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Life Events and Social Rhythms in Bipolar Spectrum Disorders: A Prospective Study

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This study examined the social zeitgeber theory, which suggests that affective symptoms are caused by life events disrupting vulnerable individuals' social and circadian rhythms. Undergraduate participants were selected based on a 2-phase screening process, including a semistructured diagnostic interview. The final sample consisted of 101 bipolar spectrum participants and 100 demographically matched normal controls. Participants who completed up to 3 follow-up visits, approximately every 4 months, as part of a longitudinal study were included in the current study. Life events did not predict social rhythm regularity and social rhythm regularity inconsistently predicted affective symptoms. However, life events, particularly social rhythm disruption (SRD) events, did predict depressive symptoms and episodes, and less consistently predicted hypo(manic) symptoms and episodes. Thus, the current study obtained mixed support for social zeitgeber theory.

DESPITE ITS PUBLIC HEALTH significance and high prevalence (4.4% of a nationally representative U.S. sample were affected by a bipolar spectrum disorder; Merikangas et al., 2007), bipolar I disorder is understudied compared to other mental health disorders (Hyman, 2000). Even fewer studies have focused on bipolar spectrum disorders (i.e., bipolar II disorder, cyclothymia). This is particularly surprising given that bipolar spectrum disorders are more prevalent than bipolar I disorder in the community (Merikangas et al., 2007). Moreover, 15% to 50% of cyclothymic individuals may subsequently develop bipolar I or II disorders (American Psychiatric Association [APA], 2000; Shen, Alloy, Abramson, & Grandin, in press). The primary aim of the current study was to test several hypotheses derived from the social zeitgeber theory of mood disorders to better understand the etiology of mood episodes, such as their underlying mechanisms and triggers, in individuals with bipolar spectrum disorders.

Evidence suggests that life events precede the onset of both depressive and hypomanic/ manic symptoms and episodes in individuals with bipolar disorder (see Alloy et al., 2005; Johnson, 2005; Johnson & Roberts, 1995; Paykel, 2001, for reviews). Ehlers, Frank, and Kupfer (1988) proposed the social zeitgeber theory to explain how life events may trigger unipolar depressive symptoms. This theory proposes that life events can disrupt social zeitgebers (i.e., external/social time cues that function to entrain circadian rhythms, such as people we engage in activities with, alarm clocks, etc.), which

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causes disruptions in the regularity of our daily activities (e.g., meal times, getting out of bed), as well as our circadian rhythms (e.g., sleep-wake cycle, body temperature, cortisol secretion), and as a result, causes depressive symptoms in vulnerable individuals (see Figure 1) (Ehlers et al., 1988).

This theory was, in part, derived from the substantial evidence that depressed individuals have irregular biological rhythms (e.g., Thase, Jindal, & Howland, 2002). Recent findings suggest that the theory may also apply to hypomanic and manic episodes, and thus, to bipolar disorder as well (for a review, see Grandin, Alloy, & Abramson, 2006). Specifically, Malkoff-Schwartz and colleagues (1998, 2000) reported that life events associated with social rhythm disruptions were more likely to be present prior to manic episodes than control periods in a bipolar I sample. Moreover, social rhythm irregularity predicted a shorter time to onset of major depressive and hypomanic or manic episodes in a prospective study (Shen et al., in press). Finally, Interpersonal and Social Rhythm Therapy (IPSRT), an intervention designed to maintain regular daily rhythms and manage potential precipitants of rhythm dysregulation, was shown to buffer against future bipolar episodes in a bipolar I sample (Frank et al., 2005).

This research suggests that individuals with mood disorders may exhibit less social rhythm regularity and that social rhythm disruptions may contribute to affective symptoms in these individuals. However, the findings to date are inconclusive with respect to the validity of the social zeitgeber model, due to limitations in the literature such as the use of cross-sectional designs, small sample sizes, and short assessment intervals (Grandin et al., 2006). Thus, the primary aim of the current study was to examine three of the hypothesized pathways of the social zeitgeber theory in a prospective, longitudinal, controlled study of a large sample of bipolar spectrum and demographically matched normal control individuals. Specifically, we hypothesized that: (1) life events, particularly social rhythm disruption (SRD) events, would predict participants' social rhythm regularity; (2) life events, particularly SRD events, would predict participants' affective symptoms; and (3) social rhythm irregularity would predict participants' affective symptoms (see Figure 1). In addition, we predicted that each of these relationships would be stronger in the bipolar spectrum group than the control group.

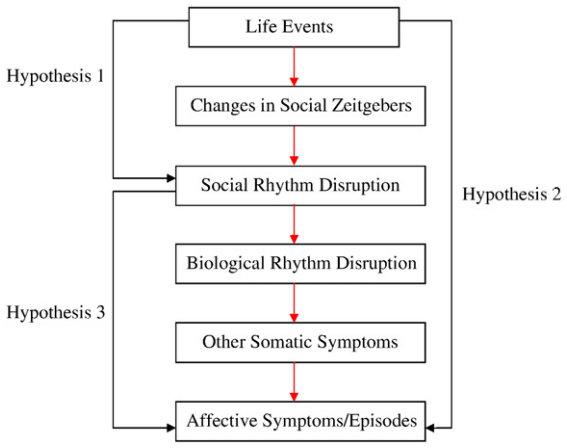

FIGURE 1 The Social Zeitgeber Theory and Main Hypotheses of the Current Study.

Methods

PARTICIPANTS

Participants for this study were recruited from the Temple University site of the Longitudinal Investigation of Bipolar Spectrum (LIBS) Project (Alloy et al., 2008; Nusslock et al., 2007; Shen et al., in press) and were selected based on a two-phase screening process. In Phase I, approximately 7,000 students at Temple University completed the revised General Behavior Inventory (GBI; Depue, Krauss, Spoont, & Arbisi, 1989). Students who met initial GBI screening criteria ($n = 2737$) were invited to participate in Phase II, which consisted of a semistructured diagnostic interview using an expanded version of the Schedule for Affective Disorders and Schizophrenia–Lifetime interview (SADS-L; Endicott & Spitzer, 1978). Students meeting *DSM-IV* (APA, 1994) criteria or Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) for bipolar II disorder or cyclothymia were invited to participate in the study.[1] These individuals were categorized as bipolar spectrum participants ($N = 137$). Individuals who did not meet criteria for any disorder in their lifetime were recruited as normal controls ($N = 127$). Normal control participants were matched at the baseline visit on a case-by-case basis to bipolar spectrum participants on age, sex, and ethnicity. We included participants in the current study who completed the baseline visit.

The final sample for this study consisted of 201 students (131 female, 70 male), 101 bipolar spectrum participants and 100 normal controls, ranging in age from 18 to 24 ($M = 19.8 \pm 1.8$ years). A higher proportion of females than males (65% and 35%, respectively) in this study is consistent with prior

[1] Students who already met criteria for bipolar I disorder were not invited to participate in the longitudinal study because one important goal of the larger LIBS Project was to predict conversion to bipolar I status among bipolar spectrum participants.

research indicating a preponderance of women in bipolar II samples (Amsterdam, Brunswick & O'Reardon, 2002; Cassano, Akiskal, Savino, Musetti, & Perugi, 1992; Depue et al., 1989), given that our sample of bipolar spectrum individuals received mostly bipolar II diagnoses (64 bipolar II, 37 cyclothymia). The ethnic composition of the final sample was diverse: 54% Caucasian, 25% African-American, 4% Hispanic, 5% Asian, 12% other.

MEASURES

Self-Report Screening Inventory (Phase I). The GBI (Depue et al., 1981, 1989) is a self-report questionnaire used here to identify potential bipolar spectrum and normal participants for Phase II of the study. The GBI has good internal consistency (α's = .90–.96), test-retest reliability (r's = .71–.74), adequate sensitivity (.78) and high specificity (.99) for bipolar spectrum conditions (Depue et al., 1981, 1989). The revised GBI (Depue et al., 1989) has 73 items, each of which captures either a depressive (D scale), hypo(manic) or biphasic (HB scale) symptom. The GBI has been extensively validated in college, psychiatric outpatient, and offspring of bipolar I patient samples (Depue et al., 1981, 1989). The LIBS project used the case-scoring method and cutoff scores recommended by Depue et al. (1989) to identify potential bipolar ($D \geq 11$; $HB \geq 13$) and normal ($D < 11$; $HB < 13$) participants. These criteria were based on Depue et al.'s (1989) findings and a pilot study in which high and low GBI classifications, using these cutoffs, were validated against diagnoses derived from SADS-L interviews (Alloy et al., 2008).

Diagnostic Screening Interview (Phase II). An expanded SADS-L interview (Endicott & Spitzer, 1978) was used to assess the occurrence, duration, and severity of symptoms of mood, anxiety, substance abuse, eating, psychotic, and other disorders over participants' lifetime. The expansion of the original SADS-L is described elsewhere (Alloy et al., 2008; Francis-Raniere, Alloy, & Abramson, 2006). This modified version of the SADS-L has yielded kappas $\geq .95$ for major depression diagnoses and $\geq .90$ for all unipolar depressive diagnoses based on 80 jointly rated interviews (Alloy & Abramson, 1999; Alloy et al., 2000). An interrater reliability study based on 105 jointly rated SADS-L interviews for the LIBS project yielded kappas $> .96$ for bipolar spectrum diagnoses (Alloy et al., 2008).

Prospective diagnostic measure. An expanded SADS–Change interview (exp-SADS-C; Spitzer & Endicott, 1978) assessed the presence or absence of affective episodes and symptoms throughout each 4-month prospective assessment. The exp-SADS-C was expanded to allow the derivation of *DSM-IV* as well as RDC diagnoses (Alloy et al., 2008; Francis-Raniere et al., 2006). In the present study, depressive episodes were included if they met criteria for either *DSM-IV* or RDC major depression or for RDC minor depression. Hypomanic episodes were included if they met criteria for either *DSM-IV* or RDC hypomanic episode. Similarly, episodes that met either *DSM-IV* or RDC criteria for mania were included as manic episode.[2] In addition, depressive and (hypo)manic symptoms from the exp-SADS-C were counted at each follow-up visit to yield a symptom count for each participant. Interrater reliability (Francis-Raniere et al., 2006) for the exp-SADS-C in joint ratings of 60 interviews from the LIBS Project was good ($k > .80$). In a validity study, participants dated their symptoms on the exp-SADS-C with at least 70% accuracy compared with daily symptom ratings made over a 4-month interval (Francis-Raniere et al., 2006).

Life events. The Life Event Scale (LES) used in the LIBS project is an expansion of the earlier 134-item LES (Alloy & Clements, 1992; Needles & Abramson, 1990) to include positive events as well as negative ones. The expanded LES contains 193 events and was designed to reduce ambiguous and redundant events as well as exclude items that reflected obvious symptoms of depression or (hypo) mania. The LES has good reliability and validity (Francis-Raniere et al., 2006; Metalsky & Joiner, 1992; Needles & Abramson, 1990; Safford, Alloy, Abramson, & Crossfield, 2007).

At each follow-up visit, after participants completed the LES, they were interviewed by a trained research assistant (who was blind to the participants' group status) as a reliability and validity check on the occurrence and dates of the events reported on the LES, as well as to provide objective ratings of the events. A recent study found that participants correctly recalled 100% of the major events using the LES followed by this Life Event Interview (LEI), when compared to a daily life event list created by the participant prospectively over a month (Alloy & Abramson, 1999). The interrater reliability of 40 LEIs yielded an average correlation of .89 between interviewers for the dating of these

[2] Although participants with a cyclothymia diagnosis had not exhibited any episodes of major depression in their lifetime prior to the start of the LIBS Project, some of these individuals did experience their first onset of major depression during the LIBS Project follow-up and thus, converted to bipolar II status. Similarly, some participants with either bipolar II or cyclothymia diagnoses at the outset of the LIBS Project developed a first onset manic episode during the follow-up period and, thus, converted to bipolar I status. Shen et al. (in press) present data on these conversion rates. Thus, we use the term "(hypo)manic" throughout the article to refer to episodes or symptoms of either hypomania or mania.

events (Francis-Raniere et al., 2006). Each event on the LES was assigned an *a priori*, objective impact rating (before the start of the LIBS Project), but these impact ratings could be adjusted by the interviewer based on the degree of contextual threat the event posed for that individual participant, similar to the Brown and Harris (1978) Life Events and Difficulties Schedule (LEDS). In making these ratings, the interviewer was trained to consider the context of the event, as well as individual differences among participants, in making the objective impact ratings. For example, the event impact of "fighting with my mother" can vary based on the intensity, frequency, duration, and content of the fight. Thus, the interviewer was trained to probe all events extensively to elucidate these details. The impact of events was rated by the interviewer on a 5-point scale from 0 (*no/slight impact*) to 4 (*extreme impact*), and these objective impact ratings were routinely discussed by LIBS Project interviewers as a group to obtain consensus ratings. Events associated with a moderate, major, or extreme negative impact for the participant as rated by the interviewer (a rating of a 2, 3 or 4) were classified as "negative events."

The LEI also assesses the extent to which a life event disrupted participants' daily routine. This rating scale ranges from 1 (*little to no effect*) to 4 (*marked effect*) and relies on the degree of disruption in the sleep-wake cycle (i.e., change in either the time at which the participant went to bed or woke up irrespective of the amount of actual sleep). It was modeled after the first standardized assessment of such social rhythm disruption (SRD) events (Frank, Malkoff-Schwartz, Sherrill, & Anderson, 1999). Events rated as SRD events had a score of a 2, 3, or 4 on this scale, similar to the guidelines used in other studies (Malkoff-Schwartz et al., 1998, 2000). A severe SRD event had a score of a 3 or 4 on this scale. The LEI also measures the total amount of sleep lost due to an event. This rating scale ranges from 1 (*little or no sleep loss*), or losing less than an hour of sleep, to 5 (*extreme sleep loss*), or losing ≥ 7 hours of sleep. Life events associated with at least an hour of sleep loss (a rating of a 2, 3, 4, or 5 on the LEI) were categorized as "sleep loss" events. Thus, whereas sleep loss events were events that led to an actual decrease in the amount of sleep, SRD events could be associated with a decrease, an increase, or no change in the number of hours of sleep. For example, an individual who usually goes to bed at midnight and wakes at 7 A.M., but due to a life event, goes to bed at 3 A.M. and wakes at 10 A.M. would be counted as having an SRD event, but not a sleep loss event.

Lifestyle regularity. The Social Rhythm Metric (SRM) (Monk, Flaherty, Frank, Hoskinson, & Kupfer, 1990) was designed to quantify participants' typical daily social rhythm patterns, and therefore, targets activities that only occur habitually or regularly. This measure consists of 17 daily activities, 15 specified (i.e., "Get out of Bed," "Have lunch," "Physical Exercise") and 2 individualized write-in items. The daily version of the SRM was found to be moderately consistent (i.e., $r = .44$) and valid (e.g., participants on vacation have considerably lower SRM scores) in a group of 50 healthy controls (Monk et al., 1990; Monk, Kupfer, Frank, & Ritenour, 1991). Other evidence for the validity of the SRM is derived from a study that found SRM scores are correlated positively with other indices of social rhythm stability (Monk, Petrie, Hayes, & Kupfer, 1994).

For this study, a modified version of the SRM (M-SRM) was used to assess the frequency with which participants performed activities at approximately the same time (±45 minutes) over each month of the follow-up period. A regular activity was defined as occurring within 45 min of the same time, every day. A score of 0 ("activity was performed regularly two or less times per week"), 1 ("activity was performed regularly three or more times per week"), or 2 ("activity was performed regularly every day per week") was assigned to each item of the M-SRM. An M-SRM score was calculated by averaging across the scores for each item of every month of the follow-up period. Thus, higher M-SRM scores represent a higher frequency of activities performed regularly. The M-SRM showed moderate consistency ($r = .61$–$.62$) across the 4-month follow-up periods in this study (Shen et al., in press).

Procedure

After the two-phase screening process, participants were invited back for follow-up visits every 4 months. Only the first three follow-up visits (i.e., F1, F2, F3) for each participant were included in this study. At each follow-up visit, participants completed the M-SRM and LES and were also interviewed with the exp-SADS-C and the LEI for each 4-month interval. These visits were conducted by two separate interviewers, one to administer the exp-SADS-C and the other to administer the LEI, in order to minimize potential interviewer bias. Participants were paid $50 for each follow-up visit, which took approximately 3 hours.

Results

PRELIMINARY ANALYSES

Sample Characteristics. The final sample included 100 normal controls and 64 individuals with bipolar II disorder and 37 individuals with cyclothymia. The control group met criteria for no

psychiatric diagnoses, other than 9 (8.2%) participants who had a lifetime diagnosis of a specific phobia. Lifetime prevalence of alcohol or drug abuse or dependence in the bipolar spectrum group was 32.4% (19.4% alcohol and 21.3% drug). Other lifetime cormorbid diagnoses in the bipolar group were: generalized anxiety disorder (10.2%), panic disorder (4.6%), specific phobia (18.2%), social phobia (13.9%), agoraphobia (5.6%), obsessive-compulsive disorder (6.5%), posttraumatic stress disorder (10.2%), any eating disorder (7.4%), and attention-deficit/hyperactivity disorder (6.5%).

Average age of onset for any depressive episode was 13.33 years old ($SD = 6.97$) and for hypomanic episode 12.53 years ($SD = 7.78$). Among individuals with a bipolar spectrum disorder, 30.8% experienced a major or minor depressive episode during the follow-up duration of this study ($M = 589.94$ days, $SD = 279.85$), and 17% experienced hypomanic or manic episodes. On average, the bipolar spectrum participants experienced 1.98 depressive episodes ($SD = 2.16$) over this study's duration and 2.69 hypomanic or manic episodes ($SD = 3.63$).

Group Equivalence. Demographic variables (age, sex, ethnicity) were examined to confirm the equivalence of the control and bipolar groups. The groups did not differ significantly on sex ($\chi^2_{1,\ N=201} = 0.06$, $p = .81$), age ($\chi^2_{8,\ N=201} = 4.47$, $p = .88$), or ethnicity coded as a dichotomous variable (i.e., non-White versus White; $\chi^2_{1,\ N=201} = 0.54$, $p = .97$). The means and standard deviations of all study variables are presented in Tables 1 and 2.

We conducted analyses of variance (ANOVAs) to determine whether the groups differed in each follow-up period on M-SRM scores as well as (hypo)manic (HYPO) and depressive (DEP) symptom scores obtained from the SADS-C interview. As expected, the bipolar group reported higher HYPO and DEP scores, but less social rhythm regularity, at each follow-up visit compared to the control group (see Table 1). We also conducted ANOVAs for each category of life events at each of the follow-up visits to determine potential differences between the groups (see Table 2). As expected, the bipolar group reported more total life events, as well as specific categories of life events (i.e., negative, SRD, sleep loss), than the control group at each of the follow-up visits with one exception. At F3, the two groups did not differ significantly in the total number of events experienced (see Table 2).

Table 2
Means and Standard Deviations of Life Event Categories by Group

Categories of Events	Control Group Mean (SD)	Bipolar Group Mean (SD)	F (df)
F1 Total	24.40 (13.96)	39.88 (26.26)	25.84 (1,190)**
F2 Total	20.89 (14.80)	27.24 (17.82)	6.61 (1,173)*
F3 Total	22.69 (20.94)	27.65 (25.31)	1.81 (1,157)
F1 Negative	7.17 (6.44)	19.67 (17.75)	42.22 (1,193)**
F2 Negative	6.32 (7.23)	12.31 (11.24)	17.64 (1,173)**
F3 Negative	4.90 (5.11)	11.45 (12.24)	18.84 (1,153)**
F1 SRD	2.34 (2.79)	7.86 (9.72)	28.34 (1,190)**
F2 SRD	2.66 (3.97)	4.64 (6.21)	6.36 (1,173)*
F3 SRD	2.12 (3.32)	5.17 (6.22)	14.45 (1,153)**
F1 Sleep Loss	1.95 (2.13)	7.11 (9.25)	28.15 (1,190)**
F2 Sleep Loss	1.76 (3.12)	3.44 (5.60)	6.21 (1,176)*
F3 Sleep Loss	1.14 (1.50)	4.35 (5.63)	23.32 (1,153)**

Note. Degrees of freedom (df) vary for each variable due to participant attrition and incomplete data. F1 = First Follow-up visit. F2 = Second Follow-up visit. F3 = Third Follow-up visit. SRD = Social Rhythm Disruption.
* $p < .05$.
** $p < .01$.

Table 1
Means and Standard Deviations of Symptoms and Social Rhythm Regularity by Group

	Control Group Mean (SD)	Bipolar Group Mean (SD)	F (df)
F1 HYPO Symptoms	0.26 (0.64)	1.58 (2.16)	32.99 (1,189)**
F2 HYPO Symptoms	0.23 (0.69)	1.34 (2.11)	22.71 (1,174)**
F3 HYPO Symptoms	0.28 (0.81)	1.21 (2.03)	14.45 (1,156)**
F1 DEP Symptoms	1.46 (1.89)	6.58 (4.72)	97.10 (1, 189)**
F2 DEP Symptoms	1.91 (2.67)	5.35 (4.37)	39.98 (1, 173)**
F3 DEP Symptoms	1.65 (1.93)	5.16 (4.62)	39.21 (1, 155)**
F1 M-SRM Score	19.67 (5.28)	17.77 (5.87)	5.05 (1,171)*
F2 M-SRM Score	19.87 (5.57)	17.88 (6.07)	4.11 (1,139)*
F3 M-SRM Score	20.19 (5.50)	17.94 (6.27)	4.63 (1,127)*

Note. Degrees of freedoms (df) vary for each variable due to participant attrition and incomplete data. F1 = First Follow-up visit. F2 = Second Follow-up visit. F3 = Third Follow-up visit. HYPO = Hypo(manic) symptoms from the SADS-C interview. DEP = Depressive symptoms from the SADS-C interview. M-SRM = Modified-Social Rhythm Metric.
* $p < .05$.
** $p < .01$.

Analyses of Demographic Variables and Study Variables of Interest. We conducted ANOVAs to determine whether any demographic characteristics were associated with any study variable over the follow-up period. Ethnicity was significantly associated with M-SRM scores at F1 ($F_{1,151} = 7.81$, $p = .01$), F2 ($F_{1,123} = 13.56$, $p < .01$), and F3 ($F_{1,111} = 7.42$, $p = .01$), such that the non-White group reported significantly higher scores than the White group. Thus, when M-SRM scores were used as the dependent variables in regression analyses, ethnicity was entered as a predictor. Age and gender were not significantly related to M-SRM, DEP, and HYPO scores at any of the follow-up visits (all p's $> .05$). We also found that total life events at F1 ($F_{1,186} = 5.23$, $p = .02$) and negative events at F1 ($F_{1,188} = 5.24$, $p = .02$) were significantly associated with age. Thus, when these variables were the dependent variables in regression analyses, age was entered as a predictor. Ethnicity and gender were not significantly related to any category of life events at any of the follow-up visits (all p's $> .05$).

MAIN ANALYSES

Hypothesis 1: Life Events and Social Rhythm Regularity. Linear regression analyses were conducted to examine whether the total number of events, as well as each category of life events (i.e., negative, SRD, sleep loss), predicted M-SRM scores (see Table 3). There was not a significant association of total number or any category of life events at the first follow-up visit (F1) with M-SRM scores at the second follow-up visit (F2) or of the total number or any category of events at F2 with M-SRM scores at F3 (see Table 3). Additionally, there were no significant Group × Event (i.e., total, negative, SRD, sleep loss) interactions for any of these analyses (all p's $> .05$). These findings suggest that life events did not significantly predict social rhythm regularity.

Table 3
Categories of Life Events Predicting to Subsequent Social Rhythm Scores

Categories of Events	Social Rhythm Scores		
	ΔR^2	B	*t* (df)
F2 Total	.01	–.02	–0.84 (131)
F3 Total	.01	–.05	–1.36 (117)
F2 Negative	.00	–.02	–0.38 (131)
F3 Negative	.02	–.10	–1.71 (116)
F2 Sleep Loss	.00	.08	0.71 (129)
F3 Sleep Loss	.00	–.11	–0.71 (117)
F2 SRD	.01	.11	1.13 (129)
F3 SRD	.01	–.13	–1.16 (117)

Note. Degrees of freedom (df) vary for each variable due to participant attrition and incomplete data. F1 = First Follow-up visit. F2 = Second Follow-up visit. F3 = Third Follow-up visit. SRD = Social Rhythm Disruption events. ΔR^2 = Change in the proportion of variance accounted for in the model by the main effect.

Table 4
Categories of Life Events Predicting to Depressive and (Hypo) manic Symptoms

Categories of Events	Depressive Symptoms			(Hypo) Manic Symptoms		
	ΔR^2	B	*t* (df)	ΔR^2	B	*t* (df)
F2 Total	.03	.03	2.45 (163)**	.00	.00	.19 (159)
F3 Total	.06	.06	3.50 (146)**	.04	.02	2.44 (148)*
F2 Negative	.08	.09	4.33 (165)**	.01	–.10	1.16 (161)
F3 Negative	.08	.12	4.14 (145)**	.05	.04	2.94 (145)**
F2 Sleep Loss	.07	.23	4.43 (163)**	.02	.05	1.81 (159)
F3 Sleep Loss	.05	.19	3.23 (146)**	.03	.06	2.11 (146)*
F2 SRD	.07	.19	3.88 (163)**	.01	.02	.94 (159)
F3 SRD	.05	.17	3.14 (147)**	.01	.03	1.27 (145)

Note. HYPO = Hypo(manic) symptoms from the SADS-C interview. F1 = First Follow-up visit. F2 = Second Follow-up visit. F3 = Third Follow-up visit. SRD = Social Rhythm Disruption events. ΔR^2 = Change in the proportion of variance accounted for in the model by the main effect.
* $p < .05$.
** $p < .01$.

Hypothesis 2: Life Events and Affective Symptoms/Episodes. We conducted five separate analyses to examine this hypothesis. Although each analysis was planned, to reduce the possibility of finding chance associations between events and symptoms/episodes, we used a Bonferroni corrected p-value of .01 (or .05/5). Linear regression analyses were conducted to examine whether the total number of events, as well as each category of life events (i.e., negative, SRD, sleep loss), significantly predicted subsequent bipolar symptoms. Each category of life events predicted DEP scores at the next follow-up, such that participants who reported more life events also had more subsequent depressive symptoms (see Table 4). In contrast, there was a less consistent predictive association of life events and HYPO scores. Specifically, there was only a significant predictive relationship of negative events at F2 with HYPO scores at F3 (see Table 4). There were also no significant Group × Event category interactions in predicting to DEP or HYPO scores, suggesting that the life event-depressive and hypo (manic) symptom relationships were consistent across groups (all p's $> .01$).

We also performed Cox regression survival analyses to examine the length of time to the bipolar spectrum individuals' first (hypo)manic and depressive episodes as a function of the number of life events reported prior to this episode. We entered group (bipolar II vs. cyclothymic), age, gender, and

ethnicity on the first step and the number of events prior to bipolar spectrum individuals' first episode (or the number of events over the study duration for bipolar spectrum participants who did not experience an episode) on the second step. We found that the total number of life events marginally predicted time to bipolar spectrum individuals' first depressive episode due to our Bonferroni corrected *p*-value, but not to their first (hypo)manic episode (see Table 5). Thus, there was a trend for bipolar spectrum participants who experienced more life events to have a shorter time to onset of their first depressive episode. Negative, SRD, and sleep loss events did not significantly predict time to depressive or (hypo)manic episodes (see Table 5).

Next, we conducted matched *t*-tests to compare the number of (hypo)manic and depressive symptoms before a SRD event versus after the SRD event. These analyses included 118 SRD events (29 reported by normal controls and 89 reported by bipolar spectrum participants). Bipolar spectrum participants reported significantly more depressive symptoms after than before a SRD event (t_{88}=-2.77, p<.01, d=.17), but not more (hypo)manic symptoms (t_{88}=-0.56, p>.01, d=.06). Participants in the control group reporting SRD events did not experience a significant change in their depressive symptoms or their (hypo)manic symptoms from before to after an SRD event (all p's>.01).

We also conducted matched *t*-tests to compare the number of life events prior to bipolar mood episodes to the number of events experienced during control periods for each bipolar participant that experienced an episode (within-subjects analyses). Control periods were episode-free periods that occurred 1 year after or 1 year before the bipolar mood episode to be as comparable as possible with respect to season of the year. If this was not possible (because the 1 year before or after the episode also contained an episode), then the control period was chosen to be an episode-free period either 4 weeks after the bipolar episode or 4 weeks prior to the 8-week period before the bipolar episode. Bipolar individuals who experienced a depressive episode during the study experienced more SRD, but not total and negative, events within the 8 weeks prior to onset of the depressive episode than during 8 week control periods (see Table 6a). With respect to (hypo)manic episodes, there were no significant differences between the pre-episode and control periods in the number of total life events, SRD, and negative events (see Table 6b).

Finally, we conducted matched *t*-tests to examine whether the number of life events reported prior to bipolar mood episodes differed significantly from the number of events reported by bipolar participants who did not experience a bipolar mood episode over the study duration (between-subjects analyses). Episodic (bipolar spectrum individuals who had an episode during the follow-up period) and nonepisodic (bipolar spectrum individuals who did not have an episode during the follow-up period) bipolar spectrum participants were matched based on gender, ethnicity, and age in that hierarchical order. For nonepisodic bipolar spectrum participants, we used the episode dates of their matched bipolar spectrum individual with an episode to achieve a "before episode" count of events. We found that episodic bipolar spectrum participants showed a trend to experience more SRD, but not more total or negative, events prior to depressive episodes than nonepisodic bipolar spectrum participants (see Table 7). However, this

Table 5
Cox Regression Survival Analyses Predicting Time to Affective Episodes as a Function of Life Events for Bipolar Spectrum Participants

Categories of Events	Time to Depressive Episode			Time to (Hypo) manic Episode		
	Exp(B)	Wald	*p*	Exp(B)	Wald	*p*
Total	.99	5.69	.02	1.00	2.24	.14
Negative	.99	1.91	.17	1.00	0.70	.40
SRD	.99	0.79	.38	.99	1.33	.25
Sleep Loss	1.00	0.04	.84	1.00	0.02	.90

Note. F1=First Follow-up visit. F2=Second Follow-up visit. F3=Third Follow-up visit. SRD=Social Rhythm Disruption.

Table 6

a. Matched *t*-Tests of Life Events prior to Depressive Episodes versus Non-episodic, Control Periods for Bipolar Spectrum Participants

Categories of Events	Control Period Mean (SD)	Depressive Period Mean (SD)	*t* (df)	*d*
Total Number	11.79 (8.63)	13.09 (8.97)	–0.65 (32)	.19
SRD	1.82 (1.76)	3.61 (3.39)	–3.42 (32)**	.59
Negative	6.00 (6.05)	7.27 (6.14)	–1.06 (32)	.26

b. Matched *t*-Tests of Life Events prior to (Hypo)manic Episodes versus Non-Episodic, Control Periods for Bipolar Spectrum Participants

Categories of Events	Control Period Mean (SD)	(Hypo)Manic Period Mean (SD)	*t* (df)	*d*
Total Number	15.06 (11.22)	12.12 (7.24)	0.90 (16)	.25
SRD	3.53 (3.26)	1.88 (1.58)	2.01 (16)	.49
Negative	8.06 (6.16)	5.35 (4.78)	1.43 (16)	.39

Note. df=Degrees of freedom. SD=Standard Deviation. SRD=Social Rhythm Disruption. *d*=Cohen's *d*, a measure of effect size.
** p<.01.

Table 7
Matched *t*-Tests of Life Events prior to Depressive Episodes for Episodic versus Non-episodic Bipolar Spectrum Participants

Categories of Events	Non-episodic Bipolars Mean (SD)	Episodic Bipolars Mean (SD)	*t* (df)	*d*
Total	13.39 (16.04)	12.77 (9.04)	0.18 (30)	.03
SRD	2.16 (3.02)	6.94 (5.78)	–3.91 (30)*	.70
Negative	7.19 (12.01)	3.52 (3.47)	1.61 (30)	.29

Note. df = Degrees of freedom vary as not every episodic bipolar was able to have a non-episodic bipolar match. SD = Standard Deviation. SRD = Social Rhythm Disruption. *d* = Cohen's *d*, a measure of effect size.
* $p < .05$.

relationship did not hold for the number of SRD events reported prior to hypo(manic) episodes ($t_{16} = 1.17$, $p = .26$, $d = .28$).

Hypothesis 3: Social Rhythm Regularity and Affective Symptoms/Episodes. Linear regression analyses were conducted to examine whether participants' M-SRM scores significantly predicted bipolar symptoms at the next follow-up. Less social rhythm regularity at F2 was associated with greater depressive symptoms at F3 ($R^2 = .257$, $t_{117} = -2.60$, $p < .01$); however, this relationship did not hold for M-SRM scores at F1 and DEP scores at F2. There were no significant relationships of social rhythm regularity (M-SRM scores) and subsequent HYPO scores (all *p*'s > .05). There were also no Group × M-SRM score interactions in predicting bipolar symptoms (all *p*'s > .05).

We also conducted Cox regression survival analyses to examine the length of time to the bipolar spectrum participants' first (hypo)manic and depressive episodes in the study as a function of their M-SRM scores over the study duration. We entered group, age, gender, and ethnicity on the first step and M-SRM scores on the next step. Participants' social rhythm regularity did not significantly predict the time to first depressive episode (Exp[B] = .99, *Wald* = 0.61, $p = .43$) or first (hypo)manic episode (Exp[B] = .99, *Wald* = 2.44, $p = .12$). Thus, retrospective report of social rhythms did not predict the time to bipolar spectrum participants' affective episodes.

Discussion

The primary goal of this study was to examine social zeitgeber theory (Ehlers et al., 1988; Ehlers, Kupfer, Frank, & Monk, 1993) as a potential explanation for affective symptoms and episodes in individuals with bipolar spectrum disorders. Specifically, we investigated three causal associations postulated by this theory: (1) whether the occurrence of life events, and particular types of events, predict social rhythm regularity; (2) whether life events were associated with, and temporally predicted, affective symptoms and episodes; and (3) whether social rhythm regularity predicted affective symptoms and episodes (see Figure 1). Overall, we obtained mixed support for the theory.

Contrary to social zeitgeber theory, the present findings suggested that life events did not predict an individual's subsequent social rhythm regularity (Hypothesis 1). These findings may be limited in that they relied on retrospective report of social rhythm regularity. The use of objective measures of social rhythm regularity (i.e., actigraphy, a measure of physical activity) or daily assessments of rhythms would have been preferable but difficult to do in a long-term longitudinal study such as this (Jones, Hare, & Evershed, 2005). Further, given that this is the first study to examine this association in a bipolar sample (Grandin et al., 2006), there is no empirical context in which to evaluate these results. However, one study found that bipolar spectrum participants rated life events as social rhythm disruptive and most (83%) of these events were rated as disruptive by the study investigators as well (Malkoff-Schwartz et al., 1998). These data suggest that objective raters found bipolar individuals' life events to be strongly linked to social rhythm disruptions.

Consistent with social zeitgeber theory, life events prospectively predicted bipolar symptoms and mood episodes as well as showed a trend ($p < .05$) to predict the time to onset of first depressive episodes (Hypothesis 2). Moreover, there was a significant increase in depressive symptoms from before to after an SRD event for both normal controls and bipolar spectrum individuals. However, it should be noted that this relationship had a small effect size, suggesting that only certain SRD events may trigger depressive symptoms. Yet, bipolar spectrum individuals also experienced more SRD events (but not more total or negative events) before depressive episodes compared to equivalent control periods. Further, bipolar spectrum individuals who had a depressive episode onset experienced more SRD events (but again, not more total or negative events) before their depressive episodes as compared to other bipolar spectrum individuals who did not have a depressive episode onset. These findings suggest that SRD events may have more of an impact than other negative life events on depressive symptoms in individuals with bipolar spectrum conditions. Other studies have concluded that manic episodes were particularly likely to be precipitated by SRD events (Malkoff-Schwartz et al., 1998, 2000), but the findings from this study suggest that bipolar spectrum individuals' depressive symptoms and episodes may also be particularly vulnerable to, or perhaps triggered by, SRD events.

Unfortunately, these relationships with SRD events did not hold for (hypo)manic symptoms and episodes in the present study (Hypothesis 2). The failure to replicate previous findings that SRD events predict (hypo)manic symptoms and episodes (Malkoff-Schwartz et al., 1998, 2000) may be due to low variability of hypomanic symptoms in the current study. Specifically, the bipolar spectrum group in the current study only experienced an average of 1 to 2 (hypo)manic symptoms in each follow-up period, which may not have allowed for enough power to detect significant associations (see Table 2). This explanation seems particularly likely given that many of the nonsignificant findings involving hypo(manic) symptoms were in the expected direction. Alternatively, the association of (hypo)manic symptoms and life events has only been consistently documented in samples with severe forms of bipolar disorder (i.e., bipolar I disorder with full-blown mania; Malkoff-Schwartz et al., 1998, 2000). Thus, perhaps individuals with less severe forms of mania (i.e., bipolar II disorder, cyclothymia) are less vulnerable to socially disruptive life stress on hypomanic symptoms. Similarly, depressive episodes in less severe bipolar individuals may also be different than those of bipolar I individuals, in that they are less likely to be "mixed" with hypomanic symptoms, but more similar to those of unipolar major depressed individuals. Indeed, consistent with the present study's findings, Haynes, McQuaid, Ancoli-Israel, and Martin (2006) also found that individuals with unipolar major depression were more susceptible to the effects of SRD events on sleep-wake rhythms than were normal controls. This may explain Malkoff-Schwartz et al.'s (1998, 2000) finding of lack of association between social rhythms and depressive episodes in bipolar I individuals. Finally, our findings may also be different than those of Malkoff-Schwartz et al. because the current sample was a nonclinical sample of bipolar individuals enrolled in college, suggesting a high degree of functioning, and the mean age of our sample was young.

Unexpectedly, we did *not* find that lower social rhythm regularity predicted increased depressive or (hypo)manic symptoms, nor the time to onset of bipolar spectrum participants' first depressive or (hypo)manic episode (Hypothesis 3). These null findings are particularly surprising, as a recent study found that participants' traitlike social rhythm regularity scores (assessed at baseline) significantly predicted time to their first depressive and (hypo) manic episode onset (Shen et al., in press). The contradiction in these findings may be because the current study had a 50% smaller sample size and, thus, much less power to observe this association. It is also possible that the retrospective self-report of social rhythms over long time intervals in the current study limited the results. For example, a *post hoc* analysis of our data showed that the bipolar spectrum individuals reported nearly twice as many SRD and sleep loss events (18.30% and 15.44% of their total number of life events, respectively) than the normal controls (10.44% and 7.58% of their total number of life events, respectively) over the study duration. These data suggest a relationship between social rhythm disruptions and affective symptoms and indicate that self-reported social rhythms may be biased compared to interview based scoring of social rhythm disruption events. Further, although several studies, including this one, found that bipolar spectrum individuals tend to have low social rhythm scores compared to normal controls (i.e., Ashman et al., 1999; Jones, 2004; Szuba, Yager, Guze, Allen, & Baxter, 1992), this is the first study to prospectively assess the association between social rhythms and affective symptoms in a bipolar spectrum sample.

Limitations of this study also should be noted. The most important limitation is that our measure of social rhythms (M-SRM) required retrospective report and, thus, it needs to be further validated against daily reports of social rhythms. However, the fact that the bipolar spectrum group exhibited less regular social rhythms than the normal control group on the M-SRM at each follow-up in this study and that M-SRM scores predicted time to onset of depressive and (hypo)manic episodes in the Shen et al. (in press) study suggest that it does have validity. Second, the M-SRM may have been subject to reporting biases associated with depressive or (hypo)manic symptoms. Perhaps bipolar spectrum participants tend to report less regular activities than normal participants. However, one would expect this bias to be reflected in the individualized write-in items. Shen et al. (in press) showed that the number of regular write-in items reported by the two groups did not differ significantly. In addition, there were no group differences in the frequency of activities reported as regular. Therefore, it is unlikely that reporting biases associated with bipolar symptoms contributed to the present findings. We also were not able to control for baseline symptoms in our follow-up analyses. Finally, our bipolar spectrum participants were a young, relatively high functioning, nonclinical sample enrolled in college. Thus, the generalizability of the present findings to other bipolar samples remains to be determined.

The current study yields mixed support for social zeitgeber theory. There was a consistent prospective association of life events and mood symptoms and

episodes. Specifically, there seemed to be a unique relationship between SRD events and depressive symptoms and episodes. However, there was not a consistent association between life events and social rhythm scores or between social rhythm scores and mood symptoms and episodes. Several explanations were proposed to explain these null findings. Yet, the findings with SRD events, such as the increase of depressive symptoms from before to after an SRD event, offer quite promising support for social zeitgeber theory. Thus, we believe that this evaluation of social zeitgeber theory highlights the importance of continuing research in this area. In particular, additional prospective, longitudinal studies are needed to further test the causal pathways hypothesized in social zeitgeber theory, but such studies would benefit from using measures of social rhythms that do not require retrospective recall (e.g., daily assessment of social rhythms).

Future research would also benefit from better understanding whether certain life events that disrupt social rhythms are more robust triggers of mood episodes. Additionally, such events could vary based on specific, individual differences. Developing a better understanding of the mechanisms and specificity of life events as triggers for individuals with bipolar disorder could be instrumental in buffering against and/or preventing opposed to against future mood episodes. However, the current findings do highlight the importance of events that change individuals' social routines, more so than events that are perceived to be negative. Therefore, disruptive events and changes in the daily schedules of individuals with bipolar disorder should be carefully monitored and other preventative strategies (i.e., compensating to ensure little or no sleep loss, avoiding stressful triggers/situations, utilizing self-soothing or distress tolerance skills) should be employed to maintain mood stability. In short, the current study suggests that maintaining a regular, daily schedule may be beneficial in reducing the depressive symptoms experienced by individuals with bipolar disorder.

References

Alloy, L. B., & Abramson, L. Y. (1999). The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Conceptual background, design, and methods. *Journal of Cognitive Psychotherapy: An International Quarterly*, *13*, 227–262.

Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., Kim, R. S., & Lapkin, J. B. (2000). 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*, *109*, 403–418.

Alloy, L. B., Abramson, L. Y., Urosevic, S., Walshaw, P. D., Nusslock, R., & Neeren, A. M. (2005). The psychosocial context of bipolar disorder: Environmental, cognitive, and developmental risk factors. *Clinical Psychology Review*, *25*, 1043–1075.

Alloy, L. B., & Clements, C. M. (1992). Illusion of control: Invulnerability to negative affect and depressive symptoms after laboratory and natural stressors. *Journal of Abnormal Psychology*, *101*, 234–245.

Alloy, L. B., Abramson, L. Y., Walshaw, P. D., Cogswell, A., Grandin, L. D., Hughes, M. E., Iacoviello, B. M., Whitehouse, W. G., Urosevic, S., Nusslock, R., & Hogan, M. E. (2008). Behavioral Approach System (BAS) and Behavioral Inhibition System (BIS) sensitivities and bipolar spectrum disorders: Prospective prediction of bipolar mood episodes. *Bipolar Disorders*, *10*, 310–322.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed., rev.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th Ed.-Text Rev.). Washington, DC: Author.

Amsterdam, J. D., Brunswick, D. J., & O'Reardon, J. (2002). Bipolar disorder in women. *Psychiatric Annals*, *32*, 397–404.

Ashman, S. B., Monk, T. H., Kupfer, D. J., Clark, A. H., Myers, F. S., Frank, E., & Leibenluft, E. (1999). Relationship between social rhythms and mood in patients with rapid cycling bipolar disorder. *Psychiatry Research*, *86*, 1–8.

Brown, G. W., & Harris, T. O. (1978). Social origins of depression: A study of psychiatric disorder in women. New York: Free Press.

Cassano, G. B., Akiskal, H. S., Savino, M., Musetti, L., & Perugi, G. (1992). Proposed subtypes of Bipolar II and related disorders: With hypomanic episodes (or cyclothymia) and with hyperthymic temperament. *Journal of Affective Disorders*, *26*, 127–140.

Depue, R. A., Krauss, S., Spoont, M. R., & Arbisi, P. (1989). General Behavior Inventory identification of unipolar and bipolar affective conditions in a nonclinical university population. *Journal of Abnormal Psychology*, *98*, 117–126.

Depue, R. A., Slater, J., Wolfstetter-Kausch, H., Klein, D., Goplerud, E., & Farr, D. (1981). A behavioral paradigm for identifying persons at risk for bipolar depressive disorder: A conceptual framework and five validation studies (Monograph). *Journal of Abnormal Psychology*, *90*, 381–437.

Ehlers, C. L., Frank, E., & Kupfer, D. J. (1988). Social zeitgebers and biological rhythms. *Archives of General Psychiatry*, *45*, 948–952.

Ehlers, C. L., Kupfer, D. J., Frank, E., & Monk, T. H. (1993). Biological rhythms and depression: The role of zeitgebers and zeitstörers. *Depression*, *1*, 285–293.

Endicott, J., & Spitzer, R. A. (1978). A Diagnostic Interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, *35*, 837–844.

Francis-Raniere, E. L., Alloy, L. B., & Abramson, L. Y. (2006). Depressive personality styles and bipolar spectrum disorders: Prospective tests of the event congruency hypothesis. *Bipolar Disorders*, *8*, 382–399.

Frank, E., Kupfer, D. J., Thase, M. E., Mallinger, A. G., Swartz, H. A., Fagiolini, A. M., Grochocinski, V., Houck, P., Scott, J., Thompson, W., & Monk, T. (2005). Two-year outcomes for interpersonal and social rhythm therapy in individuals with bipolar I disorder. *Archives of General Psychiatry*, *62*, 996–1004.

Frank, E., Malkoff-Schwartz, S., Sherrill, J. T., & Anderson, B. (1999). *Social Rhythm Disruption (SRD) criteria*. Pittsburgh: Western Psychiatric Institute and Clinic.

Grandin, L. D., Alloy, L. B., & Abramson, L. Y. (2006). The social zeitgeber theory, circadian rhythms, and mood disorders: Review and evaluation. *Clinical Psychology Review*, *26*, 679–694.

Haynes, P. L., McQuaid, J. R., Ancoli-Israel, S., & Martin, J. L. (2006). Disrupting life events and the sleep-wake cycle in depression. *Psychological Medicine*, *36*, 1363–1373.

Hyman, S. E. (2000). Goals for research on bipolar disorder: the view from NIMH. *Biological Psychiatry*, *48*(6), 436–441.

Johnson, S. L. (2005). Life events in bipolar disorder: Towards more specific models. *Clinical Psychology Review*, *25*, 1008–1027.

Johnson, S. L., & Roberts, J. E. (1995). Life events and bipolar disorder: Implications from biological theories. *Psychological Bulletin*, *117*, 434–449.

Jones, S. E. (2004). Psychotherapy of bipolar disorder: a review. *Journal of Affective Disorders*, *80*, 101–114.

Jones, S. E., Hare, D. J., & Evershed, K. (2005). Actigraphic assessment if circadian activity and sleep patterns in bipolar disorder. *Bipolar Disorders*, *7*, 1–11.

Malkoff-Schwartz, S., Frank, E., Anderson, B., Sherril, J. T., Siegel, L., Patterson, D., & Kupfer, D. J. (1998). Stressful life events and social rhythm disruption in the onset of manic and depressive bipolar episode. *Archives of General Psychiatry*, *55*, 702–707.

Malkoff-Schwartz, S., Frank, E., Anderson, B. P., Hlastala, S. A., Luther, J. F., Sherril, J. T., Houck, P. R., & Kupfer, D. J. (2000). Social rhythm disruption and stressful life events in the onset of bipolar and unipolar episodes. *Psychological Medicine*, *30*, 1005–1016.

Merikangas, K. R., Ames, M., Cui, L., Stang, P. E., Ustun, T. B., Von Korff, M., & Kessler, R. C. (2007). The impact of comorbidity of mental and physical conditions on role disability in the US adult household population. *Archives of General Psychiatry*, *64*, 1180–1188.

Metalsky, G. I., & Joiner, T. E. (1992). Vulnerability to depressive symptomatology: A prospective test of the diathesis-stress and casual mediation components of the hopelessness theory of depression. *Journal of Personality and Social Psychology*, *63*, 667–675.

Monk, T. H., Flaherty, J. F., Frank, E., Hoskinson, K., & Kupfer, D. J. (1990). The Social Rhythm Metric: An instrument to quantify the daily rhythms of life. *Journal of Nervous and Mental Disease*, *178*, 120–126.

Monk, T. H., Kupfer, D. J., Frank, E., & Ritenour, A. M. (1991). The Social Rhythm Metric (M-SRM): Measuring daily social rhythms over 12 weeks. *Psychiatry Research*, *36*, 195–207.

Monk, T. H., Petrie, S. R., Hayes, A. J., & Kupfer, D. J. (1994). Regularity of daily life in relation to personality, age, gender, sleep quality, and circadian rhythms. *Journal of Sleep Research*, *3*, 196–205.

Needles, D. J., & Abramson, L. Y. (1990). Positive life events, attributional style, and hopelessness: Testing a model of recovery from depression. *Journal of Abnormal Psychology*, *99*, 156–165.

Nusslock, R., Abramson, L. Y., Harmon-Jones, E., Alloy, L. B., & Hogan, M. E. (2007). A goal-striving life event and the onset of bipolar episodes: Perspective from the Behavioral Approach System (BAS) dysregulation theory. *Journal of Abnormal Psychology*, *116*, 105–115.

Paykel, E. S. (2001). The evolution of life events research in psychiatry. *Journal of Affective Disorders*, *62*, 141–149.

Safford, S. M., Alloy, L. B., Abramson, L. Y., & Crossfield, A. G. (2007). Negative cognitive style as a predictor of negative life events in depression-prone individuals: A test of the stress-generation hypothesis. *Journal of Affective Disorders*, *99*, 147–154.

Shen, G. C., Alloy, L. B., Abramson, L. Y., & Sylvia, L. G. (2008). Social rhythm regularity and the onset of affective episodes in bipolar spectrum individuals. *Bipolar Disorders*, *10*, 520–529.

Spitzer, R. A., & Endicott, J. (1978). *Schedule for Affective Disorders and Schizophrenia – Change version*. Biometrics Research, New York State Psychiatric Institute.

Spitzer, R. L., Endicott, J., & Robins, E. (1978). Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry*, *35*, 773–782.

Szuba, M. P., Yager, A., Guze, B. H., Allen, E. M., & Baxter, L. R. (1992). Disruption of social circadian rhythms in major depression: A preliminary report. *Psychiatry Research*, *42*, 221–230.

Thase, M. E., Jindal, R., & Howland, R. H. (2002). Biological aspects of depression. In I. H. Gotlib, & C. L. Hammen (Eds.), *Handbook of depression* (pp. 192–218). New York: Guilford Press.

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