Behavioral Approach System (BAS)–Relevant Cognitive Styles and Bipolar Spectrum Disorders: Concurrent and Prospective Associations

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The authors examined concurrent and prospective associations of behavioral approach system (BAS)–relevant and non-BAS-relevant cognitive styles with bipolar spectrum disorders. Controlling for depressive and hypomanic/manic symptoms, 195 individuals with bipolar spectrum disorders scored higher than 194 demographically similar normal controls on BAS sensitivity and BAS-relevant cognitive dimensions of performance concerns, autonomy, and self-criticism, but not on behavioral inhibition system sensitivity and non-BAS-relevant dimensions of approval seeking, sociotropy, and dependency. Moreover, group differences on autonomy fully mediated the association between higher BAS sensitivity and bipolar status. In addition, only BAS-related cognitive dimensions predicted the likelihood of onset of depressive and hypomanic/manic episodes among the bipolar individuals over a 3.2-year follow-up, controlling for initial symptoms and past history of mood episodes. Higher autonomy and self-criticism predicted a greater likelihood of hypomanic/manic episodes, and higher autonomy predicted a lower likelihood of major depressive episodes. In addition, autonomy mediated the associations between BAS sensitivity and prospective hypomanic/manic episodes. These findings suggest that individuals with bipolar spectrum disorders may exhibit a unique profile of BAS-relevant cognitive styles that influence the course of their mood episodes.

Keywords: bipolar spectrum disorder, behavioral approach system (BAS), cognitive styles

Bipolar spectrum disorders are prevalent (4.4% of a nationally representative U.S. sample; Merikangas et al., 2007) and often produce significant impairment such as poorer academic achievement, erratic work history, divorce, suicide, and substance abuse (e.g., Angst, Stassen, Clayton, & Angst, 2002; Conway, Compton, Stinson, & Grant, 2006; Goodwin & Jamison, 1990; Grant et al., 2004; Nusslock, Alloy, Abramson, Harmon-Jones, & Hogan, 2008; Quackenbush, Kutcher, Robertson, Boulos, & Chaban, 1996; Strakowski, DelBello, Fleck, & Arndt, 2000). These disor-

ders appear to form a spectrum of severity from the milder subsyndromal cyclothymia, to bipolar II disorder, to full-blown bipolar I disorder (e.g., Akiskal, Djenderedjian, Rosenthal, & Khani, 1977; Akiskal, Khani, & Scott-Strauss, 1979; Cassano et al., 1999; Depue et al., 1981; Goodwin & Jamison, 1990). Moreover, milder forms of bipolar disorder often progress to the more severe forms (e.g., Akiskal et al., 1977, 1979; Shen, Alloy, Abramson, & Grandin, 2008), providing support for the spectrum concept.

Recently, there has been increasing interest in psychosocial processes in the onset, course, and treatment of bipolar spectrum disorders (see Alloy, Abramson, Neeren, et al., 2006; Alloy, Abramson, Urosevic, Bender, & Wagner, 2009; Alloy et al., 2005; Alloy, Abramson, Walshaw, Keyser, & Gerstein, 2006; Alloy, Abramson, Walshaw, & Neeren, 2006, for reviews). Psychological processes important in unipolar depression have been extended to research on bipolar depression specifically and bipolar spectrum disorders in general (see Alloy, Abramson, Neeren, et al., 2006; Alloy et al., 2005; Alloy, Abramson, Walshaw, Keyser, & Gerstein, 2006; Alloy, Abramson, Walshaw, & Neeren, 2006; Cuellar, Johnson, & Winters, 2005; Johnson & Kizer, 2002, for reviews). In particular, cognitive theories of unipolar depression (e.g., Abramson, Metalsky, & Alloy, 1989; Beck, 1967, 1987) have been

extended to bipolar disorder to address whether maladaptive cognitive styles similar to those seen among unipolar depressed individuals are also observed among bipolar individuals and predict the expression or course of bipolar disorder. Recent reviews of research on cognition in bipolar disorder (Alloy, Abramson, Neeren, et al., 2006; Alloy et al., 2005, 2009; Alloy, Abramson, Walshaw, Keyser, & Gerstein, 2006; Alloy, Abramson, Walshaw, & Neeren, 2006) concluded that individuals with bipolar spectrum disorders exhibit underlying cognitive patterns as negative as those of unipolar depressed persons overall but with certain unique characteristics (see below). However, the degree of negativity of the observed cognitive styles of bipolar individuals depends in part on their current mood state and on whether the cognitive style assessment uses explicit or implicit measures.

In addition, there is some evidence that cognitive styles also predict the course of bipolar disorder, alone or in combination with relevant life events. However, these findings are also mixed. Whereas two studies (e.g., Johnson & Fingerhut, 2004; Johnson, Meyer, Winett, & Small, 2000) found that negative cognitions predicted subsequent depressive, but not manic, symptoms in bipolar I samples, Scott and Pope (2003) reported that negative self-esteem was the most robust predictor of any type of relapse (depressive or hypomanic) at 12-month follow-up in hypomanic bipolar patients.

Studies testing whether Beck's (1987) sociotropic and autonomous cognitive styles combine with congruent stressful events (interpersonal events for sociotropic individuals and achievement events for autonomous individuals) to predict bipolar symptoms have also been mixed. In a bipolar I sample, Hammen, Ellicott, and Gitlin (1992) observed that the Sociotropy × Negative Interpersonal Events interaction, but not the Autonomy × Negative Achievement Events interaction, predicted subsequent symptom severity (although Hammen, Ellicott, Gitlin, & Jamison, 1989, only obtained a trend for this effect). In contrast, in a bipolar spectrum sample, Francis-Raniere, Alloy, and Abramson (2006) found that autonomous/self-critical/perfectionistic cognitive styles interacted with congruent negative events to predict increases in depressive symptoms and with congruent positive events to predict increases in hypomanic symptoms over follow-up.

Finally, two additional longitudinal studies examined attributional style and dysfunctional attitudes as predictors of bipolar symptoms. Among both unipolar and bipolar spectrum participants, Alloy, Reilly-Harrington, Fresco, Whitehouse, and Zechmeister (1999) found that a negative attributional style for negative events interacted with later negative events to predict longitudinal increases in depressive symptoms and that a positive attributional style for positive events interacted with later positive events to predict longitudinal increases in hypomanic symptoms. Dysfunctional attitudes did not predict symptom changes combined with life events. On the other hand, Reilly-Harrington, Alloy, Fresco, and Whitehouse (1999) reported that initial negative attributional styles, dysfunctional attitudes, and negative self-referent information processing each interacted with subsequent negative life events to predict increases in both depressive and hypomanic symptoms within a bipolar spectrum sample. Given that bipolar I and II individuals (in Reilly-Harrington et al., 1999) had a course of disorder that included major depressive episodes, they may have been more responsive to negative life events than were the bipolar individuals without major depression (MD) in the Alloy et al. study.

Behavioral Approach System–Relevant Cognitive Styles and Bipolar Disorder

One factor that may have contributed to the mixed findings with regard to the cognitive style–bipolar disorder concurrent and prospective associations is the particular types of cognitive styles examined. The behavioral approach system (BAS) is a psychobiological system that regulates approach behavior and appetitive motivation in response to goals and rewards (e.g., Davidson, 1999; Gray, 1981, 1982). When relevant external (e.g., an attractive goal object) or internal (e.g., expectancies of goal attainment) cues activate the BAS, the person increases movement toward attaining goals and cognitive activity (e.g., planning, self-efficacy, hope) aimed at promoting goal attainment. BAS activation has been associated with hope, elation, and happiness (Depue & Iacono, 1989; Gray, 1994). On the basis of work on BAS-mediated motivation, affect, and behavior, Depue and colleagues (Depue & Iacono, 1989; Depue, Krauss, & Spoont, 1987) proposed a BAS dysregulation theory of bipolar disorder (see also Alloy et al., 2009; Johnson, 2005; Urosevic, Abramson, Harmon-Jones, & Alloy, 2008, for updates and reviews of evidence for this theory). According to this theory, bipolar individuals have a hypersensitive BAS that becomes dysregulated easily. Bipolar individuals' hypersensitive BAS is hypothesized to respond with extremely elevated affect, high energy, and excessive goal seeking and self-confidence (i.e., hypomanic/manic symptoms) in response to BAS activation-relevant events involving themes of reward incentive, goal striving, and attainment, and with depressed affect, low energy, anhedonia, and hopelessness (i.e., depressive symptoms) in response to BAS deactivation-relevant events such as definite failure or nonattainment of goals (Depue & Iacono, 1989; Depue et al., 1987; Fowles, 1988, 1993; Urosevic, Abramson, Harmon-Jones, & Alloy, 2008). BAS sensitivity is a construct of hyperresponsiveness to incentive stimuli at all levels of analysis, including, but not limited to, cognition. From a BAS perspective, individuals with bipolar spectrum disorders should exhibit cognitive styles specific to the themes of high drive/incentive motivation associated with high BAS sensitivity. Indeed, high BAS sensitivity may be a temperament that contributes to the development of BAS-relevant cognitive styles (Alloy et al., 2009), and BAS-relevant cognitive styles may at least partially mediate the association between high BAS sensitivity and mood episodes. Some of the mixed findings in the studies examining the bipolar–cognitive style association may be attributable to the failure to examine BAS-relevant styles specifically.

Several studies have obtained results consistent with distinctive BAS-relevant cognitive and personality styles in individuals with bipolar disorder. Rosenfarb, Becker, Khan, and Mintz (1988) reported that only remitted unipolar depressed women exhibited higher dependency than controls, whereas both remitted bipolar and unipolar depressed women exhibited higher self-criticism than controls on the Depressive Experiences Questionnaire (DEQ; Blatt, D'Afflitti & Quinlan, 1976). Both Scott, Stanton, Garland, and Ferrier (2000) and Goldberg, Gerstein, Wenze, Welker, and Beck (2008) also found that the perfectionism (performance evaluation), but not the approval by others, subscale of the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978) distinguished remitted or manic bipolar patients from normal controls. With current clinical state controlled, Lam, Wright, and Smith

(2004) found that bipolar disorder patients scored higher than unipolar depressed patients only on goal-attainment dysfunctional attitudes (e.g., "A person should do well at everything"), but not on dependent or achievement dysfunctional attitudes. In addition, higher goal-attainment attitudes were associated with increased past hospitalizations for bipolar episodes in general and for mania in particular. In a later study, Wright, Lam, and Newsom-Davis (2005) found that whereas a positive mood induction led to a decrease of goal-attainment dysfunctional attitudes among unipolar depressed patients, bipolar disorder patients did not exhibit this reduction in goal-attainment attitudes following the same positive mood induction. Moreover, Lozano and Johnson (2001) found that an achievement-striving style predicted manic, but not depressive, symptoms in a 6-month follow-up of bipolar I patients. Finally, as described above, Francis-Raniere et al. (2006) found that a BAS-relevant cognitive style involving high autonomy, self-criticism, and performance focus predicted increases in hypomanic symptoms in interaction with style-congruent positive events and increases in depressive symptoms in interaction with style-congruent negative events over follow-up in individuals with cyclothymic and bipolar II disorders. Thus, these studies suggest that the cognitive styles of bipolar individuals may be marked by the BAS-relevant traits of autonomy, perfectionism, self-criticism, and goal striving, whereas there is less consistent evidence for the sociotropic, dependent, or approval-seeking cognitive styles in bipolar individuals that one typically sees in unipolar depression (see Zuroff, Mongrain, & Santor, 2004, for review).

The Present Investigation

This study examined the cross-sectional and prospective associations of BAS-relevant cognitive styles with bipolar spectrum disorders among participants in the Longitudinal Investigation of Bipolar Spectrum (LIBS) Project (Alloy et al., 2008; Nusslock, Abramson, Harmon-Jones, Alloy, & Hogan, 2007; Shen et al., 2008). We compared a large sample of individuals with bipolar spectrum disorders to demographically similar normal controls at baseline on self-report measures of cognitive/personality styles commonly used in the unipolar depression literature (DAS [Weissman & Beck, 1978]; Sociotropy-Autonomy Scale [SAS; Beck, Epstein, Harrison, & Emery, 1983]; and DEQ [Blatt et al., 1976]). We hypothesized that controlling for current depressive and hypomanic/manic symptoms, bipolar spectrum individuals would score higher than controls on BAS sensitivity and the BAS-relevant cognitive style dimensions of performance concerns, autonomy, and self-criticism, but not on the non-BAS-relevant dimensions of approval seeking, sociotropy, and dependency. Moreover, we hypothesized that the group differences on BAS-relevant cognitive styles would partially mediate group differences on BAS sensitivity. We also examined whether the cognitive styles assessed at Time 1 predicted prospective onsets of hypomanic/manic and depressive episodes over follow-up among bipolar spectrum participants. We hypothesized that controlling for Time 1 levels of hypomanic/manic and depressive symptoms and past history of mood episodes, BAS-relevant cognitive styles would be more likely to predict prospective onsets of mood episodes than would non-BAS-relevant styles. Finally, we predicted that BAS-relevant cognitive styles would partially mediate the prospective prediction of mood episodes by BAS sensitivity. Predictions that

BAS-relevant cognitive styles would mediate BAS sensitivity effects were novel and had never been examined before.

Method

Participants and Procedure

Participants were from the LIBS Project and were selected based on a two-phase screening procedure. In Phase I, approximately 20,500 students ages 18 to 24 years old at Temple University and the University of Wisconsin were administered the revised General Behavior Inventory (GBI; Depue, Krauss, Spoont, & Arbisi, 1989) to identify potential bipolar spectrum and control participants. Students who met the initial GBI criteria (see *Measures*, below) for either the bipolar spectrum or the control group were eligible for Phase II. In Phase II, 1,730 participants were administered a semi-structured diagnostic interview with an expanded Schedule for Affective Disorders and Schizophrenia—Lifetime (exp-SADS–L; Endicott & Spitzer, 1978) interview. Students who met *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [*DSM–IV*]; American Psychiatric Association, 1994) and/or Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) for bipolar II or cyclothymia were eligible for the LIBS Project.[1] On the basis of the exp-SADS–L interview, control participants had no lifetime history of any Axis I psychopathology, with the exception that they could have a specific phobia. In recruiting control participants, we took steps to ensure that the bipolar and control groups were similar on age, gender, and ethnicity.

Of the 285 eligible bipolar and 308 eligible control participants following Phase II, 227 (79.6%) bipolar spectrum (164 bipolar II and 63 cyclothymic) and 227 (73.7%) control participants completed the Time 1 assessment of the longitudinal study. Of these individuals, 195 bipolar spectrum (152 bipolar II, 43 cyclothymic) and 194 control participants also completed the initial symptom assessment and comprised the current study sample (see Table 1 for demographic characteristics). The bipolar and control groups did not differ on age, gender, or ethnicity. Among the bipolar participants, 30 (15.4%) were receiving formal treatment (medication and/or psychotherapy) at the outset of the study, and 31 (15.9%) progressed to a bipolar I diagnosis (had a first onset of mania) over the follow-up. Thirty-two (74.4%) of the 43 cyclothymic participants progressed to a bipolar II diagnosis (had a first onset of MD) during the follow-up. The current sample was representative of the Phase I screening sample on demographics and did not differ from Phase II–eligible individuals who did not participate on demographics, diagnosis, treatment history, and GBI scores. Participants completed several measures of cognitive style at Time 1 and self-report measures of depressive and hypomanic/manic symptoms at an initial symptom assessment (see *Measures*, below). In addition, a subset of participants (130 bipolar, 160 control) completed the Behavioral Inhibition System/Behavioral Activation System Scales (BIS/BAS Scales; Carver & White,

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[1] Participants who met criteria for bipolar I disorder were excluded because an aim of the LIBS Project was to examine the understudied "softer" bipolar conditions and to identify risk factors that predicted progression to bipolar I status over time.

Table 1
Demographic Characteristics of the Study Sample

Demographics	Bipolar spectrum ($n = 195$)	Normal control ($n = 194$)
Age (in years)	19.74 (1.71)	19.66 (1.79)
Sex	61.5% female	59.3% female
Ethnicity		
Caucasian	70.8%	71.6%
African American	15.4%	14.9%
Hispanic	3.6%	3.1%
Asian	3.1%	3.6%
Native American	0.5%	0.5%
Other	6.6%	6.3%

Note. Mean ages are reported with standard deviations in parentheses.

1994) at Time 1.[2] Bipolar spectrum individuals who completed Time 1 and the initial symptom assessment, entered the prospective phase of the LIBS Project, and were not in a mood episode at Time 1 ($n = 167$) were included in the longitudinal analyses. During the prospective follow-up, participants were administered an expanded Schedule for Affective Disorders and Schizophrenia—Change diagnostic interview (exp-SADS–C; Spitzer & Endicott, 1978) approximately every 4 months to assess the occurrence of mood episodes.[3] Prospective analyses were based on an average of 38.1 ± 19.1 (range = 73) months of follow-up.

Measures

Revised General Behavior Inventory (GBI). The revised GBI (Depue et al., 1981, 1989) assesses chronic affective disorders in the general population. It contains 73 items that measure the frequency, intensity, and duration of core bipolar experiences on two subscales: Depression (D) and Hypomania and Biphasic (HB) items combined. As recommended by Depue et al. (1989), we used the case-scoring method to identify potential bipolar spectrum and control participants at the Phase I screening. Only items rated a 3 (*often*) or 4 (*very often or almost constantly*) on the GBI 4-point frequency scale counted toward the score on each subscale. On the basis of cutoffs recommended by Depue et al. (1989), participants who scored greater than or equal to 11 on the D scale and greater than or equal to 13 on the HB scale were identified as potential bipolar participants, whereas those who scored below these cutoffs formed a potential normal group. These criteria were based on Depue et al.'s (1989) findings and a pilot study for the LIBS Project in which these cutoffs were validated against diagnoses derived from exp-SADS–L interviews. The GBI has good internal consistency (αs = .90–.96), test–retest reliability (rs = .71–.74), high specificity (.99), and adequate sensitivity (.78) for bipolar spectrum conditions (Depue et al., 1981, 1989). Also, it has been validated extensively in college, psychiatric outpatient, and offspring of bipolar I patient samples (Depue et al., 1981, 1989).

Expanded SADS–L diagnostic interview. The exp-SADS–L (Endicott & Spitzer, 1978) is a semistructured diagnostic interview that assesses current and lifetime history of Axis I disorders. The original SADS–L was expanded in several ways for the LIBS Project: (a) probes were added to allow for *DSM–IV* as well as RDC diagnoses; (b) questions were added to better capture the nuances of episodes and frequency and duration of symptoms for

depression, hypomania, mania, and cyclothymia; (c) the order of interview questions was changed to increase the interview's efficiency and comprehension; and (d) sections were added to assess eating disorders, attention-deficit/hyperactivity disorder, and acute stress disorder; additional probes were added in the anxiety disorders section; and organic rule-out and medical history sections were appended. An interrater reliability study based on 105 jointly rated exp-SADS–L interviews yielded κs $\geq$ .96 for bipolar spectrum diagnoses. Extensively trained research assistants, unaware of participants' Phase I GBI scores, conducted the interviews. Training consisted of approximately 200 hours of reading and didactic instruction, watching videotaped interviews, role playing, discussing case vignettes, and extensive practice conducting live interviews with supervision and feedback. Consensus *DSM–IV* and RDC diagnoses were determined by a three-tiered standardized diagnostic review procedure involving senior diagnosticians and an expert psychiatric diagnostic consultant.

Expanded SADS–C diagnostic interview. Prospective onsets of mood episodes were assessed with the exp-SADS–C diagnostic interview administered approximately every 4 months during the follow-up. The exp-SADS–C was used to assess onsets, remissions, and relapses and recurrences of diagnosable episodes of Axis I disorders, including *DSM–IV* and RDC MD and hypomanic/manic episodes, during each prospective interval. Interviewers were masked to participants' cognitive style scores, BIS/BAS scores, Phase I GBI scores, and Phase II diagnostic status. The SADS–C was expanded like the SADS–L. In addition, features of the Longitudinal Interval Follow-Up Evaluation (Shapiro & Keller, 1979) were added to the exp-SADS–C to systematically track the course of symptoms and episodes during the follow-up. The exp-SADS–C inquired about the presence of each symptom on a daily basis during the prospective interval. Interrater reliability (Alloy et al., 2008) for the exp-SADS–C in joint ratings of 60 interviews was good ($\kappa > .80$). In a validity study, participants dated their symptoms on the exp-SADS–C with at least 70% accuracy compared with daily symptom ratings made over a 4-month interval (Alloy et al., 2008). Nusslock et al. (2007) provided further details about exp-SADS–L and exp-SADS–C mood episode diagnoses.

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[2] The BIS/BAS Scales were administered along with other measures not used in the present analyses at Time 1. Since participants had a right to refuse any portion of the longitudinal study, only a subset of the longitudinal sample completed this extensive set of measures. There were no differences between participants who did and did not complete the BIS/BAS Scales on diagnosis, age, gender, ethnicity, GBI, or cognitive styles scores.

[3] Although prospective assessments were planned every 4 months, some participants missed planned assessments for a variety of reasons (e.g., out of the country). In such cases, the next assessment was completed as soon as possible, and it covered the time since the previously completed assessment. Most participants who missed a planned assessment did not actually attrit from the study. Over the first 4 years of follow-up, there was 10.7% attrition across the bipolar and control groups. The majority (71%) of attrition occurred within the first 8 months of follow-up because the major reason for attrition was the time commitment required by the study, which participants discovered within the first couple of assessments. The likelihood of attrition among participants who made it to the fifth follow-up assessment was very low.

Self-report symptom measures. The original Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) was used to assess initial levels of depressive symptoms. The BDI has been validated in student samples, and the internal and test–retest reliabilities are good in both clinical and nonclinical samples (Beck, Steer, & Garbin, 1988). Initial levels of hypomanic/manic symptoms were assessed with the Halberstadt Mania Inventory (HMI; Alloy et al., 1999). This 28-item self-report measure is modeled after the BDI and assesses the affective, motivational, cognitive, and somatic symptoms of (hypo)mania. Like the BDI, the HMI asks participants to choose one of four statements graded in severity that best describes their experience, for example, "I do not feel particularly happy," "I feel happy, " "I feel so happy and cheerful it's like a high," or "I am bursting with happiness and I'm on top of the world." The HMI has good internal consistency (α = .82) and has demonstrated convergent validity with the Minnesota Multiphasic Personality Inventory's (Hathaway & McKinley, 1951) Mania scale (r = .32, p < .001), as well as discriminant validity with its Depression scale (r = −.26, p < .001) and the BDI (r = −.12, p < .001; Alloy et al., 1999). The HMI also correlated (r = .46) with hypomanic symptoms rated from the exp-SADS–C interview in the LIBS Project (Alloy et al., 2008) and had an internal consistency of α = .78. Finally, the HMI also shows expected changes as cyclothymic individuals cycle through hypomanic, euthymic, and depressed mood states (Alloy et al., 1999).

Cognitive style measures. The DAS, Form A (Weissman & Beck, 1978), is a 40-item questionnaire that assesses dysfunctional beliefs regarding concerns about others' approval and performance expectations on 7-point scales ranging from *totally agree* to *totally disagree*. Two DAS factors have been extracted that are hypothesized to be related to the sociotropic and autonomous subscales of the SAS and the dependent and self-critical subscales of the DEQ (Cane, Olinger, Gotlib, & Kuiper, 1986; Segal, Shaw, & Vella, 1989): Approval by Others (AO—10 items: e.g., "My value as a person depends greatly on what others think of me") and Performance Evaluation/Perfectionism (PE—15 items: e.g., "If I fail partly, it is as bad as being a complete failure"), respectively. We viewed PE as a BAS-relevant dimension of dysfunctional attitudes but AO as not relevant to BAS sensitivity. In the current sample, alphas for the PE and AO subscales were .89 and .78, respectively. Both factors have shown good construct validity (Francis-Raniere et al., 2006; Segal et al., 1989).

The SAS (Beck et al., 1983) is a 60-item questionnaire designed to assess Beck's (1987) depressive personality modes, with 30 items each on the Sociotropy (e.g., "I am afraid of hurting other people's feelings") and Autonomy (e.g., "It is more important to get a job done than to worry about other people's reactions") subscales. Autonomy assesses valuing of achievement, mobility, and freedom from control and is BAS relevant, whereas Sociotropy measures valuing of attachment and fears of abandonment and rejection by others and is not BAS relevant. Each item is rated on 5-point scales (0%, 25%, 50%, 75%, and 100%). The Sociotropy and Autonomy scales have shown good internal consistency (α = 0.90 and 0.93, respectively) and high retest reliability (Beck et al., 1983; Zuroff et al., 2004). In the present sample, the alphas for Sociotropy and Autonomy were .93 and .92, respectively. The Sociotropy scale also has high concurrent validity with other measures of dependency and affiliation (Clark, Beck, & Brown, 1992), whereas the Autonomy scale is moderately correlated with an autonomy subscale of the Personality Research Form (Clark et al., 1992).

The DEQ (Blatt et al., 1976) is 66 items, rated on 7-point scales (from *strongly disagree* to *strongly agree*), and has three factors measuring the depressive personality styles hypothesized by Blatt et al. (1976): Dependency, Self-Criticism, and Efficacy. We used only the Dependency and Self-Criticism subscales in this study. Self-Criticism (e.g., "I have a difficult time accepting weaknesses in myself") is BAS relevant, whereas Dependency (e.g., "Without support from others who are close to me, I would be helpless") is not. We used the DEQ factor scores as recommended by Blatt et al. The DEQ has shown high internal and retest reliability (Blatt et al., 1976; Zuroff, Moskowitz, Wielgus, Powers, & Franko, 1983). In this sample, alphas for Dependency and Self-Criticism were .64 and .87, respectively. The factors have shown good construct validity as well (Zuroff et al., 2004).

BIS/BAS measure. The BIS/BAS Scales (Carver & White, 1994) quantify individual differences in sensitivity of the BIS and BAS and are the most frequently used self-report measures for this purpose. The scales include twenty 4-point items, ranging from *strongly disagree* to *strongly agree,* and consist of one BIS subscale, and three BAS subscales: Reward Responsiveness (RR), Drive, and Fun-Seeking (FS). The BIS scale has seven items and assesses sensitivity to potential punishment cues, for instance, "If I think something unpleasant is going to happen, I usually get pretty 'worked up'." The BAS RR scale has five items that assess positive responses to rewards, such as "When I get something I want, I feel excited and energized." The Drive scale has four items that index vigor and persistence in pursuit of rewards, such as "When I want something, I usually go all-out to get it." The FS scale has four items that index willingness to impulsively approach rewards, such as "I will often do things for no other reason than that they might be fun." Carver and White (1994) reported internal consistencies (alphas) from .59 to .74 for the BIS/BAS Scales, and Urosevic, Abramson, Harmon-Jones, Donovan, et al. (2008) found good test–retest reliabilities and stabilities in both bipolar spectrum and normal samples. In this sample, the alphas were higher and were .75 for BIS, .81 for BAS Total, .81 for BAS Drive, .72 for BAS FS, and .66 for BAS RR. Confirmatory factor analyses of the BIS/BAS Scales have confirmed the latent structure of one BIS scale and three correlated BAS subscales (Campbell-Sills, Liverant, & Brown, 2004; Carver & White, 1994). Numerous studies support the construct validity of the BIS/BAS Scales, including their relation to asymmetrical prefrontal cortical activity, affect, personality traits, and performance on reaction time and learning tasks involving incentives (e.g., Alloy, Abramson, Walshaw, Cogswell, et al., 2006; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997; Zinbarg & Mohlman, 1998).

Results

Table 2 displays the bivariate correlation matrix for all variables. Some of these correlations are directly relevant to the study hypotheses and are discussed below. However, several other patterns of correlations are worth noting. Overall, the BAS-relevant (PE, Autonomy, Self-Criticism) cognitive styles correlated with each other more strongly than they did with

Table 2
Correlations Among Study Variables

Variable	DIAG	BDI	HMI	PE	AO	AUT	SOC	SC	DEP	BAS	D	FS	RR	BIS	MD	HYP/MA
DIAG	—	.60***	.05	.48***	.17***	.31***	.05	.62***	.09	.22***	.18***	.24***	.10	.11	.66**	.41***
BDI		—	−.06	.53***	.32***	.11	.32***	.62***	.26***	.11	.05	.14**	.07	.21***	.52***	.52***
HMI			—	−.06	−.19***	.17***	−.13**	−.04	−.22***	.15**	.19***	.14**	.02	−.19***	−.06	.09
PE				—	.51***	.36***	.11	.70***	.22***	.08	.11	.06	.04	.21***	.38***	.39***
AO					—	−.21***	.63***	.35***	.54***	−.03	−.08	−.02	.06	.35***	.25***	.15**
AUT						—	−.07	.31***	−.28***	.37***	.34***	.33***	.20***	−.13**	.18***	.31***
SOC							—	.39***	.76***	.05	−.04	.06	.11	.42***	.07	.01
SC								—	.15**	.19***	.18***	.18***	.09	.18***	.46***	.57***
DEP									—	−.02	−.09	.01	.06	.45***	.20***	.04
BAS										—	.81***	.81***	.72***	.05	.12	.28***
D											—	.48***	.39***	−.09	.08	.21***
FS												—	.37***	−.08	.18***	.29***
RR													—	.31***	.03	.14**
BIS														—	.20***	.13**
MD															—	.60***
HYP/MA																—

Note. Correlations were rounded to the second decimal place. DIAG = normal (0), cyclothymic (1), or bipolar II (2) status; BDI = Beck Depression Inventory; HMI = Halberstadt Mania Inventory; PE = Performance Evaluation subscale from the Dysfunctional Attitudes Scale; AO = Approval by Others subscale from the Dysfunctional Attitudes Scale; AUT = Autonomy subscale from the Sociotropy-Autonomy Scale; SOC = Sociotropy subscale from the Sociotropy-Autonomy Scale; SC = Self-Criticism subscale from the Depressive Experiences Questionnaire; DEP = Dependency subscale from the Depressive Experiences Questionnaire; BAS = Total subscale from the Behavioral Inhibition System/Behavioral Activation System Scales (BIS/BAS Scales); D = Drive subscale from the BIS/BAS Scales; FS = Fun-Seeking subscale from the BIS/BAS Scales; RR = Reward Responsiveness subscale from the BIS/BAS Scales; BIS = BIS subscale from the BIS/BAS Scales; MD = prospective onset of major depression (0 = no, 1 = yes); HYP/MA = prospective onset of hypomanic or manic episode (0 = no, 1 = yes).
** $p < .05$.   *** $p < .01$.

the non-BAS-relevant (AO, Sociotropy, Dependency) cognitive styles, which also intercorrelated with each other. Depressive (BDI) and hypomanic/manic (HMI) symptoms showed more positive correlations with the BAS-relevant dimensions than the non-BAS-relevant dimensions of each cognitive style measure, with the exception of the correlation between the BDI and the Autonomy vs. Sociotropy scales of the SAS. The three subscales of the BAS Scale intercorrelated moderately with each other. Thus, we examined both BAS Total and each of the BAS subscales in our main analyses.

BAS Relevance of Cognitive Style Dimensions

In describing the cognitive style measures, we proposed that certain cognitive style dimensions (PE, Autonomy, Self-Criticism) are BAS relevant and that other dimensions (AO, Sociotropy, Dependency) are not BAS relevant based on their item content. Before testing the study hypotheses, we first sought to verify empirically this categorization of the cognitive style dimensions. The simple correlations in Table 2 show that BAS sensitivity tended to correlate more strongly and positively with the BAS-relevant than the non-BAS-relevant cognitive dimensions, whereas BIS sensitivity correlated more strongly with the non-BAS-relevant dimensions. However, these simple correlations did not control for participants' current mood state. Thus, we conducted a series of hierarchical regression analyses in which we examined whether BIS sensitivity and BAS sensitivity were associated with each of the cognitive style dimensions, controlling for levels of depressive (BDI) and hypomanic/manic symptoms (HMI). Hierarchical regression has the advantage that it allows for the examination

of the unique contribution of predictors. Table 3 displays the results of these analyses. As expected, controlling for symptom levels, higher BAS scores were significantly associated with higher DAS PE, SAS Autonomy, and DEQ Self-Criticism scores, but not with DAS AO, SAS Sociotropy, and DEQ Dependency scores. On the other hand, controlling for symp-

Table 3
Associations Between BAS and BIS Sensitivities and Cognitive Style Dimensions, Controlling for Depressive (BDI) and Hypomanic/Manic (HMI) Symptoms

Cognitive style	BAS		BIS	
	β	t	β	t
DAS PE	.134	2.55***	.098	1.84
DAS AO	−.010	−0.17	.241	4.30****
SAS AUT	.328	5.72****	−.139	−2.31**
SAS SOC	.075	1.29	.331	6.04****
DEQ SC	.166	3.35****	.033	0.66
DEQ DEP	.013	0.21	.376	6.82****

Note. Each row and BAS or BIS column in Table 3 represents a separate analysis. BAS = behavioral approach system from the BIS/BAS Scales; BIS = behavioral inhibition system from the BIS/BAS Scales; BDI = Beck Depression Inventory; HMI = Halberstadt Mania Inventory; DAS = Dysfunctional Attitudes Scale; PE = Performance Evaluation subscale; AO = Approval by Others subscale; SAS = Sociotropy-Autonomy Scale; AUT = Autonomy subscale; SOC = Sociotropy subscale; DEQ = Depressive Experiences Questionnaire; SC = Self-Criticism subscale; DEP = Dependency subscale.
** $p < .05$.   *** $p < .01$.   **** $p < .001$.

toms, higher BIS scores were significantly associated with higher DAS AO, SAS Sociotropy, and DEQ Dependency scores but were not related to DAS PE and DEQ Self-Criticism scores. Higher BIS was associated significantly with lower SAS Autonomy scores. Thus, our initial categorization of the cognitive style dimensions with respect to BAS relevance was supported.

Diagnostic Group Differences on Cognitive Style Dimensions and BAS Sensitivity

Next, we proceeded to test the first hypothesis. Diagnostic group was more strongly correlated (see Table 2 for the correlations) with DAS PE than AO, $t(1) = 5.75$, $p < .001$; with SAS Autonomy than Sociotropy, $t(1) = 3.35$, $p < .001$; and with DEQ Self-Criticism than Dependency, $t(1) = 8.08$, $p < .001$. Although consistent with our first hypothesis, these differences in correlations did not control for current symptom levels. To examine group differences in cognitive styles not attributable to associations between levels of depressive and hypomanic/manic symptoms and cognitive styles, we conducted hierarchical regression analyses in which the various cognitive style scores were regressed onto BDI and HMI scores in Step 1 and diagnostic group in Step 2. Table 4 presents the results of these analyses, as well as the means and standard deviations of the cognitive style scores for each group. As hypothesized, controlling for depressive and hypomanic/manic symptoms, the bipolar spectrum group scored significantly higher than the control group on DAS PE, SAS Autonomy, and DEQ Self-Criticism. The groups did not differ on DAS AO, SAS Sociotropy, or DEQ Dependency. We also conducted hierarchical regression analyses to examine diagnostic group differences in BAS and BIS sensitivities, controlling for concurrent symptom levels (see Table 4).[4] Controlling for depressive and hypomanic/manic symptoms, the bipolar spectrum group scored significantly higher than the control group on BAS Total, BAS Drive, and BAS FS, but not on BAS RR or BIS.

Mediation of BAS Sensitivity–Bipolar Disorder Association by Cognitive Styles

Given that the bipolar and control groups differed significantly on BAS sensitivity (BAS Total, D, and FS) and on the BAS-relevant cognitive styles (PE, Autonomy, and Self-Criticism), we next examined whether the group differences in BAS sensitivity were mediated, at least in part, by the group differences on BAS-relevant cognitive styles. According to Baron and Kenny (1986), to demonstrate mediation, we need to show that (a) BAS sensitivity (Total, Drive, and FS) predicts the potential mediators (PE, Autonomy, and Self-Criticism), (b) the potential mediators predict diagnostic group, (c) the potential mediators still predict diagnostic group with BAS sensitivities controlled, and (d) the associations between BAS sensitivities and diagnostic group are significantly smaller when the mediators are controlled. Steps 1 and 2 of Baron and Kenny's approach have already been demonstrated above.

We conducted a series of hierarchical logistic regressions in which diagnostic group was regressed on BDI and HMI scores in

Step 1, a BAS score in Step 2, and a BAS-relevant cognitive style (the mediator) in Step 3. Given that only a subset of participants completed the BIS/BAS Scales, we first reconducted the hierarchical regression analyses examining the association between diagnostic group and the BAS-relevant cognitive styles, controlling for BDI and HMI scores (shown in Table 4) within this subsample to be sure the significant relationships still held in the smaller sample. Diagnostic group was significantly related to the BAS-relevant cognitive styles within the smaller sample ($ps < .01$), so we proceeded with the mediation analyses.

SAS Autonomy was significantly associated with diagnostic group, controlling for BAS Total (Wald = 10.93, $p < .001$, odds ratio [OR] = 1.06, confidence interval [CI] = 1.02–1.10), BAS Drive (Wald = 11.74, $p < .001$, OR = 1.06, CI = 1.03–1.10), and BAS FS (Wald = 11.84, $p < .001$, OR = 1.06, CI = 1.03–1.10), and these BAS sensitivities were no longer associated with group with SAS Autonomy controlled (ps changed from .028 to .298 for BAS Total, from .064 to .494 for BAS Drive, and from .014 to .131 for BAS FS). The Sobel tests for mediation were significant ($ps < .01$). Thus, SAS Autonomy fully mediated the BAS sensitivity–diagnostic group associations. Although DAS PE and DEQ Self-Criticism were still significantly associated with diagnostic group, controlling for symptom levels and BAS sensitivities (DAS PE: Wald = 7.50, $p < .006$, OR = 2.04, CI = 1.22–3.40, with BAS Total controlled; Wald = 6.31, $p < .012$, OR = 1.90, CI = 1.15–3.13, with BAS Drive controlled; Wald = 7.80, $p < .005$, OR = 2.06, CI = 1.24–3.41, with BAS FS controlled; DEQ Self-Criticism: Wald = 18.18, $p < .001$, OR = 3.17, CI = 1.87–5.39, with BAS Total controlled; Wald = 17.37, $p < .001$, OR = 3.03, CI = 1.80–5.10, with BAS Drive controlled; Wald = 19.96, $p < .001$, OR = 3.41, CI = 1.99–5.85, with BAS FS controlled), PE and Self-Criticism did not mediate the BAS sensitivity–diagnostic group associations (BAS Total, Drive, and FS were still significantly associated with group, controlling for PE and Self-Criticism). In sum, Autonomy was a mediator of the BAS–diagnostic group associations, but PE and Self-Criticism were not.

Cognitive Styles as Predictors of Prospective Mood Episodes

To examine whether any of the cognitive style dimensions predicted the likelihood of onset of mood episodes among bipolar spectrum participants, we conducted a series of hierarchical logistic regression analyses with the occurrence (yes–no) of MD and hypomanic/manic episodes during the follow-up as the dependent variables. Bipolar participants currently in a mood episode at Time 1 were excluded from these analyses to ensure that episodes were truly prospective. In each logistic regression ($n = 167$ for these analyses), the length of follow-up (in days) was entered in Step 1, past history of MD or hypomanic/manic episodes was entered in Step 2, initial depressive (BDI scores) and hypomanic/manic (HMI scores) symptoms were entered together in Step 3, and a cognitive style score was entered in Step 4. We included a past history of MD or hypomanic/manic episodes as a control variable to account

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[4] Alloy et al. (2008) presented similar diagnostic group differences in BAS and BIS scores based on a smaller subset of the LIBS Project sample.

Table 4
Diagnostic Group Differences in Cognitive Style Dimensions and BAS and BIS Sensitivities, Controlling for Depressive (BDI) and Hypomanic/Manic (HMI) Symptoms

Cognitive style and BIS/BAS	Bipolar		Normal		Group differences		
	M	SD	M	SD	β	t	p
DAS PE	47.07	15.78	33.86	10.42	.245	4.98	.001
DAS AO	39.53	10.48	36.86	8.67	−.034	−0.61	.54
SAS AUT	103.73	11.75	94.50	11.68	.407	7.46	.001
SAS SOC	93.60	18.19	86.76	15.82	.052	0.93	.36
DEQ SC	0.52	0.93	−0.79	0.89	.392	8.86	.001
DEQ DEP	−0.67	0.94	−0.88	0.82	−.014	−0.25	.80
BAS Total	40.88	5.31	37.97	5.12	.266	3.53	.001
BAS D	11.48	2.32	10.49	2.39	.203	2.67	.008
BAS FS	12.41	2.38	11.01	2.24	.289	3.88	.001
BAS RR	16.99	1.99	16.43	1.92	.121	1.56	.12
BIS	20.85	3.67	20.11	3.12	.033	0.43	.66

Note. The means and standard deviations shown are not adjusted for concurrent depressive and hypomanic/manic symptoms; however, the group differences shown from the regression analyses do control for symptom levels. BAS = behavioral approach system from the BIS/BAS Scales; BIS = behavioral inhibition system from the BIS/BAS Scales; D = Drive subscale; FS = Fun-Seeking subscale; RR = Reward Responsiveness subscale; BDI = Beck Depression Inventory; HMI = Halberstadt Mania Inventory; DAS = Dysfunctional Attitudes Scale; PE = Performance Evaluation subscale; AO = Approval by Others subscale; SAS = Sociotropy-Autonomy Scale; AUT = Autonomy subscale; SOC = Sociotropy subscale; DEQ = Depressive Experiences Questionnaire; SC = Self-Criticism subscale; DEP = Dependency subscale.

for any effects of past mood episodes on the prospective occurrence of new mood episodes. Table 5 displays the results of these analyses.

As shown in Table 5, SAS Autonomy was the only cognitive style that significantly predicted the likelihood of onset of MD, controlling for length of follow-up, past MD, and initial depressive and hypomanic/manic symptoms. Bipolar participants with higher

Autonomy scores were less likely to develop a MD episode than those with lower Autonomy scores. Both SAS Autonomy and DEQ Self-Criticism significantly predicted onset of hypomanic/manic episodes, controlling for length of follow-up, past hypomanic/manic episodes, and initial symptoms. Higher Autonomy and Self-Criticism scores both predicted a greater likelihood of hypomanic/manic episode occurrence.

Table 5
Hierarchical Logistic Regressions Predicting Likelihood of MD and HYP/MA Episode Onset Controlling for Length of Follow-Up, Past History of Mood Episodes, and Initial Depressive (BDI) and Hypomanic/Manic (HMI) Symptoms

BAS-relevant styles				Non-BAS-relevant styles			
Cognitive style	Wald	OR	CI	Cognitive style	Wald	OR	CI
Dependent variable: MD onset							
DAS PE	0.40	1.12	0.79–1.58	DAS AO	0.22	0.93	0.67–1.28
SAS AUT	5.35**	0.94	0.88–0.99	SAS SOC	0.61	0.98	0.94–1.03
DEQ SC	2.74*	0.71	0.47–1.06	DEQ DEP	0.51	0.88	0.61–1.26
Dependent variable: HYP/MA onset							
DAS PE	0.53	1.13	0.81–1.59	DAS AO	0.31	0.91	0.66–1.26
SAS AUT	6.08***	1.12	1.02–1.22	SAS SOC	0.42	0.98	0.94–1.03
DEQ SC	4.02**	1.55	1.31–1.81	DEQ DEP	2.31	0.74	0.50–1.09

Note. Odds ratios (ORs) less than 1.00 indicate a negative association between the predictor and mood episode onset. MD = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [*DSM–IV*]) or Research Diagnostic Criteria (RDC) major depression episode; HYP/MA = *DSM–IV* or RDC hypomanic or manic episode; BDI = Beck Depression Inventory; HMI = Halberstadt Mania Inventory; BAS = behavioral approach system; Wald = Wald statistic; CI = confidence interval; DAS = Dysfunctional Attitudes Scale; PE = Performance Evaluation subscale; AO = Approval by Others subscale; SAS = Sociotropy-Autonomy Scale; AUT = Autonomy subscale; SOC = Sociotropy subscale; DEQ = Depressive Experiences Questionnaire; SC = Self-Criticism subscale; DEP = Dependency subscale.
* p < .10.   ** p < .05.   *** p < .01.

BAS Sensitivity–Prospective Mood Episodes Associations: Mediation by Cognitive Styles

Alloy et al. (2008) previously found that controlling for follow-up time and initial depressive and hypomanic/manic symptoms, higher BAS Total and RR scores predicted a greater likelihood of and shorter time to onset of hypomanic/manic episodes in the LIBS Project bipolar sample. Likewise, higher BAS RR and BIS scores marginally predicted a greater likelihood of and shorter time to onset of MD episodes, controlling for time in study and initial symptoms. Given that Alloy et al. did not also control for past history of mood episodes, we reconducted these analyses also controlling for past history of MD or hypomanic/manic episodes, respectively. Consistent with our prior findings, both BAS Total (Wald = 4.24, p < .04, OR = 1.14, CI = 1.01–1.29) and BAS RR (Wald = 4.27, p < .04, OR = 1.35, CI = 1.02–1.78) predicted a greater likelihood of onset of prospective hypomanic/manic episodes, controlling for past hypomanic/manic episodes and initial symptoms. BIS (Wald = 3.21, p < .075, OR = 1.13, CI = 0.99–1.29) continued to marginally predict a greater likelihood of prospective MD episodes, controlling for past MD episodes and initial symptoms, but BAS RR was no longer a marginal predictor of MD episodes.

Thus, we tested whether the Time 1 cognitive styles that predicted prospective onsets of MD or hypomanic/manic episodes (namely, SAS Autonomy and DEQ Self-Criticism) mediated the predictive associations between BAS Total and RR and the prospective onset of hypomanic/manic episodes and between BIS and the prospective onset of MD episodes. Following Baron and Kenny (1986), to examine mediation, we added the SAS Autonomy or DEQ Self-Criticism score on the last step of the hierarchical logistic regression analyses predicting the likelihood of onset of MD or hypomanic/manic episodes. Time in days (Step 1), past history of MD or hypomanic/manic episodes (Step 2), Time 1 BDI and HMI scores (Step 3), and either BAS Total, BAS RR, or BIS (Step 4) were entered on the previous steps before adding in the potential mediator (SAS Autonomy or DEQ Self-Criticism) on Step 5. With BAS Total or RR controlled, SAS Autonomy significantly predicted the likelihood of onset of hypomanic/manic episodes (Wald = 5.37, p < .02, OR = 1.14, CI = 1.02–1.26, with BAS Total; Wald = 6.20, p < .02, OR = 1.15, CI = 1.03–1.28, with BAS RR), and these BAS sensitivities no longer predicted hypomanic/manic episodes with SAS Autonomy controlled. The Sobel tests for mediation indicated that Autonomy was a mediator of BAS sensitivities (ps < .05, one-tailed). DEQ Self-Criticism no longer predicted prospective onsets of hypomanic/manic episodes significantly with BAS Total or RR controlled; thus, it did not mediate these BAS sensitivities. SAS Autonomy also significantly predicted a lower likelihood of onset of MD even with BIS (Wald = 3.82, p = .05, OR = 0.95, CI = 0.91–1.00) controlled, but SAS Autonomy did not mediate the association between BIS sensitivity and prospective onset of MD (because BIS sensitivity continued to significantly predict onset of MD even with SAS Autonomy controlled).

Discussion

Given the success of cognitive models of unipolar depression in elucidating important cognitive processes in the onset, course, and treatment of depression, there has been much interest in applying these models to bipolar spectrum disorders. However, the evidence for the occurrence of negative cognitive styles independent of current mood state in individuals with bipolar disorders and for the role of such cognitive patterns in predicting mood symptoms and episodes in the course of bipolar disorder is mixed (Alloy, Abramson, Neeren, et al., 2006; Alloy et al., 2005, Alloy, Abramson, Walshaw, Keyser, & Gerstein, 2006; Alloy, Abramson, Walshaw, & Neeren, 2006; Cuellar et al., 2005). The present study was designed to examine whether the concurrent and prospective associations between cognitive styles and bipolar spectrum disorders would be more consistent for a subset of cognitive styles that are BAS relevant.

On the basis of a BAS dysregulation model of bipolar disorders (e.g., Alloy et al., 2008, 2009; Depue & Iacono, 1989; Depue et al., 1987; Johnson, 2005; Urosevic, Abramson, Harmon-Jones, & Alloy, 2008), we hypothesized that individuals with bipolar spectrum disorders would differ from normal control individuals on BAS-relevant, but not non-BAS-relevant, cognitive styles and that the group differences on BAS-relevant styles would at least partially mediate group differences in BAS sensitivity. The findings were supportive of the first hypothesis and partially supportive of the second. As expected, the correlations between diagnostic group and the BAS-relevant cognitive styles were significantly greater than the correlations between diagnostic group and the non-BAS-relevant styles from the same measures. Moreover, controlling for concurrent levels of depressive and hypomanic/manic symptoms, bipolar participants exhibited significantly higher scores than controls on the BAS-relevant cognitive dimensions of performance evaluation (DAS), autonomy (SAS), and self-criticism (DEQ). However, they did not differ from controls on the non-BAS-relevant dimensions of approval by others (DAS), sociotropy (SAS), and dependency (DEQ). That the associations between bipolar status and elevated scores on BAS-relevant cognitive dimensions held despite controlling for concurrent depressive and hypomanic/manic symptoms suggests that symptomatic state is unlikely to provide a plausible explanation for the group differences. Indeed, higher state levels of hypomanic symptoms (HMI) were associated with higher autonomy and lower need for approval by others, sociotropy, and dependency. In contrast, with state hypomanic symptoms controlled, a tendency to experience recurrent hypomania/mania as reflected in a bipolar diagnosis was not associated with lower scores on the BAS-irrelevant dimensions of approval by others, sociotropy, and dependency.

In addition, our findings are consistent with several other studies also reporting that individuals with disorders in the bipolar spectrum only exhibit dysfunctional cognitive patterns with BAS-relevant features (Goldberg et al., 2008; Lam et al., 2004; Rosenfarb et al., 1988; Scott et al., 2000; Wright et al., 2005). Thus, individuals with bipolar disorders may exhibit a unique profile of cognitive styles consistent with the goal-striving, drive, and incentive motivation associated with high BAS sensitivity (Alloy, Abramson, Neeren, et al., 2006; Alloy et al., 2005, Alloy, Abramson, Walshaw, Keyser, & Gerstein, 2006; Alloy, Abramson, Walshaw, & Neeren, 2006; Johnson, 2005), but not dependency, approval-seeking, and attachment attitudes typically observed among individuals with unipolar depression (Zuroff et al., 2004). Interestingly, and consistent with the past unipolar depression findings, we did observe that higher state levels of depressive

symptoms (BDI) were associated significantly with higher scores on all of the cognitive style dimensions except SAS Autonomy.

Moreover, our findings also go beyond prior research on BAS-relevant cognitive styles in bipolar disorder by examining whether individuals' BAS-related cognitive styles mediated their elevated scores on BAS sensitivity. Consistent with the mediation hypothesis, bipolar spectrum participants scored higher on self-reported BAS sensitivity (Total, Drive, and FS) than the controls, and higher BAS sensitivity was significantly associated with elevated scores on the three BAS-relevant cognitive styles that differentiated the bipolar and control groups (DAS PE, SAS Autonomy, and DEQ Self-Criticism). In addition, we found that autonomy fully mediated the association between bipolar status and BAS sensitivity. In contrast, although performance evaluation and self-criticism continued to be significantly associated with bipolar status controlling for BAS sensitivity, neither mediated the bipolar status–BAS sensitivity association. Although these findings are cross-sectional and not able to demonstrate causal relationships, they are consistent with the idea that high BAS sensitivity contributes to the development of an autonomous cognitive style, which, in turn, may contribute risk for bipolar disorder. Whereas high BAS sensitivity may also contribute to self-critical and perfectionistic cognitive styles and these styles are related to bipolar status, they did not seem to provide a mechanism by which BAS sensitivity is associated with bipolarity (at least as measured here).

We also evaluated whether BAS-related cognitive styles were more likely than non-BAS-relevant styles to predict the onset of mood episodes among bipolar spectrum participants during a 3.2-year prospective follow-up and whether BAS-relevant cognitive styles mediated predictive associations between BAS sensitivity and prospective mood episodes. Consistent with hypothesis, some of the BAS-related cognitive styles predicted significantly the likelihood of onset of major depressive and hypomanic/manic episodes, controlling for initial levels of depressive and hypomanic/manic symptoms and past history of mood episodes. None of the non-BAS-related cognitive dimensions predicted mood episode onsets in our bipolar sample. Whereas higher levels of self-criticism and autonomy predicted a greater likelihood of hypomanic/manic episode onset, higher autonomy was associated with a smaller likelihood of major depressive episode onset. Again, the fact that some BAS-related cognitive dimensions predicted the likelihood of mood episode onset prospectively, controlling for initial symptom levels and past history of mood episodes, suggests that the prospective associations between BAS-relevant cognitive styles and mood episodes are not readily attributable to residual symptoms or scarring by past episodes associated with elevated cognitive styles. However, it should be noted that the associations between autonomy and self-criticism and prospective mood episode onsets were small to moderate in magnitude. It may be that these BAS-relevant cognitive styles would have greater predictive power for bipolar mood episode onsets in combination with BAS-relevant life events (see Alloy et al., 2009; Urosevic, Abramson, Harmon-Jones, & Alloy, 2008).

It is interesting that SAS Autonomy predicted a greater likelihood of hypomanic/manic episodes but a smaller likelihood of major depressive episodes. This particular finding raises the intriguing possibility that some BAS-relevant cognitive styles may not always be maladaptive. Research suggests that bipolar disorder is characterized by high levels of both impairment and achievement (see Nusslock et al., 2008). Thus, an important question is what psychological traits or mechanisms are associated with achievement among individuals with bipolar spectrum disorders. It is possible that some BAS-related cognitive styles, such as high autonomy, also contribute to BAS-mediated adaptive outcomes, such as positive goal striving and achievement. Future research needs to test this speculation.

Although autonomy and self-criticism predicted the likelihood of mood episode onsets prospectively, only autonomy mediated the predictive association between BAS sensitivity and prospective hypomanic/manic episodes. Indeed, self-criticism no longer predicted hypomania/mania with BAS sensitivity controlled. Thus, autonomy mediated the associations between BAS sensitivity and both bipolar diagnosis (cross-sectional analyses) and hypomania/mania onset (prospective analyses). The prospective mediation findings for autonomy are particularly noteworthy because prospective data allow for a stronger test of mediation than do cross-sectional analyses. These prospective findings for autonomy provide further support for the idea that high BAS sensitivity may contribute to the development of an autonomous cognitive style, which, in turn, increases risk for bipolar disorder and hypomanic/manic episodes. Given that an autonomous cognitive style as measured by the SAS involves an emphasis on individualistic achievement, this finding is consistent with Lozano and Johnson's (2001) report that an achievement-striving style predicted manic symptoms in a 6-month follow-up of bipolar I patients. Moreover, our results suggest that an autonomous cognitive style mediates the effects of a temperament characterized by high drive and incentive motivation on bipolarity.

Study Strengths and Limitations

This investigation has several strengths. These include the inclusion of a large sample of individuals with bipolar spectrum disorders and demographically similar normal controls, the use of standardized diagnostic interviews and criteria, interviewers masked to cognitive style and BIS/BAS scores, a prospective longitudinal design, conservative statistical tests of the study hypotheses, and an examination of whether elevated BAS sensitivity among bipolar individuals is mediated by BAS-relevant cognitive styles.

However, it is important to recognize this investigation's limitations as well. First, the study sample consisted of undergraduates, who, although ethnically and socioeconomically diverse, may not be representative of community or clinical samples. Replication of our findings in a community sample with bipolar spectrum disorders and in samples with more severe bipolar I disorder is important. However, bipolar II and cyclothymia tend to be understudied relative to bipolar I disorder and are often risk factors for the progression to bipolar I disorder (e.g., Shen et al., 2008), suggesting the value of the present study as well. Second, cognitive styles were assessed with self-report instruments only. Although the self-report measures chosen for this study are reliable and valid assessments of cognitive style, future tests of associations between BAS-related cognition and bipolar disorder may benefit from use of task-based measures of cognition as well. Similarly, although the BIS/BAS Scales have been validated against behavioral (Zinbarg & Mohlman, 1998) and neurobiological (Harmon-Jones &

Allen, 1997; Sutton & Davidson, 1997) indices of BAS sensitivity, future studies of the relationship between cognitive styles and BAS sensitivity would also benefit from use of multiple indicators of BAS (e.g., electroencephalography).

Conclusions

Taken together, the present findings suggest that individuals with disorders in the bipolar spectrum may be characterized by a unique profile of cognitive styles that are relevant to and may mediate BAS sensitivity. Such BAS-related cognitive styles may also influence the course of bipolar disorder and contribute some degree of vulnerability to onsets of affective episodes among bipolar individuals. In conclusion, this investigation suggests that a BAS dysregulation model of bipolar disorder may be promising for understanding the nature of cognitive functioning in bipolar disorder and warrants further study.

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Call for Nominations

The Publications and Communications (P&C) Board of the American Psychological Association has opened nominations for the editorships of **Experimental and Clinical Psychopharmacology, Journal of Abnormal Psychology, Journal of Comparative Psychology, Journal of Counseling Psychology, Journal of Experimental Psychology: Human Perception and Performance, Journal of Personality and Social Psychology: Attitudes and Social Cognition, PsycCRITIQUES, and Rehabilitation Psychology** for the years 2012–2017. Nancy K. Mello, PhD, David Watson, PhD, Gordon M. Burghardt, PhD, Brent S. Mallinckrodt, PhD, Glyn W. Humphreys, PhD, Charles M. Judd, PhD, Danny Wedding, PhD, and Timothy R. Elliott, PhD, respectively, are the incumbent editors.

Candidates should be members of APA and should be available to start receiving manuscripts in early 2011 to prepare for issues published in 2012. Please note that the P&C Board encourages participation by members of underrepresented groups in the publication process and would particularly welcome such nominees. Self-nominations are also encouraged.

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Deadline for accepting nominations is January 10, 2010, when reviews will begin.