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Impulsivity and Behavior-Dependent Life Events Mediate the Relationship of Reward Sensitivity and Depression, but Not Hypomania, Among at-Risk Adolescents

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Both reward sensitivity and impulsivity are related to the development and course of bipolar spectrum disorders (BSDs) and have been implicated in other disorders and negative functional outcomes such as substance abuse, obesity, suicidal behaviors, and risk-taking. Furthermore, according to the transactional component of the Behavioral Approach System (BAS)/reward hypersensitivity theory of BSDs, people with reward hypersensitivity should experience more BAS-relevant events, and thus, are more vulnerable to mood symptoms and episodes via stress generation. Impulsivity may exacerbate stress generation in individuals at risk for BSDs based on exhibiting reward hypersensitivity. The current study examined whether impulsivity explained the generation of stress and subsequent mood symptoms beyond what is explained by reward sensitivity alone. Participants were 131 Moderate BAS and 216 High BAS sensitivity adolescents (M = 18.43 years, SD = 1.40), who completed baseline measures of reward sensitivity and impulsivity, as well as follow-up measures of life events and mood symptoms. Results from linear regression analyses indicated that higher baseline impulsivity predicted behavior-dependent, but not behavior-independent, life events. Furthermore, path analyses suggested that the effect of BAS group on depression symptoms at next follow-up was partly explained via the indirect effect of impulsivity and negative behavior-dependent life events. We did not find these effects for behavior-independent or positive-dependent events or for prediction of hypomanic symptoms. The findings suggest that impulsivity may account for stress generation of negative events that precede depression.

Keywords: impulsivity; stress generation; bipolar spectrum; behavioral approach system; life event

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BIPOLAR SPECTRUM DISORDERS (BSDs) are one of the leading causes of disability worldwide (Miklowitz & Johnson, 2006), involving significant impairment in social and occupational functioning, and occurring at about a 4% rate in the United States (Merikangas & Pato, 2009). BSDs are diagnosed when the individual has experienced a manic or hypomanic episode, which includes high mood, increased energy and restlessness, decreased need for sleep, racing thoughts and risky/impulsive behaviors (American Psychiatric Association [APA], 2013).

Researchers have utilized Gray's (1975) two biobehavioral systems perspective when conceptualizing mood disorders. Specifically, Gray proposed two

neurobiological systems that control one's sensitivities to perceived reward (behavioral activation system or BAS) and punishment (behavioral inhibition system or BIS). Elaborating on this theory, the Behavioral Approach System (BAS)/reward hypersensitivity theory of BSDs (Alloy & Abramson, 2010; Alloy, Nusslock, & Boland, 2015; Alloy, Olino, Freed, & Nusslock, 2016; Depue & Iacono, 1989; Depue, Krauss, & Spoont, 1987; Johnson, Edge, Holmes, & Carver, 2012; Nusslock & Alloy, 2017; Urosević, Abramson, Harmon-Jones, & Alloy, 2008) proposes that people who are highly reward sensitive experience *excessive* approach motivation in response to potential rewards. As a result, they react to BAS-activating events, particularly events relating to goal-striving and attainment, excessively (Johnson et al., 2000, 2008; Nusslock, Abramson, Harmon-Jones, Alloy, & Hogan, 2007). This, in turn, leaves them vulnerable to developing hypomanic/manic episodes. Likewise, individuals with a hypersensitive BAS are hypothesized to respond with an excessive decrease or down regulation in approach motivation in response to BAS-deactivating events (e.g., irreconcilable failures and losses), leading to depressive episodes. Consistent with the BAS/reward hypersensitivity theory, researchers have found that reward hypersensitivity is a key predictor of the initial onset and course of BSDs (Alloy, Bender, et al., 2012; Alloy, Urošević, et al., 2012; see Alloy et al., 2015, 2016; Nusslock & Alloy, 2017, for reviews).

The reward hypersensitivity theory of BSD also includes a transactional component. According to the stress generation theory of unipolar depression (Hammen, 1991; Liu & Alloy, 2010), people who are vulnerable to depression exhibit certain cognitive traits (e.g., negative beliefs about themselves and the world, personality characteristics such as being more withdrawn) that will likely influence their behaviors. These behaviors, in turn, lead them to experience more negative life events that precede depressive episodes. In other words, they not only react more strongly to negative life events, but exhibit specific cognitive or personality traits that may lead them to experience more of these same negative events. Similar stress-generation processes are proposed to also occur in those vulnerable to BSDs. Individuals with high reward sensitivity are hypothesized to be exposed to more BAS-activating and -deactivating life events (through stress generation processes) as well as react to such events more strongly (Alloy et al., 2015, 2016; Urosević et al., 2008). Thus, just as people with unipolar depression have characteristics and behaviors that lead them to experience more negative life events (see Liu & Alloy, 2010, for review), there also is evidence that people with BSDs, or who are vulnerable to BSDs, based on the BAS/reward model, experience more BAS-activating and -deactivating life events via stress generation (Boland et al., 2016; Urošević et al., 2010).

One factor that may account for stress generation in the BAS model is impulsivity, which is a heterogeneous and multifaceted construct, but is generally defined as the tendency to act rashly, without forethought (Sharma, Markon, & Clark, 2014). Impulsivity is related to, but distinct from, reward sensitivity, and is associated with many maladaptive outcomes (e.g., binge drinking, risky behaviors, suicidal behaviors, ADHD, obesity, etc.) that contribute to injuries and death, as well as poor physical health (Viner et al., 2012). However, like reward hypersensitivity, impulsivity is closely linked to BSDs. Of note, impulsive and risk-taking behaviors are included in the diagnostic criteria for hypomanic and manic episodes (APA, 2013), and so impulsivity frequently co-occurs with BSDs. Indeed, individuals with BSDs exhibit higher levels of impulsivity, even when euthymic (Brunborg, Johnsen, Mentzoni, Molde, & Pallesen, 2011; Suhr & Tsanadis, 2007; Swann, Anderson, Dougherty, & Moeller, 2001) than those without bipolar disorders. Moreover, increased levels of impulsiveness predict more severe BSDs (Alloy, Urošević, et al., 2012; Kwapil et al., 2000; Najt et al., 2007; Swann et al., 2009), faster time to onset of hypomanic or manic episodes (Ng et al., 2016), and greater stress generation for people with BSDs (Molz et al., 2013). Furthermore, the average age of onset of BSDs coincides with the peak in incidence of risky behaviors in one's early 20s (Viner et al., 2012). Finally, impulsivity also is associated with and predicts substance abuse, including comorbid BSD and substance abuse (Alloy et al., 2009; De Wit & Richards, 2004; Littlefield, Stevens, & Sher, 2014).

Impulsivity may exacerbate the stress-generating effects of high reward sensitivity. For example, Molz et al. (2013) found that both impulsivity and hostility significantly predicted the likelihood of experiencing more BAS-activating and deactivating events, above and beyond what could be explained by a BSD diagnosis. These findings support the hypothesis that people who are more impulsive may be more likely to engage in impulsive reward-seeking behaviors. Similarly, the finding that impulsivity predicts greater BAS-deactivating events supports the evidence demonstrating a link between increased impulsivity and various negative outcomes in general. Furthermore, Nusslock et al. (2008) found that individuals with BSDs who were higher on impulsivity were more likely to experience goal-related stress generation (e.g., more goal achievement failures). Thus, there

is evidence that impulsivity may add prediction of the experience of BAS-relevant life events, above and beyond what can be explained through reward sensitivity alone.

The aims of the current study were to examine whether impulsivity was associated with the generation of stress, above and beyond BAS/reward sensitivity. Additionally, we examined whether behavior-dependent stress incurred through higher impulsivity would be associated with affective symptoms. Specifically, we hypothesized that individuals in the high BAS group would have higher levels of impulsivity than the moderate BAS group at baseline (Hypothesis 1). We also predicted that those with higher impulsivity would experience more negative behavior-dependent events, and fewer positive behavior-dependent events at the next follow-up (Hypothesis 2), and that impulsivity would be associated more strongly with behavior-dependent than behavior-independent events (Hypothesis 3). In turn, we predicted that this pathway of impulsivity leading to negative behavior-dependent events would partly explain (i.e., mediate) the relationship between BAS status and depressive mood symptoms at the next follow-up, and that impulsivity leading to positive behavior-dependent events would partly explain the relationship between BAS status and (hypo)manic mood symptoms at next follow-up (Hypothesis 4; see Figure 1). These mediation models are theoretically driven based on the BAS/reward hypersensitivity model that states that positive BAS-activating life events should predict (hypo)mania, while negative BAS-deactivating life events should predict depression (see Nusslock & Alloy, 2017, for review).

Methods

PARTICIPANTS AND PROCEDURES

Participants in this study came from the Teen Emotion and Motivation (TEAM) Project (Alloy, Bender, et al., 2012), a prospective longitudinal study examining predictors of first onset and course of BSDs within a reward hypersensitivity framework. Screening for the project included two phases. After signing informed consent or assent (for participants under 18), 9,991 Temple University undergraduates (aged 18–19) and Philadelphia public high school students (aged 14–19) completed Carver and White's (1994) Behavioral Inhibition System/Behavioral Activation System Scale (BIS/BAS) and the Sensitivity to Punishment/Sensitivity to Reward Questionnaire (SPSRQ; Torrubia, Ávila, Moltó, & Caseras, 2001). Those scoring in the 40^{th} – 60^{th} percentile (moderate) or the 85^{th} – 100^{th} percentile (high) on both the BAS-Total subscale from the BIS/BAS and the Sensitivity to Reward subscale from the SPSRQ were invited to participate in the second part of screening. The justifications for using those scoring in the moderate range of reward sensitivity as a comparison for the High BAS group were twofold: (a) moderate BAS is closer to the population mean, thus making it a more appropriate comparison for the high BAS group, and (b) low BAS scores are associated with unipolar depression (Alloy et al., 2016; Depue & Iacono, 1989).

During the second screening phase (Phase II), participants underwent diagnostic interviewing using an expanded Schedule for Affective Disorders and Schizophrenia–Lifetime interview (exp SADS-L; Endicott & Spitzer, 1978; Alloy et al., 2008). Participants meeting lifetime Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision (*DSM-IV-TR;* APA, 2000) or Research Diagnostic Criteria (RDC; Endicott & Spitzer, 1978) for any psychotic disorder were excluded. Participants meeting criteria for BSD at screening were not excluded from the current study, although Alloy, Bender, et al. (2012) excluded them from the original sample as the goal of Project TEAM was to predict first onset of BSD (manic/hypomanic episodes). Further details regarding screening and eligibility criteria have been described elsewhere (Alloy, Bender, et al., 2012). Participants continue to be followed every 6

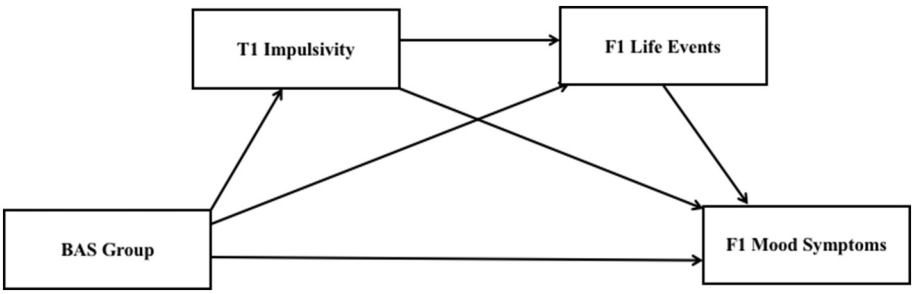

FIGURE 1 Serial Mediation Model of Effect of BAS Risk Status on Subsequent Mood Symptoms via Impulsivity and Life Events *Note*. BAS = Behavioral Approach System; T1 = Baseline; F1 = next follow-up

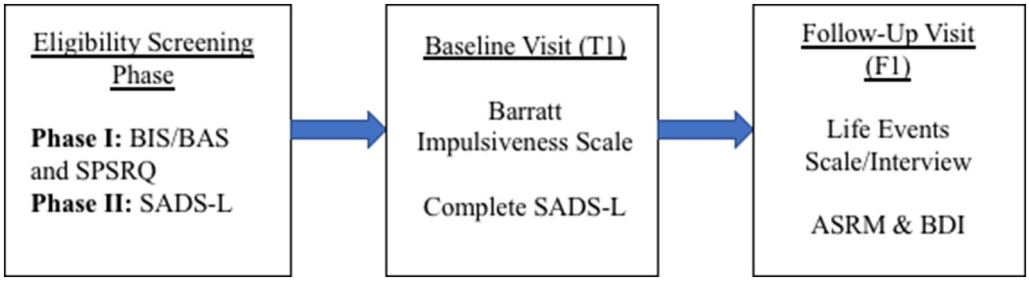

FIGURE 2 Schedule of Assessments and Study Timepoints *Note*. BIS/BAS = Behavioral Inhibition/Behavioral Activation Scale; SADS-L = Schedule for Affective Disorders and Schizophrenia – Lifetime; ASRM = Altman Self Rating Mania scale; BDI = Beck Depression Inventory

months, and complete a range of behavioral, self-report, and diagnostic measures. A flow chart summarizing the measurement schedule and timeline of this study is provided in Figure 2.

The current study includes 347 participants who had completed a baseline visit and at least one follow-up visit (because not all participants completed *both* depression and [hypo]mania symptom questionnaires, of these 347 participants, 340 were included in the depression analyses, and 344 in the [hypo]mania analyses). Thus, participants in the current study were 131 moderate reward sensitivity (Mod BAS) and 216 high reward sensitivity (High BAS) individuals (63% female, 57% white, mean age = 18.43 [SD = 1.40]). There was no difference between the groups on race, $\chi^2(7) = 10.42$, $p = .17$, or age, $t(244.63) = -1.67$, $p = .10$; however, they did significantly differ on gender, $\chi^2(1) = 6.01$, $p = .01$, with significantly more females than males in the Mod BAS group.[1] Furthermore, the current study's sample did not differ significantly from the Phase II sample on race, $\chi^2(7) = 7.12$, $p = .42$, gender, $\chi^2(1) = .13$, $p = .572$, or age, $t(663) = -.27$, $p = .78$. Average time to first follow-up was 335 days (approximately 11 months). Table 1 shows the demographic characteristics and study variable means and standard deviations of the current sample.

MEASURES

The Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) Scale

During Phase I screening, participants completed Carver and White's (1994) BIS/BAS scale, a widely used self-report measure of trait levels of the behavioral activation and inhibition systems. The measure consists of 20 Likert items (1 = *strongly disagree*, 4 = *strongly agree*). The BAS scale measures BAS-relevant constructs such as goal-striving, fun-seeking, and reward-responsiveness. The total BAS score was used to determine eligibility for the Moderate and High BAS groups. The total BAS score had acceptable internal consistency in the Phase I screening sample (α = .80), and has been shown to have acceptable retest reliability (Meyer, Johnson, & Winters, 2001).

Table 1
Sample Demographics and Variable Means and Standard Deviations (N=347)

Demographics	Mean (or %)	SD
Female	62.8%	–
Race		
Caucasian	56.8%	–
African American	23.9%	–
Asian	10.1%	–
Bi/Multiracial	2.3%	–
Other	3.2%	–
Not Reported	2.6%	–
Age at Phase II	18.43	1.40
BAS Category (Moderate)	37.8%	–
Mediator and Outcome Variables		
Barratt Total*	65.29	10.57
Pos Dep**	14.67	4.95
Pos Indep**	0.33	0.59
Neg Dep**	9.60	6.41
Neg Indep**	3.37	2.50
BDI **	5.54	6.08
ASRM**	5.05	3.86

Note. *Collected once at Time I, **Reflects score at next available follow-up time point; Barratt Total = Barratt Impulsiveness Scale; Pos = Positive Life Events; Neg = Negative Life Events; Dep = behavior-dependent life events; Indep = behavior-independent life events; BDI = Beck Depression Inventory; ASRM = Altman Self-Rating Mania scale

[1] Due to the significant gender imbalance between High and Moderate BAS groups, we ran moderation analyses to examine if gender and BAS group interacted to predict our dependent variables of interest (ASRM, BDI, Barratt, and life events). Results from these analyses indicated that there were no significant interactions (all *p*s = ns), which suggests that the associations in our path analyses do not differ by gender.

Sensitivity to Punishment Sensitivity to Reward Questionnaire (SPSRQ; Torrubia et al., 2001)
The SPSRQ also captures reward inhibition/activation sensitivities. It is a self-report measure consisting of 48 "yes" or "no" questions. Scores from the 24-item reward subscale were used to determine eligibility for the Moderate and High BAS groups, along with BAS total scores. Torrubia et al. (2001) report acceptable internal consistency (αs = .75–.83) and good retest reliability for both subscales. The SR subscale had acceptable internal consistency (α = .76) in our Phase I sample.

Expanded Schedule for Affective Disorders and Schizophrenia-Lifetime and Change (SADS-L/SADS-C; Endicott & Spitzer, 1978; Alloy et al., 2008)
This interview was used to diagnose lifetime and current Axis I disorders based on *DSM-IV-TR* and RDC criteria. Intraclass correlations of interrater reliability are generally high (ICC = .6 or greater for 82% of diagnostic subscales; Endicott & Spitzer, 1978). Expanded versions of the interviews were used in this study to allow for *DSM-IV-TR* as well as RDC diagnoses and to probe additional information regarding mood episode duration and severity. These interviews were administered by trained diagnosticians, who were blind to BAS risk group. The expanded SADS-L was administered at baseline (Time 1) to assess lifetime psychopathology and the expanded SADS-C was administered at each 6-month follow-up to assess new diagnoses since the last interview. Interrater reliability for the SADS-L was good for both depression and bipolar diagnoses (κ > .90 and >.96, respectively; Alloy et al., 2000, 2008). Similarly, interrater reliability for the SADS-C was κ > .80 (Alloy et al., 2008).

Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995)
Participants completed the BIS-11 at the Time 1 baseline visit. The BIS-11 is a 30-item self-report questionnaire with Likert items (1 = *rarely/never*, 4 = *almost/always*) that includes trait components of impulsivity. The questionnaire consists of three second-order factors: attentional impulsivity ("I don't pay attention"), motor impulsivity ("I act on the spur of the moment"), and nonplanning impulsivity ("I act 'on impulse'"). Patton et al. (1995) reported adequate internal consistencies among a variety of populations, including undergraduates, psychiatric patients, and patients with substance use disorders (α = .79–.83). In our baseline sample, reliability ranged from good to poor across the subscales (α = .80 for total, .71 for attentional, .67 for motor, .56 for nonplanning). We used the Barratt total score as our measure of impulsivity in our analyses.

Life Events Scale/Life Events Interview (LES, LEI; Francis-Raniere, Alloy, & Abramson, 2006)
Starting at the first follow-up, participants completed the LES, which consists of 193 major and minor events across a wide range of domains relevant to adolescents and young adults. Participants were asked to report if any of these events happened to them since the time of their last interview. These events were coded a priori by three independent researchers (including principal investigators and senior research staff; Urošević et al., 2010). Events were categorized into BAS-activating (including goal-striving and attainment), deactivating (including goal failures and losses), or non-BAS-relevant, and included events in domains such as family, school, romantic and interpersonal relationships, and job (α's = .79–.94; Urošević et al., 2010). A subset of the BAS-activating events qualified as goal attainment events, involving successful attainment of a goal that the participant previously was trying to achieve. The LES captures both positive and negative, as well as major and minor life events. Additionally, events were categorized as either behavior-dependent (e.g., "Received a final grade of A in a class") or behavior-independent (e.g., "Friend got in serious trouble with the law"). Stress generation processes should only occur for events that are dependent on individuals' behavior (Liu & Alloy, 2010). The LEI, which is interviewer-administered, then was completed with the participant to assess whether life events endorsed on the LES qualified based on manualized event-definition criteria. The interviewers, who were fully trained before administering the interview in this study, followed an Event Specific Criteria and Probes (ESCP) manual, which listed specific definitions and criteria for each event. In this way, the LEI ensured that events reported on the LES were not double counted, minimized the potential of dating errors, and omitted events that did not qualify based on the criteria. Both the LES and LEI have acceptable reliability and validity (Alloy et al., 2006; Francis-Raniere et al., 2006). In the current study, a dichotomous "yes/no" occurrence variable was generated for each individual life event item (e.g., yes = 1, no = 0). Then, for each event "type" (e.g., positive-dependent, negative-dependent, etc.) the sum of the yes occurrences was calculated. Follow-up life events consist of all events occurring from the baseline (T1) visit up until the date of the next follow-up visit.

Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979)
The BDI is a 21-item self-report measure assessing depressive symptoms in a variety of domains

(affective, cognitive, motivational, etc.). It is widely used to assess depressive symptoms in both clinical and nonclinical samples, as well as treatment outcome in clinical samples. It has consistently shown good internal and retest reliability in nonclinical samples (Beck, Steer, & Garbin, 1988). In our Phase II sample, $\alpha = .89$. Participants completed this measure at the next follow-up visit and were instructed to answer the BDI based on how they were feeling in the past week.

Altman Self-Rating Mania Scale (ASRM; Altman, Hedeker, Peterson, & Davis, 1997)

The ASRM is a self-report measure consisting of 5 Likert scale items (ranging from 0–4) that capture symptoms of (hypo)mania including elevated mood, increased self-confidence, decreased need for sleep, heightened psychomotor activity, and talkativeness. A single total score is calculated by summing the items. The measure is highly correlated with other clinical and self-report measures of (hypo)mania (Altman et al., 2001), and had acceptable internal consistency in our Phase II sample ($\alpha = .75$). Participants completed this measure at the next follow-up visit and were instructed to answer the ASRM based on how they were feeling in the past week.

STATISTICAL ANALYSES

First, we tested whether the BAS risk groups differed on key study variables, including impulsivity as hypothesized (Hypothesis 1), using *t*-tests. Next, we examined whether impulsivity predicted behavior-dependent, but not behavior-independent, life events using multivariate linear regressions, while controlling for BAS risk group (Hypotheses 2 and 3). To test the hypothesis that High BAS individuals would experience higher levels of prospective mood symptoms as a result of increased stress generation caused by greater impulsivity, we conducted serial mediation analyses (Hypothesis 4; see Figure 1) using MPlus Version 7 (Muthén & Muthén, 2007). Specifically, we included negative dependent and independent events in the models predicting prospective depression symptoms, and we included positive dependent and independent events in the model predicting (hypo)mania. These models were theoretically based on the BAS/reward hypersensitivity model that posits that negative events should precede depression, whereas positive events should precede (hypo)mania. We used a maximum likelihood estimator, bootstrapping with 5,000 resamples and a 95% confidence interval. When predicting to (hypo)manic and depressive symptoms, we controlled for baseline levels of symptoms (e.g., ASRM or BDI, respectively). Additionally, we controlled for the effects of opposite type and valence of life events (e.g., when including negative dependent events in the model, we included total positive and negative independent events as covariates). All analyses controlled for age at baseline, gender, race, and number of days until first follow-up.

Results

INITIAL ANALYSES (HYPOTHESIS 1)

Demographic data and bivariate correlations of key study variables are presented in Tables 1 and 2. Total Barratt and total BAS scores were significantly correlated ($r = .30$, $p < .01$). We first tested whether the two BAS groups differed on key study variables. As hypothesized, relative to Moderate BAS participants, individuals in the High BAS

Table 2
Bivariate Correlations Among Covariates and Symptoms and Life Event Outcomes

	Gender	Race	Age	Days	T1 BRRT	F1 NegDep	F1 NegInd	F1 PosDep	F1 PosInd	F1 ASRM
Gender										
Race	.01									
Age	-.05	-.14*								
Days	.03	.01	-.06							
T1 BRRT	.02	-.14*	.12*	.01						
F1 NegDep	.15*	.11**	-.01	.06	.19**					
F1 NegInd	.12*	-.04	-.00	.16**	.09	.46**				
F1 PosDep	.12*	.05	.11*	.16**	-.03	.41**	.42**			
F1 PosInd	.08	.02	-.03	.16**	.07	.32**	.38**	.24**		
F1 ASRM	-.02	.12*	-.07	-.02	.03	.13*	.02	.14*	.03	
F1 BDI	.13*	.07	-.13*	-.03	.23**	.45**	.25**	.07	.11*	.05

Note. *p < .05, **p < .01; Days = # of days to first follow-up; T1 BRRT = Barratt Impulsiveness Scale – total score at baseline; F1 NegDep = Negative dependent events at follow-up; F1 NegInd = negative independent events at follow-up; F1 PosDep = positive dependent events at follow-up; F1 PosInd = positive independent events at follow-up; F1 ASRM = Altman Self Rating Mania scale total score at follow-up; F1 BDI = Beck Depression Inventory at follow-up.

group had significantly higher levels of Time 1 impulsivity (High BAS M = 67.48, SD = 10.58; Mod BAS M = 61.73, SD = 9.59; t[339] = 5.04, p < .01; Cohen's d = .57). The High BAS group also reported more depressive and hypomanic symptoms at both baseline and follow-up, than did Moderate BAS individuals (T1 ASRM: High BAS M = 6.67, SD = 4.06, Mod BAS M = 5.03, SD = 3.74, t[343] = 3.72, p < .01, Cohen's d = .42; T1 BDI: High BAS M = 7.92, SD = 7.04, Mod BAS M = 6.03, SD = 6.79, t[340] = 2.44, p = .02, Cohen's d = .27; Follow-up ASRM: High BAS M = 5.67, SD = 3.94, Mod BAS M = 4.02, SD = 3.50, t[330] = 3.88, p < .01, Cohen' d = .44; Follow-up BDI: High BAS M = 6.34, SD = 6.48, Mod BAS M = 4.22, SD = 5.11, t[302.07] = 3.28, p < .01, Cohen's d = .36). Additionally, the High BAS group experienced significantly more behavior-dependent events at next follow-up (Positive-dependent: High BAS M = 15.24, SD = 5.06, Mod BAS M = 13.75, SD = 4.63, t[345] = 2.74, p < .01, Cohen's d = .31; Negative-dependent: High BAS M = 10.39, SD = 6.84, Mod BAS M = 8.30, SD = 5.42, t[321.12] = 3.15, p < .01, Cohen's d = .34). The groups did not significantly differ on the number of behavior-independent events at next follow-up (Positive-independent: High BAS M = 0.35, SD = .64, Mod BAS M = 0.29, SD = .50, t[321.60] = 1.00, p = .32, Cohen's d = .10; Negative-independent: High BAS M = 3.52, SD = 2.55, Mod BAS M = 3.11, SD = 2.40, t[345] = 1.48, p = .14, Cohen's d = .17). Due to the low incidence of positive-independent life events, we did not include these events as an outcome variable in our analyses. Thus, only positive and negative behavior-dependent, and negative-independent life events are included as dependent variables in the subsequent analyses.

STRESS GENERATION ANALYSES (HYPOTHESES 2 AND 3)

Results from linear regressions showed that total Barratt impulsivity score was a significant predictor of both negative and positive dependent events, but not negative independent events. In the first block, we included BAS group, age at baseline, gender, race, time to follow-up, and life event covariates. We then included Barratt impulsivity in the next block. Of the covariates, race and total positive and negative-independent events at follow-up were significantly associated with negative dependent events at follow-up (standardized Bs = .12 - .31, all ps < .01). BAS group, age, time to follow-up and negative total events at follow-up were significantly associated with positive dependent events at follow-up (standardized Bs = .11 - .45, all ps < .05). Consistent with our second hypothesis, higher levels of T1 impulsivity predicted more negative dependent events at the next follow-up (standardized B = .18, t[332] = 3.69, p < .01 R^2 = .34), but predicted fewer positive dependent events at the next follow-up (standardized B = -.17, t[332] = -3.34, p < .01, R^2 = .29). Although the addition of impulsivity to the negative and positive dependent events regression models accounted for a small portion of additional variance explained ($\triangle R^2$ = .03, $\triangle R^2$ = .02, respectively), change statistics indicate that impulsivity significantly added to the model's predictive ability, F(1, 332) = 13.61, p < .01; F(1, 332) = 11.17, p < .01.

Consistent with Hypothesis 3, impulsivity did not predict negative independent events (standardized B = .03, t[332] = .63, p = .53).

SERIAL MEDIATION ANALYSES (HYPOTHESIS 4)

Specific Indirect Effects

Two specific indirect effects were estimated for each of the serial mediation models: the relationship between BAS group and BDI/ASRM via Barratt total scores, and via life event type (negative-dependent, positive-dependent, and negative-independent). Results indicated that neither impulsivity nor life event type significantly mediated the relationship between BAS group and ASRM/BDI on their own (standardized Bs = -.00 - .26, all ps = ns).

Path Analyses

Path analyses indicated that High BAS participants experienced significantly higher levels of Time 1 impulsivity, which predicted an increased number of negative, behavior-dependent events at first follow up. Subsequently, these events predicted higher BDI scores at the follow-up. The indirect effect of BAS group on BDI score at follow-up was significant via impulsivity at T1 and negative dependent events at the follow-up (standardized B = .19, CI = .08, .37; see Figure 3A). The direct effect of BAS group on depressive symptoms was no longer significant with impulsivity and negative behavior-dependent events in the model (standardized B = .13, t[331] = 1.69, p = .09). The predictors explained about 42% of the variance in follow-up BDI scores (R^2 = .42, p < .01), although the variance explained in impulsivity and negative dependent events was relatively smaller (R^2 = .10, R^2 = .33, ps < .01, respectively). Although more negative independent events also predicted higher BDI scores at follow-up (standardized B = .20, t[331] = 3.62, p < .01), we did not observe a significant indirect effect of BAS risk group on BDI via impulsivity and negative independent events (Figure 3B). Overall, the predictors explained about

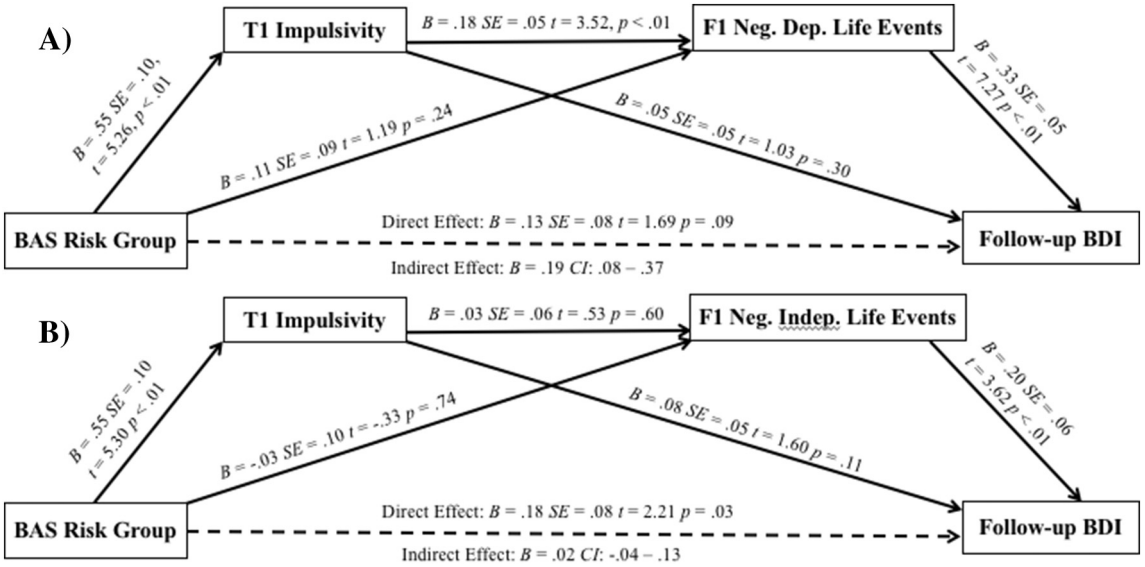

FIGURE 3 Path analyses showing Direct and Indirect Effects of BAS Risk Status on depression symptoms via impulsivity and A) negative-dependent, and B) negative-independent Life Events *Note*. *B* = standardized coefficient; *SE* = bootstrapped standard error; *CI* = bootstrapped 95% confidence interval; T1 = baseline; F1 = next follow-up

39% of the total variance in follow up BDI scores (R^2 = .39, p < .01), while the variance accounted for in impulsivity and negative independent events was about 10% and 30%, respectively (R^2 = .10, R^2 = .30, ps < .01). A number of covariates also were significantly associated with our main outcomes in the models predicting depression. For example, baseline BDI scores were associated with follow-up BDI scores (standardized *B*s = .50–.53, *p*s < .01) and being younger at baseline was associated with higher BDI scores at follow-up (standardized *B*s = -.11, *p*s = .01). Race also was significantly associated with impulsivity scores, such that being non-White was related to lower impulsivity (standardized *B*s = -.10 – -0.13, *p*s = .01). Additionally, race was negatively associated with negative independent events (standardized *B* = -.10, *p* = .03), but positively associated with negative dependent events (standardized *B* = .12, *p* = .03). Generally, life event covariates were positively associated with the life event dependent variables (negative dependent and independent). Gender was not significantly associated with any dependent variables. Despite these significant associations, age, gender and race individually contributed a very small proportion to the total variance of each outcome variable (all $\triangle R^2$s < .02).

High BAS participants also experienced lower levels of positive behavior-dependent events via impulsivity, which did not significantly influence the occurrence of hypomanic symptoms at follow-up (Figure 4). However, the direct effect of BAS group on ASRM scores was significant (standardized *B* = .26, *t*[335] = 2.24, *p* = .03), which supports the BAS-activation component of the reward hypersensitivity theory of BSDs. Although the indirect effect was not significant, the predictors explained a relatively small, but significant

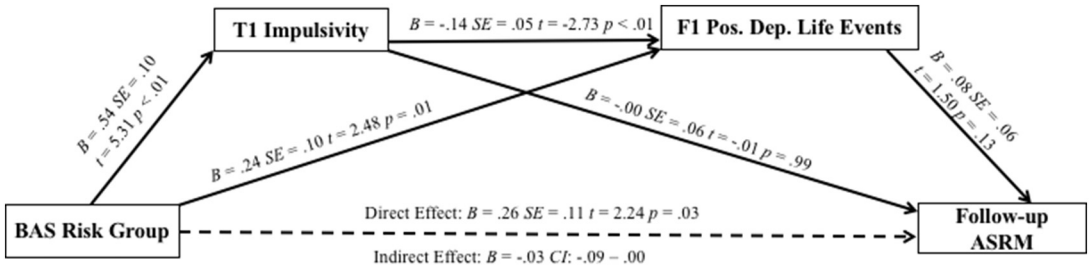

FIGURE 4 Path analyses showing Direct and Indirect Effects of BAS Risk Status on (hypo)mania symptoms via impulsivity and positive-dependent Life Events *Note*. *B* = standardized coefficient; *SE* = bootstrapped standard error; *CI* = bootstrapped 95% confidence interval; T1 = baseline; F1 = next follow-up

proportion of variance in follow-up ASRM scores (R^2 = 0.19, $p < .01$). The only covariate that was significantly associated with follow-up ASRM scores was baseline ASRM scores (standardized $B = .36$, $p < .01$). Being non-White was associated with fewer positive independent events at follow up (standardized $B = -.12$, $p = .02$). Again, age, gender, and race individually contributed a very small proportion to the total variance of each outcome variable (all $\triangle R^2$s $\leqq .02$).

Discussion

The research to date has supported the idea that impulsivity and reward sensitivity both confer predictive risk in the development and severity of BSDs and the occurrence of stressful life events. However, few studies have examined their combined role in relation to these negative outcomes. The aim of the current study was to examine whether people at risk for BSD based on reward hypersensitivity would be more susceptible to subsequent mood symptoms based on the stress generating effects of impulsivity. Results indicated medium size differences between high and moderate reward sensitive groups, such that those exhibiting higher reward sensitivity also had higher levels of impulsivity, and a greater number of behavior-dependent life events at next follow-up. Although the effect of impulsivity and positive dependent events on hypomanic symptoms was not significant, there was a moderate indirect effect of BAS group on depressive symptoms through impulsivity and negative dependent events. Overall, the results suggest that High BAS individuals may be more susceptible to depression in part because of greater levels of impulsivity, which leads to greater BAS-deactivating stress generation. Our findings support the hypothesis that individuals exhibiting reward hypersensitivity also demonstrate increased levels of impulsivity that leads to subsequent stress generation. Specifically, these personality traits seem to explain stress generation of negative events that precede depressive symptoms.

Our findings showed that impulsivity was negatively associated with positive behavior-dependent events, which did not subsequently influence the experience of (hypo)manic symptoms. There is a growing body of literature supporting the idea that impulsivity is linked to subsequent (hypo) manic symptoms and episodes in BSDs (Giovanelli, Hoerger, Johnson, & Gruber, 2013; Ng et al., 2016; Swann, Steinberg, Lijffijt, & Moeller, 2008). Although we did not find a similar effect regarding positive behavior-dependent events preceding hypomania symptoms, these results are not surprising. There is evidence that a subset of positive life events related to goal attainment are associated with the development of (hypo)manic symptoms and episodes (Johnson et al., 2000, 2008). Furthermore, stress generation of BAS-activating events may be more related to features of reward sensitivity (drive, reward responsiveness) and less related to impulsivity in general. In fact, evidence shows that impulsivity increases goal failures (Nusslock et al., 2008), which could relate more to subsequent depressive symptoms and episodes. Thus, our finding that impulsivity predicted fewer positive dependent events supports this idea that highly impulsive individuals may experience more goal failures, and not more goal attainment life events. Additionally, Johnson et al. (2013) found that individuals vulnerable to mania have deficits in controlling their reactions specifically to positive emotions. Thus, this "affective" component of impulsivity may be more associated with one's reactions to life events and subsequently influence mood symptoms. Despite evidence that affective states might play a role in influencing impulsive actions that precede hypomania, the BIS-11 and other self-report measures of impulsivity characterize impulsivity as a trait (Strakowski et al., 2010; Whiteside & Lynam, 2001). Indeed, there is evidence that different aspects of impulsivity change together over time, peak in early adolescence, and remain stable from around age 13 to 14 (Littlefield, Stevens, Ellingson, King, & Jackson, 2016). This suggests that not only are different impulsivity "types" correlated, but they follow similar developmental trajectories and tend to stabilize towards the end of this period. The current study examined specifically how trait impulsivity as measured by the BIS-11 affects stress-generation and subsequent mood symptoms. However, it may be possible that different affective states (e.g., depression vs. mania) interact differently with trait impulsiveness in real-life behavior, which could explain why certain studies have found differences in levels of impulsivity associated with mood state (e.g., hypomania) versus inter-episode periods in BSDs (Johnson et al., 2013; Strakowski et al., 2010). The current study's findings provide evidence of impulsivity's role in stress generation; however, when placed in the context of this broader literature, it becomes necessary to further investigate the many components of impulsivity to inform our understanding of each component's role in stress generation and psychopathology.

Although impulsivity and reward sensitivity individually contribute to the onset and course of BSDs, it is important to note that they are overlapping constructs. For instance, although they are distinct, often measures of reward sensitivity and impulsivity are used interchangeably (Russo, Leone, Lauriola, &

Lucidi, 2008). However, the current findings have important implications for how we understand the differences between these two constructs. To an extent, reward hypersensitivity may be adaptive particularly as it relates to behavior-dependent positive events like reaching a goal or gaining a reward. On the other hand, impulsivity may explain more of the behavior-dependent negative events like goal failures. This is particularly evident in our finding that impulsivity was negatively associated with positive dependent life events. More research is needed to tease apart the individual components of these constructs and how they are related to BSDs and other negative outcomes.

This study had several important strengths. First, we utilized a longitudinal design with two distinct time points, allowing us to assess the predictive ability of baseline impulsivity for subsequent life events and mood symptoms and to examine the life events as mediators of the impulsivity—mood symptom predictive associations. Second, by examining stress generation in a high-risk, nonclinical sample, we helped elucidate particular mechanisms that may contribute to BSD onset. Third, our evaluation of life events relied on both self-report and objective interviewer-based methods. Finally, our sample was racially and socioeconomically diverse, which allowed us to identify small but significant associations between race and life events. Although our findings generally support literature suggesting that African-American adolescents have higher stress exposure than White adolescents (Boardman & Alexander, 2011), and that stressful life events occur at a higher rate among racial minority groups (Hatch & Dohrenwend, 2007), there remain important questions about the role of race in stress generation processes. For example, we also found that being non-White was associated with lower levels of baseline impulsivity, which may indicate that stress generation processes may occur via different mechanisms among different racial groups. Thus, the intersection of race, stress generation, and mood symptoms is an important area for future research.

Nonetheless, there also are several limitations to this study. First, the life events interview contains more dependent event items than independent event items. Indeed, we were unable to adequately examine the effect of positive-independent life events in our analyses, due to the low incidence of these types of events. This may be because "stressful" life events are generally more related to one's own behavior; however, the nonsignificant findings regarding independent events should be interpreted with caution. Second, the ASRM (Altman et al., 1997) has several limitations, including that it is relatively short, and thus, does not account for the full spectrum of (hypo) manic symptoms. Additionally, although evidence suggests that it is highly correlated with clinician ratings of (hypo)mania (Altman et al., 2001), there is the possibility that participants misinterpret certain items. Thus, the findings from the hypomania analyses also should be interpreted with caution. Third, the individuals in this study were from a nonclinical high school and college-aged sample, so the results may not be generalizable to a clinical population. Finally, because of extended time between follow-ups, we decided to use life events and symptoms collected at the same follow-up visit. Although the design was mostly prospective, as number of life events were calculated based on the entire period from baseline to follow-up, and symptoms were assessed in the 1-week period prior to the follow-up visit, we cannot definitively make causal claims.

In conclusion, the present results support the idea that individuals at risk for BSDs exhibit impulsive personality characteristics that leave them vulnerable to experiencing more negative stress generation and subsequent depression. The absence of unique indirect effects of impulsivity or life event type alone on the occurrence of mood symptoms underscores the importance of the combined role of impulsivity and stress generation in predicting future depression, specifically. Future directions should involve further examining the role of impulsivity and its incorporation into the Reward Hypersensitivity Theory of BSDs. Specifically, parceling out the separate components of impulsivity will broaden our understanding of impulsivity's distinct role in the generation of stress and mood symptoms. Additionally, although this study focused on BAS sensitivity, there is evidence that negative behavior-dependent events and rumination may explain the link between impaired cognitive control and depression and anxiety (Snyder & Hankin, 2016). Thus, the role of the behavioral inhibition system, which is involved in sensitivity to punishment, should also be considered in future examinations of models of stress generation and internalizing psychopathology. Furthermore, results from such research may improve interventions targeting impulsive or risky behaviors either leading up to or in response to stressful life events in individuals prone to BSDs.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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