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Aggression and Impulsivity as Predictors of Stress Generation in Bipolar Spectrum Disorders

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Abstract

Background—Some evidence suggests that individuals with bipolar spectrum disorders (BSD) generate stressful life events, contributing to a more severe course of disorder. A recent update to the Behavioral Approach System (BAS) dysregulation theory of BSD highlights the need to investigate anger as approach motivation. Although research has shown that individuals with BSD generate stress, it is unclear whether personality traits characteristic of BSD, such as aggression and impulsivity, are related to this stress generation.

Methods—The current longitudinal study employed multilevel modeling to examine stress generation in a sample of 104 individuals with BSD and 96 healthy controls. We examined rates of BAS-deactivating, BAS-activating, and Anger-evoking life events over a period of up to 4.5 years as a function of levels of aggression and impulsivity.

Results—Individuals with BSD reported significantly higher numbers of dependent Anger-evoking events and BAS-deactivating events, but not dependent BAS-activating events, than controls. Trait levels of hostility and impulsivity predicted all types of events, although bipolar diagnosis remained a significant predictor of BAS-deactivating and Anger-evoking events. Limitations: The life events measures were not designed to assess Anger-evoking events; further research should replicate these findings and develop more finely tuned assessments of stressful anger events. In addition, the sample was not a clinical sample.

Conclusions—This study adds to the literature on stress generation in BSD; trait level personality differences predict stress generation, beyond bipolar diagnosis. This also further

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establishes the importance of including anger-evoking events in the BAS model of BSDs and stress generation.

Keywords

bipolar spectrum disorders; stress generation; aggression; impulsivity

Bipolar spectrum disorders (BSDs) are estimated to affect 4.4% of American adults (Merikangas et al., 2007), and are associated with tremendous personal and societal costs. Individuals with BSDs may experience impairment across a variety of domains, including social disruption, unemployment, substance abuse, familial discord, and suicide attempts (Depp et al., 2012; Merikangas et al., 2007; Judd et al., 2008; Oquendo et al., 2000).

The Behavioral Approach System (BAS) hypersensitivity theory of BSDs (Alloy & Abramson, 2010; Depue & Iacono, 1989; Depue et al., 1987; Urosevic et al., 2008) proposes that individuals with and at risk for BSDs may experience dysregulation of the BAS, a proposed neurobiological system thought to underlie goal-striving and reward-seeking motivation and behavior, and associated emotions such as elation and anger (Gray, 1982, 1994; Depue & Collins, 1999). Among individuals at risk for BSDs, dysregulation of the BAS is thought to lead to excessive approach motivation and behavior in response to rewards or goal-relevant life events, leading to hypomanic/manic symptoms. Conversely, negative life events, particularly failures, obstacles to goals, and losses, may result in excessive down-regulation of the BAS, resulting in the symptoms of depression (Depue & Iacono, 1989). According to this theory, beyond biological and cognitive vulnerability to BSDs, the presence of triggering life events is important for the development of mood episodes.

Research has investigated the impact of life events on the course of BSDs (see Alloy et al., 2005 see Alloy et al., 2009a; Bender & Alloy, 2011; Johnson, 2005 for reviews). Individuals with BSDs experiencing high stress have been shown to have a 4.5-fold increase in risk of relapse compared to those experiencing low stress (Ellicott et al., 1990), and a prospective study of individuals with BSDs demonstrated nearly a 4-fold increase in negative, stressful life events prior to relapse (Hunt et al., 1992). More specifically, stressful life events that occur independent of the person's behavior have been linked to mood episode onset among individuals with BSDs (Aronson & Shukla, 1987), and individuals with BSDs have reported significantly more negative, independent life events prior to episode onset than individuals with unipolar depression (Perris, 1984).

More recent research has examined polarity-specific effects of life events on mood symptoms in individuals with bipolar spectrum disorders. Negative life events have been shown to predict increases in depressive symptoms among individuals with Bipolar I disorder (Johnson et al., 2008). Further research suggests that negative cognitive style may also moderate this relationship. Three studies (Alloy et al., 1999; Francis-Raniere et al., 2006; Reilly-Harrington et al., 1999) have demonstrated that negative life events are only predictive of prospective increases in depression among bipolar spectrum individuals with depressogenic cognitive styles.

Life events also have been associated with onset and course of manic or hypomanic episodes and symptoms (see Alloy et al., 2005; 2009; Bender & Alloy, 2011; Johnson, 2005 for reviews). Goal attainment events specifically, as opposed to positive events in general, predicted increases in manic symptoms but not depressive symptoms in individuals with Bipolar I disorder (Johnson et al., 2000; 2008). Similarly, goal-striving, pre-attainment life events also predicted hypomanic episodes among individuals with BSDs, as Nusslock and

colleagues (2007) demonstrated that university students with BSDs were significantly more likely to experience hypomanic, but not depressive, episodes when studying for and taking final exams compared to a control period, and this relationship was moderated by BAS-sensitivity. Given that the final exam period is a time associated with goal-striving among university students, this finding suggests that goal striving may be associated with onset of hypomanic episodes, particularly for individuals with high BAS-sensitivity.

Previous research has suggested that individuals with bipolar spectrum disorders may not only be more reactive to stressful events, but may also experience more of these events (Bender et al., 2010; Urošević et al., 2010). The stress generation theory of unipolar depression proposes that individuals with or at risk for depression have cognitive and interpersonal characteristics that cause future negative life events, which, in turn, may precipitate increases in depressive symptoms (Hammen, 1991; 2006). Consistent with this model, numerous prospective studies have demonstrated that individuals with or at risk for depression experienced increased numbers of negative life events that are dependent on their behavior, even during periods of remission (see Liu & Alloy, 2010 for a review).

More recently, an extension of the stress generation theory has been applied to BSDs (Bender et al., 2010; Urošević et al., 2010). Similar to the way in which individuals with or at risk for depression may experience more dependent negative life events as a result of their symptomatology, cognitive characteristics, or interpersonal behaviors, it has been proposed that individuals with BSDs may also experience increased numbers of dependent life events. More specifically, the event generation model of BSDs suggests that individuals with bipolar spectrum disorders may not only generate negative life events that put them at risk for a depressive episode, but may also generate increased BAS-activating events, which may put them at risk for (hypo)mania.

To date, only two studies have investigated a stress generation model of BSDs. Urošević and colleagues (2010) compared individuals with bipolar II disorder and cyclothymia to healthy controls, and consistent with a stress generation model, demonstrated that individuals in the bipolar spectrum experienced higher rates of BAS-activating and BAS-deactivating events, but not goal-attainment events. Further, the number of BAS-activating events predicted the number of BAS-deactivating events only for individuals with bipolar spectrum disorders. These findings suggest that individuals with BSDs may engage in more goal-striving pursuits, but may subsequently experience more failures, than healthy individuals. Bender and colleagues (2010) demonstrated that individuals in the bipolar spectrum experienced higher numbers of dependent life events overall compared to healthy controls. Additionally, hypomanic symptoms predicted increases in negative interpersonal events and positive and negative achievement events, providing further evidence that bipolar symptomatology may increase the rates of both BAS-activating and BAS-deactivating stressors, and that specific types of events within these categories may be particularly relevant to BSDs.

However, within the broad categories of BAS-activating and BAS-deactivating events, there may be specific types of life events that are relevant to BSDs. Although BAS activation traditionally has been conceptualized as underlying positive affect, more recent research has linked BAS activation with anger as well. Anger, although negatively valenced, is associated with approach motivation (see Carver & Harmon-Jones, 2009 for review) as it is theorized to arise from blockage of movement towards a desired goal (Berkowitz, 1993). This hypothesis has received support in psychophysiological studies, as both anger and approach motivation have been associated with increased left frontal EEG activation (Harmon-Jones & Sigelman, 2001; Harmon-Jones et al., 2002). In recognition of these findings, researchers have begun to call for more in-depth investigation into anger-evoking events as a subclass of

BAS-activation events that may also precipitate (hypo)mania, particularly, irritable (hypo)mania (Urosevic et al., 2008).

Given that irritability is a key feature of BSDs in both (hypo)manic (Hanwella & de Silva, 2011) and depressive states (Benazzi & Akiskal, 2005; see Lara et al., 2006 for review), and that risk for BSDs has been associated with elevated trait anger (Cooke & Jones, 2009), the role of anger-evoking events in the course of BSDs demands further study. Research has evidenced differential levels of stress generation in individuals with high versus low levels of trait aggression (e.g., Rudolph et al., 2000), suggesting that for those high in anger and its behavioral correlate, aggression, stress generation may be a major concern.

Similarly, impulsivity is hypothesized to be a core feature of BSDs (Swann et al., 2009), and appears to be a trait-like personality characteristic that is elevated and stable across the phases of bipolar illness (e.g., Swann et al., 2001). Moreover, impulsivity is associated with risky, reward-seeking behaviors that may have negative consequences, and it predicts comorbid substance use problems among bipolar spectrum individuals (Alloy et al., 2009b), as well as a worse course of bipolar spectrum disorder (Kwapil et al., 2000) including conversion to full-blown Bipolar I disorder (Alloy et al., 2012). Consequently, impulsivity is another trait associated with BSDs that may contribute to the generation of BAS-activating and anger-evoking events. Consistent with this possibility, Nusslock and colleagues (2008) found that bipolar spectrum individuals with relatively low impulsivity showed lower achievement-related stress generation as compared to those with high levels of impulsivity.

Although research has shown that individuals with BSDs experience higher numbers of BAS-relevant life events (Bender et al., 2010; Urosevic et al., 2010), it remains unclear whether it is bipolar symptoms themselves or other characteristics associated with BSDs that contribute to the generation of BAS-relevant events. Indeed, cognitive and personality styles associated with unipolar depression have been found to predict stress generation even in the absence of depressive symptoms (see Liu & Alloy, 2010 for a review). Thus, personality traits associated with BSDs, such as impulsivity and aggression, may also have effects on life event generation that have yet to be elucidated, over and beyond bipolar mood symptoms.

The current study examined whether personality characteristics were associated with generation of BAS-activating events and BAS-deactivating events, above and beyond the presence of a bipolar diagnosis. We hypothesized that individuals with higher levels of aggression would experience increased dependent anger-evoking events specifically, as well as dependent BAS-activating life events generally, but not more dependent BAS-deactivating events. We also hypothesized that high levels of trait impulsivity would predict heightened levels of dependent BAS-activating, BAS-deactivating, and anger-evoking events, over and above a bipolar spectrum diagnosis. Finally, we hypothesized that those with a more severe bipolar diagnosis in our sample (i.e. Bipolar II) would experience heightened levels of these dependent life events compared to participants with a less severe (Cyclothymia or Bipolar NOS) diagnosis, but that this effect would be mediated by trait aggression and/or impulsivity. In order to investigate generation of reward-related BAS activating events independently from anger-evoking BAS-activating events, we examined them separately in our analyses, although both are hypothesized to tap the same neurobiological BAS system (Urosevic et al., 2008; 2010).

Methods

Participants and Procedures

The current study uses a sample from the Longitudinal Investigation of Bipolar Spectrum Disorders Project (LIBS; Alloy et al., 2008; 2012). Participants for the LIBS Project were recruited from Temple University and the University of Wisconsin-Madison through a two-stage screening process. In phase I, 20,500 individuals were screened with the General Behavior Inventory (GBI; Depue et al., 1989), a first-stage case identification measure, to assess whether they would be eligible for the bipolar spectrum group or the normal control group. Based on GBI cutoffs (see measures), eligible participants were invited for the diagnostic screening phase of the study. As this study was designed to examine predictors of onset of first manic episode, only participants who met criteria for Diagnostic and Statistical Manual of Mental Disorders 4th Edition (*DSM-IV*; American Psychiatric Association, 1994) or Research Diagnostic Criteria (RDC; Spitzer et al., 1978) diagnoses of Bipolar II, Bipolar NOS, or Cyclothymia were invited to participate in the longitudinal part of the study and comprised the bipolar spectrum group. Participants who did not meet criteria for any *DSM-IV* or RDC Axis I psychiatric disorder and had no family history of BSDs were asked to participate in the control group. Following the two-stage screening process, a total of 206 participants met criteria and were included in the bipolar spectrum group (age $M = 19.6$, $SD = 1.6$; 62.56% female; 68.9% Caucasian) and 208 participants were included in the normal control group ([age $M = 19.7$, $SD = 1.5$; 59.22% female; 72.8% Caucasian] Alloy et al., 2008).

After the screening processes, participants completed a Time 1 baseline assessment and then were interviewed every 4 months ($M=5.30$, $SD=3.83$). During these assessments, mood symptoms, expanded SADS-Change diagnostic interviews (Spitzer & Endicott, 1978), and life events that occurred between interviews were assessed. In order to assess the occurrence and impact of positive and negative life events in the past 4 months, participants completed a self-report questionnaire (Life Events Scale, [LES; Francis-Raniere et al., 2006]) and semi-structured interview (Life Events Interview [LEI; Francis-Raniere et al., 2006]) modeled after the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978).

The present study used a subsample of the LIBS project sample and included participants who completed a minimum of two 4-month visits and a maximum of thirteen visits ($M=5.37$, $SD=3.28$). Given our hypothesis regarding severity of diagnosis, we divided the bipolar spectrum group into two distinct groups to determine if the stress generation hypothesis was evident in a stepwise function. For the current study, we only included participants who had full data on all of our variables of interest. Therefore, the final sample included 200 participants, with 72 in the Bipolar II group, 32 in the Cyclothymia/Bipolar NOS group, and 96 in the normal control group. The bipolar sample as a whole was 68.9% Caucasian, 13.1% African-American, 5.1% Hispanic, 3.6% Asian, 0.5% Native American, and 8.2% Other. The normal control sample was 72.8% Caucasian, 12.1% African-American, 3.4% Hispanic, 4.4% Asian, 0.5% Native American, and 6.8% Other. In addition, all three groups did not differ from each other on age, gender, race, or months in the study.

Measures

General Behavior Inventory (GBI; Depue et al., 1989). The GBI is a 73-item, self-report questionnaire, in which participants rate bipolar symptoms with (intensity and duration) on a 4-point scale (1=never, 4=very often). The GBI was validated by findings indicating similar rates of mood disorders in a large sample as in the general population, high specificity between mood disorders, and high correlations with interview ratings (Depue et al., 1989). We employed the GBI cutoffs recommended by Depue and colleagues (1989), whereby

participants who scored ≥ 11 on the Depression scale as well as ≥ 13 on the Hypomanic-Biphasic scale were potentially eligible for the bipolar spectrum group, whereas participants below these cutoffs on both scales were potentially eligible for the control group.

Schedule for Affective Disorders and Schizophrenia-Lifetime (SADS-L; Endicott & Spitzer, 1978). In Phase II of the screening process, diagnostic interviews were conducted using an expanded version of the SADS-L to establish diagnosis based on *DSM-IV* or RDC criteria. The SADS-L is a well-validated diagnostic interview that has high inter-rater reliability (Endicott & Spitzer, 1978), including in the LIBS Project ($\kappa > .96$ for bipolar spectrum diagnoses; Alloy et al., 2008; 2012).

Life Events Scale (LES; Francis-Raniere et al., 2006). At each 4-month prospective assessment, participants first completed the self-report LES that assesses 193 a priori defined life events that span various domains (e.g., employment, family, education, relationships). The LES assesses both negative and positive events ranging in severity from major life events to more minor events. The LES includes a number of BAS-activating, BAS-deactivating, and anger-evoking events that were categorized prior to administration, as discussed below. The LES demonstrates good reliability and validity (Francis-Raniere et al., 2006).

Life Events Interview (LEI; Francis-Raniere et al., 2006). Following completion of the LES, participants were administered the LEI, a semi-structured interview designed to serve as a reliability and validity check on the LES-reported events, facilitate precise dating of events, and allow for contextual information to be collected. Interview based life events scales are designed to assess the objective nature of events as an attempt to combat reporter bias (Monroe & Roberts, 1990). As such, the LEI is accompanied by an Event Specific Criteria and Probes (ESCP) manual to provide clear definitions of the criteria for each event. Events reported on the LES that did not meet the event definition criteria were disqualified by the interviewers and were not counted in event scores. Interviewers were trained and tested on their reliability prior to interviewing participants in this study. The standards employed in this study reduced inaccuracies in determining the timing of events, and potential double-reporting or omitted events. The reliability and validity of the LEI are well established (Francis-Raniere et al., 2006; Alloy et al., 2006).

BAS-Activation and BAS-Deactivation Ratings—As reported by Urosevic et al. (2010), each of the LES events were a priori rated by three independent researchers on BAS-Activation (i.e., the extent the life event category involves goal-striving, opportunity to obtain goals/rewards, or opportunity to remove obstacles to these goals/rewards) and BAS-Deactivation (i.e., the extent the life event category triggers cessation of approach and/or involves failure to obtain rewards/goals) using a 4-point Likert scale (that included anchors of not at all, somewhat, very, and extremely). These three independent raters had good reliability ($\alpha = .79$ for BAS-Activation, $\alpha = .94$ for BAS-Deactivation). Any discrepancies in ratings of “0” (i.e., “not at all”) on this dimension were resolved by consensus and, in ambiguous situations, erred towards inclusion in this category. For the current study, the BAS-Activation and Deactivation ratings were dichotomized and a sum of each type was created for each prospective assessment for each participant. Participants included in this study reported an average of 7.72 BAS-Activating events ($SD = 11.52$) and 3.12 BAS-Deactivating events ($SD = 6.25$) per follow-up interview.

Anger-Evocation Ratings—For the current study, life events also were rated by four independent coders (different raters than those who rated the BAS-Activating events) on the extent of anger-evocation according to the expanded BAS-dysregulation model postulated by Urosevic et al. (2008). Following this theory, life events were coded on how anger-

evoking they would be for the average person, on a scale from 0 to 2. Coders were asked to include events that may evoke “revenge,” or the opportunity to “remove an obstacle” (Urosevic et al., 2008). The inter-rater reliability coefficient was good ($\alpha = .845$). In order to test whether anger-evoking life events are a different kind of BAS-Activating event, all BAS-Activating events that qualified as anger-evoking were removed from the BAS-Activating list. This left 90 BAS-Activating events rather than the original 129, as 39 of the original BAS-Activating events were categorized as anger-evoking. Consistent with the BAS-Activating events, for the current study, the Anger-Evocation ratings were dichotomized and a sum of these events was created for each prospective assessment for each participant. Participants included in this study reported an average of 4.22 Anger-Evoking events per follow-up interview ($SD = 7.42$).

Aggression Questionnaire (AQ; Buss & Perry, 1992). The AQ is a 29-item self-report questionnaire designed to assess trait levels of aggression. Participants completed this measure at the Time 1 baseline visit by answering each question using a five point Likert scale from “extremely uncharacteristic of me” to “extremely characteristic of me.” The AQ consists of four factors: Physical Aggression, Verbal Aggression, Anger, and Hostility. In addition, a total score for the AQ is the sum of the factor scores. This measure has shown good retest reliability as well as concurrent validity with other measures of trait aggression, including peer nomination of physical aggression (Buss & Perry, 1992). Internal consistency was strong in the current sample as well (Total scale $\alpha = .914$, Physical $\alpha = .797$, Verbal $\alpha = .819$, Anger $\alpha = .789$, Hostility $\alpha = .821$).

Impulsive Nonconformity Scale (INS; Chapman et al., 1984). The INS is a 51-item true/false questionnaire designed to assess trait levels of impulsive behavior. Participants completed this self-report measure at the baseline visit. Items include, “When I want something, delays are unbearable” and “When I start out in the evening, I seldom know what I’ll end up doing.” The INS has good internal consistency (α 's = .79-.84; Alloy et al., 2006; Chapman et al., 1984) and 6-week retest reliability ($r = .84$; Chapman et al., 1984). The internal consistency in the current sample was $\alpha = .86$. Chapman et al. (1984) found that high scorers on the INS were more likely to endorse antisocial, psychotic, depressive, and hypomanic/manic symptoms than a control group. Moreover, higher INS scores predicted prospective substance abuse problems in individuals with BSDs and mediated BSD-substance abuse comorbidity (Alloy et al., 2009b) and predicted progression to Bipolar I disorder among individuals with bipolar spectrum conditions (Alloy et al., 2012).

Results

Preliminary Analyses

Bipolar spectrum participants did not differ significantly from control participants on gender ($\chi^2(1) = .013$, $p = .91$), age ($t(42522) = .20$, $p = .84$), or race (white vs. non-white; $\chi^2(1) = .01$, $p = .94$). As would be expected, bipolar spectrum participants experienced more depressive and hypomanic episodes, as well as a greater number of days spent in mood episodes than control participants (all p 's < .001). There were no significant differences between groups in time spent in the study ($t(412.93) = .43$, $p = .67$). There were also no gender differences in episodes experienced. We found that BSD participants had significantly higher scores on impulsivity and aggression: INS ($t(201.31) = 9.03$, $p < .001$), AQ Physical ($t(201.77) = 3.16$, $p < .01$), AQ Verbal ($t(177.84) = 4.40$, $p < .001$), AQ Anger ($t(201.77) = 9.03$, $p < .01$), AQ Hostility ($t(196.17) = 7.12$, $p < .001$), AQ Total ($t(196.98) = 5.15$, $p < .001$). Table 1 displays the correlations between all study variables.

Main Analyses

To test the main hypotheses, we employed Hierarchical Linear Modeling (HLM; Raudenbush & Bryk, 2002) because it can statistically account for the nested nature of the dataset, as we examined trait level variables (aggression, impulsivity) predicting to numbers of dependent life events within, as well as between, subjects. HLM analyzes within-subject variation in the levels of, and relationships between, trait variables and life events and over multiple time points as well as between-subject effects of diagnostic category, gender, aggression, and impulsivity. Furthermore, we used HLM rather than Ordinary Least Squares Regression (OLS) because we would violate the OLS assumption of independence of residuals.

We used data from a minimum of two and a maximum of thirteen prospective visits. Our final sample had an average of 5.37 follow-up visits per person ($SD = 3.28$). We controlled for total days spent in mood episode in all analyses, given the aforementioned research showing that mood episodes are often associated with increases in life events. We also controlled for gender and site differences in all models, as these variables did affect some results. We first examined diagnostic (healthy control vs. bipolar spectrum) differences in the reporting of events. Bipolar spectrum individuals reported significantly higher numbers of dependent anger-evoking events ($b = 2.02, p < .01$) and dependent BAS-deactivating events ($b = 1.42, p < .01$), but not dependent BAS-activating events ($b = -0.394, p = .66$), than controls.

AQ Hostility and INS were both found to predict greater numbers of dependent BAS activating events experienced (see Table 2 top portion for full HLM models). Of note, bipolar diagnosis did not predict numbers of dependent BAS activating events in any of these models once aggression or impulsivity was controlled. AQ Hostility and INS were both found to predict greater numbers of dependent BAS-Deactivating events experienced (see Table 2 middle portion for full HLM models). AQ Physical also marginally predicted the number of BAS-Deactivating events. Bipolar diagnosis remained significant in most analyses, was marginally significant in the INS model, and was not a significant predictor in the AQ Anger model.

Dependent anger-evoking events were predicted by AQ Total, AQ Anger, AQ Hostility, AQ Physical, and INS scores (see Table 2 bottom portion for full HLM models). Bipolar diagnosis remained significant in all analyses except the AQ Hostility model, where it was marginally significant. These differences could signify that the effect of bipolar diagnosis may be accounted for by other trait measures.

Interactions between Trait Variables and Group

In order to better understand these results, we also examined interactions between diagnostic group (bipolar spectrum vs. healthy control) and trait variables that significantly predicted types of life events. However, there were no significant interactions between diagnostic group and aggression or impulsivity in predicting any type of life events.

Finally, we examined these effects within the bipolar sample. Without accounting for personality level factors, bipolar diagnostic group (Bipolar II vs. Cyclothymia/Bipolar NOS) did not predict dependent BAS-activating ($b = -0.06, p = .69$), BAS-deactivating ($b = 0.12, p = .86$) or anger-evoking events ($b = 0.12, p = .54$). When we examined whether personality factors predicted numbers of dependent life events in the bipolar sample, we found that INS significantly predicted all three types of dependent events (see Table 3 top, middle, and bottom portions), while controlling for bipolar diagnostic group. In addition, AQ Hostility significantly predicted dependent BAS-Deactivating (see Table 3 middle portion) and Anger-Evoking events (Table 3 bottom portion), and marginally predicted

dependent BAS-Activating events (Table 3 top portion), while controlling for bipolar diagnostic group. There were no significant interactions between bipolar subgroup and either aggression or impulsivity in predicting the three types of life events.

Discussion

Consistent with our hypotheses, individuals with bipolar spectrum disorders not only evidenced higher levels of aggression and impulsivity compared to control participants, but also experienced more dependent anger-evoking and BAS-deactivating life events. Interestingly, individuals with BSDs did not differ from controls in terms of number of dependent non-anger BAS-activating life events. This finding is particularly interesting in light of previous findings that individuals with BSDs experience higher rates of BAS-activation life events (Bender et al., 2010; Urosevic et al., 2010). Given that previous work has not examined anger-relevant separately from reward-relevant BAS-activating life events, it may be that previous findings may specifically reflect bipolar - control differences in anger-relevant BAS-activating life events, as anger/irritability and reward sensitivity are both characteristic of individuals with and at risk for BSDs (Cooke & Jones, 2009; Alloy et al., 2012, respectively). Furthermore, the associations between bipolar status and BAS-activating life events did not hold after controlling for aggression and impulsivity, suggesting that elevated levels of these personality traits among individuals with bipolar spectrum disorders may specifically contribute to stress generation. However, it is also important to note that these constructs have considerable overlap. Behavioral activation, aggression, and impulsivity, while all thought to be uniquely important in those with bipolar disorders, are not orthogonal constructs. More research should be conducted on the ways in which these constructs overlap and which aspects of these constructs are most important for stress generation in individuals with BSD.

Consistent with hypotheses, the current study demonstrated that several subscales of the AQ, specifically anger, hostility, and physical aggression as well as total aggression, predicted the experiencing of anger events. Previous research has demonstrated that outward expressions of anger may evoke “reciprocal hostile mood,” from others (Hokanson & Butler, 1992), which may be conceptualized (as in the current study) as an anger-evoking event. Furthermore, hostility has been shown to predict same-day increases in dependent (primarily interpersonal) stressful events (Sahl et al., 2009). AQ subscales also prospectively predicted increases in BAS-activating non-anger events. This is consistent with previous conceptualizations of anger as an approach motivation (Harmon-Jones & Sigelman, 2001) as well as with research that has demonstrated strong perceptual similarities between anger and determination (Harmon-Jones et al., 2011). Bipolar diagnosis predicted increased anger-evoking events after controlling for anger, physical aggression, and total aggression, but did not significantly predict anger events after controlling for hostility. This may suggest that hostility associated with BSDs may drive an elevated frequency of dependent anger-evoking events. Although this study employed a prospective design, we cannot definitively conclude that hostility in particular causes the generation of anger-evoking events; there may be other factors as yet unaccounted for.

Impulsivity also predicted increases in anger events, consistent with previous research that suggests that impulsivity is likely to result in peer rejection or hostility (Denson et al., 2011) or goal-achievement failures (Nusslock et al., 2008), both of which may be anger-evoking types of events. Impulsivity also significantly predicted non-anger BAS-activating life events. This finding is consistent with previous findings that individuals high in impulsivity may be more inclined to impulsively pursue rewards (Zapolski et al., 2010), which could lead to increases in BAS-activating life events (Urosevic et al., 2008). Furthermore, controlling for impulsivity, bipolar diagnosis no longer significantly predicted anger-

evoking life events. Again, this may suggest that impulsivity associated with BSDs, rather than other symptoms or characteristics, may play a key role in the generation of anger-evoking events. Unsurprisingly, as both aggression and impulsivity are often associated with a variety of negative outcomes (Barlow et al., 2000; Lewis et al., 2009, respectively), both AQ hostility and INS also predicted BAS-deactivating events. Together, these findings are consistent with previous research that has suggested that personality traits may be predictors of stress generation (Hammen, 2006; Hankin, 2010). Although bipolar diagnosis may indeed be associated with a life course characterized by more stressful life events, these findings suggest that personality characteristics associated with bipolar disorder such as hostility and impulsivity must not be overlooked when examining life event generation. Furthermore, these aspects of bipolar disorder have been linked to suicidality in a number of studies (e.g., Oquendo et al., 2000, 2004), furthering our conclusion that aggression and impulsivity are factors associated with a more severe course of bipolar disorder.

The current study is among the first to examine the effects of personality factors on stress generation in a bipolar sample, as well as to separate anger-evoking from BAS-activating life events. There were several strengths, including a large sample that allowed the examination of bipolar diagnostic group differences as well as comparing the bipolar group to a healthy control group. This study examined several years of longitudinal follow-up data, and employed multilevel modeling in order to test the effects of diagnostic group and personality over time without violating statistical assumptions. The current study also has some limitations. The life events measures used in these analyses were not created to examine the differences in types of BAS-activating events; therefore, there may be some issue in parsing out these different life event types. We also found varying results with respect to BAS-deactivating life events. We believe this is due to the wide variability in types of BAS-deactivating events included. Further research should examine subcategories of BAS-deactivation events. Finally, it remains to be seen whether these results can be generalized to a more conventional clinical sample. These individuals were college-aged, and not treatment-seeking. Research on stress generation and personality factors in BSDs should be replicated in clinical samples.

In conclusion, the current study demonstrates that there are trait level personality differences that are associated with stress generation, beyond bipolar diagnosis. The most robust findings included that high levels of impulsivity and some forms of aggression predicted increases in anger- and BAS-activating events. This study further establishes the importance of including anger-evoking events in the BAS model of BSDs and stress generation, and adds to the existing literature showing that anger is an approach emotion, as well as particularly important in BSDs. Treatment implications include specifically targeting impulsivity and hostility in patients with BSDs, and perhaps also the anger and aggression that contributes to a more stressful life.

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Table 1

Inter-correlations of study variables

	Bipolar diagnosis	INS	AQ Physical	AQ Verbal	AQ Anger	AQ Hostility	AQ Total	Anger events	BAS Act events
INS	0.504 ***								
AQ Physical	0.196 **	0.446 ***							
AQ Verbal	0.286 ***	0.431 ***	0.423 ***						
AQ Anger	0.380 ***	0.482 ***	0.458 ***	0.537 ***					
AQ Hostility	0.419 ***	0.405 ***	0.376 ***	0.339 ***	0.550 ***				
AQ Total	0.318 ***	0.468 ***	0.518 ***	0.588 ***	0.595 ***	0.559 ***			
Anger events	0.323 ***	0.396 ***	0.215 **	0.149 *	0.259 ***	0.348 ***	0.252 ***		
BAS Act events	0.077	0.253 ***	0.092	0.130 †	0.078	0.106	0.163 *	0.311 ***	
BAS Deact events	0.270 ***	0.388 ***	0.229 **	0.140 *	0.191 **	0.341 ***	0.173 *	0.489 ***	0.295

Note.

† $p < .10$,

* $p < .05$,

** $p < .01$,

*** $p < .001$

AQ = Aggression Questionnaire; INS = Impulsive Nonconformity Scale

Table 2

HLM Models Predicting to Dependent Events

Individual level	BAS-Activating Events					
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Site	-1.834	-1.885	-0.675 *	-1.600 †	-1.800 *	-1.498
Gender	-0.520	-0.765	-0.840	-0.661	-0.560	-0.630
Bipolar diagnosis	-0.749	-1.050	-0.555	-0.622	-0.541	-1.400
Mood episode days	0.005 *	0.005 *	0.005 *	0.005 *	0.005 *	0.005 *
AQ Total	0.047 †					
AQ Hostility		0.174 ***				
AQ Anger			0.050			
AQ Verbal				0.132		
AQ Physical					0.082	
INS						0.155 **
Observation level						
RPA	1.447 ***	1.450 ***	1.446 ***	1.447 ***	1.448 ***	1.460 ***
BAS-Deactivating Events						
Individual level	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Site	0.012	-0.087	-1.675 *	0.098	-0.065	0.172
Gender	-0.164	-0.469	-0.840	-0.231	-0.056	-0.120
Bipolar diagnosis	1.401 **	1.462 *	-0.555	1.478 **	1.387 **	0.707 †
Mood episode days	0.002 *	0.002	0.005 *	0.002 *	0.001 *	0.001
AQ Total	0.018					
AQ Hostility		0.248 ***				
AQ Anger			0.050			
AQ Verbal				0.037		
AQ Physical					0.079 †	
INS						0.125 **
Observation level						

Individual level	BAS-Activating Events					
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
RPA	0.120 [†]	-0.208 [*]	1.446 ^{***}	0.120 [†]	0.121 [†]	0.124 [†]
Anger-Evoking Events						
Individual level	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Site	-0.300	-0.371	-0.171	-0.054	-0.266	-0.030
Gender	-0.212	-0.438	-0.713	-0.435	-0.078	-0.219
Bipolar diagnosis	1.688 ^{**}	1.14 [†]	1.525 [*]	1.963 ^{**}	1.866 ^{***}	0.958 [*]
Mood episode days	0.001	0.001	0.001	0.002	0.001	0.001
AQ Total	0.050 [*]					
AQ Hostility		0.174 ^{***}				
AQ Anger			0.162 [*]			
AQ Verbal				0.028		
AQ Physical					0.115 [*]	
INS						0.148 ^{**}
Observation level						
RPA	-0.554 ^{***}	-0.550 ^{***}	-0.555 ^{***}	-0.553 ^{***}	-0.578 ^{***}	-0.576 ^{***}

Note.

[†] $p < .10$,

^{*} $p < .05$,

^{**} $p < .01$,

^{***} $p < .001$

AQ = Aggression Questionnaire; INS = Impulsive Nonconformity Scale; RPA = Regular Prospective Assessment

Table 3

HLM Models Predicting to Dependent Events in BSD sample

Individual level	BAS-Activating Events					
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Site = TU	-0.594	-0.760	-0.285	-0.361	-0.507	-0.256
Female	-0.785	-0.108	-0.969	-0.958	-0.831	-0.738
BD II	0.833	0.631	0.800	0.943	0.622	0.707
Mood episode days	0.005 *	0.005 *	0.006 *	0.006 *	0.006 *	0.005 *
AQ Total	0.043					
AQ Hostility		0.155 †				
AQ Anger			-0.015			
AQ Verbal				0.132		
AQ Physical					0.087	
INS						0.153 *
Observation level						
RPA	1.584 ***	1.590 ***	1.583 ***	1.583 ***	1.584 ***	1.582 ***
BAS-Deactivating Events						
Individual level	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Site = TU	0.463	-0.003	0.422	0.523	-0.507	-0.256
Female	0.045	-0.086	-0.116	0.001	-0.831	-0.738
BD II	0.032	-0.516	-0.028	-0.003	0.622	0.707
Mood episode days	0.002 *	0.001 †	0.002 *	0.002 *	0.006 *	0.005 *
AQ Total	0.008					
AQ Hostility		0.173 **				
AQ Anger			0.066			
AQ Verbal				-0.027		
AQ Physical					0.087	
INS						0.153 *
Observation level						
RPA	0.232 †	0.239 †	0.231 †	0.231 †	1.584 ***	1.582 ***

Individual level	BAS-Activating Events					
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Individual level	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Site = TU	-0.087	-0.350	0.077	0.279	0.411	5788.000
Female	0.178	-0.262	-0.404	-0.124	-0.293	-0.001
BD II	-0.259	-0.538	-0.275	-0.255	-0.416	-0.463
Mood episode days	0.001	0.001	0.001	0.001	0.001	0.001
AQ Total	0.055 [†]					
AQ Hostility		0.210 ^{***}				
AQ Anger			0.164 [†]			
AQ Verbal				0.046		
AQ Physical					0.001	
INS						0.135 ^{**}
Observation level						
RPA	-0.700 ^{***}	-0.687 ^{***}	-0.706 ^{***}	-0.755 ^{***}	-0.722 ^{***}	-0.684 ^{***}

Note.

[†] $p < .10$,

* $p < .05$,

** $p < .01$,

*** $p < .001$

BD II = bipolar II disorder diagnosis; AQ = Aggression Questionnaire; INS = Impulsive Nonconformity Scale; RPA = Regular Prospective Assessment