

Bipolar Spectrum–Substance Use Co-Occurrence: Behavioral Approach System (BAS) Sensitivity and Impulsiveness as Shared Personality Vulnerabilities

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Bipolar disorders and substance use disorders (SUDs) show high co-occurrence. One explanation for this co-occurrence may be common personality vulnerabilities involved in both. The authors tested whether high behavioral approach system (BAS) sensitivity and impulsiveness are shared personality vulnerabilities in bipolar spectrum disorders and substance use problems and their co-occurrence in a longitudinal study of 132 individuals on the bipolar spectrum and 153 control participants. At Time 1, participants completed the Behavioral Inhibition System/BAS Scales and the Impulsive Nonconformity Scale. Substance use problems were assessed via the Michigan Alcoholism Screening Test and the Drug Abuse Screening Test at 4-month intervals for 1 year. Participants with bipolar disorder had higher rates of lifetime SUDs and substance use problems during the follow-up, relative to control participants. In line with hypotheses, higher BAS sensitivity and impulsiveness predicted bipolar status and increased substance use problems prospectively. BAS total, BAS Fun Seeking, and impulsiveness mediated the association between bipolar spectrum status and prospective substance use problems, with impulsiveness as the most important mediator. High BAS sensitivity and impulsiveness may represent shared personality vulnerabilities for both disorders and may partially account for their co-occurrence.

Keywords: bipolar spectrum, substance use, behavioral approach system (BAS) sensitivity, impulsiveness

Theory and evidence have accumulated on the role of long-term personality differences in vulnerability to clinical disorders. The hypothesized behavioral functions of neurobiological systems have provided a way of conceptualizing associations between personality traits and various forms of psychopathology. For example, the reinforcement sensitivity theory (RST) of personality is a broad theory of personality rooted in research and theory on the biological bases of learning and motivation. In Gray's 1982 version, RST posited three systems: (a) the fight–flight system (FFS), (b) the behavioral approach system (BAS), and (c) the behavioral inhibition system (BIS). The FFS was posited to be sensitive to

unconditional aversive stimuli; the BAS was posited to be sensitive to conditioned appetitive stimuli; and the BIS was posited to be sensitive to conditioned aversive stimuli, extreme novelty, high-intensity stimuli, and innate fear stimuli. In 2000, Gray and McNaughton revised RST. Now, the FFS was referred to as the fight–flight–freeze system (FFFS), it was posited to be sensitive to all aversive stimuli, BAS was posited to mediate reactions to all appetitive stimuli, and the BIS was now posited to not mediate reactions to conditioned aversive stimuli but to resolve goal conflicts between BAS and FFFS as well as BAS–BAS and FFFS–FFFS conflicts (Corr, 2008). In addition to providing a broad theory of personality, RST suggests new ways of thinking about the causes and consequences of clinical disorders. Extreme FFFS sensitivity is posited to underlie disorders such as phobia and panic; extreme BIS sensitivity is posited to underlie disorders such as generalized anxiety and obsessive–compulsive disorder. Extreme BAS sensitivity is posited to underlie addictive behaviors and the appetitive component of mania (Corr, 2008), which is part of bipolar disorder.

Considerable evidence documents high co-occurrence (*comorbidity*) of bipolar spectrum disorders and substance use disorder (SUDs), even controlling for substance-induced mood disorders and the presence of other Axis I disorders (e.g., Conway, Compton, Stinson, & Grant, 2006; Regier et al., 1990; Wilens et al.,

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2004). Moreover, SUDs may exhibit even higher co-occurrence with bipolar disorder than with other mood or anxiety disorders (Conway et al., 2006; Grant et al., 2004). For example, in a recent nationally representative sample of U.S. adults, the lifetime prevalence of any SUD was 37.5% among participants with mania versus 20.7% among participants with unipolar major depression and 19.1% among participants with any anxiety disorder (Conway et al., 2006). Such bipolar–substance use co-occurrence is mutually detrimental to the course, consequences, and treatment of each condition (Dalton, Cate-Carter, Mundo, Parikh, & Kennedy, 2003; Salloum & Thase, 2000; Strakowski et al., 2007). The high co-occurrence between bipolar disorders and SUDs could reflect (a) attempts to self-medicate bipolar mood symptoms with substance use (e.g., Raimo & Schuckit, 1998), (b) changes in brain functioning leading to mood symptoms as a consequence of repeated substance use, and (c) common vulnerabilities or risk factors involved in both disorders. The present study is designed to examine this last possibility. Specifically, in this study, we investigated the idea that the bipolar spectrum and substance use share the common personality vulnerabilities of enhanced BAS sensitivity and impulsiveness.

BAS Hypersensitivity Theory of Bipolar Disorder and SUD

As briefly noted above, RST posits that in regulating approach behavior to attain rewards and goals, the BAS is activated by external (e.g., an attractive goal object) or internal (e.g., expectancies of goal attainment) signals of reward (e.g., Depue & Collins, 1999; Fowles, 1987; Gray, 1991). When these cues activate the BAS, the person increases movement toward attaining goals and cognitive activity (e.g., hope, self-efficacy, planning) aimed at promoting goal attainment. Hope, elation, and happiness are hypothesized to be associated with BAS activation (Depue & Iacono, 1989; Gray, 1994). Recent work also documents a link between anger and activation of the BAS (Carver, 2004; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001), particularly when people have a high expectancy of success for rectifying the anger-provoking situation (Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003). The underlying neural substrate of BAS has been hypothesized to involve dopaminergic systems, particularly the mesolimbic dopaminergic pathways with projections from the ventral tegmental area to the nucleus accumbens, amygdala, and prefrontal cortex. This is also one of the critical pathways hypothesized to underlie the positively reinforcing effects of drugs of abuse (e.g., Chambers, Taylor, & Potenza, 2003; Chiara, 1995; Goldstein & Volkow, 2002; Jentsch & Taylor, 1999). In addition, diverse work has converged on the conclusion that activation of the left frontal cortex is a key component of the neural circuitry implementing BAS function (e.g., Coan & Allen, 2004; Davidson, Jackson, & Kalin, 2000; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997; Sobotka, Davidson, & Senulis, 1992).

Depue and colleagues (Depue & Iacono, 1989; Depue, Krauss, & Spont, 1987) proposed a BAS hypersensitivity theory of bipolar disorder (see also Urosevic, Abramson, Harmon-Jones, & Alloy, 2008, for an update and review of evidence for this theory). According to the BAS theory, individuals vulnerable to bipolar disorder exhibit an overly sensitive BAS that is hyperreactive to relevant cues. A hyperresponsive BAS can lead to excessive BAS

activity in response to BAS activation-relevant events involving themes of reward incentive, goal striving and attainment, and anger evocation. Excessive activation of the BAS in vulnerable individuals is hypothesized to be reflected in (hypo)manic symptoms such as euphoria, excessive goal seeking, decreased need for sleep, distractibility, irritability, excessive self-confidence, and optimism (Depue & Iacono, 1989; Fowles, 1993; Urosevic et al., 2008). According to this view, the high co-occurrence between SUDs and bipolar disorder may be due, in part, to bipolar individuals' excessive pursuit of rewarding stimuli such as drug-induced highs. In contrast, according to the theory, depressive symptoms such as sadness, low energy, anhedonia, psychomotor retardation, hopelessness, and low self-confidence reflect a shut-down of behavioral approach or excessive deactivation of the BAS in response to BAS deactivation-relevant events such as definite failure or nonattainment of goals (Depue et al., 1987; Fowles, 1993). A key prediction, then, from the BAS hypersensitivity model is that individuals who have a highly sensitive BAS should be vulnerable to both (hypo)mania and depressive states, that is, to bipolar spectrum disorders.

Much evidence supports the BAS hypersensitivity model of bipolar spectrum disorders. Individuals with bipolar spectrum disorders or exhibiting or prone to hypomanic symptoms show elevated scores on self-reported BAS sensitivity (Alloy et al., 2008; Carver & White, 1994; B. Meyer, Johnson, & Carver, 1999; B. Meyer, Johnson, & Winters, 2001). In addition, high self-reported BAS sensitivity predicted levels of positive affect and hypomanic symptoms among students over 17 days in a daily diary study (T. D. Meyer & Hofmann, 2005), increases in manic symptoms over 6 months in a recovered bipolar I sample (B. Meyer et al., 2001), a 6 times greater likelihood of a lifetime bipolar spectrum disorder diagnosis among students (Alloy et al., 2006), and a shorter time to onset of hypomanic and manic episodes over 3.5 years in a prospective follow-up of a bipolar spectrum sample (Alloy et al., 2008). Also consistent with the theory, low levels of BAS activation have distinguished individuals with current and recovered major depression from controls (Campbell-Sills, Liverant, & Brown, 2004; Kasch, Rottenberg, Arnow, & Gotlib, 2002; Pinto-Meza et al., 2006) and predicted a worse course of depression (Kasch et al., 2002; McFarland, Shankman, Tenke, Bruder, & Klein, 2006).

Life events hypothesized to be BAS activation relevant, such as goal-attainment (Johnson et al., 2000, 2008) and goal-striving (Nusslock, Abramson, Harmon-Jones, Alloy, & Hogan, 2007) events, triggered (hypo)manic symptoms or episodes, respectively, in individuals with bipolar disorders. Further, BAS-relevant cognitive styles involving goal striving, perfectionism, and autonomy characterize individuals with bipolar disorders (Alloy et al., in press; Lam, Wright, & Smith, 2004; Scott, Stanton, Garland, & Ferrier, 2000) and, in a bipolar spectrum sample, prospectively predicted increases in hypomanic symptoms in combination with congruent positive events and increases in depressive symptoms in combination with congruent negative events (Francis-Raniere, Alloy, & Abramson, 2006). Similarly, the BAS-relevant personality trait of achievement striving predicted increases in manic symptoms over a 6-month follow-up in a bipolar I sample (Lozano & Johnson, 2001). Finally, relative left frontal cortical activity (as measured by electroencephalograph, or EEG), a neurobiological index of BAS activity, is increased in mania or proneness to

hypomania (Harmon-Jones et al., 2002; Kano, Nakamura, Matsuoka, Lida, & Nakajima, 1992) and decreased in unipolar and bipolar depression (Allen, Iacono, Depue, & Arbisi, 1993; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1990, 1991). Moreover, increased left frontal cortical activity in response to reward incentives in a challenging task distinguished individuals with bipolar spectrum disorders from control participants (Harmon-Jones et al., 2008).

Similarly, several theorists (Dawe & Loxton, 2004; Dawe, Gullo, & Loxton, 2004; Franken & Muris, 2006; Franken, Muris, & Georgieva, 2006) hypothesized that heightened reward sensitivity or drive (i.e., BAS) contributes to substance use and addiction. For example, it has been suggested that individuals with *reward deficit syndrome*, who have lower levels of dopamine concentration in neural pathways, are more responsive to the reinforcing effects of rewarding stimuli including drugs and, thus, are more prone to abusing drugs (Blum et al., 2000). Given that high BAS activation leads to approach behavior in situations involving potentially rewarding stimuli and drugs of abuse have rewarding properties, high BAS activation should lead to greater substance use. High BAS sensitivity should also be associated with greater responsiveness to conditioned cues associated with substance use (Kambouropoulos & Staiger, 2001, 2004) and to using drugs to enhance positive affect or euphoria (Colder & O’Conner, 2002). Thus, a second prediction from the BAS hypersensitivity model is that individuals with a hypersensitive BAS should be vulnerable to greater problems with substance use. Moreover, BAS hypersensitivity should contribute, at least in part, to the high co-occurrence found between bipolar disorders and substance use.

Although less extensive than the work on BAS and bipolar disorders, growing evidence also supports the BAS hypersensitivity theory for substance use. Cross-sectional and retrospective studies show consistent associations between high self-reported BAS sensitivity and increased substance use and SUDs (Franken & Muris, 2006; Franken et al., 2006; Johnson, Turner, & Iwata, 2003; Knyazev, 2004; Loxton & Dawe, 2001, 2006). Behavioral task measures of BAS sensitivity also differentiate heavy or binge drinkers from light drinkers (Colder & O’Conner, 2002; Palfai & Ostafin, 2003) and drinking for enhancement reasons from drinking for coping or social reasons (Colder & O’Conner, 2002). High BAS sensitivity as measured by self-report or behavioral task is also predictive of greater cravings, intention to drink, and positive affective responses in alcohol cue reactivity paradigms (Franken, 2002; Kambouropoulos & Staiger, 2001). Few studies have examined EEG frontal cortical asymmetry in association with substance use, and the results of these studies have been mixed (Hayden et al., 2006; Zinser, Fiore, Davidson, & Baker, 1999). However, an important limitation of most of these studies is that the researchers only examined EEG cortical activity in the resting state. According to the BAS perspective, increased left frontal cortical activity, as an index of BAS, should be observed particularly when approach or incentive motivation is engaged. Thus, consistent with the BAS theory, anticipation of reward (smoking) was associated with increased relative left frontal EEG cortical activity among smokers, and this BAS activation was most pronounced in smokers with the greatest motivation to smoke (those recently withdrawn from nicotine; Zinser et al., 1999).

Thus, much empirical work is consistent with the BAS theory. However, a major shortcoming of this work is that all of the studies

on the BAS–substance use association and most of the studies on the BAS–bipolar disorder association are cross-sectional or retrospective. Such designs are not ideal for testing whether BAS sensitivity provides vulnerability to bipolar mood episodes and substance use. Moreover, to our knowledge, no study has examined whether BAS sensitivity mediates the co-occurrence between bipolar disorder and substance use.

Impulsiveness: A Second Common Personality Vulnerability for Substance Use and Bipolar Disorders

One of the strongest and most consistent personality predictors of substance use is impulsiveness, a tendency toward behavior that is rash, lacks planning and foresight, and occurs without reflection or deliberation (Dawe & Loxton, 2004). Individual differences in impulsiveness and related behavioral tendencies (e.g., sensation seeking) are associated concurrently with substance misuse and prospectively predict SUDs (Acton, 2003; Dawe et al., 2004). Consistent with recent RST discussions of BAS and impulsiveness (Corr, 2008; Pickering & Smillie, 2008), investigators have emphasized that impulsiveness is not a unidimensional construct and contains at least two important and separable but correlated dimensions (Dawe & Loxton, 2004; Dawe et al., 2004): BAS or reward sensitivity (discussed above) and rash impulsiveness (RI) or rash, unplanned, impulsive behavior that occurs without consideration of the consequences. Indeed, recent factor analytic studies show that questionnaire measures of RI (e.g., Eysenck Impulsiveness Scale [Eysenck & Eysenck, 1991], Barrett Impulsivity Scale [Patton, Stanford, & Barratt, 1995], Cloninger Novelty-Seeking Scale [Cloninger, Przybeck, Svrakic, & Wetzel, 1994], Zuckerman Sensation-Seeking Scales [Zuckerman, 1994]) load with each other on a separate factor from measures of BAS and reward sensitivity (e.g., BAS from the BIS/BAS Scales [Carver & White, 1994] and the Sensitivity to Reward scale from the Sensitivity to Punishment Sensitivity to Reward Questionnaire [Torrubia, Avila, Molto, & Caseras, 2001]), although the two factors are correlated. This RI component is associated with deficits in executive functions of the prefrontal cortex that underlie inhibitory control (Chambers et al., 2003; Dawe et al., 2004), and such PFC executive dysfunction has also been linked to substance misuse (e.g., Bechara & Damasio, 2002; Bechara, Dolan, & Hindes, 2002).

Recent integrative models of addiction (Chambers et al., 2003; Dawe & Loxton, 2004; Dawe et al., 2004; Goldstein & Volkow, 2002; Jentsch & Taylor, 1999) suggest that BAS sensitivity and RI act in tandem to contribute to substance use. RI and its associated executive dysfunction lead to the inability to curb substance use once reward (drug)-cued approach behavior (mediated by BAS reward sensitivity or drive) has been initiated. Individuals with high BAS sensitivity may find it particularly difficult to control goal-striving and reward-seeking behaviors, including substance use, and this may be even more likely if they are also high in RI with its concomitant poor inhibitory control. Thus, RI may be a second risk factor for problematic substance use that enhances the effects of heightened BAS sensitivity.

Impulsiveness has also been found to relate to bipolar disorders. Self-report measures of RI are elevated in participants with bipolar spectrum disorders and stable across phases of illness (Swann, Anderson, Dougherty, & Moeller, 2001; Swann, Dougherty, Paz-

zaglia, Pham, & Moeller, 2004). In addition, behaviorally assessed RI is high during manic states (Swann, Pazzaglia, Nicholls, Dougherty, & Moeller, 2003) and even further elevated in manic individuals with co-occurring SUDs (Swann et al., 2004). Thus, impulsiveness may be a second common vulnerability factor for both substance use and bipolar disorder and may also mediate their co-occurrence.

RI and BAS sensitivity may also interact to affect bipolar disorders. Individuals with hypomanic personality who were low on impulsiveness were least likely to develop bipolar disorder and experienced better outcomes (e.g., fewer arrests) in a longitudinal follow-up (Kwapil, Miller, Zinser, Chapman, & Eckblad, 2000). Similarly, although students with bipolar spectrum disorders exhibited academic impairment relative to controls, the subgroup that scored high on the BAS Drive subscale but low on impulsiveness was protected from such impairment (Nusslock, Alloy, Abramson, Harmon-Jones, & Hogan, 2008).

The Present Study

Thus, the goal of the present 1-year longitudinal study was to examine BAS sensitivity and impulsiveness as predictors of bipolar disorder status and substance use (alcohol and drugs) and their co-occurrence in a sample of late adolescents and young adults with bipolar spectrum disorders and demographically matched control participants. Despite the high prevalence of comorbidity between bipolar spectrum disorders and substance use, studies have yet to examine the potential role of BAS sensitivity and impulsiveness in their co-occurrence. We hypothesized that both high self-reported BAS sensitivity and high self-reported impulsiveness (a) would be associated with lifetime bipolar spectrum and SUD diagnosis (Hypothesis 1), (b) would predict substance use problems prospectively (Hypothesis 2), and (c) would mediate the co-occurrence of bipolar disorder and substance use (Hypothesis 3). We also explored whether high impulsiveness mediated or moderated the association of high BAS sensitivity with bipolar disorder status, substance use, and their co-occurrence.

Method

Participants

Participants were part of a larger prospective longitudinal study of the psychosocial, cognitive, and biological predictors of the course of bipolar spectrum disorders. They were selected via a two-stage screening process. In Phase I, approximately 20,500 18- to 24-year-old students at a large northeastern university and a large midwestern university completed the revised General Behavior Inventory (GBI; Depue et al., 1981). Those who met the GBI cutoff scores recommended by Depue et al. (1981; see the *Measures* section below) to identify individuals on the bipolar spectrum and control participants were invited to participate in Phase II, which involved an interview with the expanded Schedule for Affective Disorders and Schizophrenia–Lifetime (exp-SADS-L; Endicott & Spitzer, 1978) diagnostic interview. Diagnoses were assigned on the basis of the *Diagnostic and Statistical Manual for Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) and/or Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978). Those receiving a diagnosis of bipolar II

disorder or cyclothymia were invited to participate in the longitudinal study. Participants with a lifetime history of mania were excluded from the final sample, as a goal of the larger project was to assess risk factors for the development of full-blown bipolar I disorder in those with milder bipolar spectrum conditions. During Phase II, lifetime history of alcohol and substance abuse and dependence was also assessed via the exp-SADS-L.

Control participants were recruited and matched with participants on the bipolar spectrum on age, sex, and ethnicity. Only those control participants with no lifetime history of Axis I psychopathology (with the exception of specific phobia)¹ and no family history of mood disorders were included in the final sample. Informed consent was obtained from all participants prior to their participation in the study.

The final sample consisted of 149 participants with bipolar II disorder (56 men, 93 women) and 57 participants with cyclothymia (23 men, 34 women), with a mean age of 19.6 years (± 1.6 years). Among participants with bipolar spectrum disorders, the ethnic composition was 68.9% Caucasian, 13.1% African American, 5.1% Hispanic, 3.6% Asian, 0.5% Native American, and 8.2% other. Only 31 of the participants with bipolar spectrum disorders (15%) had sought treatment (medication or psychotherapy) prior to their entry into the study, whereas 63 (30.6%) sought treatment during the follow-up. Of those who sought treatment after inclusion in the study, 32 (15.5%) received medication, 26 (12.6%) received psychotherapy only, and 7 (3.4%) were hospitalized (of these, 2 received other treatment as well). The final control sample consisted of 208 participants (86 men, 122 women) with a mean age of 19.7 years (± 1.5 years). The ethnic composition of the control participants was 72.8% Caucasian, 12.1% African American, 3.4% Hispanic, 4.4% Asian, 0.5% Native American, and 6.8% other.

Only participants with complete data on impulsivity and BAS sensitivity were included in the present analyses, resulting in a final sample for this study of 132 participants with bipolar spectrum disorders (98 bipolar II, 34 cyclothymia) and 153 control participants. Demographic and clinical characteristics of the current sample are presented as a function of diagnosis in Table 1. Overall, 59.6% ($n = 170$) were women. Participant ages ranged from 18 to 24 years with a mean age of 19.65 years (± 1.74 years). Gender, age, and ethnicity did not differ significantly by diagnosis, $\chi^2(1, N = 285) = 0.094, p = .760$; $t(211) = -0.095, p = .924$; and $\chi^2(5, N = 213) = 3.40, p = .639$, respectively.

¹ During the end of the recruitment period of the Longitudinal Investigation of Bipolar Spectrum Project sample, it was becoming difficult to complete the recruitment of control participants matched to the participants in the bipolar group on age, sex, and ethnicity, who had no history of any Axis I disorders. We found that some of the control participants who matched the participants with bipolar disorder on demographics had a prior history of a single specific phobia (e.g., of spiders). Thus, we decided to allow control participants with a history of one specific phobia into the control group. Control participants with a specific phobia represented a small proportion (about 5%) of the total control group. From an RST perspective, the presence of some control participants with specific phobia could lead the control group to score higher than the bipolar group on BIS sensitivity. However, this was not the case: The two groups did not differ on BIS sensitivity (see Table 1).

Table 1
Sample Demographic and Clinical Characteristics ($N = 285$)

Characteristic	Control	Bipolar spectrum
<i>n</i>	153	132
Age in years	19.64 (1.84)	19.66 (1.63)
Gender (female)	58.8%	60.6%
Ethnicity		
Caucasian	77.9%	75.0%
African American	8.8%	14.0%
Hispanic	3.5%	4.0%
Asian	1.8%	1.0%
Native American	0.0%	1.0%
Other	8.0%	5.0%
BAS total	37.85*** (5.12)	40.96*** (5.34)
BAS Drive	10.42*** (2.38)	11.57*** (2.34)
BAS Reward Responsiveness	16.41* (1.93)	19.98* (1.98)
BAS Fun Seeking	10.99*** (2.26)	12.42*** (2.38)
BIS	20.15 (3.14)	20.83 (3.75)
Impulsive Nonconformity	9.50*** (6.56)	17.42*** (8.63)
Lifetime substance diagnosis (any) ^a		
Yes		24.5%
No		75.5%
MAST-DAST average	1.91*** (0.89)	2.53*** (1.26)

Note. Means are presented with standard deviations in parentheses. MAST = Michigan Alcoholism Screening Test; DAST = Drug Abuse Screening Test.

^a Individuals with a lifetime history of substance abuse or dependence were screened out of the normal control group. Thus, lifetime history of substance dependence is not reported for the normal control group.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Procedure

At Time 1, participants completed the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979), the Halberstadt Mania Inventory (HMI; Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999), the BIS/BAS Scales (Carver & White, 1994), and the Impulsive Nonconformity Scale (IN; Chapman et al., 1984). Approximately every 4 months over the prospective follow-up period, participants were interviewed with an expanded SADS-Change diagnostic interview (exp-SADS-C) to assess the occurrence of major depressive, hypomanic, and manic episodes over the past 4 months. In addition, at each 4-month interval, participants completed the Michigan Alcoholism Screening Test (MAST; Selzer, 1971) and the Drug Abuse Screening Test (DAST; Skinner, 1992). The present study was based on data from the first three follow-up periods (1 year). All participants were reimbursed for their time as follows: \$5 for Phase I completion of GBI; \$25 for Phase II completion of exp-SADS-L; \$80 for completion of all baseline measures at first regular prospective assessment; \$50 for all subsequent regular prospective assessments.

Measures

GBI (Depue et al., 1981; Depue, Krauss, Spont, & Arbisi, 1989). The GBI is a 73-item self-report questionnaire designed as a cost-effective and rapid first-stage screening method for identifying individuals with bipolar spectrum disorders. Items assess the intensity, duration, and frequency of bipolar symptoms. The inventory has two subscales: a Depression subscale (GBI-D)

and a combined Hypomanic and Biphasic subscale (GBI-HB). Participants rate the frequency with which each symptom occurs on a 4-point scale, with 1 representing *never* and 4 representing *very often or almost constantly*. In line with Depue et al. (1981, 1989), we used a case scoring method, in which one point was added to the total score for all items rated 3 (*often*) or 4 (*very often*). Participants who exceeded Depue et al.'s (1981) recommended cutoff scores (GBI-D ≥ 11 ; GBI-HB ≥ 13) were considered potential participants for the bipolar group and those who scored below these cutoffs were considered potential control participants; members of both of these groups were invited to participate in Phase II. In past research (Depue et al., 1989), the GBI has shown good internal consistency ($\alpha = .90-.96$), high specificity ($r = .99$), and adequate sensitivity ($r = .78$) and test-retest reliability ($r = .71-.74$). In addition, it has shown good discriminant validity, correctly identifying those with affective disorders among those with nonaffective disorders (Mallon, Klein, Bornstein, & Slater, 1986) and has been validated in a variety of samples, including university students, individuals with a parental history of bipolar disorder, and outpatients (Depue et al., 1981, 1989; Klein, Depue, & Slater, 1986).

Exp-SADS-L (Endicott & Spitzer, 1978). The exp-SADS-L is a semistructured diagnostic interview that assesses current and lifetime history of Axis I disorders, including lifetime history of alcohol and substance abuse and dependence. To increase reliability and accuracy in diagnosing bipolar spectrum disorders, we expanded the SADS-L and SADS-C (see below for an overview of the exp-SADS-C) interviews in consultation with Akiskal, Angst, Clayton, Endicott, and Gruenberg, who are experts on bipolar disorders. The following alterations were made to the exp-SADS-L:

1. Additional probes were included to allow for the assignment of *DSM-IV* diagnoses in addition to research diagnostic criteria diagnoses.
2. In the Depression, Mania, Hypomania, and Cyclothymia sections, the number of items was expanded and additional probes were incorporated to capture details regarding episodes and frequency and duration of symptoms.
3. Additional probes were added to assess the precise number of days and percentage of waking hours participants felt depressed, euphoric, or irritable in the Depression, Mania, and Hypomania sections, respectively.
4. Probes in the Depression, Mania, Hypomania, and Cyclothymia sections were improved on the basis of Depue's (1985) Behavioral Variability Interview.
5. In the Cyclothymia section, items were added to assess the frequency, duration, and switch rapidity of the depressive and hypomanic periods.
6. Probes were added to examine the extent to which participants' changes in behavior were noted by people in their lives.
7. For each symptom item, a 5-point scale was used (0–4), with a rating of 3 representing the cutoff score for the clinical presence of the symptom.

8. Sections assessing past episodes of depression, mania, and hypomania were placed immediately following their corresponding current sections to enhance clarity and facilitate comprehension.
9. Sections were added assessing eating disorders, ADHD, and acute stress disorder.
10. Additional probes were included in the anxiety disorders section.
11. An organic rule-out module and a medical history section were appended.

The exp-SADS-L interview has demonstrated good interrater reliability for diagnoses of major depressive episodes ($\kappa_s > .95$) and all unipolar diagnoses ($\kappa_s > .90$) on the basis of 80 jointly rated interviews in a previous study (Alloy et al., 2000) and for bipolar spectrum diagnoses ($\kappa_s > .96$) in the current project on the basis of 105 jointly rated interviews. Interviews were conducted by research assistants who had completed over 200 hr of training and were blind to participants' Phase I GBI scores, BIS/BAS Scales scores, and IN scores. Consensus *DSM-IV* and Research Diagnostic Criteria diagnoses were determined by a three-tiered standardized diagnostic review procedure involving senior diagnosticians and an expert psychiatric diagnostic consultant, Alan Gruenberg.

BIS/BAS Scales (Carver & White, 1994). The BIS/BAS Scales are the self-report measures most widely used to assess sensitivity of the BIS and BAS. The scales consist of 20 items that are rated on a 4-point Likert-type scale (1 = *strongly disagree*, 4 = *strongly agree*) and comprise three BAS subscales and one BIS subscale. The BAS Reward Responsiveness subscale has five items designed to assess positive response to reward stimuli. The BAS Drive subscale has four items indicative of persistence in pursuit of reward. The BAS Fun Seeking subscale contains four items indicative of willingness to approach novel and rewarding stimuli. All subscales have adequate internal consistency ($\alpha_s = .66-.76$) and test-retest reliability ($r_s = .59-.69$; Carver & White, 1994). The BIS/BAS Scales have demonstrated construct validity; they have been associated with prefrontal cortical activity, affect, personality traits, and performance on reaction-time and learning tasks involving incentives (Colder & O'Conner, 2004; Harmon-Jones & Allen, 1997; Kambouropoulos & Staiger, 2004; Sutton & Davidson, 1997; Zinbarg & Mohlman, 1998). The items on the BAS Scales did not overlap in content with the items from the exp-SADS-L and exp-SADS-C interviews used to assess the (hypo)manic symptom criteria for a bipolar spectrum disorder diagnosis or with items on our self-report measure of (hypo)manic symptoms (HMI; see below). Moreover, Alloy et al. (2008, Table 2) reported that the correlations between the HMI and the BAS total and BAS subscales ranged from .038 to .214, corresponding to a maximum of 4.6% of shared variance. Thus, the BAS scales appear to measure a distinct construct of behavioral approach, sufficiently distinct from (hypo)manic symptoms themselves.

IN (Chapman et al., 1984). The IN consists of 51 true-false items that tap impulsive behavior. Items include "When I want something, delays are unbearable," and "I avoid trouble whenever I can" (reverse scored). The IN has good internal consistency ($\alpha_s = .79-.84$; Alloy et al., 2006; Chapman et al., 1984) and

6-week retest reliability ($r = .84$; Chapman et al., 1984). Chapman et al. (1984) found that individuals who scored high on the IN were more likely to endorse antisocial, psychotic, depressive, and hypomanic/manic symptoms than were those in a control group. Moreover, Alloy et al. (2006) found that individuals high in BAS sensitivity exhibited higher IN scores than did individuals with moderate BAS sensitivity.

BDI (Beck et al., 1979). The BDI is a 21-item self-report scale that assesses affective, motivational, cognitive, and somatic symptoms of depression. Participants select among four statements graded in severity, which are weighted from 0 to 3, with 3 representing the highest level of symptom severity. Total scores range from 0 to 63. The BDI has demonstrated good internal consistency, retest reliability, and concurrent validity with clinical depression ratings in both clinical ($r = .72$) and nonclinical ($r = .60$) samples. Furthermore, it has been validated in samples of undergraduates (Beck, Steer, & Garbin, 1988).

HMI (Alloy et al., 1999). The HMI is a 28-item self-report questionnaire, modeled after the BDI, designed to assess current affective, motivational, cognitive, and somatic symptoms of (hypo)mania. As with the BDI, participants select one of four statements representing differing degrees of symptom severity (e.g., *I do not feel particularly happy, I feel happy, I feel so happy and cheerful it's like a high, and I am bursting with happiness and I'm on top of the world*). In the Longitudinal Investigation of Bipolar Spectrum Project, the HMI demonstrated construct validity and was correlated ($r = .46$) with hypomanic symptoms reported on the exp-SADS-C interview (Alloy et al., 2008). Moreover, as would be expected, cyclothymic individuals reported HMI scores consistent with their current mood state, reporting significantly more hypomanic symptoms on the HMI when they were in hypomanic states ($M = 23.9$) than when they were in depressed ($M = 15.7$) or euthymic states ($M = 18.8$). In a sample of 1,282 undergraduates (Alloy et al., 1999), the HMI showed high internal consistency ($\alpha = .82$), adequate convergent validity with the mania scale of the Minnesota Multiphasic Personality Inventory ($r = .32, p < .001$), and adequate discriminant validity both with the Minnesota Multiphasic Personality Inventory Depression Scale ($r = -.26, p < .001$) and with the BDI ($r = -.12, p < .001$).

Exp-SADS-C (Spitzer & Endicott, 1978). The exp-SADS-C interview was administered at 4-month intervals over the prospective follow-up period. It was used to assess onset, duration, and severity of symptoms and episodes of Axis I psychopathology, including major depressive, manic, and hypomanic episodes. Interviewers were blind to the diagnostic status of participants, as well as to their GBI, BIS/BAS Scales, and IN scores. The SADS-C was expanded in the same manner as the SADS-L. In addition, it also incorporated features of the Longitudinal Interval Follow-up Evaluation (Shapiro & Keller, 1979). This allowed detailed tracking of the course of symptoms and episodes over each 4-month period. In the current project, the exp-SADS-C demonstrated good interrater reliability ($\kappa > .80$) in joint ratings of 60 interviews. Moreover, in a validity study, participants reported the dates of their symptoms on the exp-SADS-C with at least 70% accuracy relative to daily symptom ratings made over a 4-month interval.

MAST (Selzer, 1971). The MAST is a commonly used self-report questionnaire designed to screen for alcohol use problems. It has 24 items assessing symptoms of alcohol abuse and dependence. Items are given a score of 0, 1, 2, or 5 and are summed to

yield total MAST scores ranging from 0 to 53, with higher scores indicating greater alcohol use problems. The MAST has shown good reliability (Marion, Fuller, Johnson, Michels, & Diniz, 1996) and sensitivity (Maisto, Connors, & Allen, 1995), adequate internal consistency (Conley, 2001), construct validity (Conley, 2001), and good concurrent validity with *DSM-IV* diagnostic criteria (Watson, Detra, Fox, & Ewing, 1995). The MAST was administered regularly at 4-month intervals, and participants were asked to provide separate responses for each individual month. Cronbach's α for dichotomized initial MAST scores in the present sample was .64, which falls within the range reported in a recent reliability meta-analysis of the MAST (Shields, Howell, Potter, & Weiss, 2007).

DAST (Skinner, 1992). The DAST is a self-report questionnaire designed to assess drug use problems. It consists of 28 yes-no items indicative of symptoms of drug use and dependence. Items are weighted 0 or 1, and responses are summed to yield a total score ranging from 0 to 28, with higher scores indicating higher levels of drug use problems. The DAST has shown good validity and reliability (Cocco & Carey, 1998; El-Bassel et al., 1997; Lin, Lee, Pan, & Hu, 2003; McCann, Simpson, Ries, & Roy-Byrne, 2000). Furthermore, it has been validated for use in both psychiatric and community settings (Cocco & Carey, 1998; El-Bassel et al., 1997; Klamen & Miller, 1997; Lin et al., 2003). As with the MAST, the DAST was administered at each follow-up assessment and yielded monthly ratings of drug use problems. Cronbach's α for dichotomized initial DAST scores in the present sample was .65.

Given that we were interested in the prediction of general substance use problems, rather than of any one particular substance, MAST and DAST scores were combined to obtain an overall measure of substance use problems. Specifically, we calculated mean MAST scores across the first three follow-up assessments and mean DAST scores across the same three follow-up assessments. We then averaged MAST and DAST scores to create a composite score representing general substance use problems over the 1-year follow-up period.

Results

Diagnostic Group Differences in BAS Sensitivity, Impulsiveness, and Substance Use Problems

Table 1 presents means and standard deviations of BAS total, BAS subscale, BIS, IN, and average MAST and DAST composite

scores. Participants in the bipolar group evidenced significantly higher BAS total, $t(278) = -4.95, p < .001$; BAS Drive, $t(280) = -4.06, p < .001$; BAS Reward Responsiveness, $t(281) = -2.41, p = .017$; and BAS Fun Seeking, $t(283) = -5.20, p < .001$, scores than did control participants. Participants in the bipolar group also scored significantly higher on the IN than did control participants, $t(283) = -8.76, p < .001$. There were no significant group differences on BIS scores, $t(283) = -1.67, p = .097$. Finally, participants in the bipolar group had significantly higher average MAST and DAST composite scores over the 1-year follow-up, $t(275) = -4.79, p < .001$.

Associations Among BAS Sensitivity, BIS Sensitivity, and Impulsiveness

Pearson correlations were conducted to examine whether the BAS/BIS Scales and IN assess unique or overlapping constructs. Intercorrelations between BAS total, BAS subscales, BIS, and IN scores are presented in Table 2. BAS total and IN scores were significantly positively correlated ($r = .41, p < .001$), but the correlation was moderate and corresponds to only 16.81% shared variance. Thus, consistent with their theoretical origins, these instruments appear to measure overlapping but distinct constructs. Likewise, IN scores were significantly positively correlated with BAS Drive ($r = .35, p < .001$) and BAS Fun Seeking ($r = .49, p < .001$) scores, but again these correlations were moderate and correspond to only 12.25% and 24.01% shared variance, respectively. BIS scores were negatively correlated with IN scores ($r = -.14, p = .018$) but were not correlated with BAS total ($r = .03, p = .637$) or any of the BAS subscales, except for BAS Reward Responsiveness ($r = .275, p < .001$).

Lifetime and Prospective Associations Between Bipolar Spectrum Diagnosis and Substance Use

We first examined the association between lifetime history of SUDs and bipolar spectrum diagnosis. In line with previous research, we expected higher rates of lifetime SUDs among those with bipolar spectrum disorders compared with control participants. Because lifetime history of SUD was an exclusion criterion for control participants in the longitudinal study, we were unable to compare rates of lifetime SUDs by diagnostic status in our analysis sample. Instead, we examined lifetime prevalence rates of SUDs among those who completed our Phase II diagnostic inter-

Table 2
Intercorrelations Between the Behavioral Approach System (BAS) Scale, BAS Subscales, Impulsive Nonconformity, and Behavioral Inhibition System (BIS) Scale

Scale	1	2	3	4	5	6
1. BAS total	—					
2. BAS Drive	.83***	—				
3. BAS Reward Responsiveness	.74***	.42***	—			
4. BAS Fun Seeking	.82***	.51***	.41***	—		
5. Impulsive Nonconformity	.41***	.35***	.11	.49***	—	
6. BIS	.03	-.09	.28***	-.08	-.14*	—

* $p < .05$. *** $p < .001$.

view. Specifically, from the Phase II sample, we compared rates of lifetime SUDs among participants with bipolar spectrum disorders (bipolar I, II, or cyclothymia; $n = 204$), those with a history of major depressive episode(s) but no (hypo)mania ($n = 251$), and those without a history of either (hypo)mania or major depression ($n = 449$). Consistent with results reported in the literature, rates of lifetime SUDs differed significantly among those with bipolar spectrum disorder (28.4%), those with a history of major depressive episode(s) but no (hypo)mania (21.1%), and those without a history of any mood disorder (14.5%), $\chi^2(2, N = 904) = 18.02, p < .001$. Pairwise comparisons revealed that participants with bipolar spectrum disorders had significantly higher rates of SUDs than those without a history of (hypo)mania or major depression, $\chi^2(1, N = 653) = 17.87, p < .001$. Participants with bipolar spectrum disorders also showed higher rates of SUDs than individuals with histories of major depression only, and this effect approached significance, $\chi^2(1, N = 455) = 3.27, p = .07$.

We also examined the association between bipolar spectrum status and prospective substance use problems (as measured by the combined average of monthly MAST and DAST scores), controlling for lifetime SUD. Despite gender differences in mean combined MAST–DAST scores, gender was not a significant predictor of prospective substance use problems in any of our regression models and, therefore, was not included as a covariate in final analyses. A linear regression revealed that bipolar spectrum status significantly predicted prospective substance use problems ($\beta = .164, p = .005$), controlling for lifetime history of SUD. Thus, compared with control participants, bipolar spectrum individuals exhibited elevated rates of substance use, both lifetime SUDs and prospective substance use problems. Given the demonstrated association between bipolar spectrum disorders and substance use problems in this sample, we next tested our remaining hypotheses, namely, whether BAS sensitivity and IN scores represent shared vulnerabilities for both and whether they can account for their co-occurrence.

Associations of BAS Sensitivity and Impulsiveness With Bipolar Spectrum Status

Logistic regression analyses were conducted to examine the hypothesis that BAS sensitivity and IN scores would be related to bipolar diagnostic status. Results are presented in Table 3. We did not control for any demographic variables, as none were predictive of bipolar spectrum status. BAS total scores were associated sig-

nificantly with diagnostic status, with higher levels of BAS sensitivity among those with bipolar spectrum disorder relative to control participants. We also examined the contribution of each of the three BAS subscales (Reward, Drive, and Fun Seeking) to the prediction of bipolar spectrum status and found that all three were associated significantly with diagnostic status, with higher levels of sensitivity among those with a bipolar spectrum disorder relative to control participants. Controlling for initial depressive (BDI) and hypomanic (HMI) symptom levels, we found that BAS total and BAS Fun Seeking continued to be associated significantly with bipolar spectrum status ($B = .084, SE B = .038, p = .028$, and $B = .202, SE B = .081, p = .012$, respectively). However, BAS Drive became marginally associated with bipolar diagnostic status ($B = .142, SE B = .082, p = .083$), and BAS Reward Responsiveness was no longer associated ($B = .065, SE B = .090, p = .470$). BIS was marginally associated with diagnostic status and became nonsignificant after controlling for initial symptom levels ($B = .016, SE B = .055, p = .774$). Logistic regression analyses also revealed that IN scores were associated significantly with diagnostic status, with higher IN scores among participants in the bipolar group than among control participants. This finding remained significant even after controlling for initial symptom levels ($B = .078, SE B = .545, p < .001$).

BAS Sensitivity and Impulsiveness as Predictors of Prospective Substance Use Problems

Next, we conducted a series of hierarchical linear regression analyses to examine whether BAS sensitivity and IN scores predicted prospective substance use problems over the 1-year follow-up period. Again, we controlled for lifetime SUD diagnosis in our analyses. Results are presented in Table 4. As hypothesized, BAS total scores and IN scores each significantly predicted prospective substance use problems over the follow-up, controlling for lifetime SUD diagnosis. With regard to the BAS subscales, BAS Fun Seeking significantly predicted prospective substance use problems; however, the BAS Reward Responsiveness subscale, BAS Drive subscale, and BIS scores did not.

Examination of the Interaction Between BAS Sensitivity and Impulsiveness

Given that BAS and IN scores do represent distinct constructs in our data, we explored whether IN scores moderated the association

Table 3
Logistic Regression Models of Associations of the Behavioral Approach System (BAS) Scale Total, BAS Subscales, Behavioral Inhibition System (BIS) Scale, and Impulse Nonconformity With Bipolar Spectrum Status

Scale	β	SE β	Wald	OR	95% CI	p
BAS total	.12	.03	21.78	1.12	1.07–1.18	<.001
BAS Fun Seeking	.27	.06	24.29	1.31	1.18–1.46	<.001
BAS Drive	.20	.05	14.42	1.22	1.10–1.35	<.001
BAS Reward Responsiveness	.16	.06	6.44	1.17	1.04–1.32	.01
BIS	.07	.04	3.52	1.07	1.00–1.14	.06
Impulsive Nonconformity	.13	.02	49.17	1.14	1.10–1.18	<.001

Note. OR = odds ratio; CI = confidence interval.

Table 4
Summary of Linear Regression Models of BAS, BAS Subscales, BIS, and Impulsive Nonconformity Predicting MAST-DAST Average Over the 1-Year Follow-Up

Predictor	B	SE B	β	t	p
Model 1					
Step 1					
Lifetime SUD	0.77	0.24	.22	3.20	<.01
Step 2					
Lifetime SUD	0.73	0.24	.21	3.07	<.01
BAS total	0.04	0.01	.18	2.75	<.01
Model 2					
Step 1					
Lifetime SUD	0.91	0.24	.25	3.79	<.001
Step 2					
Lifetime SUD	0.82	0.23	.23	3.50	<.01
BAS Fun Seeking	0.11	0.03	.24	3.77	<.001
Model 3					
Step 1					
Lifetime SUD	0.90	0.24	.25	3.74	<.001
Step 2					
Lifetime SUD	0.89	0.24	.25	3.69	<.001
BAS Drive	0.04	0.03	.09	1.40	.16
Model 4					
Step 1					
Lifetime SUD	0.78	0.24	.22	3.25	<.01
Step 2					
Lifetime SUD	0.77	0.24	.22	3.21	<.01
BAS Reward	0.06	0.04	.10	1.55	.12
Model 5					
Step 1					
Lifetime SUD	0.91	0.24	.25	3.79	<.001
Step 2					
Lifetime SUD	0.92	0.24	.25	3.82	<.001
BIS	0.02	0.02	.07	1.03	.31
Model 6					
Step 1					
Lifetime SUD	0.86	0.24	.24	3.55	<.001
Step 2					
Lifetime SUD	0.62	0.25	.17	2.54	.012
Impulsiveness	0.03	0.01	.24	3.55	<.001

Note. SUD = substance use disorder; BAS = Behavioral Approach System Scale; BIS = Behavioral Inhibition System Scale; MAST = Michigan Alcoholism Screening Test; DAST = Drug Abuse Screening Test; Fun Seeking = Fun Seeking subscale; Drive = Drive subscale; Reward = Reward Responsiveness subscale. In Model 1, $R^2 = .047$ and $F(1, 208) = 10.25$ for Step 1 ($p = .002$); $\Delta R^2 = .034$ and $\Delta F(1, 207) = 7.586$ for Step 2 ($p = .006$). In Model 2, $R^2 = .063$ and $F(1, 213) = 14.36$ for Step 1 ($p = .000$); $\Delta R^2 = .059$ and $\Delta F(1, 212) = 14.24$ for Step 2 ($p = .000$). In Model 3, $R^2 = .062$ and $F(1, 210) = 13.99$ for Step 1 ($p = .000$); $\Delta R^2 = .009$ and $\Delta F(1, 209) = 1.953$ for Step 2 ($p = .164$). In Model 4, $R^2 = .048$ and $F(1, 211) = 10.559$ for Step 1 ($p = .001$); $\Delta R^2 = .011$ and $\Delta F(1, 210) = 2.402$ for Step 2 ($p = .123$). In Model 5, $R^2 = .063$ and $F(1, 213) = 14.361$ for Step 1 ($p = .000$); $\Delta R^2 = .005$ and $\Delta F(1, 212) = 1.059$ for Step 2 ($p = .305$). In Model 6, $R^2 = .056$ and $F(1, 211) = 12.614$ for Step 1 ($p = .000$); $\Delta R^2 = .053$ and $\Delta F(1, 210) = 12.606$ for Step 2 ($p = .000$).

between BAS and (a) bipolar spectrum status or (b) prospective substance use problems. Each of the previously tested models predicting bipolar status or prospective substance use problems was reanalyzed with predictors entered into the model in the following order: lifetime history of SUD diagnosis entered in Step 1 (when prospective substance use problems was the dependent variable); BAS, a BAS subscale, or BIS entered in Step 2; IN

scores entered in Step 3; and the interaction term (BAS \times IN, a BAS Subscale \times IN, or BIS \times IN) entered in Step 4. Results for these tests of moderation are presented in Tables 5 and 6, with results predicting bipolar spectrum status presented in Table 5 and results predicting prospective substance use presented in Table 6. In no case were any of the interaction terms significant, indicating that BAS and IN did not interact to predict either bipolar spectrum status or substance use problems, over and above the prediction afforded by either construct alone.

Shared Vulnerability: Test of a Mediation Model

In summary, the findings above suggest (a) that bipolar spectrum status is associated with prospective substance use, (b) that BAS sensitivity and impulsiveness both are associated with bipolar spectrum status, and (c) that BAS sensitivity and impulsiveness both are associated with prospective substance use. Therefore, we were able to test our final hypothesis that the positive association between bipolar spectrum status and prospective substance use problems might be attributable in part to the shared personality factors of BAS sensitivity and impulsiveness. Such mediation hypotheses are often tested using the causal steps approach popularized by Baron and Kenny (Baron & Kenny, 1986) or the product-of-coefficients approach developed by Sobel (Sobel, 1982, 1986). Both procedures use parametric techniques that assume multivariate normality of the sampling distribution of total and specific indirect effects, which can be problematic except in very large samples (MacKinnon, Rose, Chassin, Presson, & Sherman, 2000; Preacher & Hayes, 2008). The present study used a bootstrapping approach to multiple mediation that does not impose such an assumption, a process that simultaneously increases power and maintains reasonable control over the Type I error rate (Preacher & Hayes, 2008). Bootstrapping is a nonparametric resampling technique that empirically generates an approximation of the sampling distribution. In the case of multiple mediation models, sampling distributions of total and indirect effects are empirically generated by selecting a subsample, with replacement, of the full data set and then calculating indirect effects in the repeated subsamples. The procedure yields point estimates and percentile confidence intervals for indirect and total effects. In the present study, bootstrap percentile confidence intervals were further improved using bias-correction and acceleration, as recommended by Preacher and Hayes (2008). Lifetime history of SUD was entered as a covariate in each model. Controlling for lifetime history of SUD represents a conservative test of our hypothesis, because BAS sensitivity and/or IN score may represent vulnerabilities to lifetime SUDs as well as to prospective substance use problems.

Table 7 presents point estimates and bias-corrected and accelerated bootstrap confidence intervals based on 5,000 bootstrap samples, for both simple and multiple mediator models of our hypotheses. Tests of simple indirect effects (Table 7, top part) indicated that the relationship between bipolar spectrum diagnosis and prospectively assessed substance use problems was significantly mediated by BAS total, BAS Fun Seeking, and IN scores but not by BAS Drive, BAS Reward Responsiveness, or BIS scores. Conclusions drawn from an examination of the bias-corrected and accelerated confidence intervals were consistent with results of a traditional product-of-coefficients approach: the Sobel test statistic was 1.81 for BAS total ($p = .07$), 2.86 for BAS

Table 5
Logistic Regression Models With BAS, BAS Subscales, IN, and the Interaction of BAS and BAS Subscales With IN Predicting Bipolar Spectrum Status

Predictor	β	SE	Wald	OR	95% CI
Model 1					
Step 1					
BAS total	.114	.025	20.936	1.121***	1.067–1.177
Step 2					
BAS total	.052	.028	3.355	1.053	0.996–1.113
IN	.119	.020	36.188	1.126***	1.083–1.170
Step 3					
BAS total	.050	.028	3.199	1.052	0.995–1.111
IN	.118	.020	0.020	35.96***	1.083–1.170
BAS \times IN	.002	.004	0.287	1.002	0.995–1.009
Model 2					
Step 1					
BAS Fun Seeking	.265	.055	23.089	1.303***	1.170–1.452
Step 2					
BAS Fun Seeking	.086	.064	1.782	1.09	0.961–1.236
IN	.120	.020	34.206	1.127***	1.083–1.173
Step 3					
BAS Fun Seeking	.087	.064	1.836	1.09	0.962–1.236
IN	.119	.021	33.487	1.126***	1.082–1.172
Fun Seeking \times IN	.006	.008	0.593	1.006	0.990–1.023
Model 3					
Step 1					
BAS Drive	.205	.053	14.893	1.228***	1.106–1.363
Step 2					
BAS Drive	.087	.061	2.064	1.091	0.969–1.230
IN	.124	.019	40.972	1.132***	1.090–1.176
Step 3					
BAS Drive	.086	.061	1.982	1.09	0.967–1.228
IN	.124	.019	40.671	1.132***	1.089–1.175
Drive \times IN	.002	.008	0.086	1.002	0.987–1.018
Model 4					
Step 1					
BAS Reward	.148	.063	5.615	1.16*	1.026–1.311
Step 2					
BAS Reward	.118	.070	2.839	1.125	0.981–1.291
IN	.129	.019	47.584	1.138***	1.097–1.181
Step 3					
BAS Reward	.118	.070	2.841	1.125	0.981–1.289
IN	.130	.019	47.71	1.139***	1.097–1.181
Reward \times IN	.006	.009	0.371	1.006	0.987–1.024
Model 5					
Step 1					
BIS	.058	.035	2.745	1.06	1.026–1.311
Step 2					
BIS	.130	.042	9.381	1.139**	0.981–1.291
IN	.144	.020	53.094	1.155***	1.097–1.181
Step 3					
BIS	.130	.043	9.371	1.139**	0.981–1.289
IN	.144	.020	52.429	1.155***	1.097–1.181
BIS \times IN	.000	.005	0.021	0.999	0.987–1.024

Note. OR = odds ratio; 95% CI = 95% confidence interval for odds ratio; BAS = Behavioral Approach System Scale; BIS = Behavioral Inhibition System Scale; IN = Impulsive Nonconformity Scale; Fun Seeking = Fun Seeking subscale; Drive = Drive subscale; Reward = Reward Responsiveness subscale. In Model 1, Block 1, $\chi^2(1, N = 280) = 23.657, p = .000$. In Model 1, Block 2, $\chi^2(1, N = 280) = 45.250$ for Step 1 ($p = .000$) and $\chi^2(2, N = 280) = 68.907$ for the model ($p = .000$). In Model 1, Block 3, $\chi^2(1, N = 280) = 0.287$ for Step 1 ($p = .592$) and $\chi^2(3, N = 280) = 69.195$ for the model ($p = .000$). In Model 2, Block 1, $\chi^2(1, N = 285) = 42.613$ ($p = .000$). In Model 2, Block 2, $\chi^2(1, N = 285) = 42.613$ for Step 1 ($p = .000$) and $\chi^2(2, N = 285) = 68.413$ for the model ($p = .000$). In Model 2, Block 3, $\chi^2(1, N = 285) = 0.597$ for Step 1 ($p = .440$) and $\chi^2(3, N = 285) = 69.010$ for the model ($p = .000$). In Model 3, Block 1, $\chi^2(1, N = 285) = 16.083$ ($p = .000$). In Model 3, Block 2, $\chi^2(1, N = 285) = 52.199$ for Step 1 ($p = .000$) and $\chi^2(2, N = 285) = 68.282$ for the model ($p = .000$). In Model 3, Block 3, $\chi^2(1, N = 285) = 0.087$ for Step 1 ($p = .768$) and $\chi^2(3, N = 285) = 68.369$ for the model ($p = .000$). In Model 4, Block 1, $\chi^2(1, N = 283) = 5.785$ ($p = .016$). In Model 4, Block 2, $\chi^2(1, N = 283) = 63.007$, for Step 1 ($p = .000$) and $\chi^2(2, N = 283) = 68.792$ for the model ($p = .000$). In Model 4, Block 3, $\chi^2(1, N = 283) = 0.368$ for Step 1 ($p = .544$) and $\chi^2(3, N = 283) = 69.160$ for the model ($p = .000$). In Model 5, Block 1, $\chi^2(1, N = 285) = 2.88, p = .095$. In Model 5, Block 2, $\chi^2(1, N = 285) = 73.762$, for Step 1 ($p = .000$) and $\chi^2(2, N = 295) = 68.792$ for the model ($p = .000$). In Model 5, Block 3, $\chi^2(1, N = 285) = 0.021$ for Step 1 ($p = .884$) and $\chi^2(3, N = 285) = 76.572$ for the model ($p = .000$).

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 6
 Summary of Linear Regression Models of BAS Total, BAS Subscales, BIS, Impulsive Nonconformity and the Interaction of BAS, BAS Subscales, and BIS With Impulsive Nonconformity Predicting MAST-DAST Average Over the 1-Year Follow-Up, Controlling for Lifetime History of Substance Use

Model 1		Model 2		Model 3		Model 4		Model 5		
	β	t	β	t	β	t	β	t	β	t
Step 1										
Lifetime SUD	.201**	2.927	.238**	3.536	.238**	3.536	.203**	2.975	.237**	3.488
Step 2										
Lifetime SUD	.192**	2.818	.240***	3.565	.214**	3.245	.201**	2.065	.234**	3.455
BAS	.171*	2.522	.066	.979	.232**	3.515	.083	1.214	.096	1.413
Step 3										
Lifetime SUD	.147*	2.112	.173*	2.541	.179**	2.638	.142*	2.065	.174*	2.517
BAS	.089	1.187	.097	1.465	.155*	2.062	.050	.750	.009	.126
IN	.192*	2.488	.252***	3.671	.159*	2.043	.229**	3.293	.231**	3.094
Step 4										
Lifetime SUD	.146*	2.096	.172*	2.531	.177*	2.612	.142*	2.050	.174*	2.505
BAS	.088	1.171	.100	1.496	.156*	2.066	.052	.770	.008	.115
IN	.190*	2.449	.250***	3.640	.155*	1.981	.229**	3.277	.230**	3.059
BAS × IN	.022	.326	-.022	.746	.034	.518	.040	.603	.010	.882

Note. SUD = substance use disorder; BAS = Behavioral Approach System Scale; BIS = Behavioral Inhibition System Scale; IN = Impulsive Nonconformity Scale; Fun Seeking = Fun Seeking subscale; Drive = Drive subscale; Reward = Reward Responsiveness subscale. In Model 1, $R^2 = .041$ and $F(1, 203) = 8.569$ for Step 1 ($p = .004$); $\Delta R^2 = .029$ and $\Delta F(1, 202) = 6.359$ for Step 2 ($p = .012$); $\Delta R^2 = .028$ and $F(1, 201) = 6.188$ for Step 3 ($p = .014$); $\Delta R^2 = .000$ and $\Delta F(1, 200) = 0.106$ for Step 4 ($p = .745$). In Model 2, $R^2 = .057$ and $F(1, 208) = 12.505$ for Step 1 ($p = .001$); $\Delta R^2 = .004$ and $\Delta F(1, 207) = 0.959$ for Step 2 ($p = .329$); $\Delta R^2 = .058$ and $F(1, 206) = 13.473$ for Step 3 ($p = .000$); $\Delta R^2 = .000$ and $\Delta F(1, 205) = 0.105$ for Step 4 ($p = .746$). In Model 3, $R^2 = .057$ and $F(1, 208) = 12.505$ for Step 1 ($p = .001$); $\Delta R^2 = .053$ and $\Delta F(1, 207) = 12.354$ for Step 2 ($p = .001$); $\Delta R^2 = .018$ and $\Delta F(1, 206) = 4.176$ for Step 3 ($p = .042$); $\Delta R^2 = .001$ and $\Delta F(1, 205) = 0.268$ for Step 4 ($p = .605$). In Model 4, $R^2 = .041$ and $F(1, 206) = 8.849$ for Step 1 ($p = .003$); $\Delta R^2 = .007$ and $\Delta F(1, 205) = 1.473$ for Step 2 ($p = .226$); $\Delta R^2 = .048$ and $F(1, 204) = 10.847$ for Step 3 ($p = .001$); $\Delta R^2 = .002$ and $\Delta F(1, 203) = .364$ for Step 4 ($p = .547$). In Model 5, $R^2 = .056$ and $F(1, 205) = 12.163$ for Step 1 ($p = .001$); $\Delta R^2 = .009$ and $\Delta F(1, 204) = 1.998$ for Step 2 ($p = .159$); $\Delta R^2 = .042$ and $\Delta F(1, 203) = 9.574$ for Step 3 ($p = .002$); $\Delta R^2 = .000$ and $\Delta F(1, 202) = .022$ for Step 4 ($p = .882$).
 * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 7
Simple and Multiple Mediation of the Indirect Effects of Bipolar Spectrum Diagnosis on Prospective Substance Use Problems Through the Behavioral Approach System (BAS) Scale, BAS Subscales, Impulsive Nonconformity Scale, and Behavioral Inhibition System (BIS) Scale

Mediator or mediation model	Point estimate	BCa 95% CI	
		Lower	Upper
Simple indirect effects			
BAS total	0.0899*	0.0029	0.2194
Fun Seeking	0.1391*	0.0415	0.2815
Reward	0.0296	-0.0171	0.1168
Drive	0.0264	-0.0317	0.1189
Impulsiveness	0.1747*	0.0418	0.1738
BIS	0.0140	-0.0226	0.0842
Multiple indirect effects			
Model 1			
Indirect effects			
BAS total	0.0493	-0.0526	0.1636
Impulsiveness	0.1401*	0.0008	0.3525
Total	0.1895*	0.0353	0.3993
Contrasts			
BAS total vs. Impulsiveness	-0.0908	-0.3539	0.1013
Model 2			
Indirect effects			
Fun Seeking	0.0966	-0.0039	0.2287
Impulsiveness	0.1118	-0.0282	0.3454
Total	0.2084*	0.0587	0.4289
Contrasts			
Fun Seeking vs. Impulsiveness	-0.0152	-0.2951	0.1905
Model 3			
Indirect effects			
Drive	0.0010	-0.0798	0.0744
Impulsiveness	0.1748*	0.0342	0.3970
Total	0.1757*	0.0309	0.3916
Contrasts			
Drive vs. Impulsiveness	-0.1738	-0.4178	-0.0016
Model 4			
Indirect effects			
Reward Responsiveness	0.0151	-0.0341	0.0943
Impulsiveness	0.1711*	0.0372	0.3905
Total	0.1862*	0.0364	0.4186
Contrasts			
Reward Responsiveness vs. Impulsiveness	-0.1560	-0.3620	-0.0121
Model 5			
Indirect effects			
BIS	0.0248	-0.0058	0.1103
Impulsiveness	0.1964*	0.0523	0.4127
Total	0.2212*	0.0622	0.4528
Contrasts			
BIS vs. Impulsiveness	0.1717*	0.0258	0.3829

Note. BCa = bias corrected and accelerated bootstrapping confidence intervals that include correction for median bias and skew. Confidence intervals containing zero are interpreted as being not significant.

* $p < .05$.

Fun Seeking ($p = .004$), and 3.21 for IN ($p = .001$). Sobel tests for all other simple mediation models were nonsignificant.

Bootstrapped tests of simultaneous multiple indirect effects were conducted to determine the unique ability of each putative mediator to account for the effects of bipolar diagnosis on prospective substance use problems. In other words, we examined whether the relationship between bipolar disorder and substance use problems was significantly mediated by (a) BAS total, BAS subscales, or BIS, even after controlling for IN scores, and (b) IN,

even after controlling for the effects of BAS total, BAS subscales, or BIS. Results are presented in Table 7. When entered simultaneously with IN score, BAS total and BAS Fun Seeking no longer significantly mediated the bipolar spectrum-prospective substance use relationship. Impulsiveness, however, remained as a significant mediator even when controlling for the simultaneous specific indirect effects through BAS, BAS Reward Responsiveness, BAS Drive, and BIS. The sum of the specific indirect effects (i.e., total indirect effects) was significant in all multiple mediation models.

When contrasting specific indirect effects within each model, no significant differences were found between BAS total or BAS Fun Seeking and impulsiveness. Significant differences did emerge between specific indirect effects of (a) BAS Drive and impulsiveness and (b) BAS Reward Responsiveness and impulsiveness. A contrast of the specific indirect effects of BIS and impulsiveness was also significant. Of note, all confidence intervals constructed for interaction terms between the BIS/BAS Scales (BAS total, BAS subscales, or BIS) and IN contained zero, indicating nonsignificant interactions (consistent with the nonsignificant interaction effects shown in Tables 5 and 6). Moreover, inclusion of interaction terms in the multiple mediation models did not change the significance or direction of relationships between other variables.

Discussion

Our aim in the present study was to examine whether high BAS sensitivity and/or impulsiveness represent shared vulnerabilities to bipolar spectrum disorders and substance use problems. Consistent with prior evidence of high co-occurrence between bipolar disorders and SUDs (e.g., Conway et al., 2006; Grant et al., 2004), our initial analyses revealed that individuals with bipolar spectrum disorders were at elevated risk for lifetime SUDs and that they exhibited higher levels of prospective substance use problems. Specifically, in the large screening sample, individuals diagnosed with bipolar disorder (bipolar I, bipolar II, or cyclothymia) exhibited higher rates of lifetime SUDs than did those with either a history of unipolar major depression or no history of mood episodes. In addition, in the main analysis sample, bipolar spectrum status (bipolar II or cyclothymia) also prospectively predicted increased substance use problems over the 1-year follow-up, even after controlling for lifetime history of SUDs. Thus, the consistently reported association between bipolar disorder and SUD was conceptually replicated in our sample and extended with the more powerful prospective design. These results also allowed us to test the shared vulnerability hypothesis.

Consistent with Hypothesis 1, RST (Gray, 1982; Gray & McNaughton, 2000), and the BAS dysregulation model (Depue & Iacono, 1989; Depue et al., 1987; Urosevic et al., 2008), individuals with bipolar spectrum disorders, as compared with controls, exhibited significantly higher self-reported BAS sensitivity, as measured by BAS total, Drive, Fun Seeking, and Reward Responsiveness scores. This finding is consistent with the prior results of Alloy et al. (2008); in that study, high self-reported BAS sensitivity also predicted shorter time to onset of hypomanic/manic episodes among individuals with bipolar spectrum disorders. Our analyses revealed that BAS sensitivity (specifically, BAS total and Fun Seeking scores) continued to significantly predict bipolar spectrum diagnosis, even after controlling for initial symptom levels. High self-reported impulsiveness also predicted bipolar spectrum status, controlling for initial symptom levels. Thus, results are consistent with the hypothesis that both BAS sensitivity and impulsiveness may confer vulnerability to bipolar spectrum disorders.

Consistent with Hypothesis 2 and RST, both high self-reported BAS sensitivity and impulsiveness predicted greater substance use problems over the follow-up. As with bipolar spectrum status, it was again BAS total and Fun Seeking scores that specifically predicted prospectively assessed substance use problems, after

controlling for lifetime SUD. It is important to note that controlling for lifetime history of SUDs represents a conservative test of our hypotheses, in that BAS sensitivity and/or impulsiveness may represent vulnerabilities to lifetime SUDs as well as to prospective substance use problems. Thus, results support our second hypothesis that BAS sensitivity and impulsiveness confer vulnerability to substance use problems. That the same BAS scale scores predicted both bipolar diagnosis and substance use further underscores a possible shared personality vulnerability between the two disorders.

In contrast with findings on BAS, findings on the role of BIS in bipolar disorder and SUD expression have been mixed (Alloy et al., 2008). In the present sample, BIS sensitivity did not predict either bipolar spectrum status or substance use problems over the follow-up. Our results add to research suggesting that BIS may not play a critical role in the expression of bipolar disorder and SUD. However, it is important to note that our measure of BIS (Carver & White, 1994) was based on the 1982 version of RST and probably best assesses punishment sensitivity. It will be interesting to assess whether yet-to-be-developed measures of the revised RST's BIS may explain variance in bipolar disorder and SUDs.

With respect to our mediational hypotheses, BAS total, BAS Fun Seeking, and impulsiveness each predicted increased prospective substance use problems after controlling for lifetime history of SUD and bipolar spectrum status. Both bootstrapping methods and Sobel tests of mediation indicated that BAS total, BAS Fun Seeking, and impulsiveness all mediated the association between bipolar spectrum status and substance use problems. Thus, all three study hypotheses received support. Moreover, our bootstrapped tests of simultaneous multiple indirect effects indicated that BAS total and Fun Seeking no longer mediated the bipolar–substance use association controlling for impulsiveness, whereas impulsiveness continued to mediate bipolar–substance use co-occurrence controlling for BAS and BIS. BAS sensitivity (total or subscales) and impulsiveness did not interact to predict bipolar spectrum status, substance use problems, or their co-occurrence. Our mediational findings are consistent with the idea that although existing theory and research and the correlations between the BAS and IN instruments used in this study suggest that BAS sensitivity and impulsiveness are separate but overlapping constructs, it is the component of BAS sensitivity shared with impulsiveness that appears to be most important in mediating the co-occurrence of bipolar disorder with substance use.

It is of interest that in most analyses, BAS Fun Seeking scores were the strongest predictor among BAS total and subscale scores. Whereas BAS Drive assesses persistence in striving for goals and BAS Reward Responsiveness assesses responsiveness to obtained rewards (or consummatory, postgoal positive affect), the BAS Fun Seeking subscale assesses specifically the component of BAS sensitivity relating to willingness to approach new and potentially rewarding experiences (items include “I’m always willing to try something new if I think it will be fun” and “I crave excitement and new sensations”). Of the three BAS subscales, perhaps the BAS Fun Seeking subscale best captures anticipatory, pregoal positive affective processes that activate the nucleus accumbens (Knutson & Wimmer, 2007), a structure rich in dopamine, a neurochemical implicated in BAS sensitivity (Depue & Collins, 1999). It is important to note that the accumbens and dopamine

have been found to be involved in drug addiction in animal research (Berridge, 2007).

To our knowledge, this is the first study to demonstrate that impulsiveness and BAS sensitivity mediate the association between bipolar spectrum disorders and prospectively assessed substance use problems. Findings are consistent with the hypothesis that high BAS sensitivity and impulsiveness represent shared vulnerabilities to both bipolar disorder and substance use problems, with the component of BAS sensitivity that overlaps with impulsiveness (i.e., BAS Fun Seeking) being most important in this shared vulnerability. Although the strength of association between the tested variables was modest, it is important to note that the shared vulnerability hypothesis likely explains only a portion of bipolar spectrum–substance use co-occurrence. As noted earlier, other potential explanations include the self-medication hypothesis and the neuropsychological effects of repeated substance use over time.

Notable study strengths include the prospective assessment of substance use problems, the large sample size, interviewers blind to BIS/BAS Scales and impulsivity scores, and a unique contribution to the literatures on both RST and bipolar spectrum–substance use co-occurrence. Several study limitations should be noted as well. First, this study relied on self-report measures of BAS, impulsiveness, and substance use problems; future research should adopt a multimethod, multimeasure approach to the assessment of these constructs. In particular, it may be beneficial to assess additional dimensions of substance use, such as frequency and amount of use over the follow-up. A second limitation is that associations between bipolar spectrum diagnosis and BAS sensitivity and impulsiveness were examined cross-sectionally, although we previously demonstrated that BAS sensitivity predicted time to onset of prospectively assessed manic and hypomanic episodes (Alloy et al., 2008). Further studies are needed to determine whether BAS sensitivity and impulsiveness prospectively predict first lifetime onsets of bipolar disorder and SUD, to more precisely test the shared vulnerability hypothesis. Finally, in the present study, we examined prospective prediction of general substance use problems in an undergraduate sample. Participants experienced relatively low levels of mood symptomatology and substance use problems. Although it is noteworthy that shared vulnerability effects were demonstrated even in this relatively high-functioning sample, further research is needed to replicate findings in other samples.

In conclusion, the present study adds to a growing body of research implicating both BAS sensitivity and impulsiveness in bipolar and SUDs. Results support the central importance of heightened impulsiveness and components of BAS sensitivity that overlap with impulsiveness in both bipolar disorders and SUDs. Moreover, findings are consistent with the shared personality vulnerability hypothesis and indicate that the hypothesis warrants further evaluation. Understanding the role of these personality vulnerabilities may be especially important, as they represent potential points of behavioral intervention.

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