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Generation of Life Events in Bipolar Spectrum Disorders: A Re-examination and Extension of the Stress Generation Theory

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Abstract

The extent to which stress generation occurs in bipolar spectrum disorders (BSD) is not well understood. The present study examined whether 75 BSD participants experienced elevated rates of behavior-dependent life events, as compared with 88 normal control participants. Within the BSD group, we also examined whether depressive or hypomanic symptoms prospectively predicted increases in various types of negative and positive life events. Results indicated that BSD participants experienced overall increases in behavior-dependent events over the follow-up, as compared with normal controls. At the symptom level, the event generation process occurred in more specific event domains. Results suggest that the stress generation theory of unipolar depression can be extended to BSD and that the type of generated events may be polarity-specific.

Keywords

bipolar spectrum disorder; stress generation; life events; hypomania; depression

Bipolar disorder (BD) affects an estimated 0.5%–3.5% of the world population (Grant et al., 2005; Miklowitz & Johnson, 2006). It can result in a wide range of psychosocial impairments including marital distress, financial problems, and substance use, as well as heightened risk of suicide and erratic work history (Judd et al., 2005; Kessler et al., 2006; Miklowitz & Johnson, 2006; Weinstock, Keitner, Ryan, Solomon, & Miller, 2006). Within the bipolar category, cyclothymia, bipolar II, and bipolar I disorders appear to exist on a continuum or spectrum of severity, and milder forms often progress to the more severe forms (Akiskal, Djenderedjian, Rosenthal, & Khani, 1977; Akiskal, Khani, & Scott-Strauss, 1979; Cassano et al., 1999; Depue et al., 1981; Goodwin & Jamison, 1990; Klein, Depue, & Slater, 1985; Shen, Alloy, Abramson, & Sylvia, 2008). Much of the research on BD has focused on individuals diagnosed with bipolar I disorder. This is somewhat surprising, given that bipolar spectrum disorders (BSD) are more prevalent than bipolar I disorder in mental

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health clinics, and typically persist for years without the symptom-free periods observed in more severe bipolar disorders (Depue et al., 1981; Howland & Thase, 1993). As BSD is relatively prevalent and increases risk for more severe bipolar I disorder, it is important to better understand the mechanisms underlying its maintenance and progression. Thus, we consider BSDs in this article, and the possible role of stress generation processes in maintaining and worsening bipolar symptoms.

Studies are beginning to identify important psychosocial factors affecting the etiology and course of bipolar illness. Research examining the impact of life events on symptom expression, timing, and severity of bipolar affective episodes has been especially promising (Alloy et al., 2005). Of particular interest is the idea that the impact of events is polarity-specific, with systematic differences existing between event-related predictors of (hypo)mania and depression (Johnson, 2005a; Johnson et al., 2008). Studies on bipolar depression suggest that event triggers are comparable to those identified in unipolar depressed samples, and often involve severe negative life events (Johnson et al., 2008). In contrast, well-controlled studies have not as consistently implicated severe negative life events as precipitants of (hypo)mania. Instead, evidence suggests that individuals with or at risk for BSD may be more vulnerable to (hypo)mania after (a) goal-striving or achievement-related events, and (b) exposure to reward-relevant stimuli (Alloy, Bender, Wagner, Abramson, & Urošević, 2009; Johnson, 2005b; Johnson et al., 2000; Nusslock, Abramson, Harmon-Jones, Alloy, & Hogan, 2007; Urošević, Abramson, Harmon-Jones, & Alloy, 2008). A recent review of the behavioral approach system (BAS; a motivational system underlying goal-striving and reward responsiveness) dysregulation theory of BSD indicated that reward-related and goal-related stimuli may take the form of *either* negative or positive events (Urošević et al., 2008). In other words, depending on the context and the individual's appraisal of the event, both achievement failures and successes could potentially trigger hypomania/manic symptoms.

Life events, therefore, appear to frequently precipitate subsequent bipolar affective symptoms. But, it is unclear whether the opposite directional relationship also holds true, i.e., whether bipolar symptomatology plays a role in the generation of subsequent life events. The stress generation theory in unipolar depression posits that depressed and recently remitted individuals actively shape their own environments and, in doing so, generate future stressful events (Hammen, 1991, 2006). The resulting life stress may elicit or exacerbate depressive symptoms, potentially leading to a full-blown depressive relapse. Thus, a transactional relationship exists between stress and depression, such that depressed individuals may actually generate stress in addition to reacting to it.

A stress generation model, therefore, implies that depressed and recently remitted individuals will experience increased rates of *dependent* negative life events over time. Dependent events are those that are caused fully or in part by the individual's own behavior. Examples of dependent negative life events include an unwanted termination of a romantic relationship or a job loss due to poor performance. In contrast, *independent* negative life events are those occurring wholly outside the individual's realm of control. Examples of independent events include the death of a family member, a natural disaster, some medical illnesses, or a car theft. Supporting the stress generation theory, prospective studies on unipolar depression consistently find elevations in rates of dependent, but not independent, stressors among a wide range of depressed samples, including clinically depressed patients (Bottonari, Roberts, Kelly, Kashdan, & Ciesla, 2007; Chun, Cronkite, & Moos, 2004; Hammen, 1991; Harkness, Monroe, Simons, & Thase, 1999), depressed undergraduates (Cui & Vaillant, 1997; Joiner, Wingate, Gencoz, & Gencoz, 2005; Potthoff, Holahan, & Joiner, 1995; Shih, 2006), and community samples with varying degrees of depressive symptomatology (Daley, Hammen, Burge, & Davila, 1997; Davila, Bradbury, Cohan, &

Tochluk, 1997; Davila, Hammen, Burge, & Paley, 1995; Holahan, Moos, Holahan, Brennan, & Schutte, 2005; Jones, Beach, & Forehand, 2001; Nelson, Hammen, Daley, Burge, & Davila, 2001; Wingate & Joiner, 2004). It is important to note that these stress generation studies have focused largely on the domain of interpersonal stress, and the theory has frequently been tested in female-only samples (Daley et al., 1997; Davila et al., 1995; Hammen, 1991, 2006; Nelson et al., 2001). In mixed gender studies, results have indicated that the process may occur exclusively in females (e.g., Davila et al., 1997; Safford, Alloy, Abramson, & Crossfield, 2007) or that it may operate differently across genders (Rudolph et al., 2000; Shih, 2006). Several mixed gender stress generation studies do not substantively address the issue of gender differences at all (Bottonari et al., 2007; Hankin, Kassel, & Abela, 2005; Harkness et al., 1999; Joiner, Wingate, Gencoz et al., 2005).

Stress generation research suggests that formerly depressed individuals may continue to show increased rates of dependent stressors, even during remitted periods (Daley et al., 1997; Hammen, 1991). This finding is important for two reasons. First, to demonstrate that the phenomenon reflects more than simply acute behavioral manifestations of symptom-related functional impairment (e.g., secondary to irritability, anhedonia, and impaired concentration), stress generation must also be observed outside periods of diagnosable depression. Second, the presence of stress generation outside of diagnosable episodes is important to the theory's purported ability to explain relapses or recurrences over time. That is, for stress generation to precipitate relapse, it must by definition also occur between diagnosable episodes.

Although the interpersonal stress generation hypothesis is robustly supported in various dysphoric and depressed female samples, surprisingly little parallel work has been conducted within bipolar samples. Hammen (1991) did not find evidence of stress generation among individuals with bipolar I disorder, but the sample was small and comprised female-only and treatment-seeking participants. Furthermore, the study design may have been insufficiently sensitive to the specific types of life events potentially involved in BSD stress generation, in that the life events catalogued were exclusively negative and primarily interpersonal in nature.

Since Hammen's (1991) original report, two additional studies have evaluated stress generation in bipolar disorder. Reilly-Harrington, Alloy, Fresco, and Whitehouse (1999) did not find support for the stress generation hypothesis in a sample of individuals with bipolar I disorder, bipolar II disorder, or cyclothymia. The study was limited by its use of self-report checklists rather than interview methods to assess life events, and by its relatively short 1-month follow-up period. The events assessed were mostly negative in nature, again raising questions about the sensitivity of the design to possible stress generation-like processes in BSD. A study on the impact of childhood stressful life events on BSD found limited support for the stress generation model: A bipolar spectrum diagnosis did not predict increased overall rates of dependent events occurring after age at onset, although postonset maltreatment events were elevated in comparison with a matched control group (Grandin, Alloy, & Abramson, 2007). In this study, stress was assessed via a retrospective self-report checklist that measured negative or stressful life experiences only.

Although not explicitly testing the stress generation hypothesis, two prospective longitudinal studies have examined the impact of independent versus dependent stressful events on bipolar disorder in adolescents. One study found that in the adolescent offspring of bipolar parents, negative dependent but not independent events predicted onset of an episode of mood disorder (Wals et al., 2005). The relationship between dependent stress and subsequent mood episodes was fully accounted for by baseline depression and anxiety symptoms, supporting the idea that initial symptoms may predict subsequent dependent

stress (Wals et al., 2005). Only 4% of the offspring had actually developed a bipolar disorder by the time of the follow-up period, however, so it is unclear whether these findings generalize to individuals with diagnosable bipolar disorder. A second study examined independent and dependent negative life stressors as predictors of mood symptoms among bipolar adolescents participating in a treatment outcome study (Kim, Miklowitz, Biuckians, & Mullen, 2007). Overall, neither independent nor dependent episodic events predicted subsequent depressive or hypomanic symptoms. The researchers found that some chronic interpersonal stressors predicted attenuated symptom improvement. As the study involved participation in both psychosocial and psychopharmacological treatments, it may not fully reflect the naturalistic relationship between stress and symptomatology.

As noted above, distinct lines of research are converging on the idea that some types of positive events may also be influential in the course of BSD (Alloy et al., 2009; Johnson, 2005a, b; Urošević et al., 2008). Such findings suggest that a comprehensive stress generation theory of BSD may need to incorporate positive life events, as well as those traditionally categorized as stressful. Although positive life events do not fall under the general rubric of "stress generation," they may be clinically relevant in light of research suggesting that such events could heighten risk for hypomania/manic episodes. In other words, increased generation and experiencing of some types of positive events may also place strain (i.e., "stress") on the affective stability or regulatory systems of individuals with BSD (Alloy et al., 2009; Johnson, 2005a, b; Urošević et al., 2008). In the context of BSD, therefore, a *psychosocial event generation* model may prove more relevant than a strictly stress-focused model.

Despite the paucity of existing research on event generation in BSD, there remains a strong clinical and theoretical rationale for hypothesizing its existence. Because the process may operate differently in BSD than in unipolar depression, methodologies that allow us to detect stress generation in unipolar depression may fail to detect or actually obscure it in BD. Moreover, life events research on BSD repeatedly cites stress generation as a limitation confounding interpretation of directional causality (e.g., Alloy et al., 2005; Cohen, Hammen, Henry, & Daley, 2004) and as an important area for future research (Johnson, 2005a; Urošević et al., 2008). The aim of the present study was to investigate whether the stress generation model of unipolar depression could be refined and extended in a BSD sample. First, we examined whether individuals with BSD experienced increased rates of dependent events as compared with a healthy control group. Among those with BSD, we next examined whether depressive or hypomanic symptoms at an initial time point prospectively predicted various types of dependent life events at a subsequent time point. Building on past research and to maximize sensitivity to the stress generation process in a bipolar sample, we proposed a psychosocial event generation hypothesis using events categorized across two dimensions: valence (negative/positive) and content (achievement/interpersonal). We expected to find polarity-specific effects, such that depressive symptoms would predict negative interpersonal, but not achievement, events, whereas hypomanic symptoms would predict positive *and* negative achievement events, but not interpersonal ones. We further expected that the generation of life events would occur even outside periods of full-blown affective episodes. Finally, based on past research demonstrating interpersonal stress generation in depressed females, we expected to find gender-specific effects, with females more likely to generate events in the negative interpersonal domain.

Method

Participants and Procedures

Participants were drawn from the Temple University site of the Temple-Wisconsin Longitudinal Investigation of Bipolar Spectrum Disorders (LIBS) Project. The LIBS Project

investigated psychosocial, cognitive, and biological predictors of the course of bipolar spectrum disorders.[1] Participants were selected for the overall study via a two-phase screening process. In phase 1, approximately 7,500 individuals, aged 18–24 years, completed the General Behavior Inventory (Depue & Iacono, 1989). Based on General Behavior Inventory cutoffs (see measures), 12.3% of the phase 1 sample were potentially eligible for the bipolar spectrum group, and 65% were potentially eligible for the healthy control group. Six hundred and eighty-four potentially eligible participants completed a phase 2 diagnostic interview, approximately 2 weeks to 4 months after phase 1.

The main eligibility criterion for the bipolar spectrum group was a current or lifetime Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV; American Psychiatric Association, 1994) or Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978) diagnosis of bipolar II disorder, cyclothymia, or bipolar not otherwise specified (NOS). The bipolar NOS category was comprised of individuals who had experienced (a) hypomanic episode(s) but no diagnosable depressive episodes, (b) cyclothymic mood pattern with periods of affective disturbance not meeting duration criteria for hypomanic and depressive episodes, or (c) hypomanic and depressive episodes not meeting frequency criteria for diagnosis of cyclothymia. Participants were ineligible for the bipolar spectrum group if they had experienced one or more Diagnostic and Statistical Manual of Mental Disorders 4th edition (DSM-IV) or Research Diagnostic Criteria (RDC)-defined manic episodes, thus meeting diagnostic criteria for bipolar I disorder.[2] Main exclusion criteria for the normal control group were as follows: (a) current or past diagnosis of any Axis I psychopathology as defined by DSM-IV or RDC (with the exception of a specific phobia) and (b) family history of mood disorder. One hundred thirty-seven of 384 (35.7%) of those potentially eligible for the BSD group met criteria for a BSD (90 for bipolar II disorder and 47 for cyclothymia/bipolar NOS), and 127 of 300 (40.2%) participants met criteria for the healthy control group. The final LIBS Project sample (110 bipolar spectrum participants and 110 eligible matched normal controls) was representative of the large phase 1 screening sample with respect to gender, age, and ethnicity.

Of the 220 participants who entered the longitudinal phase of the study, 163 had complete data relevant to study hypotheses. The 57 excluded participants did not significantly differ from the 163 included participants on the basis of the following: gender ($\chi^2[2] = .68$, $p = .71$); age ($t[218] = .06$, $p = .95$); ethnicity ($\chi^2[4] = 2.78$, $p = .60$); or bipolar spectrum diagnosis ($\chi^2[2] = 2.35$, $p = .31$). The final sample for the present study thus included 75 bipolar spectrum participants (49 female, 26 male) and 88 normal controls (60 female, 28 male). Of the bipolar spectrum participants, 64.0% ($N = 48$) met criteria for bipolar II disorder (36 women, 12 men) and 36.0% ($N = 27$) received diagnoses of cyclothymia or bipolar NOS (13 women, 14 men).

As part of the general LIBS Project, all participants completed measures of mood symptoms and life events approximately every 4 months at each regular prospective assessment. Data for the present study were drawn from the first two prospective assessments completed by each participant, hereafter called time 1 (T1) and time 2 (T2). Prior to entering the study, all participants provided written informed consent.

[1]For an examination of diagnostic group differences in life events from a Behavioral Approach System perspective, using a separate subset of participants from the LIBS Project, (see Urošević et al., 2010).

[2]The purpose of this exclusion criterion was related to one goal of the LIBS Project, which involved predicting first onset of manic episodes.

Measures

General Behavior Inventory (GBI)—The GBI (Depue, Krauss, Spoont, & Arbisi, 1989) is a self-report questionnaire used during the phase 1 screening process to identify potential bipolar spectrum participants and normal controls. The GBI has been extensively validated among a range of populations, including undergraduates, psychiatric outpatients, and relatives of bipolar I probands (Depue et al., 1989; Klein, Depue, & Slater, 1985b). Psychometric properties of the instrument are strong, with a reported internal consistency of α's = 0.90–0.96, test-retest reliability of r's = 0.71–0.74, adequate sensitivity (0.78), and excellent specificity (0.99) for bipolar spectrum disorders (Depue et al., 1981, 1989). Discriminant validity has also been good (.88 in discriminating between those with and without affective disorders; Mallon, Klein, Bornstein, & Slater, 1986). The revised GBI utilized in the present study contains 73 items, each designed to assess various experiences related to depressive, hypomania/manic, or biphasic symptoms on the dimensions of intensity, duration, and frequency. The respondent provides a rating on a 4-point Likert scale, ranging from 1 (*not at all*) to 4 (*very often or almost constantly*). As recommended by Depue and colleagues (1989), an item received a score of one point when rated as 3 (*often*) or 4 (*very often or almost constantly*) on the scale. Points were then summed to obtain two subscores, one for depression items (GBI-D score) and another for (hypo)mania and biphasic items (GBI-HB score). This scoring method yields GBI scores that represent the number of symptomatic experiences satisfying the criteria of duration, intensity, and frequency.

Based on Depue et al.'s (1989) findings, the following cutoff scores were used to identify potential bipolar spectrum and normal control participants: GBI-D score of ≥11 and GBI-HB score of ≥13 for potential bipolar spectrum participants, and GBI-D scores<11 and GBI-HB scores<13 for normal controls. A pilot study for the LIBS Project validated this high-GBI and low-GBI group assignment procedure against diagnoses obtained via Schedule for Affective Disorders and Schizophrenia-Lifetime interviews (see Alloy et al., 2008).

Schedule for Affective Disorders and Schizophrenia-Lifetime (SADS-L)—In phase 2 of the screening process, diagnostic interviews were conducted using an expanded version of the SADS-L (Endicott & Spitzer, 1978) semistructured diagnostic interview (exp-SADS-L). The exp-SADS-L assesses the occurrence, duration, and severity of symptoms related to mood, anxiety, eating, psychotic, and substance use disorders over the lifetime. Modifications made to the instrument for use in the LIBS Project included: (a) additional probes to allow for the assignment of DSM-IV diagnoses as well as RDC diagnoses; (b) additional items inquiring about affective symptoms, developed to better capture nuances of episodes as well as frequency and duration of symptoms; and (c) additional sections on eating disorders, attention deficit hyperactivity disorder, acute stress disorder, medical history, family history, and organic rule-out conditions.

The interviews were conducted by extensively trained interviewers who were blind to participants' phase 1 GBI scores. Interviews were audiotaped to obtain consensus diagnoses and to monitor interrater reliability. A three-tiered consensus diagnostic procedure was employed for the LIBS Project, using an expert psychiatric consultant as the third diagnostic tier. Interrater reliability generated by this procedure was high: kappas for major depressive disorder diagnoses based on 80 jointly rated interviews were >.95, while an interrater reliability study based on 105 jointly rated interviews yielded κ>.96 for bipolar spectrum diagnoses (Alloy et al., 2008).

Schedule for Affective Disorders-Change (SADS-C; Endicott & Spitzer, 1978)—An expanded SADS-C (exp-SADS-C) interview was used to assess affective symptoms

and episodes throughout the 4-month prospective assessment period. The exp- SADS-C is a semistructured interview that inquires about occurrence, duration, and severity of symptoms of mood, anxiety, psychotic, and substance use disorders since the prior assessment. The version used in the current study was expanded to allow for assignment of both DSM-IV and RDC diagnoses (see also Alloy et al., 2008; Francis-Raniere, Alloy, & Abramson, 2006). Exp-SADS-C interviewers were also blind to participants' phase 1 GBI results and SADS-L diagnoses. Based on 60 jointly rated LIBS Project interviews, interrater reliability for the exp-SADS-C was strong (κ>.80; Francis-Raniere et al., 2006). Results of a validity study for the LIBS Project indicated that 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). For the present study, a depressive or hypomanic symptom counted toward the symptom total if it received a minimum severity rating of three, which corresponds to a rating of clinically significant. Depressive and hypomanic symptom counts were then calculated for each of the two time points in the present study.

Life Events Scale (LES) and Life Events Interview (LEI)—Life events were assessed using a combination of self-report and interview measures. At each regular prospective assessment, participants completed the self-report LES. The LES employed in the LIBS Project is an expanded version of an earlier 134-item LES (Alloy & Clements, 1992; Needles & Abramson, 1990). The expanded version includes items assessing positive life events as well as negative ones. Each of the 193 items in this expanded version was carefully designed to minimize ambiguity and redundancy, and items were eliminated if deemed to directly reflect symptoms of affective disturbance. Both the original and expanded versions of the LES have demonstrated good reliability and validity (Francis-Raniere et al., 2006; Metalsky & Joiner, 1992; Needles & Abramson, 1990). Prior to the start of the study, a team of raters determined a consensus-based objective impact rating (OIR) for each LES item. The OIR was rated on a 4-point scale ranging from 0 (*no slight impact*) to 4 (*extreme impact*). Each event was also a priori categorized by the consensus team across a series of dimensions, including valence (negative/positive) and content (achievement/interpersonal).

Following completion of an LES, participants completed an LEI (Francis-Raniere et al., 2006). LEI interviewers were blind to phase 1 GBI scores, phase 2 diagnostic status, and concurrent symptoms and diagnoses obtained on the exp-SADS-C. The LEI served as a reliability and validity check on LES-reported events, facilitated precise dating of each event occurrence, and allowed for collection of contextual information. Explicit manualized probes and event definition criteria were used for each item, and any events not meeting criteria were disqualified by the interviewer. To determine the final contextualized OIR, the interviewer used the a priori determined objective rating as an anchor and could increase or decrease the a priori objective impact rating by one point, based on contextual information gathered from the LEI. In this way, contextualized objective impact ratings were obtained. Alloy and Abramson (1999) reported that participants correctly recalled 100% of major events using the combined LES and LEI procedures, when compared with a daily life event list that was prospectively generated by each participant throughout a monthlong period. Based on 40 LEIs reviewed in that study, the average interrater reliability for dating of events was .89 (Alloy & Abramson; see also Safford et al., 2007).

As the present study focuses on the active generation of life events, each LES item was categorized as dependent, independent, or other-focused. The dependent category comprised events that were most likely caused or highly influenced by the participant, such as academic probation, initiation of an important new friendship, or resolution of a significant fight or argument with family member. The independent category comprised events that were most likely beyond the participant's personal control, such as the death of a close

family member, being the victim of a random crime, medical illnesses, and natural disasters. The other-focused category comprised events that happened to other individuals in the participant's social network, and these were excluded from analyses.

For the present study, only events receiving a contextualized OIR of two (*moderate impact*) or above were counted towards the participant's event total. This criterion was applied to maximize consistency with other studies using this life events assessment procedure, and with much of the stress generation literature in general. Using the a priori established categorizations across dimensions of valence (negative/positive) and content (achievement/interpersonal), dependent LES items were assigned to one of four possible event types: negative interpersonal (e.g., "unwanted final break-up of romantic relationship with significant other"), positive interpersonal (e.g., "accepted into desired social group"), negative achievement (e.g., "not hired or promoted at job due to poor work performance, too few qualifications, etc."), and positive achievement (e.g., "achieved desired weight loss or gain"). In other words, by combining the ratings for event dependence, valence, and content, we examined events in the following categories: (a) overall independent events; (b) overall dependent events; and (c) specific dependent event categories (negative interpersonal, positive interpersonal, negative achievement, and positive achievement). Each event meeting inclusion criteria was counted, and events were summed to obtain the total number of events reported in each category over the follow-up period. Per-day event scores (i.e., frequencies) were then calculated by dividing the total number of reported events by the number of days elapsed since the prior assessment. These were finally multiplied by 100 to obtain a standardized measure of "events per 100 days."

Statistical Analyses

Within the BSD group, individuals diagnosed with bipolar II disorder did not significantly differ from those with cyclothymia/bipolar NOS on event rates or affective symptoms at either time point (magnitude of all t's <1.46, all p's>.15, except in T1 positive achievement events, $t = 1.84$, $p = .07$). Thus, to maximize power, the two groups were combined for the present analyses. Group differences were examined using chi-square and *t*-test analyses. Among BSD participants, specific event generation hypotheses were tested using a series of hierarchical linear regression analyses, with T1 depression and hypomania as the primary predictor variable and T2 events as the criterion variable. When extreme univariate outliers were removed from analyses, results remained unchanged with respect to pattern, magnitude, and significance. Thus, univariate outliers were recoded to a value representing 2.5 standard deviations from the group mean for that event category and were retained in the final analysis. Multivariate outliers were excluded from analyses (range of number of deleted data points per tested model = 0–6).

Results

Preliminary Analyses

Table 1 presents basic demographic and clinical characteristics for the present sample. Bipolar spectrum participants did not differ significantly from normal participants on the following: gender ($\chi^2[1] = .11$, $p = .75$); age ($t[159] = -.36$, $p = .72$); or ethnic group (White vs. non-White) ($\chi^2[1] = .83$, $p = .36$). As would be expected, bipolar spectrum participants experienced more depressive and hypomanic symptoms at T1 and at T2 (all p's <.01). Males and females did not differ on age ($t[87.30] = .61$, $p = .54$) or ethnic group (i.e., White vs. non-White) ($\chi^2[1] = .01$, $p = .91$). There were no gender differences in affective symptoms, except that females experienced more depressive symptoms at T2 ($t[152] = -1.96$, $p = .05$). Event rates (independent, dependent, and specific categories) did not significantly differ by gender at either time point (magnitude of all t's≤1.57, all p's≥.12). Across both time points,

neither age (magnitude of all r's≤.14, all p's≥.08) nor ethnic group (i.e., White vs. non-White; magnitude of all t's ≤ 1.70, all p's≥.09) was significantly correlated with event frequencies. Table 2 presents intercorrelations between demographic and relevant clinical variables.

Disqualified event (DNQ) rates were computed by dividing the number of objectively disqualified events by the total number of reported events over the follow-up period. At T1, normal control participants tended to have higher rates of DNQ events (t[156.92] = 1.85, p = .07); there were no group differences in T2 DNQ rates (t[161] = −.82, p = .42). DNQ rates did not significantly differ by gender: T1 (t[163] = −.22, p = .83); T2 (t[163] = −.34, p = .73). Finally, depressive and hypomanic symptoms were not correlated with concurrent DNQ rates at T1 or T2 (magnitude of all r's≤.08, all p's≥.33). Taken together, results of the DNQ event analysis suggest that our findings are not confounded by reporting bias due to diagnostic status, mood state, or gender.

Main Analyses

Diagnostic group differences in life events—Prior to examining the event generation process in specific valence and content domains, we examined whether it occurred at the overall event level. Cross-sectional analyses indicated that the BSD and normal control groups had similar rates of total independent events at T1 and T2, t(156) = −1.31, p = .19 and t(156) = −.82, p = .41, respectively, and of dependent events at T1, t(154) = −1.54, p = .13. However, bipolar participants experienced significantly higher rates of total dependent events at T2, t(154) = −2.56, p = .01. Even after controlling for initial event levels, hierarchical linear regression analyses indicated that BSD predicted increases in T2 dependent but not independent events (B = 1.36, standard error [SE] B = .67, β = .14, p = .04; and B = −.15, SE B = .32, β = .04, p = .64, respectively). When dependent events were analyzed by specific category, rates of T1 negative interpersonal events were significantly higher in bipolar spectrum participants than in normal controls: t(120.90) = −2.94, p<.01. Rates of occurrence were comparable between the two groups for all other specific dependent event categories at both time points (magnitude of all t's≤1.30, all p's≥.20). Hierarchical linear regression models controlling for specific dependent T1 event types indicated that a bipolar spectrum diagnosis did not predict changes in any of the four specific categories.

Prospective test of the relationship between T1 symptoms and T2 events—A series of hierarchical linear regressions was conducted among BSD participants to determine whether depressive or hypomanic symptoms at T1 significantly predicted T2 changes in any of the four dependent event categories. T1 event frequency in the category of interest was entered into the first step of each regression model, which allowed for a test of residual change scores as opposed to absolute event frequencies at T2. The resulting analysis represents a clearer examination of a psychosocial event generation process over time, given that variance likely attributable to chronic or baseline events is partialed out. Joiner and colleagues (Joiner, Wingate, Gencoz et al., 2005) point out that in light of the typically high association between earlier and later stress, the ability of initial symptoms to predict a residual stress term is a stringent test of the stress generation hypothesis. In the second step of each model, we entered gender. The third step comprised the primary predictor variables, T1 depression and T1 hypomania. Finally, we entered the two mean-centered interaction terms for gender with depression, and gender with hypomania.

Separate analyses were conducted for independent and dependent events. Gender significantly predicted independent events, with males experiencing higher rates of independent events at T2 (B = −1.11, SE B = .52, β = −.23, p = .04). T1 depressive

symptoms showed a trend association with T2 independent events (B = .09, SE B = .05, β = .19, p = .08). None of the other model variables predicted changes in T2 independent event rates. There was no main effect of gender or symptoms on T2 dependent events, but hypomanic symptoms tended to predict greater dependent event increases in males than in females (for the gender by hypomanic symptom interaction term, B = −1.20, SE B = .63, β = −.39, p = .06).

Table 3 presents results of analyses evaluating event generation in the negative and positive interpersonal domains. Depressive symptoms marginally predicted *decreases* in positive interpersonal events, whereas hypomanic symptoms marginally predicted increases in negative interpersonal events. Gender significantly predicted positive interpersonal events and moderated the relationship between hypomania and negative interpersonal events, such that hypomania predicted increased negative interpersonal events in men but not in women.

Table 4 presents results of analyses evaluating event generation in the negative and positive achievement domains. Hypomanic symptoms significantly predicted increased rates of positive achievement events and tended to predict increases in negative achievement events as well. T1 depressive symptoms failed to predict T2 negative or positive achievement events. There was no main effect of gender on negative or positive achievement events, nor did gender moderate any symptomevent relationship.

The role of full-blown affective episodes—The stress generation theory specifies that the generation of stress should occur during periods of severe mood symptomatology, but not exclusively so. Thus, the stress generation theory is not uniquely supported if changes in event frequencies are found to be simply correlates of concurrent fullblown affective episodes. When analyses controlled for the presence of a major depressive or hypomanic episode over the follow-up period, results remained largely unchanged. Exceptions were as follows: gender no longer moderated a relationship between hypomanic symptoms and negative interpersonal events; gender no longer significantly predicted positive interpersonal events; and the main effect of hypomanic symptoms on negative achievement events was no longer marginally statistically significant (new p = .16). Thus, hypomanic symptoms continued to significantly predict increased positive achievement events (B = .39, SE B = .15, β = .34, p = .01) and predict increased negative interpersonal events at a trend level (B = .72, SE B = .43, β = .21, p<.10). Depressive symptoms also continued to predict fewer positive interpersonal events at a trend level (B = −.09, SE B = .04, β = −.26, p = .05).

Discussion

This study examined whether the stress generation theory, which has been well supported in unipolar depression, is also relevant to bipolar spectrum disorders. To our knowledge, it is the first prospective study to extend the stress generation theory to such a sample. The potential relevance of positive events to the course of BSD led us to test a broader "psychosocial event generation hypothesis," as opposed to a more specific stress generation hypothesis.

Results of the present analyses provide some support for the psychosocial event generation hypothesis. Although there were no group differences in event rates at baseline, individuals with BSD experienced more dependent, but not independent, events over the follow-up period. In parallel, this is consistent with the original stress generation theory, which posits that individuals with a history of depression should be more likely to generate stress over time (Hammen, 1991). A bipolar spectrum diagnosis did not predict changes in rates of specific dependent event categories. The heterogeneous nature of BSD may account for the significant findings among overall dependent events, but not among specific events.

At the symptom level, event generation processes appeared to be specific to affective polarity, event content, and event valence. Contrary to our hypotheses, the interpersonal model of stress generation in depression was not strongly supported among those with BSD. That is, irrespective of gender, initial depressive symptoms did not prospectively predict increases in negative interpersonal events. Initial depressive symptoms did predict fewer positive interpersonal events at T2, but this finding did not attain conventional statistical significance. Theoretically consistent with the stress generation model of depression, initial depressive symptoms failed to predict increases in positive interpersonal or achievement events. Depressive symptoms also failed to predict the overall number of dependent events.

In contrast with depressive symptoms, hypomanic symptoms did predict increases in negative interpersonal events, particularly among males. This suggests that in our sample, hypomanic symptoms and their associated behavioral patterns may have been more disruptive to the male than the female social network. Findings, thus, reflected a male interpersonal stress generation process in hypomania, rather than a female interpersonal stress generation process in depression. As this study is the first to report a male interpersonal stress generation process in hypomania, results should be considered preliminary. If replicated, however, it will be important to explore the mechanisms by which the male and female social networks are differentially affected by hypomanic symptoms.

For both genders, initial hypomanic symptoms produced an event generation effect in the positive achievement domain, and they also tended to predict increased negative achievement events. These findings are consistent with growing evidence, suggesting that both negative *and* positive life events may be relevant to hypomanic symptom expression (Johnson, 2005a). For example, the expanded BAS dysregulation theory posits an association between the course of BSD and negative and positive achievement-relevant and reward-relevant events (Urošević et al., 2008). In a separate subset of LIBS Project data, Urošević and colleagues (in press) found that bipolar spectrum participants experienced elevated rates of events categorized as BAS-activating and BAS-deactivating, as compared with a normal control group. As the literature converges on the importance of positive and negative achievement, research should identify a theory-driven classification scheme that most precisely maps onto the mechanisms underlying the relation between life events and BSD.

Psychosocial event generation effects were essentially unchanged after controlling for the presence of major depressive or hypomanic episodes during the follow-up period. The role of gender did appear to change in some of the tested models. It is important to note that controlling for the presence of major depressive or hypomanic episodes during the follow-up period is overly conservative, given that event generation is also expected to occur during those periods. The purpose of performing this analytic step was to determine whether event generation effects were wholly accounted for by episodes, and they were not. That stress generation occurs outside such periods is important to the theory's purported ability to explain relapses or recurrences over time.

In sum, the present results support an event generation hypothesis of BSD in suggesting that individuals with BSD generate overall increases in dependent events. At the symptom level, the event generation process appears to occur in more specific event domains. Specifically, hypomanic symptoms predicted increased positive achievement events. Hypomanic symptoms also tended to predict increased negative achievement and negative interpersonal events, whereas depressive symptoms tended to predict decreased positive interpersonal events. However, these latter findings occurred only at a trend level. Gender differences emerged only in the relationship between hypomanic symptoms and negative interpersonal events.

The current study is the first of its kind and has numerous strengths. First, interpersonal and achievement stress generation processes have not yet been examined in separate relation to both depressive and hypomanic symptoms. Second, analysis of positive and achievement-oriented life events, in addition to negative and primarily interpersonal ones, afforded a more comprehensive picture of the event generation process. These methods allowed us to increase sensitivity to understudied processes in BSD. Third, both mood and life events data were collected prospectively, using rigorous standardized interviews with demonstrated high reliability and validity. In particular, the Life Events Scale/Life Events Interview uses a systematic combination of contextual and objective rating procedures. This methodology allowed us to examine DNQ rates and, thereby, rule out reporting bias as a major confound in our results. Interviewer bias was also minimized by blinding life events interviewers to diagnostic status and concurrent mood symptoms. Finally, the present results reflect conservative tests of our hypotheses, because of partialing out variance accounted for by opposite-category mood symptoms and samecategory life events at T1 (see Joiner, Wingate, Gencoz et al., 2005).

Several study limitations should be noted as well. One limitation involves the use of an undergraduate sample with BSD and relatively low levels of bipolar symptom severity; generalizability of findings to individuals with full-blown bipolar I disorder cannot be assumed. For example, it is possible that those with more severe bipolar I disorder are less likely to generate positive events and more likely to generate negative events. Future studies should also test for differences in patterns of event generation between individuals with bipolar II disorder, cyclothymia, or bipolar NOS, as the current study was underpowered for such an analysis. Our sample size of 75, though relatively large in comparison to other examinations of stress generation in BD (e.g., Hammen, 1991; Reilly-Harrington et al., 1999), may have provided insufficient power to detect smaller event generation effects or interactions. These sample size limitations, in concert with a number of results occurring at only a trend level, indicate that findings should be considered preliminary and warrant further replication. Another limitation of the present study is the challenge inherent in measuring psychosocial events and stress. These constructs are highly complex and can be conceptualized in many different ways (see Monroe, 2008). As noted above, however, we took several measures to improve accuracy and minimize bias, following the guidelines provided by Johnson (2005a). Finally, analyses included symptom and event totals at two discrete time points, allowing for examination of only one piece of a hypothesized cyclical process.

In conclusion, results provide preliminary evidence that the stress generation theory of unipolar depression may also be relevant to the course of bipolar spectrum disorders. Future research should examine whether similar findings emerge among individuals diagnosed with bipolar I disorder. It will also be important to investigate possible mechanisms of the different event generation patterns, with particular attention to behavioral response styles that precipitate the occurrence of specific types of life events. In unipolar stress generation, researchers have identified a number of potential mediators and moderators of the process, including poor interpersonal problem solving (Davila et al., 1995) or social skills (Segrin, 2001), excessive reassurance seeking (Joiner, Wingate, Gencoz et al., 2005), attachment style and personality variables (Hankin et al., 2005; Nelson et al., 2001; Shih, 2006), cognitive styles (Safford et al., 2007), and hopelessness (Joiner, Wingate, & Otamendi, 2005); it will be important to examine the role of such constructs in bipolar event generation as well. Also, as stress generation is likely a nuanced and transactional process, examining symptoms and life events across additional time points will enable us to better understand how the process unfolds over time.

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Table 1

Sample Demographics and Clinical Characteristics (N = 164)

	Normal (*N* = 87)	Bipolar (*N* = 77)
Gender		
Male	31.8%	34.2%
Female	68.2%	65.8%
Age	19.66 (1.99)	19.77 (1.84)
Ethnicity		
Caucasian	54.1%	60.3%
African American	23.5%	26.5%
Hispanic	4.7%	2.9%
Asian	7.6%	2.8%
Other	11.8%	7.4%
T1 mood symptomatology		
Depressive symptoms**	1.74 (2.29)	6.21 (4.88)
Hypomanic symptoms**	.34 (.82)	1.49 (2.02)
T2 dependent events per 100 days		
Total dependent*	4.95 (4.55)	6.95 (5.22)
Negative interpersonal	4.33 (7.33)	5.89 (7.84)
Positive interpersonal	1.45 (1.80)	1.50 (1.55)
Negative achievement	1.40 (2.13)	1.85 (2.20)
Positive achievement	3.11 (2.96)	2.80 (2.57)
T2 DNQ rates	10.32%	12.66%

Note. Means are presented with standard deviations in parentheses. Normal = healthy control group; bipolar = bipolar spectrum group (bipolar II, not otherwise specified, or cyclothymia); T1 depressive symptoms = sum of depressive symptoms from Schedule for Affective Disorders-Change (SADS-C) at time 1; T1 hypomanic symptoms = sum of hypomanic symptoms from SADS-C at time 1; T2 total dependent events = number of total events reported minus number of events objectively disqualified; T2 disqualified event (DNQ) rates = number of events objectively disqualified by interviewer, divided by total number of events reported.

* $p<.05$;

** $p<.01$.

Table 2

Intercorrelations Between Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Bipolar status	–															
2. Gender	−.03	–														
T1 events																
3. Total dependent	.12	−.06	–													
4. Negative Interpersonal	.24**	−.06	.71**	–												
5. Positive Interpersonal	.01	−.09	.67**	.48**	–											
6. Negative Achievement	.06	−.07	.58**	.40**	.29**	–										
7. Positive Achievement	−.09	−.07	.48**	.21**	.32**	.11	–									
T1 symptoms																
8. Depression	.52**	.04	.41**	.44**	.25**	.21**	.11	–								
9. Hypomania	.36**	−.03	.19*	.17*	.13	.04	.01	.46**	–							
T2 events																
1. Total dependent	.20*	−.02	.54**	.55**	.51**	.19*	.24**	.31**	.21**	–						
11. Negative Interpersonal	.10	.02	.55**	.58**	.41**	.19*	.21**	.28**	.23**	.79**	–					
12. Positive Interpersonal	.01	.12	.44**	.38**	.46**	.26**	.28**	.07	.05	.57**	.42**	–				
13. Negative Achievement	.10	−.06	.48**	.49**	.41**	.33**	.15	.25**	.31**	.49**	.50**	.27**	–			
14. Positive Achievement	−.06	−.13	.24**	.18*	.31**	.05	.35**	.04	.19*	.36**	.20*	.34**	.07	–		
T2 symptoms																
15. Depression	.43**	.16	.31**	.32**	.21**	.12	.02	.67**	.43**	.34**	.36**	.16*	.32**	−.06	–	
16. Hypomania	.30**	.05	.14	.13	.05	.07	−.04	.48**	.67**	.03	.10	−.06	.24**	−.02	.49**	–

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Table 3

Time 1 Depression and Hypomania Predicting Negative and Positive Interpersonal Events Among Bipolar Spectrum Participants (N =75)

	Negative interpersonal					Positive interpersonal			
	B	**SE *B***	***β***	***t***		***B***	**SE *B***	***β***	***t***
Step 1					*Step 1*				
T1 negative interpersonal	.35	.06	.57	5.74**	T1 positive interpersonal	.24	.07	.38	3.41**
Step 2					*Step 2*				
T1 negative interpersonal	.35	.06	.57	5.70**	T1 positive interpersonal	.27	.07	.43	4.07**
Gender	.47	1.63	.03	.29	Gender	1.06	.34	.33	3.09**
Step 3					*Step 3*				
T1 negative interpersonal	.36	.07	.59	5.43**	T1 positive interpersonal	.31	.07	.48	4.43**
Gender	.68	1.62	.04	.42	Gender	1.14	.34	.36	3.34**
T1 depression	−.10	.19	−.06	−.53	T1 depression	−.07	.04	−.21	−1.83^
T1 hypomania	.75	.41	.19	1.81^	T1 hypomania	.08	.09	.10	.91
Step 4					*Step 4*				
T1 negative interpersonal	.40	.07	.65	5.82**	T1 positive interpersonal	.33	.08	.52	4.34**
Gender	−.59	1.76	−.04	−.33	Gender	1.00	.38	.31	2.63*
T1 depression	−.54	.33	−.34	−1.64	T1 depression	−.12	.07	−.38	−1.74^
T1 hypomania	2.30	.75	.59	3.07**	T1 hypomania	.15	.16	.20	.96
Gender × depression	.57	.37	.30	1.54	Gender × depression	.07	.08	.20	.92
Gender × hypomania	−2.13	.88	−.46	−2.42*	Gender × hypomania	−.10	.19	−.11	−.54

Note. SE =standard error; T1 negative/positive interpersonal =T1 sum of negative or positive interpersonal events per 100 days; bipolar status =bipolar spectrum diagnosis versus normal control; T1 depression/hypomania =sum of depressive or hypomanic symptoms from Schedule for Affective Disorders-Change interview at T1. Negative interpersonal model: *Step 1 F* = 32.89**, R^2 = .32; *Step 2 F* = 16.27**, *ΔF* = .08, R^2 =.32, ΔR^2<.01; *Step 3 F* =9.12**, *ΔF* = 1.65, R^2 =.36, ΔR^2 = .03; *Step 4 F* = 7.52**, *ΔF* = 3.14*, R^2 = .41, and ΔR^2 = .06. Positive interpersonal model: *Step 1 F* =11.65**, R^2 = .14; *Step 2 F* = 11.32**, *ΔF* = 9.55**, R^2 = .25, ΔR^2 = .11; *Step 3 F* = 6.65**, *DF* = 1.73, R^2 = .29, ΔR^2 = .04; *Step 4 F* = 4.51**, *ΔF* = .45, R^2 = .30, and ΔR^2 = .01.

*p<.05;

**p <.01;

^p<.10.

Table 4

Time 1 Depression and Hypomania Predicting Negative and Positive Achievement Events Among Bipolar Spectrum Participants (N =75)

	Negative achievement					Positive achievement			
	B	**SE *B***	***β***	***t***		***B***	**SE *B***	***β***	***t***
Step 1					*Step 1*				
T1 negative achievement	.34	.07	.52	5.04**	T1 positive achievement	.33	.14	.28	2.46*
Step 2					*Step 2*				
T1 negative achievement	.34	.07	.52	5.06**	T1 positive achievement	.33	.14	.28	2.39*
Gender	−.65	.47	−.14	−1.38	Gender	−.04	.63	−.007	−.06
Step 3					*Step 3*				
T1 negative achievement	.35	.07	.53	5.03**	T1 positive achievement	.30	.13	.26	2.28*
Gender	−.61	.47	−.13	−1.31	Gender	.02	.61	.005	.04
T1 depression	.002	.05	.003	.03	T1 depression	−.02	.06	−.03	−.26
T1 hypomania	.22	.12	.20	1.81^	T1 hypomania	.42	.15	.33	2.79**
Step 4					*Step 4*				
T1 negative achievement	.32	.07	.50	4.61**	T1 positive achievement	.29	.13	.25	2.23*
Gender	−.28	.52	−.06	−.54	Gender	.22	.67	.04	.33
T1 depression	.11	.09	.24	1.19	T1 depression	.05	.11	.09	.43
T1 hypomania	.20	.22	.18	.90	T1 hypomania	.70	.28	.55	2.53*
Gender × depression	−.15	.11	−.29	−1.43	Gender × depression	−.10	.13	−.16	−.76
Gender × hypomania	.008	.26	.006	.03	Gender × hypomania	−.40	.33	−.27	−1.22

Note. SE =standard error; T1 negative/positive achievement =T1 sum of negative or positive achievement events per 100 days; bipolar status =bipolar spectrum diagnosis versus normal control; T1 depression/hypomania =sum of depressive or hypomanic symptoms from Schedule for Affective Disorders-Change interview at T1. Negative achievement model: *Step 1* F = 25.44**, R^2 = .27; *Step 2* F = 13.84**, ΔF = 1.90, R^2 =.29, ΔR^2 =.02; *Step 3* F = 8.06**, ΔF = 1.91, R^2 = .33, ΔR^2 = .04; *Step 4* F = 5.79**, ΔF = 1.16, R^2 = .35, and ΔR^2 = .02. Positive achievement model: *Step 1* F = 6.03*, R^2 = .08; *Step 2* F = 2.97, ΔF = .003, R^2 = .08, ΔR^2 = <.001; *Step 3* F = 3.69**, ΔF = 4.14*, R^2 = .18, ΔR^2 = .10; *Step 4* F = 3.04*, ΔF = 1.59, R^2 = .22, and ΔR^2 = .04.

* p<.05;

** p <.01;

^ p<.10.