
Lifestyle Regularity and Cyclothymic Symptomatology



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The social zeitgeber theory emphasizes the importance that social rhythm regularity may play in promoting internal synchronization of circadian rhythms in individuals with or at risk for bipolar spectrum disorders. This study examined the relationship of lifestyle regularity, affective symptomatology, and sleep in 71 individuals exhibiting cyclothymic mood and behavior patterns. Participants were randomly assigned to either an experimental group in which they were encouraged to regulate their daily routines or to a control group. Participants in the experimental group were able to successfully regulate their daily schedules. Although relationships between regularity and severity of depressive symptoms, across-day variances in mood and behavior, and sleep duration were identified during baseline, increased lifestyle regularity did not differentially result in changes in these variables. © 2008 Wiley Periodicals, Inc. *J Clin Psychol* 64: 482–500, 2008.

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Introduction

Bipolar disorder has been ranked the sixth leading cause of disability among 15- to 44-year-olds worldwide (Murray & Lopez, 1996). Despite its high prevalence (4.4% of a representative U.S. sample, Merikangas et al., 2007) and associated disability (Calabrese et al., 2003), bipolar disorder has been understudied relative to other mental health disorders (Hyman, 2000).

Cyclothymia is widely considered a mild, subthreshold form of bipolar disorder. Lifetime prevalence rates of subthreshold bipolar disorder were recently reported to be 2.4% (Merikangas et al., 2007). However, higher rates of cyclothymia have been reported in mental health clinics (3–9%) and among college students (6%; Akiskal, Djenderedijan, Rosenthal, & Khani, 1977; Depue et al., 1981; Kraepelin, 1976). Cyclothymic symptoms typically are chronic and characterized by fluctuating mood

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disturbances involving periods of hypomanic symptoms alternating with periods of depressive symptoms. Individuals with cyclothymic symptoms exhibit phenomenology, longitudinal course, family history, and treatment response consistent with belonging in the bipolar spectrum, and 15–50% may go on to develop bipolar I and II disorders (Akiskal et al., 1977; Cassano et al., 1999; Depue et al., 1981; Howland & Thase, 1993; Klein, Depue, & Slater, 1986; Shen, Alloy, Abramson, & Grandin, 2008).

Although the relationship between cyclothymic symptoms and bipolar disorder has been established, the mechanisms by which individuals with cyclothymia progress to more severe bipolar disorder are poorly understood. Several mechanisms explaining the onset of bipolar episodes have been proposed (see Johnson & Roberts, 1995). Among these, the social zeitgeber theory proposes that depressive episodes may be triggered by life events that disrupt social rhythms (e.g., the time one gets up or goes to bed or eats meals, etc.) that, in turn, derail physiological homeostasis or circadian rhythm regularity (Ehlers, Frank, & Kupfer, 1988; Ehlers, Kupfer, Frank, & Monk, 1993; Howland & Thase, 1999). This theory is supported by evidence suggesting that individuals diagnosed with affective disorders have dysregulated social rhythms and circadian physiology (i.e., body temperature, hormone levels, sleep–wake cycle; Lenox, Gould, & Manji, 2002; Malkoff-Schwartz et al., 1998, 2000; Shen et al., 2008; Wever, 1979; for a review see Grandin, Alloy, & Abramson, 2006).

The term “zeitgeber” is used to describe the environmental cues that entrain circadian rhythms (Wever, 1979). More specifically, social zeitgebers (e.g., meals, meetings, exercise) have been found to play an important role in synchronizing rhythms as suggested by the social zeitgeber theory (Ehlers et al., 1988). For example, studies have linked bereavement of a spouse (loss of a social zeitgeber) to depressive symptoms and the onset of mania (Ambelas, 1979; Flaherty, Frank, Hoskinson, Richman, & Kupfer, 1987). However, it is important to note that how an individual perceives the event, and/or the severity of the event, also impacts the degree to which an event may cause social rhythm disruption (Healy & Williams, 1989; Jones, 2001; Jones, Mansell, & Waller, 2006). Other studies have shown that individuals diagnosed with bipolar disorder display social rhythm and sleep irregularities (Ashman et al., 1999; Chang, Alloy, & Abramson, 2003; Harvey, Schmidt, Scarnà, Semler, & Goodwin, 2005; Riemann, Volderholzer, & Berger, 2002; Wehr et al., 1985). Further, evidence suggests that these irregularities, and particularly shifts in one’s sleep–wake cycle, may predict bipolar episodes (Riemann et al., 2002). For example, a recent study found that deficits in sleep predicted depressive, but not manic, symptoms in a bipolar sample (Perlman, Johnson, & Mellman, 2006). Another study found that wake onset time was a robust predictor of (hypo)manic and depressive symptoms (Leibenluft, Albert, Rosenthal, & Wehr, 1996). Additionally, studies have found that participants with bipolar disorder, currently in a manic episode, experienced more life events associated with social rhythm disruptions during pre-onset episode periods than during control periods (Malkoff-Schwartz et al., 1998, 2000). Taken together, this evidence suggests that regulating disrupted circadian rhythms may promote internal synchronization of these rhythms, particularly in vulnerable individuals (Howland & Thase, 1999). Thus, individuals keeping regular sleep, meal, and exercise schedules may be able to invoke artificial control over desynchronization of their biological rhythms.

Interpersonal Social Rhythm Therapy (IPSRT) was designed to explore this potential implication of the social zeitgeber theory (Frank, Schwartz, & Kupfer,

2000). IPSRT has been shown to increase the stability of daily routines in patients with bipolar disorder over time as well as the duration of time that these individuals are episode-free in comparison with individuals in a control group (Frank et al., 1997, 2000, 2005). Other recent data suggest that greater social rhythm regularity is predictive prospectively of a lower likelihood of onset of affective episodes among bipolar spectrum individuals (Chang et al., 2003; Shen et al., 2008). Thus, regularity in daily routines may prevent bipolar episodes and symptoms.

This study investigates lifestyle regularity in young adults with cyclothymic symptoms at behavioral risk for bipolar I and II disorders. We examined the social rhythms, sleep patterns, and affective symptoms of individuals self-reporting cyclothymic symptomatology on a continuous measure of mood variability. The specific aims of this study were three-fold. First, we examined whether lifestyle regularity is related to affective symptoms in young adults reporting cyclothymic symptoms. Second, we tested whether an experimental manipulation that increased lifestyle regularity reduces affective lability in these behaviorally at-risk individuals. Finally, we assessed how sleep patterns are related to affective symptoms.

Methods

Participants

The General Behavior Inventory (GBI; Depue et al., 1981) was administered to 1,044 undergraduates at Temple University. Participants scoring above the established criteria for cyclothymia on the depressive and hypomanic/biphasic subscales of the GBI (see the "Measures" section) were invited to participate in the study. Of those screened, 128 (12.26%) met cutoffs for the study and 71 students (51 female, 20 male) agreed to participate. There were no significant differences on demographics or GBI scores between the individuals who met the study criteria but did not participate and those who did participate. The age of participants ranged from 18 to 24 ($M = 19.70$), which is ideal given that this age range is after the first peak in rates of onset of bipolar spectrum disorders (ages 15–19; e.g., Kennedy et al., 2005; Kupfer et al., 2002; Weissman et al., 1996), but close to the mean onset age of bipolar I and II disorders (18.2–20.3 years; Merikangas et al., 2007). In addition, college students are an ideal group for better understanding the impact of lifestyle regularity on affective symptoms given the likelihood of chaotic daily schedules in college (Machado, Varella, & Andrade, 1998). The ethnic composition of the final sample was diverse: 71.8% Caucasian, 11.3% African American, 1.4% Hispanic, 7.0% Asian, and 8.5% Other.

Measures

General Behavior Inventory (GBI). Potential participants were screened using the GBI, a self-report inventory designed to screen for individuals potentially in the bipolar spectrum (Depue et al., 1981). The GBI is a continuous scale of mood variability that contains 73 items that map onto one of two subscales: depression (D) or hypomania plus biphasic (HB). Based on cutoffs established in the literature, individuals with a GBI-HB score ≥ 13 and a GBI-D score ≥ 11 were invited to participate in the study (Depue, Krauss, Spont, & Arbisi, 1989; Francis-Raniere, Alloy, & Abramson, 2006). The GBI has good internal consistency ($\alpha s = .90-.96$), test-retest reliability ($r_s = .71-.74$), adequate sensitivity (.78), and high specificity (.99; Depue et al., 1981, 1989). In addition, it has been extensively validated in college, psychiatric outpatient, and offspring of patients with bipolar I disorder

samples (Depue et al., 1981, 1989; Klein et al., 1986; Mallon, Klein, Bornstein, & Slater, 1986; Reichart et al., 2004).

Beck Depression Inventory (BDI). The BDI (Beck, Rush, Shaw, & Emery, 1979) is a 21-item self-report inventory that assesses the presence and severity of affective, cognitive, motivational, and somatic symptoms of depression. It has high internal consistency, test–retest reliability, and validity with both psychiatric and nonclinical samples (Beck, Steer, & Garbin, 1988). The BDI was administered weekly and reflects an individual's symptoms of depression over the past week.

Halberstadt Mania Inventory (HMI). The HMI (Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999; Francis-Raniere et al., 2006) is a 28-item self-report inventory designed to measure the severity of current (hypo)manic symptoms and is administered and scored similarly to the BDI. The HMI assesses the presence and severity of affective, cognitive, motivational, and somatic symptoms of (hypo)mania. Halberstadt and Abramson (2008) tested the psychometric properties of the HMI in a sample of 1,282 undergraduates. They found that it had high internal consistency ($\alpha = .82$), adequate convergent validity ($r = .32, p < .001$) with the mania scale of the Minnesota Multiphasic Personality Inventory; Hathaway & McKinley, 1951) as well as discriminant validity ($r = -.26, p < .001$ with the depression scale of the Minnesota Multiphasic Personality Inventory and $r = -.12, p < .001$ with the BDI). The HMI also correlated ($r = .46$) with hypomanic symptoms rated from a structured diagnostic interview (Alloy et al., 2008). Finally, the HMI shows expected changes as cyclothymic individuals' cycle through hypomanic, euthymic, and depressed mood states (Alloy et al., 1999). The HMI was administered weekly and reflects an individual's symptoms of (hypo) mania over the past week. To avoid redundancy with the sleep items on the Modified Social Rhythm Metric (M-SRM; below), BDI and HMI scores were calculated excluding the sleep items.

Modified Social Rhythm Metric (M-SRM). The M-SRM is the Social Rhythm Metric (SRM; Monk, Flaherty, Frank, Hoskinson, & Kupfer, 1990) with additional sleep-related items from the Pittsburgh Sleep Diary (Monk, Petrie, Hayes, & Kupfer, 1994). Utilization of select components of the Pittsburgh Sleep Diary to assess sleep duration (SLP) and sleep quality, in conjunction with the SRM, has been carried out by the measure's designer in previous research studies (Monk et al., 1994). The M-SRM was administered daily over a 6-week period (2 weeks before and 4 weeks after the lifestyle regularity manipulation for the experimental group).

The self-report M-SRM quantifies an individual's typical daily social rhythm pattern by capturing the timing and frequency of 15 specific activities and events (i.e., get out of bed, have a morning beverage, exercise, eat breakfast/lunch/dinner, go to bed), and 2 write-in activities. To minimize inter-individual reporting variability, only the original 15 specified activities were used for analysis. Data for each week are analyzed as a unit so that 2 weeks of data are collected and averaged together to obtain an individual's "trait" score (Monk, Kupfer, Frank, & Ritenour, 1991). One index score is the M-SRM score (M-SRM-S), which provides a *frequency* measure of one's activities. More specifically, the M-SRM-S is calculated by adding the number of times per week that an individual performs "frequent" activities divided by the total number (frequent and nonfrequent) of activities engaged in that week. A "frequent" activity must be completed at least three times in a week. This calculation of the M-SRM-S follows the algorithm established by its designers (Monk et al., 1990, 1991).

A second regularity index (REG) was also calculated. REG is derived as a *count* of activities (0–15) that were performed regularly over the course of the week or the number of regular activities each week. A regularly performed activity was one completed ≥ 3 times/week within a 45 minutes window of that activity's average performance time. For example, if a person woke up between 7:45 a.m. and 8:30 a.m. on at least 3 days in 1 week, then this would be counted as a regular activity. The M-SRM has been found to be moderately consistent (i.e., $r = .44$, $p < .001$, between SRM scores in weeks 1 and 2) and valid (Monk et al., 1990, 1991). This measure was used to assess participants' baseline social rhythm regularity and to detect changes in social rhythm patterns after the intervention.

Modified Inventory for Behavioral Variation (MIBV). The Inventory for Behavioral Variation (IBV; Depue et al., 1981; Goplerud & Depue, 1985), based on the Personal Feelings Scale (Wessman & Ricks, 1966), assesses fluctuations in mood and behavior over time. This measure has proven to be reliable and sensitive to within-day and between-day symptom changes (Alloy, Just, & Panzarella, 1997; Goplerud & Depue, 1985). For this study, some bipolar scale descriptors were reworded to improve clarity on the modified IBV (MIBV). Participants were asked to complete this measure daily for 6 weeks.

The MIBV has two parts. Part I asks participants to list separately any positive and negative events that happened during the day. We calculated total event scores for each participant by averaging the daily good events and bad events over the study duration. Participants also rated the degree of pleasantness and unpleasantness of these experiences. Part II contains 18 primary, 10-point graduated bipolar scales describing moods, cognitions, or behaviors that represent common symptoms or features of depression or mania (e.g., energy: 1 = "utterly exhausted, entirely worn out, completely incapable of the slightest effort"; 10 = "limitless zeal, surging with energy, vitality spilling over," mood: 1 = "utter depression and gloom"; 10 = "complete elation"). Participants are asked to provide three ratings on each scale: highest level (peak) reached, lowest level (trough) reached, and average level of that symptom for that day. To obtain a total (TOT) score, we added the average raw score ratings across the 18 items. Sums of daily peak to trough differences (TOT-D) were averaged across the week to yield a within-day variability score of participants' daily mood and behavior. The variance of the sums of average ratings (TOT-A) across the week was utilized to assess across-day variability. The internal consistencies of the MIBV TOT, TOT-D, and TOT-A scores during the 2-week baseline were $\alpha s = .97, .98$, and $.96$, respectively.

Academic Performance Checklist (APC). To mask the true goals of the study, we created the APC. Participants were told that the goal of the study was to examine factors that influence one's academic performance. The APC contained 10 questions regarding academic performance including the ability to concentrate in class, complete homework, and study effectively. Participants were asked to complete this measure weekly.

Family History of Bipolar Disorder. In conclusion to the study, the first author interviewed each participant using Family History–Research Diagnostic Criteria (Andreasen, Endicott, Spitzer, & Winokur, 1977) to assess affective disorders in participants' first-degree relatives. The family history method of obtaining familial history of psychiatric disorders, although less accurate than the use of the family study method, has been found to be a reliable means of obtaining information.

Procedure

We recruited 1,044 undergraduates at Temple University through courses offering completion of the GBI as extra credit as well as via campus flyers from September, 2003, to April, 2004. Participants meeting the GBI criteria were scheduled for a visit to learn about the study and complete the consent form. Participants received \$35 or research credits for the 2-week baseline portion of the study. They were informed that they might be asked to commit to an additional 4 weeks of study participation on completion of the initial phase of the study.

Baseline Phase (Phase I)

The goal of the baseline phase was to determine participants' typical social rhythm regularity and mood lability. Thus, participants completed the M-SRM and MIBV daily for 2 weeks. Participants completed the questionnaires via a secure website and data were identified by an ID number only. To minimize the effects of diurnal variation, participants were instructed to complete these questionnaires every night between 9 p.m. and 12 a.m. or just before going to bed. If a participant could not complete these questionnaires at the assigned time, she/he was instructed to complete them before noon the next morning, but no later. Participants who did not complete the ratings in a timely fashion were reminded with a phone call or an e-mail message. Participants were instructed to complete the BDI, HMI, and APC at the end of each week in the study. All participants completed the study between January and May of 2004 depending on their start date. Given that participants' schedules may vary based on the time at which they were followed during an academic semester (i.e., during final exams, lifestyle regularity for college students may decrease), we included participants' start date as part of the randomization scheme.

After the end of the 2-week baseline phase, participants' data were reviewed. Each participant met individually with the first author (G. S.) to receive feedback on their baseline scores. Participants' social rhythms needed to be sufficiently irregular to participate in the experimental phase (Phase II) of the study. Specifically, participants who had an average baseline M-SRM-S > 4.00, were excluded from Phase II. Only one individual was excluded from Phase II based on this criterion. All other participants were invited to continue in the study and were asked to sign a second consent form at that time.

Experimental Phase (Phase II)

Participants qualifying for the experimental phase (Phase II) of the study were matched on age, sex, ethnicity, and start date and randomly assigned to either the experimental ($n = 35$) or the control ($n = 36$) group. Participants were blind to the study goals given our use of the APC measure to mask the study objectives as well as to the randomization process (i.e., participants were unaware that there were two study groups). Participants in the experimental group were asked to increase their lifestyle regularity to see if it impacted their academic performance (thus, masking our interest in mood lability). Participants in the control group were asked to continue to complete the questionnaires in an effort to better understand the factors that may influence their academic performance (thus, masking our interest in social rhythms and mood lability). Group status was assigned at the time that each participant met with the first author (G. S.) to review their baseline M-SRM

patterns. The first author *only* also then gave specific instructions based on their group status to minimize potential differences between experimenters. This session was conducted in person with the participant and was approximately 30 minutes for both study groups.

Participants randomly assigned to the experimental group were encouraged to regulate their schedules. Target times for each activity were set collaboratively with each participant, such that target times were selected based on their schedule constraints as well as their willingness to change their routine. Emphasis was placed on regulating five M-SRM items: (1) get out of bed, (2) first contact with another person, (3) start work, school, housework, volunteer activities, child or family care, (4) have dinner, and (5) go to bed. These five items were chosen as their regularity score had good values of κ (.69), sensitivity (74%), and specificity (95%) to the 17-item SRM scores (Monk, Frank, Potts, & Kupfer, 2002). For individuals already meeting regularity criteria on any of these items (i.e., six regular times per week), another activity was substituted for that individual. We discussed strategies for increasing regularity with each participant (i.e., participants were encouraged to bring food to class to eat meals at consistent times or to set their alarm clocks to the same time every morning despite the time of their first scheduled activity). Emphasis was placed on accuracy of reporting. Each participant's M-SRM was reviewed after 3 days to determine whether he/she was on track. If goal times were not being met, the participant was contacted to discuss adherence difficulties. At the end of each week, the experimental group participants received an e-mail summarizing their target and actual activity times and highlighting any disparities between them. Participants were asked about difficulties maintaining regularity and, at their request, adjustments in target times were made for the following week. The experimental group was instructed to complete their daily and weekly measures for 4 weeks.

Participants randomly assigned to the control group also met with the first author (G. S.) after the 2-week baseline period. These participants also reviewed their M-SRM patterns with the first author; however, there was no mention of regulating their schedules and/or of reviewing or changing their social rhythms. Instead, the control group was instructed to continue completing their daily and weekly measures for an additional 4 weeks.

After completing Phase II of the study, all participants (in both groups) were debriefed on the true study goals. Each participant was also interviewed using the Family History–Research Diagnostic Criteria to assess the family history of affective disorders. Participants received \$85 for completion of Phase II of the study. A bonus of \$25 was given to participants who completed all their questionnaires on time. In addition, participants missing 5 or fewer days were entered into a lottery for a chance to win an additional \$50. All study participants randomized to one of the two groups completed the study; however, only those with complete data ($n = 62$; 31 in each group) were included in the following analyses. Participants not included in the analyses owing to missing data did not differ on any demographics or study variables from those whose data were included.

Results

Data from each 2-week period were analyzed as a unit to produce three data points: Baseline (Base), Post-Experimental Phase 1 (Post 1), and Post-Experimental Phase 2 (Post 2).

Baseline Analyses

Social Rhythm Regularity and Symptomatology. Given that the two measures of lifestyle regularity (M-SRM-S and REG scores) were highly correlated ($r = .87$, $p < .01$), and parallel analyses with each measure produced highly similar findings (with few exceptions that will be noted), we only present the findings for M-SRM-S scores in detail. A Pearson correlation analysis was conducted on baseline regularity scores and affective symptom scores (BDI and HMI) to determine whether participants reporting less social rhythm regularity also reported increased levels of affective symptoms (see Table 1). Although M-SRM-S and BDI were not significantly associated, REG and BDI scores were found to be significantly negatively related ($r = -.23$, $p = .05$). Participants reporting a higher number of regularly performed activities reported fewer depressive symptoms. HMI scores were not significantly related to M-SRM-S or BDI scores (see Table 1). We also examined the correlations between social rhythm regularity (M-SRM-S) and within-day (TOT-D) and across-day (TOT-A) symptom lability. As expected, M-SRM-S scores were found to be significantly negatively correlated with across-day variances in mood and behavior (TOT-A). Increased regularity was associated with less symptom lability across the 2 weeks. Within-day variability, however, was not found to be significantly correlated with M-SRM-S or TOT-A scores (see Table 1).

Social Rhythm Regularity and Sleep Patterns. We conducted a Pearson correlation analysis to determine whether average sleep duration (SLP) and sleep variability (SLP-SD) were related to affective symptoms and symptom lability. Sleep duration variability (SLP-SD) significantly correlated with regularity (M-SRM-S), sleep duration, depressive symptoms, and across-day symptom variance scores (see Table 1).

Group Equivalency. Demographic variables (age, sex, parental education, and ethnicity) were examined to confirm the equivalency of the experimental and control groups. The groups did not differ significantly on any of these variables. Positive family history of depression and bipolarity was assessed and was found not to differ significantly between groups (72% and 74% for the control and experimental groups, respectively). Family history of substance abuse was also assessed and found not to differ significantly between groups (25% and 23% for the control and

Table 1
Intercorrelations of Baseline Measures of Regularity, Mood, and Sleep (N = 71)

Measure	1	2	3	4	5	6	7	–
1. M-SRM-S	–							
2. BDI	–.08	–						
3. HMI	.03	–.11	–					
4. TOT-A	–.32**	.31*	–.08	–				
5. TOT-D	.19	.18	.11	–				
6. SLP	–.03	–.11	–.08	–.12	–.14	–		
7. SLP-SD	–.30*	.28**	–.21	.34**	.15	–.37**	–	

Note. M-SRM-S, Social Rhythm Metric Score; BDI, Beck Depression Inventory; HMI, Halberstadt Mania Inventory; TOT-A, across-day symptom variability; TOT-D, within-day symptom variability; SLP, sleep duration; SLP-SD, standard deviation of sleep duration. BDI and HMI scores were calculated with the sleep items deleted.

* $p < .05$; ** $p < .01$.

experimental groups, respectively). Means, standard deviations, and *t*-tests were calculated for each study variable to determine equivalency between groups at baseline. Only TOT-D was found to differ significantly between the experimental and control groups ($t_{1, 70} = 2.31, p = .02$) and HMI scores approached significance ($t_{1, 70} = 1.81, p = .08$).

Experimental Analyses

Table 2 summarizes the means and standard deviations of the study variables for Base, Post 1, and Post 2 by group. Throughout the following analyses, effect size was calculated as Cohen's *d* with pooled standard deviation.

Regularity. Repeated measures analysis of variance (rANOVA) was conducted on M-SRM-S scores to determine whether participants who received the experimental manipulation were successful in regulating their schedules (see Table 2). The Greenhouse–Geisser correction was used to address the sphericity assumption

Table 2
Post 1-Experimental and Post 2-Experimental Scores of Variables of Interest ($N = 62$)

Measure	Experimental group ($n = 31$)		Control group ($n = 31$)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>M-SRM-S</i>				
Baseline	2.69	0.61	2.56	0.73
Post 1	3.96**	1.05	2.49	0.79
Post 2	4.04**	1.38	2.49	0.70
<i>BDI</i>				
Baseline	12.41	8.19	13.12	7.74
Post 1	9.28**	6.07	10.80*	7.88
Post 2	9.55**	8.64	10.93*	8.57
<i>HMI</i>				
Baseline	17.10	5.81	19.80	8.04
Post 1	14.90*	6.62	15.50**	8.49
Post 2	15.48	7.03	13.31**	7.66
<i>TOT-A</i>				
Baseline	231.11	239.03	250.74	216.35
Post 1	120.68**	106.08	165.07**	204.37
Post 2	118.85**	123.68	159.38**	278.40
<i>TOT-D</i>				
Baseline	33.98	14.77	43.26	19.85
Post 1	31.30	16.86	43.97	22.25
Post 2	31.39	18.06	40.92	20.27
<i>SLP</i>				
Baseline	6.61	0.89	6.84	1.19
Post 1	6.71	1.22	7.20	0.97
Post 2	6.86	1.49	7.17	0.98
<i>SLP-SD</i>				
Baseline	1.84	0.68	1.85	0.95
Post 1	1.39	0.60	1.54	0.66
Post 2	1.26*	0.55	1.67	0.99

Note. M-SRM-S, Social Rhythm Metric Score; BDI, Beck Depression Inventory; HMI, Halberstadt Mania Inventory; TOT-A, across-day symptom variability; TOT-D, within-day symptom variability; SLP, sleep duration; SLP-SD, standard deviation of sleep duration. BDI and HMI scores were calculated with the sleep items deleted.

** $p < .01$, for the difference from the baseline score; * $p < .05$, for the difference from the baseline score.

violation of this measure and, thus, degrees of freedom (*dfs*) are reduced owing to this correction. Group \times Time interactions were found to be significant for M-SRM-S ($F_{2, 96} = 21.74, p < .01$). The significant Group \times Time interaction was followed by tests of simple main effects. Within-subject rANOVA contrasts were used to compare Base scores with Post 1 and Post 2 scores for each group.

The experimental group Base M-SRM-S scores differed significantly from both Post 1 M-SRM-S scores ($F_{1, 30} = 47.85, p < .01, d = 1.48$) and Post 2 M-SRM-S scores ($F_{1, 30} = 28.16, p < .01, d = 1.27$). These results confirm that the frequency of regularly performed daily activities after the experimental manipulation significantly increased for this group. However, the control group Base M-SRM-S scores did not differ significantly from either Post 1 M-SRM-S scores ($F_{1, 30} = .49, p = .49, d = .09$) or Post 2 M-SRM-S scores ($F_{1, 30} = .26, p = .62, d = .10$).

These results suggest that the experimental manipulation increased lifestyle regularity in the experimental group compared with the control group (see Table 2). SRM-S scores of normal controls ($N = 293$) in another study ($M = 3.9, SD = 0.83$) were comparable to the experimental group's Post 2 scores ($M = 4.04, SD = 1.38$; Monk et al., 2002), suggesting that the experimental manipulation increased lifestyle regularity in the experimental group to a normative level.

Affective Symptomatology. Separate Group \times Time rANOVAs were conducted on BDI and HMI scores to determine whether those scores changed differentially between groups over time (see Table 2). The Time main effect ($F_{2, 120} = 8.13, p < .01, d = .76$) for the BDI was found to be significant; however, the Time \times Group interaction was not ($F_{2, 120} = .48, p = .60; d = .18$). BDI scores for both groups decreased over time. The Group \times Time interaction for the HMI was significant ($F_{2, 120} = 6.58, p < .01; d = .69$); therefore, we conducted tests of simple main effects.

Within-subject contrasts were used to compare Base HMI scores with Post 1 and Post 2 scores for each group. The experimental group Base HMI scores differed significantly from Post 1 HMI scores ($F_{1, 30} = 5.89, p < .03, d = .90$), but not from Post 2 HMI scores ($F_{1, 30} = 2.32, p < .14, d = .54$). The control group Base HMI scores differed significantly from both Post 1 HMI scores ($F_{1, 30} = 24.09, p < .001, d = 1.79$) and Post 2 HMI scores ($F_{1, 30} = 48.01, p < .001, d = 2.73$). Although both groups showed decreases in hypomanic symptoms over time, individuals who did not receive the experimental manipulation exhibited greater decreases in hypomanic symptoms; however, their HMI scores were still higher than those found in a normal sample ($M = 12.01, SD = 5.55$; see Table 2; Halberstadt & Abramson, 2008).

Symptom Lability. Separate rANOVAs were conducted on TOT-A and TOT-D scores to determine whether symptom lability across and within days changed differentially between groups over time (see Table 2). The Greenhouse–Geisser correction was also used to address the sphericity assumption violation of these measures and, thus, *dfs* are reduced. Time main effects were significant for TOT-A ($F_{2, 91} = 13.42, p < .01$) and approached significance for TOT-D ($F_{1, 89} = 2.98, p = .07$). Across-day and within-day variability decreased over time for both the experimental and the control groups. No interaction terms were found to be significant ($ds = .10$ and $.34$ for TOT-A and TOT-D, respectively).

Sleep Patterns. Separate rANOVAs were conducted on SLP and SLP-SD scores to determine whether sleep duration and sleep variability changed differentially between groups over time (see Table 2). The Greenhouse–Geisser correction was used to address the sphericity assumption violation of SLP. The Time main effects

Table 3

Experimental Group Correlations of Change Scores from Base to Post 2 for Regularity with Change Scores from Base to Post 2 for Mood and Sleep ($N = 62$)

Dependent measure	M-SRM-S Δ	REG Δ
BDI Δ	.16	-.36*
HMI Δ	-.14	-.23
TOT-A Δ	-.03	-.10
TOT-D Δ	-.16	-.43*
SLP Δ	-.41**	-.24
SLP-SD Δ	-.21	-.10

Note. M-SRM-S, Social Rhythm Metric Score; REG, Regularity Score; BDI, Beck Depression Inventory; HMI, Halberstadt Mania Inventory; TOT-A, across-day symptom variability; TOT-D, within-day symptom variability; SLP, sleep duration; SLP-SD, standard deviation of sleep duration. BDI and HMI scores were calculated with the sleep items deleted.

* $p < .05$; ** $p < .01$.

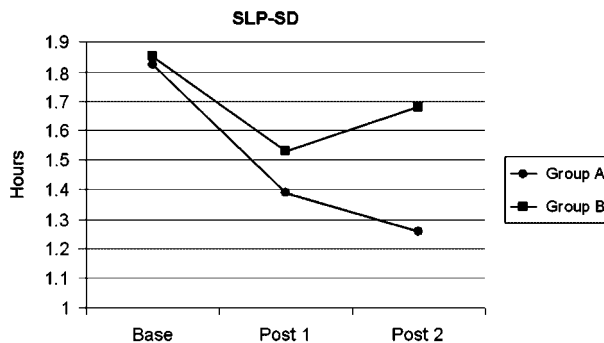


Figure 1. Level of sleep variability over time for Group A (Experimental) and Group B (Control) participants.

for SLP ($F_{2, 95} = 3.50, p = .04$) and SLP-SD ($F_{2, 118} = 9.35, p < .01$) were significant. Sleep duration increased and sleep variability decreased over time for both the experimental and control groups. No omnibus interaction terms were significant. However, a post hoc test of within-subject contrasts revealed a significant Group \times Time interaction for SLP-SD when comparing Base with Post 2 ($F_{1, 59} = 3.93, p = .05, d = .94$). SLP-SD decreased significantly for the experimental, but not for the control participants (see Figure 1).

Post-Experimental Phase Change Scores

To determine whether changes in lifestyle regularity corresponded with changes in the dependent measures, the experimental group's change scores (Post 2–Base) were calculated for each variable. A Pearson correlation analysis was used to determine whether changes in social rhythm regularity were associated with changes in depressive and hypomanic symptoms, within-day and across-day symptom lability, and sleep duration and variability (see Table 3). Increased frequency with which participants performed regular activities (M-SRM-S) from Base to Post 2 corresponded to a decrease in sleep duration over this period. A positive change in the number of regularly performed activities (REG) was significantly related to

decreased depressive symptoms and decreased variability in within-day affective symptom lability from Base to Post 2 (Table 3).

The experimental group's regularity change scores were further examined to determine whether participants who successfully increased their regularity differed systematically from those who did not on baseline measures. An increased change in regularity was not significantly related to Base GBI scores, family history of mood disorders, parental education, ethnicity, gender, or age (all $r_s < .30$). Increased regularity change scores as measured by the REG Δ were significantly related to REG ($r = -.37, p < .05$) Base scores, but no other baseline measures (all $r_s < .23$). Thus, the lower the REG Base score, the higher the change score.

Discussion

A major finding of this study is that participants self-reporting cyclothymic symptoms were able to successfully regulate their daily schedules. Additionally, no symptomatic or demographic differences were identified that differentiated those who successfully regulated their schedules from those who did not. Given that participants were effective in increasing their lifestyle regularity, the impact of these changes could be examined in relation to affective symptoms, lability, and sleep.

We found that lifestyle regularity was significantly related to depressive symptoms. Participants reporting a higher number of regularly performed activities reported less severe depressive symptoms at baseline. This finding is consistent with earlier studies conducted with bereaved elderly and clinically depressed samples (Prigerson et al., 1993; Szuba, Yager, Guze, Allen, & Baxter, 1992). However, changes in regularity scores did not significantly correspond to changes in depression scores over time. Additionally, depression scores of individuals who successfully increased their regularity did not differ significantly from individuals who did not attempt to regulate their schedules. Instead, depressive symptoms for both groups decreased over time. Although not significant, the rate of change in BDI scores was greater in those who had received the experimental manipulation.

It is possible that social rhythm changes affect depressive onsets more gradually than manic onsets (Malkoff-Schwartz et al., 1998). Thus, the 4-week follow-up may not have been long enough and changes in depressive symptoms may lag behind schedule changes. Moreover, the finding that changes in regularity did not directly correspond to changes in depressive symptoms is also consistent with earlier literature (Chang et al., 2003; Prigerson et al., 1993). Further, Frank et al. (1997, 2000) found that participants with bipolar I disorder receiving IPSRT were able to increase their lifestyle regularity, but experienced no differential changes in depressive symptoms compared with a control group. However, more recent findings suggest that IPSRT may buffer against future affective episodes (Frank et al., 2005).

An alternative explanation for the decrease in depressive symptoms in both groups is that the act of monitoring one's daily activities for both groups throughout the baseline and intervention phase was helpful in itself. Such self-monitoring could have provided participants with greater self-awareness and self-efficacy over time. Moreover, the fact that the daily monitoring of activities occurred each evening at a specified time may have induced greater rhythmicity and symptom stability among participants, and consequently, led to a decreased chance for the intervention to have a differential effect.

Unlike depressive symptoms, baseline hypomanic symptoms did not correlate with baseline lifestyle regularity scores. Ashman et al. (1999) also described an inconsistent pattern of correlations between SRM scores and hypomanic mood severity in patients with rapid cycling bipolar disorder. The heterogeneous nature of (hypo)mania may partially explain this. Unlike individuals in severe manic episodes, who may be given to impulsive, disorganized, and frenzied behavior, individuals with milder hypomania may actually be more productive with "heightened sensibilities" (Goodwin & Jamison, 1990). Perhaps intermediate levels of hypomania may be related to increased regularity, whereas low and high levels may result in disrupted patterns. Further investigation is necessary to substantiate this hypothesis.

Changes in regularity also did not correlate well with changes in hypomanic symptoms. Although the hypomania scores of individuals who were encouraged to regulate their schedules decreased from baseline to Post 1, those of the control group showed greater decreases over time. This finding was surprising and must be interpreted with caution, given that differences in HMI scores between groups approached significance at baseline. Further research is necessary to better understand this relationship.

The second aim of this study was to understand how lifestyle regularity relates to symptom lability in individuals with cyclothymic symptoms. Earlier research indicates that disruptions of internal biological rhythms may be responsible for the affective shifts experienced by bipolar spectrum individuals (Howland & Thase, 1999; Lenox et al., 2002). Given the power of social zeitgebers to entrain biological rhythms, one would expect individuals keeping to more regular weekly schedules to report less mood and behavioral lability across the week. This study's findings support this hypothesis. Specifically, higher baseline regularity scores were associated with lower across-day variances in mood and behavior. This study is the first to document this relationship in individuals exhibiting cyclothymic symptoms; however, causality cannot be established based on these results.

We hypothesized further that increased regularity would differentially result in decreased symptom lability in those receiving the experimental manipulation. However, both groups reported decreased overall symptom lability across the follow-up period. It should be noted that, given the moderate effect size ($d = .34$) obtained for within-day lability, it is possible that our sample size did not provide adequate power to detect statistically significant differences between groups. In addition, our follow-up time period may not have been long enough to detect sustained overall changes or that the regular monitoring for both groups was helpful in itself. The control group also had significantly higher within-day variability at baseline, despite randomization, which may have also contaminated the results. Although no overall group differences were found, of those receiving the experimental manipulation, change in regularity was negatively correlated with change in within-day variations in their mood. As Frank et al. (1997, 2000) suggested, this finding is important in that individuals with bipolar spectrum disorders who maintain regular schedules may ultimately have less functional impairment.

The third aim of this study was to better understand how sleep patterns relate to lifestyle regularity and affective symptoms. Wehr, Sack, and Rosenthal (1987) hypothesized that sleep impacts both regularity and affective episode onset. Indeed, sleep disruption is often an associated feature of depression (APA, 2000; Riemann et al., 2002; Wilson & Nutt, 2005). Sleep duration did not significantly

relate to depressive symptoms or lifestyle regularity in our participants, although sleep duration variability did significantly correlate with baseline regularity, depression, and across-day symptom variance scores. This suggests that consistency in sleep patterns may be a more sensitive measure than sleep duration in our participants. Increased sleep variability was associated with higher depression scores and increased mood lability across the week. These data, and our findings that participants' sleep patterns correlated more consistently with the mood indices than did social rhythm scores, may indicate a particularly important role for sleep in bipolar symptoms (see Table 1). This finding is supported by other studies that found an association between sleep regularity and bipolar symptoms and/or episodes (Harvey et al., 2005; Leibenluft et al., 1996; Perlman et al., 2006; Riemann et al., 2002; Wehr et al., 1985). For example, Benedetti et al. (2007) found that sleep deprivation alters brain activity in individuals with bipolar disorder, currently in a depressive episode. Thus, future research should further investigate the unique role of sleep regularity on bipolar symptoms and/or episodes.

In this study, sleep variability decreased significantly over time for participants who were encouraged to regulate their schedules, but not for those who did not receive the experimental manipulation. Although unrelated at baseline, increases in regularity were found to correlate with decreases in sleep duration in those receiving the experimental manipulation. However, causal conclusions regarding sleep patterns should be drawn with caution given that sleep duration was not specifically targeted in the experimental manipulation. Instead, target times were selected by participants without specific regard to sleep duration. Thus, for many participants, successfully meeting goal times also meant decreased sleep duration. This is also reflected in the decreased sleep variability over time. Although changes in sleep duration were statistically significant, average sleep changes were less than 30 minutes. The clinical significance of this change is questionable given that 30 minutes is only 7% of the total sleep duration at baseline for both groups (6.58 and 6.65 hours for the control and experimental groups, respectively). Changes in sleep duration and sleep variability were not related to changes in affective symptoms or mood lability. This may be owing to the small magnitude of sleep changes. Further studies specifically targeting sleep duration may better illuminate associations between sleep and mood than this study.

In summary, our study provided mixed support for the social zeitgeber theory in individuals with cyclothymic symptoms. Although relationships between regularity, severity of depressive symptoms, across-day variances in mood and behavior, and sleep duration variability were identified during baseline, increased lifestyle regularity did not differentially result in changes in those variables over time. However, among participants receiving the experimental manipulation, increased regularity was related to a decrease in affective symptom lability.

There are several limitations to this study. First, we only examined affective symptoms, rather than assessing diagnosed affective episodes. Earlier work found that bipolar spectrum participants with more regular schedules at baseline were less likely to experience an affective episode during a prospective follow-up than their less regular counterparts (Chang et al., 2003; Shen et al., 2008). By focusing on symptoms alone, rather than diagnosed affective episodes, it is possible that this study failed to capture clinically significant differences between the groups. The control group exhibited higher scores on several mood indices at baseline, despite our randomization procedures. This may further highlight the need for using episode

data. Moreover, the fact that our sample exhibited mild bipolar symptoms that were relatively stable at the outset may have also contributed to our null findings. Another factor that may have influenced the findings is the assessment of hypomanic symptoms with the HMI. To date, the HMI has correlated only moderately with other measures of (hypo)mania and may have insufficient sensitivity. Future studies that include more formal diagnostic interviews, several measures of hypomania and depression, or more severely symptomatic samples may be necessary to further test the social zeitgeber theory.

A second limitation of this study is that participants were monitored for daily activities for only 6 weeks. This period may be too short to observe longer-term effects in participants with cyclothymic symptoms. Given that cyclothymia has characteristically unstable mood and behavior patterns, long-term benefits of increased regularity may take longer to assess than 6 weeks. There are also potential limitations with the SRM. For example, it is a retrospective (i.e., administered at the end of the day) self-report measure that may be subject to response and/or memory bias of participants. It is also possible that the SRM does not capture an activity that is an important zeitgeber for an individual, given that 15 of the 17 items are fixed activities. Thus, our lack of an objective measure (i.e., actigraphy) of social rhythms may have contributed to the absence of group differences after the intervention. We also did not assess participants' appraisal of their environment, or their daily life events, which may also have impacted their social rhythm regularity and contributed to the null findings (Jones, 2001; Jones et al., 2006).

Our study participants were unique in that they were college students. Although diverse in both socioeconomic status and ethnicity, this group may not be representative of all individuals exhibiting cyclothymic symptoms. Although in many ways this sample is ideal because they are at an age of risk for bipolar disorder onset and tend to have variable daily schedules, the generalizability of these findings may be limited. Additionally, participants were not assessed using a formal diagnostic interview. Thus, although our sample's rates of family history of mood disorders and substance abuse were comparable to those identified in the literature for other bipolar spectrum disorders (e.g., Akiskal, Hantouche, & Allilaire, 2003; Benazzi, 2004; Perugi et al., 1998), it is possible that our sample included individuals who would not meet strict criteria for cyclothymia. The generalizability of our findings to community and/or clinical samples remains to be determined.

Despite these limitations, this study was a first step in better understanding the relationship between lifestyle regularity, symptoms, and sleep among nonpatient individuals experiencing cyclothymic symptoms. Additionally, this is the first study to prospectively assess both daily activity patterns and affective symptoms in individuals at high risk for developing bipolar disorder. Future research should expand on these preliminary results by assessing these associations with clinical or community samples followed prospectively for at least 2 months.

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