI: Ellen E. Lee)], NIA K01AG061239 (PI: Christopher N. Kaufmann)], NARSAD Young Investigator grant from the Brain and Behavior Research Foundation (PI: Ellen E. Lee), and by the Stein Institute for Research on Aging at the University of California San Diego. Dr. Malhotra is funded by NIH. The content of this paper is solely the responsibility of the authors and does not necessarily represent the official views of the NIH.

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	Mean Sadness	Mean Anger	Mean Anxiety	Mean Impulsivity	Sadness RMSSD	Anger RMSSD	Anxiety RMSSD	Impulsivity RMSSD
PSQI Total	ß = 0.07 p = 0.005	ß = 0.06 p = 0.02	ß = 0.05 p = 0.05	ß = 0.02 p = 0.55	ß = 0.05 p = 0.03	ß = 0.05 p = 0.04	ß = 0.03 p = 0.26	ß = 0.02 p = 0.40
EMA Sleep Quality	ß = -0.48 p = <0.001*	ß = -0.38 p = <0.001*	ß = -0.29 p = 0.008	ß = -0.25 p = 0.02	ß = -0.26 p = 0.02	ß = -0.17 p = 0.14	ß = 0.01 p = 0.91	ß = -0.11 p = 0.29
Total Sleep Time	ß = 0.08 p = 0.43	ß = -0.09 p = 0.39	ß = -0.01 p = 0.96	ß = -0.11 p = 0.30	ß = 0.05 p = 0.65	ß = -0.07 p = 0.51	ß = -0.07 p = 0.56	ß = -0.38 p = 0.72
EMA Sleep Quality RMSSD	ß = -0.07 p = 0.47	ß = -0.03 p = 0.76	ß = 0.08 p = 0.47	ß = 0.08 p = 0.45	ß = 0.02 p = 0.88	ß = 0.05 p = 0.69	ß = 0.29 p = 0.01	ß = 0.17 p = 0.14
Total Sleep Time RMSSD	ß = 0.05 p = 0.64	ß = 0.09 p = 0.40	ß = <0.001 p = 0.10	ß = 0.20 p = 0.07	ß = -0.04 p = 0.73	ß = 0.17 p = 0.12	ß = 0.06 p = 0.60	ß = 0.13 p = 0.21

Fig. 1. Results from linear mixed models that assess associations between overall EMA mood and sleep variables.

Values in the table reflect ß coefficients and p-values prior to FDR correction. Relationships shaded in gray indicate $p < 0.05$ prior to FDR correction. Asterisks indicate $p < 0.05$ after FDR correction. EMA = Ecological Momentary Assessment, PSQI = Pittsburgh Sleep Quality Index, RMSSD = root mean squared successive difference.

Table 1

Demographic comparison of participants with 1 year vs 2 or more years of data.

	Total Bipolar Participants			Participants with 1 year of Data			Participants with 2 or more years of Data			Statistical Test
	N	**Mean or %**	**SD**	**N**	**Mean or %**	**SD**	**N**	**Mean or %**	**SD**	
Sociodemographic Factors										
Age (years)	56	47.3	9.2	22	46.5	8.9	34	47.9	9.5	$U = 338.0$, $p = 0.55$
Sex (% female)		64			68			62		$\chi^2(1) = 0.24$, p = 0.63
Race (% Caucasian)		63			64			62		$\chi^2(1) = 0.02$, $p = 0.89$
Education (years)	54	14.5	2.1	20	13.7	1.8	34	15.0	2.1	**$U = 221.0$, $p = 0.03$**
Sleep medication use (% never)		26			32			21		$\chi^2(1) = 0.78$, $p = 0.38$
Psychopathology										
Duration of Illness (years)	42	33.0	9.8	16	34.1	8.5	26	32.3	10.7	$U = 190.0$, $p = 0.64$
Antipsychotic dose (mg/d)	48	0.6	0.8	16	0.8	0.9	32	0.6	0.8	$t(46) = -0.60$, p = 0.55
Medication load	48	3.2	2.0	16	2.9	1.9	32	3.3	2.1	$U = 222.0$, $p = 0.40$
Overall Psychopathology (BPRS)	54	38.7	8.5	21	38.2	8.0	33	39.1	8.8	$U = 322.5$, $p = 0.67$
Manic symptoms (YMRS)	54	5.9	4.9	20	5.0	5.4	34	6.4	4.6	$U = 259.5$, $p = 0.15$
Clinician-rated depressive symptoms (HAM-D)	54	14.3	7.7	20	16.1	7.1	34	13.2	8.0	$U = 264.0$, $p = 0.17$
Self-rated depressive symptoms (PHQ-9)	37	9.3	5.7	13	9.7	4.8	24	9.0	6.2	$t(35) = 0.33$, $p = 0.74$
Self-rated anxiety symptoms (BSIA)	40	7.3	5.2	13	7.2	5.5	27	7.3	5.2	$U = 172.5$, $p = 0.93$
Self-rated impulsivity (BIS-11)	42	73.2	12.3	16	77.5	9.7	26	70.5	13.2	$U = 1142.0$, $p = 0.70$
Physical Health										
BMI (Kg/m^2)	53	30.2	6.2	21	30.2	6.4	32	30.2	6.1	$U = 333.0$, $p = 0.96$
EMA Mood Rating										
Mean Sadness	56	2.6	1.1	22	2.4	1.1	34	2.7	1.1	$U = 328.0$, $p = 0.44$
Mean Anger	56	2.0	0.8	22	2.0	0.8	34	1.9	0.8	$U = 346.5$, $p = 0.64$
Mean Anxiety	56	2.7	1.0	22	2.7	1.1	34	2.6	0.9	$U = 351.0$, $p = 0.70$
Mean Impulsivity	55	2.0	0.9	22	2.1	1.0	33	1.9	0.9	$U = 221.0$, $p = 0.47$
EMA Mood Rating Variability										
Sadness RMSSD	52	0.5	0.4	20	0.5	0.3	32	0.5	0.5	$t(50) = -0.16$, p = 0.88
Anger RMSSD	52	0.6	0.5	20	0.6	0.5	32	0.5	0.5	$U = 259.5$, $p = 0.57$
Anxiety RMSSD	52	0.7	0.6	20	0.9	0.6	32	0.7	0.5	$U = 226.0$, $p = 0.22$

	Total Bipolar Participants			Participants with 1 year of Data			Participants with 2 or more years of Data			Statistical Test
	N	Mean or %	SD	N	Mean or %	SD	N	Mean or %	SD	
Impulsivity RMSSD	51	0.6	0.6	19	0.7	0.6	32	0.6	0.6	$U = 246.5$, $p = 0.49$
Sleep Measures										
Subjective Sleep Quality (PSQI Total Score)	53	9.7	4.0	21	10.2	3.6	32	9.4	4.3	$t(49) = 0.29$, p = 0.77
Mean Self-Reported EMA Sleep Quality	56	0.8	0.8	22	0.8	0.8	34	0.8	0.8	$U = 346.0$, $p = 0.64$
Actigraphic Mean Total Sleep Time (min)	56	406.6	79.1	22	381.2	84.7	34	423.1	71.9	$t(54) = -1.99$, p = 0.05
Mean Efficiency (%)	56	85.9	7.1	22	85.6	6.8	34	86.2	7.5	$U = 342.0$, $p = 0.60$
Mean Wake After Sleep Onset (min)	56	60.5	31.5	22	57.7	28.6	34	62.3	33.5	$U = 349.5$, $p = 0.68$
Mean Sleep Onset Time[a]	56	23:38	102.0	22	23:44	108.5	34	23:34	91.5	$U = 354.0$, $p = 0.74$
Sleep Variability Measures										
Self-Reported Daily Sleep Quality RMSSD	55	1.1	0.5	21	1.2	0.5	34	1.0	0.5	$t(52) = 0.92$, p = 0.37
Total Sleep Time (min) RMSSD	56	101.7	47.1	22	99.1	38.2	34	102.9	51.2	$U = 287.0$, $p = 0.61$
Efficiency (%) RMSSD	56	7.8	4.4	22	7.3	4.6	34	8.0	4.3	$U = 249.0$, $p = 0.22$
Wake After Sleep Onset (min) RMSSD	56	38.9	24.0	22	31.8	18.1	34	42.2	25.8	$U = 279.0$, $p = 0.51$
Sleep Onset Time (min) RMSSD	56	90.5	73.2	22	80.5	55.9	34	95.1	80.2	$U = 283.0$, $p = 0.56$

BMI = Body Mass Index, BPRS = Brief Psychiatric Rating Scale, BSIA = Brief Symptom Inventory – Anxiety, HAM-D = Hamilton Depression Rating Scale, RMSSD = root mean squared successive difference, PHQ-9 = Patient Health Questionnaire-9 item, PSQI = Pittsburgh Sleep Quality Index, YMRS: Young Mania Rating Scale, BIS-11: Barratt Impulsiveness Scale.

[a]Mean is written in 24hr clock and RMSSD in minutes.