HHS Public Access

Author manuscript

J Abnorm Psychol. Author manuscript; available in PMC 2015 November 27.

Published in final edited form as:

J Abnorm Psychol. 2015 November ; 124(4): 944–952. doi:10.1037/abn0000107.

Low Social Rhythm Regularity Predicts First Onset of Bipolar Spectrum Disorders Among At Risk Individuals with Reward Hypersensitivity

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Abstract

The social zeitgeber model (Ehlers, Frank, & Kupfer, 1988) suggests that irregular daily schedules or social rhythms provide vulnerability to bipolar spectrum disorders. This study tested whether social rhythm regularity prospectively predicted first lifetime onset of bipolar spectrum disorders in adolescents already at risk for bipolar disorder based on exhibiting reward hypersensitivity. Adolescents (ages 14 – 19) previously screened to have high (N = 138) or moderate (N = 95) reward sensitivity, but no lifetime history of bipolar spectrum disorder, completed measures of depressive and manic symptoms, family history of bipolar disorder, and the Social Rhythm Metric. They were followed prospectively with semi-structured diagnostic interviews every six months for an average of 31.7 (SD = 20.1) months. Hierarchical logistic regression indicated that low social rhythm regularity at baseline predicted greater likelihood of first onset of bipolar spectrum disorder over follow-up among high, but not moderate, reward sensitivity adolescents, controlling for follow-up time, gender, age, family history of bipolar disorder, and initial manic and depressive symptoms (β= −.150, Wald = 4.365, p = .037, OR = .861, 95% CI = .748 – .991). Consistent with the social zeitgeber theory, low social rhythm regularity provides vulnerability to first onset of bipolar spectrum disorder among at-risk adolescents. It may be possible to identify adolescents at risk for developing a bipolar spectrum disorder based on exhibiting both reward hypersensitivity and social rhythm irregularity before onset occurs.

Keywords

social rhythm regularity; social zeitgebers; reward sensitivity; bipolar spectrum disorders

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Disclosures

None of the authors report any conflicts of interest associated with this manuscript. All authors have approved the submission of this article.

Disruption of circadian rhythms has been proposed to be a central mechanism in the neurobiological vulnerability to bipolar spectrum disorders (BSDs) (McClung, 2007; Murray & Harvey, 2010). According to the social zeitgeber theory of mood disorders (Ehlers, Frank, & Kupfer, 1988; Grandin, Alloy, & Abramson, 2006), life events that disrupt social zeitgebers, defined as daily social rhythms or schedules (e.g., mealtimes, bedtimes, start and end of work), are hypothesized to disturb circadian rhythms, which, in turn, precipitate bipolar symptoms. Events that perturb social rhythms (e.g., causing a change in bedtime, skipping a meal) may disrupt circadian rhythms through their effects on either photic (e.g., Roenneberg & Merrow, 2007; Stetler, Dickerson, & Miller, 2004; Wever, 1989) or non-photic (e.g., Goel, 2005) cues that help to entrain circadian rhythms. Changes in daily activity patterns, whether manipulated or naturally occurring, have been found to be associated with changes in social rhythms in healthy individuals (e.g., Stetler et al., 2004).

A growing body of evidence supports the social zeitgeber model of BSDs (see Alloy, Nusslock, & Boland, 2015 for review). For example, studies have found significant associations between life event-induced social rhythm disruption and episode onset in individuals with BSDs. Malkoff-Schwartz and colleagues (1998, 2000) reported that more social rhythm disrupting events occurred in the period prior to manic episode onset than in a matched, equal duration control period not related to episode onset in a sample with bipolar I disorder. In addition, Sylvia et al. (2009) found that the occurrence of social rhythm disrupting events significantly predicted prospective onset of depressive episode recurrences in a sample of individuals with bipolar II disorder or cyclothymia. Moreover, Boland and colleagues (2012) found that individuals with BSDs exhibit an underlying sensitivity to life event-induced social rhythm disruption. Compared with demographically matched healthy controls, individuals with BSDs experienced significantly more social rhythm disruption following the experience of similar intensity and valence life events (Boland et al., 2012).

Given the hypothesized role of social zeitgebers in entraining biological rhythms, individuals with low social rhythm regularity may be at increased risk for desynchronization of circadian rhythms and, in turn, onset of bipolar mood episodes. That is, low social rhythm regularity should serve as a vulnerability to bipolar mood episodes. Several studies have used the Social Rhythm Metric (SRM; Monk, Flaherty, Frank, Hoskinson, & Kupfer, 1990) to compare individuals with BSDs and controls on self-reported regularity of their social rhythms. The SRM assesses the frequency (number of days per week) with which daily activities (e.g., getting out of bed, eating lunch, starting work or school) are performed and the degree of regularity (occurrence of each activity within 45 minutes of the average time) of these activities. Social rhythm regularity scores are calculated as the number of activities that occur at least three days per week within 45 minutes of the average time. Consistent with the social zeitgeber model, studies using different versions of the SRM find that individuals with BSDs exhibit lower social rhythm regularity than do healthy controls (Ashman et al., 1999; Jones, Hare, & Evershed, 2005; Szuba, Yager, Guze, Allen, & Baxter, 1992), even during the euthymic state (Jones et al., 2005; Shen, Alloy, Abramson, & Sylvia, 2008). And, in euthymic individuals with bipolar II or cyclothymia diagnoses, low social rhythm regularity scores on a modified SRM at baseline predict a greater likelihood and shorter time to recurrence of both major depressive and hypomanic or manic episodes over

prospective follow-up, controlling for baseline subsyndromal mood symptoms and family history of bipolar disorder (Shen et al., 2008).

Consistent with the hypothesis that low social rhythm regularity may confer vulnerability to BSDs, individuals at behavioral risk for bipolar disorder based on exhibiting hypomanic personality (Meyer & Maier, 2006) or subsyndromal bipolar symptoms (Bullock, Judd, & Murray, 2011) exhibited lower social rhythm regularity on a brief version of the SRM (SRM-5) than did participants at low behavioral risk for bipolar disorder. Alternatively, in a genetic high-risk study, Jones, Tai, Evershed, Knowles, and Bentall (2006) did not find lower social rhythm regularity on the SRM in the children of bipolar parents compared with the age- and sex-matched children of healthy control parents. It is possible that Jones et al. (2006) did not observe lower social rhythm regularity in the children of bipolar parents because children's daily activity schedules are more regimented by their parents, school, and other external constraints. Thus, there is some, but not fully consistent, evidence for low social rhythm regularity in individuals without a current bipolar disorder, but at behavioral risk for developing a BSD. However, no study to date has examined whether low social rhythm regularity prospectively predicts first lifetime onset of BSD in individuals at behavioral risk for bipolar disorder, which would provide the strongest evidence for irregular social rhythms as a vulnerability factor for BSDs. This is the goal of the present study.

We examined social rhythm regularity as a predictor of first onset of BSD in a group of adolescents previously shown to be at increased risk for BSD based on exhibiting a hypersensitive reward system and a comparison group of adolescents at low risk for BSD based on exhibiting moderate reward sensitivity. According to the reward hypersensitivity theory of bipolar disorder (e.g., Alloy & Abramson, 2010; Alloy et al., 2015; Johnson, Edge, Holmes, & Carver, 2012; Urosevic, Abramson, Harmon-Jones, & Alloy, 2008), vulnerability to BSDs is hypothesized to be the result of an overly sensitive reward system that is hyperreactive to goal- and reward-relevant cues. Much self-report, behavioral, life event, neurophysiological, and neuroimaging evidence supports the reward hypersensitivity theory of BSDs (see Alloy et al., 2015 for a recent review). Consistent with the reward model, Alloy et al. (2012) previously demonstrated that controlling for baseline hypomanic and depressive symptoms and family history of bipolar disorder, adolescents who exhibited high levels of reward sensitivity at baseline, but had no prior history of BSD, were significantly more likely to develop a first onset of a BSD over an average of 13 months of prospective follow-up than were adolescents with moderate reward sensitivity. Although high reward sensitive adolescents were three times more likely to develop a BSD than moderate reward sensitive adolescents (12.3% vs. 4.2%), the majority of the at-risk reward hypersensitive adolescents did not develop a BSD over the follow-up period (Alloy et al., 2012). Thus, it is important to examine additional factors, such as low social rhythm regularity considered to be a vulnerability to BSD from the perspective of the social zeitgeber model, which may further predict which individuals will go on to develop first onset of BSD.

Moreover, the social/circadian rhythm disruption and reward hypersensitivity models of BSD can be integrated involving bidirectional associations between reward sensitivity and

social and circadian rhythm dysregulation (e.g., Alloy et al., 2015; Murray et al., 2009; Nusslock, Abramson, Harmon-Jones, Alloy, & Coan, 2009). Indeed, there is evidence of circadian influences on reward motivation (e.g., Clark, Watson, & Leeka, 1989; Murray et al., 2009; Thayer, Takahashi, & Pauli, 1988), associations between reward-related brain activity and circadian clock genes (Forbes et al., 2012) and sleep variables (Holm et al., 2009), as well as reward hypersensitivity influences on social rhythm disruption (Boland et al., in press). These bidirectional influences of the reward and circadian systems suggest that examining social rhythm regularity as a predictor of first onset of BSD among adolescents with high reward sensitivity may prove fruitful and increase the precision of identification of adolescents at increased risk for first onset of BSD. In turn, early identification of adolescents at greatest risk for onset of BSD is important for early intervention efforts designed to prevent onset or mitigate the severity of BSD.

Consequently, the present study provides the first test of social rhythm regularity as a predictor of first lifetime onset of BSD in a sample of adolescents at increased risk for BSD based on exhibiting reward hypersensitivity and a comparison group of low-risk adolescents with moderate reward sensitivity. Consistent with the social zeitgeber model of BSDs, we hypothesized that among high, but not moderate, reward sensitive adolescents with no prior history of BSD, lower levels of social rhythm regularity would predict a greater likelihood of developing a first onset of BSD over prospective follow-up than would higher social rhythm regularity.

Method

Participants

Participants in the current study were a subset of those participating in the Teen Emotion and Motivation (TEAM) Project, a prospective longitudinal study of predictors of first onset of BSDs (Alloy et al., 2012). All participants < 18 years old provided written assent and their parents provided written consent, whereas participants ≥ 18 years old provided their own written consent. All procedures were approved by the Temple University Institutional Review Board. Adolescents (ages 14 – 19) were selected for Project TEAM based on a two-phase screening procedure. The age range of 14 – 19 was selected because it is a major “age of risk” for first onset of BSD (see Alloy et al., 2012 for review). In Phase I, students from 13 Philadelphia public high schools (Grades 9 –12, ages 14–18) and two universities (ages 17–19) were screened with two self-report reward sensitivity questionnaires: the Behavioral Inhibition System/Behavioral Activation System Scales (BIS/BAS; Carver & White, 1994) and the Sensitivity to Punishment Sensitivity to Reward Questionnaire (SPSRQ; Torrubia, Avila, Molto, & Caseras, 2001). Students scoring in the highest 15th percentile on both the BAS-Total subscale of the BIS/BAS Scales and the Sensitivity to Reward (SR) subscale of the SPSRQ formed a High BAS (reward sensitivity) group, whereas those who scored between the 40th and 60th percentiles on both measures formed the Moderate BAS (reward sensitivity) group. Of 9991 students screened in Phase I, 7.77% (n = 776) qualified for the High BAS and 4.04% (n = 404) qualified for the Moderate BAS groups.

A subset of adolescents who met the Phase I screening criteria was invited for Phase II screening (see Alloy et al., 2012 for more details on Phase II selection of participants): 244

High BAS (31.4%) and 146 Moderate BAS (36.1%) students completed Phase II. In Phase II, participants were administered the mood and psychosis disorder sections of an expanded Schedule for Affective Disorders and Schizophrenia = Lifetime (exp-SADS-L) diagnostic interview (Alloy et al., 2008; Endicott & Spitzer, 1978); the rest of the exp-SADS-L, including a family history section, was administered to the final eligible sample at baseline (Time 1). Participants also completed the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) and the Altman Self-Rating Mania Scale (ASRM; Altman, Hedeker, Peterson, & Davis, 1997) at Phase II screening to assess depressive and (hypo)manic symptoms, respectively. Exp-SADS-L interviewers were blind to participants' BAS risk group.

Participants were excluded from the final sample if they met *DSM-IV-TR* (American Psychiatric Association, 2000) or Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) diagnosis of: 1) any BSD (Bipolar I, Bipolar II, Cyclothymia, Bipolar NOS) or a hypomanic episode with onset prior to the participant's Phase I screening date, or 2) any lifetime psychotic disorder (Schizophrenia, Schizoaffective Disorder, Major Depressive Disorder with psychosis). They were not excluded if they met criteria for a non-psychotic *DSM-IV-TR* or RDC major depressive or RDC minor depressive episode with onset prior to Phase I, because prior depressive episodes without mania or hypomania may reflect unipolar depression rather than bipolar disorder. Participants also were excluded if they lacked fluency in English. Participants with a prior BSD or hypomanic episode were excluded because the main goal of Project TEAM was to examine predictors of first onset of BSD.

Of 390 participants interviewed at Phase II, 22 were excluded because they met criteria for a BSD or hypomanic episode with onset prior to their Phase I screening, 7 were excluded because they exhibited psychotic symptoms or met criteria for a psychotic disorder, and another 5 were excluded for poor English fluency. The Project TEAM final sample included 171 High BAS and 119 Moderate BAS participants (mean age = 17.44; SD = 1.56). Further details of the screening and selection criteria and evidence that the final sample was representative of both the Phase I and Phase II screening samples may be found in Alloy et al. (2012).

The present analyses were based on only the participants who also had baseline (Time 1) social rhythm regularity data needed for the current study. Thirty-three of the 171 High BAS and 24 of the 119 Moderate BAS participants in the final TEAM sample were missing Time 1 social rhythm regularity data; thus, the present analyses were based on 138 (87 F, 51 M) High BAS and 95 (68 F, 27 M) Moderate BAS participants, with mean ages at baseline of 18.11 and 17.87 years (SDs = 1.49 and 1.63), respectively. The racial breakdown of the sample was 54.2% Caucasian, 30.2% African American, 8.0% Asian or Pacific Islander, 4.5% Biracial, and 3.1% Other. Also, 8.5% were Hispanic. A family history of bipolar disorder was present in 6% of the High BAS and 11% of the Moderate BAS participants. The participants with missing social rhythm regularity data did not differ from those included on demographics, initial BAS and Sensitivity to Reward scores, or initial BDI and ASRM scores. In addition, the High BAS and Moderate BAS groups did not differ from each other on age, gender, or race/ethnicity. Table 1 presents means and SDs of the baseline BAS Total, Sensitivity to Reward, BDI, ASRM, and SRM regularity scores.

Although Boland et al. (in press) also studied the interplay between reward sensitivity and social rhythm dysregulation in the Project TEAM sample, there is no overlap between the Boland et al. (in press) and current studies. Whereas Boland et al. (in press) used interviewer-rated social rhythm disruption scores in response to actual life events that High BAS and Moderate BAS participants experienced at the first follow-up assessment to predict subsequent hypomanic and depressive symptoms, the present study uses self-reported trait social rhythm regularity at baseline on the Social Rhythm Metric to predict first onset of diagnosed BSDs.

Procedure

Participants in the final sample were invited for a baseline (Time 1) assessment and the prospective study, and additional informed consent and assent were obtained at Time 1. At Time 1, participants completed the remainder of the exp-SADS-L diagnostic interview, including family history and substance use disorders sections, and a modified SRM (Monk et al., 1990) as well as other measures not relevant to the present study. Then, they completed an exp-SADS-Change (exp-SADS-C) diagnostic interview (Alloy et al., 2008; Spitzer & Endicott, 1978) approximately every 6 months. The present analyses were based on an average of 31.7 months (SD = 20.1 months) of prospective follow-up.

Measures

Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) Scale— The BIS/BAS Scale (Carver & White, 1994) is a 20-item, self-report questionnaire that assesses trait individual differences in BIS and BAS sensitivities in general. It includes three BAS subscales and one BIS subscale and items are rated on 4-point Likert scales ranging from 1 = strongly disagree to 4 = strongly agree. A BAS-Total score also may be calculated as the sum of all BAS items. This total score was used as part of the screening criteria for selecting the BAS risk groups. The BIS/BAS scales have demonstrated internal consistency and retest reliability (Carver & White, 1994), as well as construct validity, exhibiting expected associations with affect, personality traits, and performance on reaction-time and learning tasks involving incentives (Colder & O'Conner, 2004; Kambouroupolis & Staiger, 2004; Zinbarg & Mohlman, 1998). Internal consistency of the BAS-Total score in the Phase I screening sample was $\alpha = .80$.

Sensitivity to Punishment Sensitivity to Reward Questionnaire (SPSRQ)—The SPSRQ (Torrubia et al., 2001) is another measure of trait BIS and BAS sensitivities in general that focuses on sensitivity to specific types of rewards and punishments. It contains 24 Sensitivity to Reward (SR; e.g., "Does the good prospect of obtaining money motivate you strongly to do some things?") and 24 Sensitivity to Punishment (SP; e.g., "Do you often refrain from doing something because you are afraid of it being illegal?) "yes" or "no" items. Both subscales have acceptable internal consistency, with α's = .75–.83, and retest reliabilities (Torrubia et al., 2001). The SPSRQ also exhibits construct validity in terms of expected correlations with extraversion, impulsivity, sensation seeking, and neuroticism, and associations with proneness to various personality disorders (e.g., Alloy et al., 2006; Torrubia et al., 2001). The SR subscale was used along with the BAS-T to select High

versus Moderate BAS participants. In Phase I, α for the SR scale was .76. BAS-T and SR scores correlated *r* = .40 in our Phase I sample.

Modified Social Rhythm Metric (M-SRM)—The SRM (Monk et al., 1990) assesses a person's daily social rhythm patterns. It captures the frequency and timing of specific activities that are part of a person's daily routine (e.g., getting out of bed, going to bed, mealtimes, first social contact of the day, starting work or school). The SRM includes 15 specified daily activities and two individualized write-in items. The SRM was found to be modestly consistent (*r* = .44) and valid in healthy controls (Monk et al., 1990; 1991), and it distinguishes individuals with BSDs from controls (Ashman et al., 1999; Jones et al., 2005; Shen et al., 2008; Szuba et al., 1992) and predicts recurrences of BSD mood episodes (Shen et al., 2008). In the current study, we used a slightly modified version of the SRM (M-SRM) previously used by Shen et al. (2008). The M-SRM is intended to assess more trait-like social rhythm regularity or an individual's typical social rhythm patterns. Participants were asked to endorse activities if they occurred a minimum of three times per week within 45 min of the usual time each week over the past month. The Regularity score was the number of activities endorsed as occurring three or more times within 45 min of the habitual time during the week (possible range = 0 – 17) over the past month. The M-SRM was moderately consistent over approximately eight months in a sample of late adolescent/young adult BSD and healthy control participants (*r* = .61; Shen et al., 2008). The M-SRM was given at baseline (Time 1) and exhibited good internal consistency (α= .76). In addition, we calculated a Sleep/Wake Regularity score for exploratory analyses based on the go to bed and get out of bed items.

Beck Depression Inventory (BDI)—The BDI (Beck et al., 1979) has 21 items assessing the severity of affective, cognitive, motivational, and somatic symptoms of depression over the past 30 days. It has good internal (α's = .81–.86) and retest reliability (*r*'s = .48–.86) and validity in nonclinical samples (Beck, Steer, & Garbin, 1988). In our Phase II sample, α = .87.

Altman Self Rating Mania Scale (ASRM)—The ASRM (Altman et al., 1997) has 5 items rated on 5-point Likert scales that assess five symptoms of (hypo)mania over the past 30 days: inflated self-confidence, talkativeness, elation, reduced need for sleep, and excessive activity. Items load on a single factor and ASRM scores are highly correlated with both clinical interview and other self-report measures of mania (Altman et al., 2001). In our Phase II sample, α = .75.

Schedule for Affective Disorders and Schizophrenia – Lifetime (SADS-L)—The SADS-L (Endicott & Spitzer, 1978) is a semi-structured diagnostic interview used to assess current and lifetime history of Axis I disorders. The mood disorders and psychosis sections of an expanded SADS-L (exp-SADS-L; see Alloy et al., 2008; 2012) were given during Phase II to determine eligibility for the final Project TEAM sample, with the remainder administered at baseline (Time 1) to participants in the final sample. The exp-SADS-L assessed the occurrence, duration, and severity of symptoms related to mood, anxiety, eating, substance use, and psychotic disorders over the course of an individual's lifetime.

Specific additions were made to the SADS-L to obtain project-specific information and included: 1) additional probes to allow for *DSM-IV-TR* as well as RDC diagnoses; 2) additional probes regarding mood episodes to better capture individual symptom differences, duration, and frequency of episodes; and 3) additional sections on eating disorders, ADHD, acute stress disorder, medical history, family history, and organic rule-out conditions. Project interviewers were highly trained doctoral students in clinical psychology, postdoctoral fellows, and post-baccalaureate research assistants. They were unaware of participants' BAS risk group and Phase I BIS/BAS and SPSRQ scores. The exp-SADS-L has demonstrated excellent inter-rater reliability, with $\kappa > .90$ for unipolar depression diagnoses based on 80 jointly rated interviews (Alloy et al., 2000) and $\kappa > .96$ for BSDs based on 105 jointly rated interviews (Alloy et al., 2008). Further details regarding the exp-SADS-L administration and diagnoses for Project TEAM may be found in Alloy et al. (2012).

Schedule for Affective Disorders and Schizophrenia – Change (SADS-C)—An expanded version of the SADS-C (Alloy et al., 2008; 2012; Spitzer & Endicott, 1978) diagnostic interview was used to assess prospective onsets of mood episodes and diagnoses. It was administered approximately every six months during the prospective follow-up by interviewers blinded to participants' BAS risk group, BIS/BAS and SPSRQ scores, SRM scores, and baseline diagnostic information and family history. The exp-SADS-C was expanded in the same way as the exp-SADS-L and also included features of the Longitudinal Interval Follow-up Evaluation (LIFE II; Shapiro & Keller, 1979) to track symptoms and mood episodes over follow-up. However, the exp-SADS-C inquired about the presence of every symptom of depression and hypomania and mania more frequently (daily) than does the LIFE II (weekly) during each six-month follow-up interval. Based on joint ratings of 60 interviews, interrater reliability for the exp-SADS-C was $\kappa > .80$ (Alloy et al., 2008). In addition, a validity study that compared participants' dating of symptoms on the exp-SADS-C against daily symptom ratings obtained prospectively over four months obtained 70% accuracy for exp-SADS-C symptom dating (Alloy et al., 2008). In Project TEAM, interrater reliability for BSDs on the exp-SADS-L or exp-SADS-C interviews was $\kappa = 1.0$ for Bipolar I, $\kappa = .92$ for Bipolar II, and $\kappa = .88$ for Bipolar NOS.

Data Analysis

To examine whether social rhythm regularity on the M-SRM at baseline predicted the likelihood of first onset of a BSD among High BAS as well as Moderate BAS participants, we conducted a hierarchical logistic regression analysis for each group with the occurrence (yes-no) of a BSD during follow-up as the dependent variable.[1] In each logistic regression, the length of follow-up (in days), gender, age at baseline, baseline depressive (BDI) and hypomanic (ASRM) symptoms, and family history of bipolar disorder were entered in Step 1 as covariates. Then, baseline M-SRM regularity scores were entered in Step 2. We included age and baseline depressive and (hypo)manic symptoms as covariates to control for any effects of initial differences in age and subsyndromal mood symptoms on the prospective first onset of a BSD. We also included family history of bipolar disorder as a covariate to ensure that any prediction of first onset of BSD by social rhythm regularity was above and beyond any effects of family history. Subsequently, if social rhythm regularity

was a significant predictor of BSD onset in either group, we re-conducted the relevant logistic regression analysis controlling for further possible confounds (i.e., BAS-Total scores, presence of alcohol or drug use disorders). We also explored the role of regularity of sleep and wake times specifically in accounting for any social rhythm regularity effects.

Results

Of the 138 High BAS and 95 Moderate BAS participants, 20 High BAS and 4 Moderate BAS individuals developed a first onset of BSD during follow-up. Of these 24 BSD onset cases, 15 had Bipolar II (onset of at least one hypomanic and one major depressive episode or onset of a hypomanic episode) and 9 had Bipolar NOS (onset of at least one hypomanic episode). The mean age of first onset of BSD was 20.5 years (SD = 2.8 years). Table 2 displays the correlations between all study variables for the two groups. Among High BAS participants, M-SRM Regularity scores were negatively correlated with initial BDI scores, such that higher initial depressive symptoms were associated with lower social rhythm regularity. The only other significant correlation was between participants' ages and ASRM scores, such that greater age was associated with lower initial hypomanic symptoms. For Moderate BAS participants, high baseline ASRM scores were associated with greater social rhythm regularity and greater likelihood of developing a BSD.

Table 3 presents the results of the hierarchical logistic regression analyses testing whether M-SRM Regularity at baseline predicted the likelihood of first onset of BSD among High BAS and Moderate BAS participants, controlling for time in study, age, gender, baseline depressive and hypomanic symptoms, and family history of bipolar disorder. As shown in Table 3, none of the covariates entered in Step 1 significantly predicted BSD onset among High BAS participants. However, consistent with this study's hypothesis, when entered in Step 2, baseline M-SRM Regularity scores did significantly predict first onset of BSD, controlling for all covariates. Among high BAS adolescents, those with lower Regularity scores were more likely to have a BSD onset than those with greater regularity. In contrast, among Moderate BAS participants, M-SRM Regularity scores did not predict onset of BSD, although greater initial hypomanic symptom severity on the ASRM did predict onset of BSD among Moderate BAS participants.

Potential Confounds

To be certain that the predictive association between lower social rhythm regularity and onset of BSD among High BAS participants was not attributable to adolescents with the most irregular social rhythms having the highest BAS scores, we repeated the logistic

[1]The low base rate of first onsets of BSDs in the current sample, particularly in the Moderate BAS group, contributes to relatively low statistical power. As a result, it is difficult to demonstrate a significant interaction between BAS risk status and social rhythm regularity in predicting first onsets of BSDs with six or more needed covariates using a maximum likelihood procedure, like logistic regression. When the BAS X Regularity interaction is added on the final step of the logistic regression controlling for the main effects of BAS risk and social rhythm regularity in the full sample, the interaction does not predict first onset of BSD significantly (OR = .98, *p's* = .23 – .28 depending on the number of covariates included).
On the other hand, the social zeitgeber theory does not require that low social rhythm regularity serve as a risk factor for BSD specifically among individuals with heightened reward sensitivity. However, given the bidirectional influence of the reward and circadian systems on each other, reward hypersensitivity may well potentiate a general tendency for social rhythm irregularity to contribute to BSD onset. Thus, low social rhythm regularity may predict BSD onset to a greater extent in High BAS than Moderate BAS participants, as the results in Table 3 suggest.

regression analysis for the High BAS group including BAS-Total scores as an additional, seventh covariate. This analysis revealed that M-SRM Regularity continued to predict first onset of BSD even while also controlling for BAS-Total scores (β = −.139, SE β = .072, Wald = 3.786, OR = 0.870, p = .05).

Given the association between substance use disorders and high BAS/reward sensitivity (e.g., Alloy et al., 2009; Dawe & Loxton, 2004), as well as the possibility that extensive substance use may interfere with social rhythm regularity, we also controlled for a history of alcohol and drug use disorders as a possible confounding factor in the relationship between low social rhythm regularity and prospective first onset of BSD among High BAS adolescents. Controlling for DSM-IV-TR or RDC alcohol or drug use disorders as an additional covariate, social rhythm regularity still predicted first onset of BSD among High BAS participants (β= −.150, SE β = .072, Wald = 4.354, OR = .861, p = .037).

Exploratory Analysis

Inasmuch as social rhythm regularity includes stability of the sleep/wake cycle as one of its several components, we explored whether the Sleep/Wake Regularity score derived from the M-SRM would predict onset of BSD among High BAS participants in a manner comparable to, or better than, the overall Regularity score. When we substituted Sleep/Wake Regularity for the overall Regularity scores in the analysis shown in Table 3 for High BAS participants, sleep/wake regularity predicted onset of BSD at trend level significance (β = −.743, SE β = .461, Wald = 2.603, OR = .475, p = .107). Thus, sleep/wake regularity did not predict BSD onset as well as overall social rhythm regularity.

Discussion

This study provided the first test of irregularity of social rhythms as a predictor of first onset of BSD. According to the social zeitgeber model of BSDs (Alloy et al., 2015; Ehlers et al., 1988; Grandin et al., 2006), changes in daily social rhythms or schedules lead to disruption of circadian rhythms and, in turn, onset of bipolar mood episodes. From this perspective, individuals with low regularity of social rhythms are hypothesized to be vulnerable to circadian rhythm disruption, and thus, to bipolar episode onset. Given evidence for bidirectional influences between reward sensitivity and motivation and circadian rhythms (e.g., see Alloy et al., 2015 for review), and prior findings that reward hypersensitivity predicts first lifetime onset of BSD (Alloy et al., 2012), we examined the vulnerability status of low social rhythm regularity in adolescents with high reward sensitivity compared to those with moderate reward sensitivity.

Consistent with the social rhythm irregularity vulnerability hypothesis, we found that low social rhythm regularity significantly predicted a greater likelihood of first lifetime onset of a BSD among adolescents already at risk based on exhibiting reward hypersensitivity, but not among those with moderate reward sensitivity, controlling for length of follow-up, gender, age, baseline hypomanic and depressive symptoms, and family history of bipolar disorder. This was a very conservative test of the social rhythm regularity vulnerability hypothesis because it involved a truly prospective design in adolescents with no prior history of BSD and controlled for any initial subsyndromal depressive and hypomanic symptoms

and family history of bipolar disorder. The predictive effect was maintained even when baseline reward sensitivity (BAS-T) scores and presence of alcohol and drug use disorders were controlled as additional covariates. It also was a conservative test because we used a baseline measure of social rhythm regularity as the predictor of first onset of BSD. Although the M-SRM was meant to assess more trait-like social rhythm regularity, it is impressive that this measure predicted BSD onset up to several months later. Potentially, even stronger prediction of BSD onset might be attained with repeated assessments of social rhythm regularity. Thus, this study provides the strongest evidence to date that low social rhythm regularity is a vulnerability factor for BSD, specifically among individuals with high reward sensitivity, and adds additional support for the social zeitgeber theory of BSDs.

Our results extend prior work demonstrating that low social rhythm regularity is associated with BSDs (Ashman et al., 1999; Jones et al., 2005; Shen et al., 2008; Szuba et al., 1992), as well as prior studies demonstrating that low social rhythm regularity predicts recurrences of bipolar mood episodes in individuals with BSDs (Shen et al., 2008) and that life events that disrupt social rhythms precipitate bipolar mood episodes (Malkoff-Schwartz et al., 1998; 2000; Sylvia et al., 2009). They also are consistent with prior findings that individuals at behavioral risk for BSDs exhibit low social rhythm regularity (Bullock et al., 2011; Meyer & Maier, 2006). Finally, along with Boland et al. (in press), these findings provide further evidence for a potential integration of reward hypersensitivity and social and circadian rhythm models of BSD (e.g., Alloy et al., 2015; Murray et al., 2009). In the Project TEAM sample, Boland et al. (in press) reported that High BAS individuals were more likely than Moderate BAS individuals to experience social rhythm disruption in response to the occurrence of BAS-relevant life events and that this social rhythm disruption mediated the association between the occurrence of BAS-relevant events and prospective hypomanic and depressive symptoms. The present findings build upon the Boland et al. study by demonstrating that more trait-like patterns of low social rhythm regularity also predict first onset of diagnosable BSD among individuals with reward hypersensitivity.

Our findings have potentially important clinical implications. They suggest that it may be possible to screen adolescents and identify those at risk for developing a BSD based on exhibiting both high reward sensitivity and low social rhythm regularity before onset occurs. As such, it also may be possible to develop early interventions for these at-risk adolescents targeted at either reward processing or regularizing daily schedules (Frank et al., 1997; 2005; Nusslock et al., 2009). Early identification of risk for BSDs is key to promoting more positive outcomes.

Study Strengths and Limitations

A major strength of this study is the truly prospective design with a theory-based assessment of social rhythm regularity. In addition, our study included a large, ethnically diverse community sample of adolescents, which should increase generalizability of study findings, standardized diagnostic interviews, interviewers who were blinded to social rhythm regularity and other predictors, frequent assessment intervals allowing for sensitivity in assessing mood episodes, and highly conservative tests of the study hypothesis including

controls for initial symptom levels, family history of bipolar disorder, and substance use disorders.

However, the study's limitations need to be noted as well. First, although our sample was ethnically diverse and representative of the larger adolescent community population from which it was drawn on demographics, our findings may not generalize to other community samples or to clinical samples of adolescents. Second, our assessment of social rhythm regularity was based on a self-report measure only, albeit the reliable and valid Social Rhythm Metric. Future studies would benefit from testing alternative measures of social rhythm regularity as vulnerabilities for onset of BSD. Third, although the M-SRM was designed to measure trait regularity of social rhythms, participants only completed it once at baseline. It is possible that the regularity of some individuals' social rhythms changed over follow-up and thus, readministering the M-SRM at a later time point would have allowed us to confirm that participants maintained their regularity patterns over time. Fourth, family history of bipolar disorder was measured with the family history method rather than with more accurate direct interviews with relatives. Fifth, social rhythm regularity as assessed by the Social Rhythm Metric likely involves several components, including engaging in activities that provide structure in one's life and regularity of the sleep/wake cycle, to name two. It is not clear conceptually which of these two aspects of social rhythm regularity is central to its predictive association with BSD. Although we explored whether sleep/wake regularity predicted BSD onset as well as overall regularity did, and found that it did not, future research is needed to carefully conceptualize and parse apart the key active components of social rhythm regularity. Given the important role of sleep disturbance in circadian rhythm regularity and bipolar disorders (e.g., Murray & Harvey, 2010), it is likely that other approaches to assessing regularity of the sleep/wake cycle (e.g., actigraphy, dim light melatonin onset) may yield stronger predictive associations. Sixth, given low rates of BSD onset in our sample and the consequent limited statistical power, we could not fully test the specificity of the social rhythm regularity effect to High BAS participants by examining the BAS risk group X social rhythm regularity interaction in a full logistic regression model with all covariates. Finally, this study examined self-reported social rhythm regularity as a risk factor for BSD onset, but not desynchronization or disruption of circadian rhythms itself, which is hypothesized to be the more proximal mechanism underlying vulnerability to BSDs. Future studies should investigate circadian rhythm disruption itself as a predictor of first onset of BSD, as well as whether circadian rhythm disruption mediates the predictive association between social rhythm regularity and BSD onset.

Conclusion

In summary, this is the first prospective test of social rhythm regularity as a predictor of first lifetime onset of disorders in the bipolar spectrum. Keeping in mind the study limitations, our findings support the vulnerability hypothesis of the social zeitgeber model and indicate that irregularity of social rhythms is a vulnerability for first onset of bipolar spectrum disorders among adolescents already at risk based on reward hypersensitivity.

Acknowledgments

This research was supported by National Institute of Mental Health Grants MH77908 and MH102310 to Lauren B. Alloy. The writing of this manuscript also was supported by the Office of Academic Affiliations, Advanced Fellowship Program in Mental Illness Research and Treatment, Department of Veterans Affairs.

References

Alloy LB, Abramson LY. The role of the behavioral approach system (BAS) in bipolar spectrum disorders. Current Directions in Psychological Science. 2010; 19:189–194. [PubMed: 20606725]

Alloy LB, Abramson LY, Hogan ME, Whitehouse WG, Rose DT, Robinson MS, Lapkin JB. The Temple – Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive risk for depression. Journal of Abnormal Psychology. 2000; 109:403–418. [PubMed: 11016110]

Alloy LB, Abramson LY, Walshaw PD, Cogswell A, Grandin LD, Hughes ME, Hogan ME. Behavioral approach system (BAS) and behavioral inhibition system (BIS) sensitivities and bipolar spectrum disorders: Prospective prediction of bipolar mood episodes. Bipolar Disorders. 2008; 10:310–322. [PubMed: 18271911]

Alloy LB, Abramson LY, Walshaw PD, Cogswell A, Smith J, Neeren AM, Nusslock R. Behavioral approach system (BAS) sensitivity and bipolar spectrum disorders: A retrospective and concurrent behavioral high-risk design. Motivation and Emotion. 2006; 30:143–155.

Alloy LB, Bender RE, Wagner CA, Whitehouse WG, Abramson LY, Hogan ME, Harmon-Jones E. Bipolar spectrum – substance use co-occurrence: Behavioral Approach System (BAS) sensitivity and impulsiveness as shared personality vulnerabilities. Journal of Personality and Social Psychology. 2009; 97:549–565. [PubMed: 19686007]

Alloy LB, Bender RE, Whitehouse WG, Wagner CA, Liu RT, Grant DA, Abramson LY. High behavioral approach system (BAS) sensitivity, reward responsiveness, and goal-striving predict first onset of bipolar spectrum disorders: A prospective behavioral high risk design. Journal of Abnormal Psychology. 2012; 121:339–351. [PubMed: 22004113]

Alloy LB, Nusslock R, Boland EM. The development and course of bipolar spectrum disorders: An integrated reward and circadian rhythm dysregulation model. Annual Review of Clinical Psychology. 2015; 11:213–250.

Altman EG, Hedeker D, Peterson JL, Davis JM. The Altman self-rating mania scale. Biological Psychiatry. 1997; 42:948–955. [PubMed: 9359982]

Altman FG, Hedeker D, Peterson JL, Davis JM. A comparative evaluation of three self-rating scales for acute mania. Biological Psychiatry. 2001; 50:468–471. [PubMed: 11566165]

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 2000. Text Revision

Ashman SB, Monk TH, Kupfer DJ, Clark CH, Myers FS, Frank E, et al. Relationship between social rhythms and mood in patients with rapid cycling bipolar disorder. Psychiatry Research. 1999; 86:1–8. [PubMed: 10359478]

Beck, AT.; Rush, AJ.; Shaw, BF.; Emery, G. Cognitive therapy of depression. New York, NY: Guilford Press; 1979.

Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clinical Psychology Review. 1988; 8:77–100.

Boland EM, Bender RE, Alloy LB, Conner BT, LaBelle DR, Abramson LY. Life events and social rhythms in bipolar spectrum disorders: An examination of social rhythm sensitivity. Journal of Affective Disorders. 2012; 139:264–272. [PubMed: 22381951]

Boland EM, Stange JS, LaBelle DR, Shapero BG, Weiss RB, Abramson LY, Alloy LB. Affective disruption from social rhythm and behavioral approach system (BAS) sensitivities: A test of an integration of the social zeitgeber and reward theories of bipolar disorder. Clinical Psychological Science. (in press).

Bullock B, Judd F, Murray G. Social rhythms and vulnerability to bipolar disorder. Journal of Affective Disorders. 2011; 135:384–388. [PubMed: 21708409]

Carver CS, White TL. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. Journal of Personality and Social Psychology. 1994; 67:319–333.

Clark LA, Watson D, Leeka J. Diurnal variation in the positive affects. Motivation and Emotion. 1989; 13:205–234.

Colder CAR, O'Connor RM. Gray's reinforcement sensitivity model and child psychopathology: Laboratory and questionnaire assessment of the BAS and BIS. Journal of Abnormal Child Psychology. 2004; 32:435–451. [PubMed: 15305548]

Dawe S, Loxton NJ. The role of impulsivity in the development of substance use and eating disorders. Neuroscience and Biobehavioral Reviews. 2004; 28:343–351. [PubMed: 15225976]

Ehlers C, Frank E, Kupfer DJ. Social zeitgebers and biological rhythms. Archives of General Psychiatry. 1988; 45:948–952. [PubMed: 3048226]

Endicott J, Spitzer RA. A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. Archives of General Psychiatry. 1978; 35:837–844. [PubMed: 678037]

Forbes EE, Dahl RE, Almeida JRC, Ferrell RE, Nimgaonkar VL, et al. PER2 rs2304672 polymorphism moderates circadian-relevant reward circuitry activity in adolescents. Biological Psychiatry. 2012; 71:451–457. [PubMed: 22137505]

Frank E, Hlastala S, Ritenour A, Houck P, Tu XM, Monk TH. Inducing lifestyle regularity in recovering bipolar disorder patients: results from the maintenance therapies in bipolar disorder protocol. Biological Psychiatry. 1997; 47:1165–1173. [PubMed: 9171907]

Frank E, Kupfer DJ, Thase ME, Mallinger AG, Swartz HA, Fagiolini AM, et al. Two-year outcomes for interpersonal and social rhythm therapy in individuals with bipolar I disorder. Archives of General Psychiatry. 2005; 62:996–1004. [PubMed: 16143731]

Goel N. Late-night presentation of an auditory stimulus phase delays human circadian rhythms. American Journal of Physiology – Reg. Integrat Comparative Physiology. 2005; 289:R209–R216.

Grandin LD, Alloy LB, Abramson LY. The social zeitgeber theory, circadian rhythms, and mood disorders: Review and evaluation. Clinical Psychology Review. 2006; 26:679–694. [PubMed: 16904251]

Holm SM, Forbes EE, Ryan ND, Phillips ML, Tarr JA, Dahl RE. Reward-related brain function and sleep in pre/early pubertal and mid/late pubertal adolescents. Journal of Adolescent Health. 2009; 45:326–334. [PubMed: 19766936]

Johnson SL, Edge MD, Holmes MK, Carver CS. The behavioral activation system and mania. Annual Review of Clinical Psychology. 2012; 8:243–267.

Jones SH, Hare DJ, Evershed K. Actigraphic assessment of circadian activity and sleep patterns in bipolar disorder. Bipolar Disorders. 2005; 7:176–186. [PubMed: 15762859]

Jones SH, Tai S, Evershed K, Knowles R, Bentall R. Early detection of bipolar disorder: A pilot familial high-risk study of parents with bipolar disorder and their adolescent children. Bipolar Disorders. 2006; 8:362–372. [PubMed: 16879137]

Kambouroupolis N, Staiger PK. Reactivity to alcohol-related cues: Relationship among cue type, motivational processes, and personality. Psychology of Addictive Behavior. 2004; 18:275–283.

Malkoff-Schwartz S, Frank E, Anderson B, Sherril JT, Siegel L, Patterson D, et al. Stressful life events and social rhythm disruption in the onset of manic and depressive bipolar episode. Archives of General Psychiatry. 1998; 55:702–707. [PubMed: 9707380]

Malkoff-Schwartz S, Frank E, Anderson BP, Hlastala SA, Luther JF, Sherril JT, et al. Social rhythm disruption and stressful life events in the onset of bipolar and unipolar episodes. Psychological Medicine. 2000; 30:1005–1016. [PubMed: 12027038]

McClung CA. Circadian genes, rhythms and the biology of mood disorders. Pharmacology & Therapeutics. 2007; 114:222–232. [PubMed: 17395264]

Meyer TD, Maier S. Is there evidence for social rhythm instability in people at risk for affective disorders? Psychiatry Research. 2006; 141:103–114. [PubMed: 16360217]

Monk TH, Flaherty JF, Frank E, Hoskinson K, Kupfer DJ. The social rhythm metric an instrument to quantify the daily rhythms of life. Journal of Nervous and Mental Disease. 1990; 178:120–126. [PubMed: 2299336]

Monk TH, Kupfer DJ, Frank E, Ritenour AM. The social rhythm metric (SRM): Measuring daily social rhythms over 12 weeks. Psychiatry Research. 1991; 36:195–207. [PubMed: 2017534]

Murray G, Harvey A. Circadian rhythms and sleep in bipolar disorder. Bipolar Disorders. 2010; 12:459–472. [PubMed: 20712747]

Murray G, Nicholas CL, Kleiman J, Dwyer R, Carrington MJ, et al. Nature's clock and human mood: The circadian system modulates reward motivation. Emotion. 2009; 9:705–716. [PubMed: 19803592]

Nusslock R, Abramson LY, Harmon-Jones E, Alloy LB, Coan JA. Psychosocial interventions for bipolar disorder: perspective from the behavioral approach system (BAS) dysregulation theory. Clinical Psychology: Science and Practice. 2009; 16:449–469. [PubMed: 20161456]

Roenneberg T, Merrow M. Entrainment of the human circadian clock. Cold Spring Harbor Symposium on Quantitative Biology. 2007; 72:293–299.

Shapiro, RW.; Keller, MB. Longitudinal Interval Follow-up Evaluation (LIFE). Boston, MA: Massachusetts General Hospital; 1979.

Shen GC, Alloy LB, Abramson LY, Sylvia LG. Social rhythm regularity and the onset of affective episodes in bipolar spectrum individuals. Bipolar Disorders. 2008; 10:520–529. [PubMed: 18452448]

Spitzer, RA.; Endicott, J. Schedule for Affective Disorders and Schizophrenia – Change version. Biometrics Research, New York State Psychiatric Institute; 1978.

Spitzer RL, Endicott J, Robins E. Research Diagnostic Criteria: Rationale and reliability. Archives of General Psychiatry. 1978; 35:773–782. [PubMed: 655775]

Stetler C, Dickerson SS, Miller GE. Uncoupling of social zeitgebers and diurnal cortisol secretion in clinical depression. Psychoneuroendocrinology. 2004; 29:1250–1259. [PubMed: 15288704]

Sylvia LG, Alloy LB, Hafner JA, Gauger MC, Verdon K, Abramson LY. Life events and social rhythms in bipolar spectrum disorders: A prospective study. Behavior Therapy. 2009; 40:131–141. [PubMed: 19433144]

Szuba MP, Yager A, Guze BH, Allen EM, Baxter LR. Disruption of social circadian rhythms in major depression: A preliminary report. Psychiatry Research. 1992; 42:221–230. [PubMed: 1496054]

Thayer RE, Takahashi PJ, Pauli JA. Multidimensional arousal states, diurnal rhythms, cognitive and social processes, and extraversion. Personality and Individual Differences. 1988; 9:15–24.

Torrubia R, Avila C, Molto J, Caseras X. The Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) as a measure of Gray's anxiety and impulsivity dimensions. Personality and Individual Differences. 2001; 31:837–862.

Uroševič S, Abramson LY, Harmon-Jones E, Alloy LB. Dysregulation of the behavioral approach system (BAS) in bipolar spectrum disorders: Review of theory and evidence. Clinical Psychology Review. 2008; 28:1188–1205. [PubMed: 18565633]

Wever RA. Light effects on human circadian rhythms: A review of recent Andechs experiments. Journal of Biological Rhythms. 1989; 4:161–185. [PubMed: 2519587]

Zinbarg RE, Mohlman J. Individual differences in the acquisition of affectively valenced associations. Journal of Personality and Social Psychology. 1998; 74:1024–1040. [PubMed: 9569657]

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Table 1

Means and Standard Deviations (SD) of Baseline Measures

	High BAS (N = 138)	Moderate BAS (N = 95)	*p*
BAS-T	46.17 (2.87)	37.99 (1.32)	<.001
SR	17.94 (1.88)	11.52 (1.84)	<.001
BDI	7.11 (6.50)	6.18 (6.09)	.254
ASRM	6.46 (4.01)	5.63 (3.69)	.109
M-SRM	11.05 (3.36)	11.76 (2.75)	.078

Note. Standard deviations are in parentheses. BAS-T = Behavioral Approach System – Total scores from the BIS/BAS Scales; SR = Sensitivity to Reward scores from the SPSRQ; BDI = Beck Depression Inventory scores; ASRM = Altman Self-Rating Mania Scale scores; M-SRM = Modified Social Rhythm Metric Regularity scores.

Table 2

Correlations among Study Variables

	Days	Gender	Age	Fam Hx	ASRM	BDI	M-SRM	BSD
Days	-----	.064	.063	.005	.104	−.094	.089	−.038
Gender	−.051	-----	−.066	.079	−.046	.005	.029	−.084
Age	−.188	.110	-----	.080	−.242**	−.041	−.049	−.009
Fam Hx	.014	.009	.069	-----	−.075	.050	.031	−.038
ASRM	.028	−.191	.012	−.062	-----	−.041	.053	.071
BDI	.159	.096	−.112	.163	.044	-----	−.222**	.042
M-SRM	.064	−.098	−.069	.020	.284**	.078	-----	−.098
BSD	−.032	−.100	.081	.088	.250*	.063	−.001	-----

Note. Correlations for High BAS participants are shown above the diagonal and correlations for Moderate BAS participants are shown below the diagonal.

* $p < .05$;

** $p < .01$

Days = Days in Study; Age = Age at Baseline; Fam Hx = Family history of bipolar disorder; ASRM = Altman Self-Rating Mania Scale scores; BDI = Beck Depression Inventory scores; M-SRM = Modified Social Rhythm Metric Regularity scores; BSD = Onset of a bipolar spectrum disorder (0 = No, 1 = Yes).

Table 3

Hierarchical Logistic Regressions Predicting Likelihood of Bipolar Spectrum Disorder Onset

	β	SE β	Wald	OR	95% CI	*p*
High BAS Group						
Step 1						
Days in Study	.000	.000	0.138	1.000	0.999 – 1.000	.711
Gender	−.603	.495	1.483	0.547	0.207 – 1.444	.223
Age	−.009	.170	0.003	0.991	0.710 – 1.384	.958
Fam Hx	−.069	1.121	0.004	0.934	0.104 – 8.403	.951
ASRM	.047	.062	0.557	1.048	0.927 – 1.184	.456
BDI	.015	.036	0.182	1.015	0.947 – 1.089	.670
Step 2						
M-SRM Regularity	−.150	.072	4.365	0.861	0.748 – 0.991	.037
Moderate BAS Group						
Step 1						
Days in Study	−.001	.001	0.371	0.999	0.997 – 1.002	.542
Gender	−1.080	1.163	0.862	0.340	0.035 – 3.319	.353
Age	.305	.431	0.499	1.356	0.583 – 3.156	.480
Fam Hx	1.841	1.441	1.632	6.301	0.374 – 106.18	.201
ASRM	.399	.193	4.282	1.490	1.021 – 2.173	.039
BDI	.071	.096	0.542	1.073	0.889 – 1.295	.461
Step 2						
M-SRM Regularity	−.090	.252	0.127	0.914	0.558 – 1.497	.722

Note. Odds ratios (OR) less than 1.0 indicate a negative association between the predictor and bipolar spectrum disorder onset. CI = Confidence Interval; Age = Age at baseline; Fam Hx = Family History of bipolar disorder; ASRM = Altman Self-Rating Mania Scale scores; BDI = Beck Depression Inventory scores; M-SRM Regularity = Modified Social Rhythm Metric Regularity score.