Motiv Emot (2006) 30:143–155
DOI 10.1007/s11031-006-9003-3

ORIGINAL PAPER

Behavioral Approach System (BAS) Sensitivity and Bipolar Spectrum Disorders: A Retrospective and Concurrent Behavioral High-Risk Design

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Received: 14 June 2005 / Accepted: 29 December 2005 / Published online: 15 August 2006

Abstract In this article, we tested the vulnerability hypothesis of the behavioral approach system (BAS) hypersensitivity model of bipolar disorders. We examined whether self-reported BAS sensitivity predicts lifetime bipolar spectrum diagnoses as well as symptoms and personality characteristics associated with bipolar disorder using a retrospective and concurrent behavioral high-risk design. Participants with high (HBAS; $n = 28$) or moderate (MBAS; $n = 24$) BAS sensitivity were selected and given a lifetime psychiatric diagnostic interview and self-report measures of proneness to bipolar symptoms, current symptoms, and personality characteristics relevant to bipolarity. HBAS participants were significantly and substantially more likely to have a lifetime bipolar spectrum disorder diagnosis than were MBAS participants, but did not differ from MBAS participants in their likelihood of a unipolar depression diagnosis. Also, the HBAS group exhibited higher impulsivity and proneness to hypomanic symptoms than the MBAS group, and BAS-reward responsiveness predicted hypomanic personality characteristics. Finally, high behavioral inhibition system (BIS) sensitivity was associated with proneness to and current depressive symptoms.

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Keywords Behavioral approach system · Bipolar disorders · Behavioral high-risk design · Hypomania · Depression · Behavioral inhibition system

> I'm like the Energizer bunny. I keep going and going and going. No need for sleep .... And, everything I see grabs my attention ... excites me. I want to do everything, be everything! And, I can! I feel powerful ...

This is a description of a hypomanic episode from a young woman with Bipolar II disorder. Notice that she exhibits extremely high levels of energy, goal-striving, and confidence, needs little sleep and is easily distracted, classic symptoms of hypomania. At other times, this same woman describes major depressive episodes in which she has little or no energy, loses interest in her friends, family, and activities, and experiences low self-esteem. What explains this young woman's highs and lows of mood, energy, interest, and confidence?

Several theorists (Depue & Iacono, 1989; Depue, Krauss, & Spoont, 1987; Fowles, 1988, 1993; Urosevic, Abramson, Harmon-Jones, & Alloy, 2006a) have suggested that a behavioral approach system (BAS) hypersensitivity model may explain both the hypomanic/manic and depressive episodes of individuals with bipolar spectrum disorders. According to the BAS hypersensitivity model, individuals with bipolar disorders have a hyper-sensitive BAS, a motivational system involved in goal-seeking and approach to reward, and thus, they are vulnerable to extreme fluctuations in activation and deactivation of the BAS, resulting in hypomanic/manic and depressive symptoms, respectively. In this article, we test whether self-reported sensitivity of the BAS predicts lifetime bipolar spectrum diagnoses as well as symptoms and personality characteristics associated with bipolar disorder

using a retrospective and concurrent behavioral high-risk design (Alloy, Lipman, & Abramson, 1992; Alloy et al., 2000).

BAS and bipolar disorder

In its regulation of appetitive motivation and goal-directed behavior, the BAS is activated by signals of reward and escape from or avoidance of punishment. These cues can be either external (e.g., the presence of an attractive goal object) or internal (e.g., expectancies of goal attainment). Activation of the BAS causes the person to increase cognitive activity aimed at promoting goal attainment (e.g., hope, self-efficacy, planning) and movement toward attainment of goals and is hypothesized to be associated with positive emotions such as hope, elation, and happiness (Depue & Iacono, 1989; Gray, 1994). Recent work (Carver, 2004; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001; Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003; Harmon-Jones et al., 2002) also documents a link between anger, irritability and activation of the BAS. Thus, anger and irritability, although negative emotions, may also represent approach motivational states. Indeed, Bauer et al. (1991) suggested that heightened activation or drive is a core feature of both euphoric and irritable hypomania/mania. In contrast, hypo-activation of the BAS has been associated with depression and anhedonia (e.g., Davidson, 1999; Fowles, 1988). Finally, considerable evidence indicates that relative left frontal cerebral activation is a neurobiological index of BAS activity (e.g., Davidson, Jackson, & Kalin, 2000; Harmon-Jones & Allen, 1997; Sobotka, Davidson, & Senulis, 1992; Sutton & Davidson, 1997).

According to a BAS hypersensitivity theory of bipolar disorder (Depue & Iacono, 1989; Depue et al., 1987; Urosevic et al., 2006a), individuals vulnerable to bipolar disorder may exhibit an overly sensitive BAS that is hyper-reactive to relevant cues. Such trait-like BAS hypersensitivity leads such individuals to experience great variability in their state levels of BAS activation over time and across situations. Thus, a hyper-responsive BAS can lead to excessive BAS activity in response to BAS activation-relevant events involving themes of goal striving and attainment, reward incentive, and anger-evocation, which, in turn, is reflected in hypomanic/manic symptoms such as euphoria, excessive goal seeking behavior, decreased need for sleep, irritability, distractibility, excessive self-confidence, and optimism (Depue & Iacono, 1989; Fowles, 1993; Urosevic et al., 2006a). In contrast, depressive symptoms such as sadness, low energy, anhedonia, psychomotor retardation, hopelessness, and low self-confidence reflect a shutdown of behavioral approach/engagement or excessive deactivation of the BAS in response to BAS deactivation-relevant events such as definite failure and non-attainment of goals (Depue et al., 1987; Fowles, 1988, 1993; Urosevic et al., 2006a). Thus, a key prediction deriving from the BAS hypersensitivity model is that individuals who have a highly sensitive BAS should be vulnerable to both hypomanic and depressive states, that is, to bipolar spectrum disorders.

Recent studies have provided evidence consistent with the BAS hypersensitivity model of bipolar disorder. Compared to relevant control groups, individuals exhibiting or prone to hypomanic symptoms (Carver & White, 1994; Meyer, Johnson, & Carver, 1999), diagnosed with Bipolar II and Cyclothymia (Urosevic et al., 2006b), and Bipolar I (Meyer, Johnson, & Winters, 2001) disorders show elevated scores on self-reported BAS sensitivity. High BAS sensitivity during recovery also predicted an increase in manic symptoms over 6 months in a Bipolar I sample (Meyer et al., 2001). In addition, individuals with bipolar spectrum disorders exhibit cognitive styles with distinctive BAS-relevant features of autonomy, perfectionism, and goal-striving (Alloy, Abramson, Walshaw, Whitehouse, & Hogan, 2004; Lam, Wright, & Smith, 2004). Indeed, a BAS-relevant cognitive style involving high self-standards, self-criticism, and focus on performance combined with congruent negative life events to predict prospective increases in depressive symptoms and with congruent positive events to predict prospective increases in hypomanic symptoms in a sample of Bipolar II and Cyclothymic individuals (Francis-Raniere, Alloy, & Abramson, in press). Moreover, the BAS-relevant personality trait of achievement striving predicted increases in manic symptoms over 6 months in a Bipolar I sample (Lozano & Johnson, 2001). Studies of goal appraisal and success expectancy also support the BAS hypersensitivity theory (Meyer & Krumm-Merabet, 2003; Meyer, Beevers, & Johnson, 2004). Further, mania is associated with an increase (Kano, Nakamura, Matsuoka, Iida, & Nakajima, 1992), and bipolar depression with a decrease (Allen, Iacono, Depue, & Arbisi, 1993), in relative left frontal cortical activity, a neurobiological index of BAS activation. In addition, compared to normal individuals, people prone to hypomania exhibited greater relative left frontal cortical activity to a BAS activation-relevant (anger-provoking) event (Harmon-Jones et al., 2002). Finally, two studies found that life events involving goal attainment (Johnson et al., 2000) or goal-striving (Nusslock, Abramson, Harmon-Jones, Alloy, & Hogan, 2006), hypothesized to be BAS activation-relevant, triggered hypomanic/manic symptoms or diagnosable hypomanic episodes in individuals with bipolar disorders.

Behavioral high-risk design

Although the studies reviewed support the BAS hypersensitivity model of bipolar disorders, all involve samples either already diagnosed with a bipolar disorder, currently exhibiting hypomanic symptoms, or reporting that they are prone

to hypomanic symptoms. Such samples are not ideal for testing the BAS hypersensitivity theory's vulnerability hypothesis that a highly sensitive BAS actually increases vulnerability to bipolar symptoms and diagnoses, because they employ a logically backward participant selection strategy (Just, Abramson, & Alloy, 2001) in which participants are selected on the basis of the presence vs. absence of bipolar symptoms and then compared on BAS sensitivity or other BAS-relevant characteristics. A more powerful strategy for testing the BAS vulnerability hypothesis involves use of a behavioral high-risk design (Alloy et al., 1992, 1999, 2000; Depue et al., 1981) in which participants are selected on the basis of the hypothesized psychological vulnerability to the disorder. Thus, to test whether high BAS sensitivity increases vulnerability to bipolar disorders, we would want to select individuals on the basis of relatively higher vs. lower BAS sensitivity and then compare these groups on their likelihood of exhibiting bipolar spectrum disorders during their lifetime, in a retrospective version of the design, or in the future, in a prospective version of the design. We could also compare the groups' likelihood of exhibiting concurrent bipolar symptoms and personality characteristics (concurrent version of the design).

The present study

The present study tested the BAS vulnerability hypothesis of bipolar disorder using the retrospective and concurrent versions of the behavioral high-risk design. We selected individuals with high vs. moderate levels of self-reported BAS sensitivity, blind to their symptoms, and then assessed their lifetime history of mood disorders with a semi-structured psychiatric diagnostic interview, blind to their BAS sensitivity scores. Whereas previous studies demonstrated that undergraduates selected on the basis of current hypomanic symptoms or bipolar spectrum disorders show high self-reported BAS sensitivity (Carver & White, 1994; Meyer et al., 1999, 2001; Urosevic et al., 2006b), the present study is unique in examining whether undergraduates selected on the basis of high BAS sensitivity exhibit an increased lifetime history of bipolar spectrum disorders based on diagnostic interview. In addition, we examined whether BAS sensitivity is associated with self-reported current levels and proneness to depressive and hypomanic symptoms, as well as personality features characteristic of bipolar individuals (specifically, hypomanic tendencies and impulsivity).

We chose individuals with moderate levels of BAS sensitivity, rather than those with low BAS sensitivity, to compare to high BAS sensitivity participants for three reasons. First, if we obtain differences in rates of bipolar disorders in a high BAS vs. a low BAS sensitivity group, the meaning of this difference would be unclear. Would the rate difference be due to increased vulnerability to bipolar disorder in the high BAS group or decreased vulnerability in the low BAS group? A comparison of a high BAS group to a moderate BAS group resolves this ambiguity. Second, given our limited resources for conducting diagnostic interviews with three groups, a moderate BAS comparison group provided a more conservative test of the BAS vulnerability hypothesis than a low BAS comparison group. Finally, there is evidence that low BAS sensitivity is associated with unipolar depression (Depue & Iacono, 1989; Depue et al., 1987; Fowles, 1988, 1993; Gotlib, Ranganath, & Rosenfeld, 1998; Kasch, Rottenberg, Arnow, & Gotlib, 2002). Thus, we hypothesized that relative to individuals with moderate BAS sensitivity, those with high BAS sensitivity would be more likely to meet diagnostic criteria for a lifetime bipolar spectrum disorder (Bipolar I, Bipolar II, Cyclothymia, or Bipolar Not Otherwise Specified [NOS]). We also hypothesized that high BAS sensitivity individuals would exhibit higher levels of proneness to hypomanic and depressive symptoms and impulsivity than would moderate BAS sensitivity individuals. We did not make any predictions with respect to current levels of hypomanic and depressive symptoms because, according to the BAS hypersensitivity model, current symptoms should be a function of current BAS activation levels which, in turn, are influenced by whether the individual has recently experienced BAS activation- or deactivation-relevant life events.

In addition, we examined two sets of exploratory questions. We selected participants for the high versus moderate BAS sensitivity groups on the basis of their scores on the BAS Drive (D) and Fun-Seeking (FS) subscales from the Behavioral Inhibition and Behavioral Activation Scales (BIS/BAS Scales; Carver & White, 1994) and the Sensitivity to Reward (SR) subscale from the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ; Torrubia, Ávila, Moltó, & Caseras, 2001). We did not include the BAS Reward Responsiveness (RR) subscale of the BIS/BAS Scales as a selection criterion for two reasons. First, the reliability of this subscale was low in our screening sample (see Section "Measures") and, thus, was not ideal for selection of the risk groups. Second, Davidson (1994) hypothesized that BAS sensitivity is more strongly associated with pre-goal attainment motivational states rather than post-goal reward responsiveness. Consistent with this line of reasoning, some studies suggest that the Drive and Fun Seeking subscales are more strongly associated with bipolar diagnoses and with hypomania than Reward Responsiveness (Carver & White, 1994; Meyer et al., 2001; Urosevic et al., 2006b). Consequently, our first exploratory question was whether BAS RR predicts additional variance in lifetime bipolar diagnoses and current bipolar-relevant symptoms and personality characteristics over and above the BAS measures used to select the groups. Second, some investigators have hypothesized that an overactive BAS coupled with the absence of constraint of the BAS by the BIS is associated with

hypomania/mania (Fowles, 1988, 1993; Gray, 1991). Thus, we also explored whether self-reported BIS sensitivity also contributed to the prediction of lifetime bipolar diagnoses and concurrent symptoms and personality characteristics alone or in combination with BAS sensitivity.

Method

Participants

Fifty-two Temple University undergraduates participated in this study. Students in Introductory Psychology completed a screening packet and were selected based on their scores on BAS sensitivity from the BIS/BAS Scales (Carver & White, 1994) and the SPSRQ (Torrubia et al., 2001). Those who scored in the highest quartile of the combined BAS D and FS subscales from the BIS/BAS Scales (high BAS score cut point $\geq$25), as well as in the highest quartile on the SR subscale of the SPSRQ (high BAS score cut point $\geq$ 15) were categorized as high BAS (HBAS) participants. Participants were classified as moderate BAS (MBAS) if they scored between the 40th and 60th percentiles on the BAS D and FS subscales combined (moderate BAS score cut points $\geq$ 21 and $\leq$ 23), and on the SR subscale (moderate BAS score cut points $\geq$ 10 and $\leq$ 12).

Of the 1504 students who completed a screening packet, 20.6% ($n = 311$) qualified for HBAS (12.0%; $n = 181$) or MBAS (8.6%; $n = 130$) status. Lack of resources required us to invite only a subset of all qualifying HBAS and MBAS students to participate. Thus, a random subset of those who qualified for the HBAS ($n = 40$) and MBAS ($n = 40$) groups were invited into the study. Of these 80 students contacted, 68 expressed interest and 12 (6 HBAS, 6MBAS) refused participation. We actually scheduled 52 (28 HBAS, 24 MBAS) of these students, who formed the final sample. The final sample did not differ from the overall screening sample on age ($t(1479) = 1.32$, *ns*), gender ($\chi^2 = 0.75$, *ns*) or ethnicity ($\chi^2 = 0.85$, *ns*).

The ethnic composition of the final sample was 69% Caucasian ($n = 36$), 19.2% African American ($n = 10$), 1.9% Hispanic/Latino ($n = 1$), 5.8% Asian ($n = 3$), and 3.8% Other ($n = 2$). Their mean age was 18.89 years ($SD = 0.95$) and 78.8% ($n = 41$) were women. Table 1 displays the demographic characteristics and means and SDs of the BIS/BAS and SPSRQ scores for each group. The HBAS and MBAS groups did not differ on age ($t(50) = -0.46$, *ns*), ethnicity ($\chi^2 = 0.47$, *ns*), or gender ($\chi^2 = 3.71$, *ns*). Given that they were selected based on BAS-D, BAS-FS, and SR scores, it is not surprising that the HBAS group scored significantly higher than the MBAS group on these three measures, $F(1, 51) = 25.82$, $p < .001$, $F(1, 51) = 25.74$, $p < .001$, and $F(1, 51) = 204.85$, $p < .001$, respectively (see Table 1). Although participants were not selected on the basis of BAS-RR, the HBAS group scored significantly higher than the MBAS group on this measure as well, $F(1, 51) = 4.09$, $p < .05$. The two groups did not differ on BIS, $F(1, 51) = 2.68$, $p < .11$, but the HBAS group did score higher on SP, $F(1, 51) = 4.00$, $p = .05$.

Table 1 Demographic information and questionnaire scores as a function of BAS status

	High BAS ($n = 28$)	Moderate BAS ($n = 24$)
Age	18.89 (1.07)	18.88 (0.80)
Sex	64.3% Female	87.5% Female
Ethnicity	64.3% Caucasian	70.8% Caucasian
BAS—drive	13.25 (1.74)	10.71 (1.60)***
BAS—fun seeking	13.96 (1.29)	11.54 (1.32)***
BAS—reward responsiveness	18.04 (1.73)	16.83 (2.26)*
BIS	19.43 (4.10)	20.29 (3.52)
SPSRQ—sensitivity to reward	17.36 (1.89)	11.21 (0.72)***
SPSRQ—sensitivity to punishment	9.14 (5.27)	11.67 (4.72)*
BDI	12.12 (9.55)	12.43 (8.90)
HMI	21.23 (8.77)	18.23 (7.55)
GBI—depression	6.96 (10.25)	5.04 (6.73)
GBI—hypomania/biphasic	5.39 (5.47)	3.04 (3.18)†
HP scale	23.57 (9.05)	20.42 (8.34)
IN scale	20.58 (6.36)	14.87 (6.12)**

Note. Means are reported with standard deviations in parentheses. BAS: behavioral activation system from the BIS/BAS scales; BIS: behavioral inhibition system from the BIS/BAS scales; SPSRQ: sensitivity to punishment and sensitivity to reward questionnaire; BDI: Beck Depression Inventory; HMI: Halberstadt Mania Inventory; HP scale: hypomanic personality scale; IN scale: impulsive nonconformity scale.

*$p < .05$. **$p < .01$. ***$p < .001$. †$p < .07$.

Self-report measures

BIS/BAS scales

The BIS/BAS scales were developed by Carver and White (1994) to quantify individual differences in sensitivity of the BAS and BIS and they are the most frequently used self-report measures for this purpose. The BIS/BAS scales include 20 items, each arranged on a 4-point Likert scale, ranging from "strongly disagree" to "strongly agree" and consist of one BIS subscale, and three BAS subscales: RR, D, and FS. The BIS subscale has seven items and assesses sensitivity to potential punishment cues. It includes items such as "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 reward stimuli, such as, "When I get something I want, I feel excited

and energized." The D scale has 4 items that index vigor and persistence in pursuit of a reward, with items such as, "When I want something, I usually go all-out to get it." The FS scale includes four items that index willingness to impulsively approach reward stimuli, such as, "I will often do things for no other reason than that they might be fun." All subscales have demonstrated adequate internal consistencies; α's range from .66 to .76 and 2 month test–retest reliabilities range from .59 to .69 (Carver & White, 1994). In this study, α's for BAS RR, D, FS, and BIS $=.58$, .72, .65, and .74, respectively. Numerous studies support the construct validity of the BIS/BAS scales, including their relation to prefrontal cortical activity, affect, and performance on reaction-time and learning tasks involving incentives (e.g., Harmon-Jones & Allen, 1997; Heponiemi, Keltikangas-Jarvinen, Puttonen, & Ravaja, 2003; Sutton & Davidson, 1997; Zinbarg & Mohlman, 1998).

Sensitivity to punishment and reward questionnaire

The SPSRQ (Torrubia et al., 2001) was designed to improve upon Carver and White's (1994) BIS/BAS measure in three ways: (1) it was intended to be more theoretically consistent with Gray's BIS/BAS Theory; (2) to have greater construct validity; and (3) to improve on weaknesses in the BIS/BAS scales' item content. The SPSRQ is composed of 48 "yes" or "no" items, such as, "Are you easily discouraged in difficult situations?" and "Do you have trouble resisting the temptation of doing forbidden things?" It has two subscales, each 24 items, SR and SP, designed to assess BAS and BIS sensitivity, respectively. Both subscales have acceptable levels of internal consistency, with α's $=.75$–.83 (Torrubia et al., 2001). In this study, α's for the SR and SP scales $=.75$ and .84, respectively. Three-month test–retest reliabilities are .87 for the SR scale and .89 for the SP scale (Torrubia et al., 2001). Findings also support the construct validity of the SPSRQ (Torrubia et al., 2001).

Symptom measures

The Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) is a 21-item, self-report measure that assesses the severity of cognitive, motivational, affective and somatic symptoms of depression. The BDI has been validated for student samples (Bumberry, Oliver, & McClure, 1978; Hammen, 1980) and in non-clinical samples, the internal reliability is good with α's ranging from .81 to .86 and test–retest reliabilities ranging from .48 to .86 (Beck, Steer, & Garbin, 1988). Alpha $=.91$ in this study.

Current levels of manic/hypomanic symptoms were assessed using the Halberstadt Mania Inventory (HMI; Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999). This 28-item self-report measure was chosen because it is modeled after the BDI, and similar to the BDI, it assesses the affective, cognitive, motivational and somatic symptoms of mania/hypomania. Like the BDI, the HMI asks participants to choose one of 4 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 ($\alpha = .82$), and it has demonstrated convergent validity with the MMPI-Mania scale ($r = .32$, $p < .001$), as well as discriminant validity with the MMPI-Depression scale ($r = -.26$, $p < .001$) and the BDI ($r = -.12$, $p < .001$; Alloy et al., 1999). It also shows expected changes as cyclothymic individuals cycle through hypomanic, euthymic, and depressed mood states (Alloy et al., 1999). In this study, $\alpha = .78$.

The General Behavior Inventory (GBI; Depue et al., 1981; Depue, Krauss, Spoont, & Arbisi, 1989) was included as a measure of proneness to (or more trait-like levels of) both depressive and hypomanic symptoms and is often used as a first-stage screening procedure to identify individuals likely to have bipolar spectrum disorders (e.g., Depue et al., 1981, 1989). The GBI is a self-report, 69-item measure composed of two subscales: depression (D) symptoms and hypomania plus biphasic (HB) symptoms. Items are designed to capture the frequency, duration, and intensity of mood symptoms in general, for example, "Have you had long periods in which you felt you couldn't enjoy life as easily as other people?" (D scale) and "Has your mood or energy shifted rapidly back and forth from happy to sad or high to low?" (HB scale). Individuals rate whether a given behavior describes them on a 4-point Likert scale ranging from "never or hardly ever" to "very often or almost constantly." As recommended by Depue et al. (1989), we used the case-scoring method in which 1 point was added to the total D or HB score only if an individual's response was '3' ("often") or '4' ("very often or almost constantly"). The GBI has excellent internal consistency (α's $=.90$–.96), and adequate test–retest reliability (r's $=.71$–.74). It has also demonstrated adequate sensitivity (.78) and high specificity (.99) for bipolar spectrum conditions (Depue et al., 1989). In our sample, α's $=.95$ and .88, for the D and HB scales.

Personality measures

The Hypomanic Personality Scale (HP; Eckblad & Chapman, 1986) was developed to assess premorbid temperament of individuals with bipolar disorders. Respondents rate 48 statements such as, "Sometimes ideas and insights come to me so fast that I cannot express them all" and "There are often times when I am so restless that it is impossible for me to sit still," as "true" or "false." The HP scale has a reliability coefficient of $\alpha = .87$, and test–retest reliability of .81 (Eckblad & Chapman, 1986). In this study,

$\alpha = .88$. The HP exhibits familial aggregation (Meyer & Hautzinger, 2001) and is associated with an increased likelihood of depressive and hypomanic/manic episodes (Klein, Lewinsohn, & Seeley, 1996; Kwapil et al., 2000). Over a 13-year follow-up, individuals identified as having hypomanic characteristics by the HP scale were more likely than controls to exhibit DSM hypomanic episodes (Eckblad & Chapman, 1986; Kwapil et al., 2000).

Impulsivity was indexed using Chapman's (1984) Impulsive Nonconformity Scale (IN). The IN scale consists of 51 "true" or "false" items that tap impulsive and antisocial behavior. Items include, "When I want something, delays are unbearable" and "I avoid trouble whenever I can." The IN scale has demonstrated adequate internal consistency (α's = .83–.84) and 6 week test–retest reliability ($r = .84$; Chapman, 1984). In this study, $\alpha = .79$. In addition, individuals who scored high on the IN scale were more likely to endorse antisocial, psychotic, depressive, and manic/hypomanic symptoms than a control group (Chapman, 1984).

Diagnostic interview

An expanded version of the Schedule for Affective Disorders and Schizophrenia—Lifetime (exp-SADS-L; Endicott & Spitzer, 1978) diagnostic interview was used to assess lifetime *Diagnostic and Statistical Manual of Mental Disorders* (4th edition; *DSM-IV*; American Psychiatric Association, 1994) and Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) diagnoses. In this study, only the current and past mood disorders sections (depression, mania, hypomania, cyclothymia sections) of the exp-SADS-L were administered. The SADS-L mood disorder sections were modified and expanded in the following ways: (1) probes were added to aid in the assignment of *DSM-IV* diagnoses as well as RDC diagnoses; (2) additional questions were added in the mood disorder sections to better capture the nuances of episodes and frequency and duration of symptoms; and (3) questions assessing past episodes of a given disorder immediately followed the assessment of a current episode of that disorder.

Inter-rater reliability on the exp-SADS-L has been excellent, with overall $\kappa \geq .90$ for all unipolar depressive diagnoses based on 80 jointly rated interviews (Alloy et al., 2000). For bipolar diagnoses, an inter-rater reliability study based on 105 jointly rated interviews yielded $\kappa = .96$ (Floyd et al., 2006). In this study, κ's were $\geq .90$ for all mood disorder diagnoses.

Diagnostic interviewers were blind to participants' BAS group status and BIS/BAS and SPSRQ scores. Interviewers were all clinical psychology Ph.D. students and were extensively trained in a program modeled after other training programs (Amenson & Lewinsohn, 1981; Gibbon, McDonald-Scott, & Endicott, 1981). Interviewers received extensive feedback throughout their training and during the study. Consensus *DSM-IV* and RDC diagnoses were determined by a three-tiered standardized diagnostic review procedure involving senior diagnosticians and a Ph.D. clinical psychologist. In assigning lifetime diagnoses, each participant was assigned to one of three mutually exclusive general categories based on *DSM-IV* and RDC criteria: Any Bipolar Disorder (Bipolar I, Bipolar II, Cyclothymia, or Bipolar Disorder Not Otherwise Specified [BiNOS[1]]); Any Unipolar Depression (Major Depression, Minor Depression, or Dysthymia/Intermittent Depressive Disorder [IDD]); or No Mood Disorder Diagnosis.

Procedure

As part of an Introductory Psychology course requirement, undergraduates completed a screening packet containing a consent form and the BIS/BAS and SPSRQ measures, along with other questionnaires not relevant to the current study. A random subset of individuals who qualified for HBAS or MBAS status were contacted and invited to participate in the study. Those who agreed were invited to the lab, where they received a packet of self-report questionnaires including an informed consent form, the GBI, BDI, HMI, HP, and IN. After completion of the questionnaires, participants were administered the exp-SADS-L interview. Depending on the length of the interview, some participants required two sessions (and thus, were scheduled again on a separate day) to complete the procedure. Participants were paid $25–$40 (depending on the length of the entire procedure) for their participation.

Results

Preliminary analyses

Prior to conducting tests of our hypotheses and exploratory questions, we first conducted preliminary analyses to determine whether the demographic variables were associated with any of the dependent variables. Age, gender, and ethnicity were not associated significantly with any of the dependent variables, with one exception. There was a gender difference on the IN Scale, $F(1, 50) = 5.43$, $p < .03$, such that males ($M = 5.33$, $SD = 0.78$) exhibited greater

[1] BiNOS was assigned to participants who exhibited recurrent hypomanic episodes without diagnosable depressive episodes, who exhibited a cyclothymic pattern but with hypomanic and depressive periods that did not meet minimum duration criteria for hypomanic and depressive episodes, or who showed hypomanic and depressive periods that were too infrequent to qualify for a Cyclothymia diagnosis.

Table 2 Lifetime diagnosis as a function of bas status

Diagnosis	High BAS ($n = 28$) (%)	Moderate BAS ($n = 24$) (%)	Wald	χ^2	p	Exp(B)	CI
Any bipolar disorder	14 (50.0)	2 (8.3)	8.35		.004	11.00	2.16–55.92
Bipolar II[a]	6 (21.4)	0 (0.0)		5.81	.016		
Cyclo/BiNOS	8 (28.6)	2 (8.3)	3.05		.081	4.40	0.83–23.22
Any unipolar depression	4 (14.3)	7 (29.2)	1.66		.198	0.40	0.10–1.60
Major depression	3 (10.7)	6 (25.0)	1.75		.186	0.36	0.08–1.63
Dysthymia/IDD	1 (3.6)	1 (4.2)	0.01		.911	0.85	0.05–14.39
No mood diagnosis	10 (35.7)	15 (62.5)	3.62		.057	0.33	0.11–1.03

Note. BAS: behavioral approach system; CI: confidence interval; Cyclo/BiNOS: cyclothymia or bipolar disorder not otherwise specified; IDD: intermittent depressive disorder. Participants were assigned to the three main diagnostic categories (any bipolar disorder, any unipolar depression, and no mood diagnosis) in a mutually exclusive fashion.

[a]It was not possible to run logistic regression analyses on the Bipolar II diagnosis because there were 0 participants in the MBAS group with this diagnosis. Thus, we ran χ^2 analyses instead and these are presented in the table.

impulsivity than females ($M = 5.10$, $SD = 0.44$). Therefore, we controlled for gender in all subsequent analyses involving the IN Scale.

Hypothesis 1: Lifetime diagnoses of mood disorders

Table 2 displays the lifetime mood disorder diagnoses of the HBAS and MBAS groups, as well as Wald statistics, odds ratios (Exp(B)), and confidence intervals.[2] To test the hypothesis that the HBAS group would be more likely to meet criteria for a lifetime bipolar spectrum disorder than the MBAS group, we conducted a logistic regression analysis on Any Bipolar Disorder with Group (HBAS, MBAS) as the predictor. Consistent with our hypothesis, the HBAS group was significantly and substantially more likely to have a lifetime bipolar spectrum disorder than was the MBAS group (50.0% vs. 8.3%; Exp(B) = 11.00, $p < .004$; see Table 2). Given that the groups differed significantly on their likelihood of having any bipolar spectrum disorder, we explored the specific bipolar diagnoses that were the basis of the difference. The HBAS group was significantly more likely to have a Bipolar II disorder diagnosis than the MBAS group (21.4% vs. 0%; $\chi^2 = 5.81^2$, $p < .016$; see Table 2). The two groups did not differ in their likelihood of receiving a Cyclothymia or BiNOS diagnosis, although there was a trend for the HBAS group to be more likely to receive this diagnosis (28.6% vs. 8.3%; Exp(B) = 4.40, $p = .081$).

To examine the specificity of the HBAS group's greater lifetime prevalence of bipolar spectrum disorders, we also conducted a logistic regression analysis on the Any Unipolar Depression category. The two groups did not differ in their likelihood of receiving Any Unipolar Depression diagnosis (14.3% vs. 29.2%; Wald = 1.66, Exp(B) = 0.40, $p = .198$; see Table 2).

Hypothesis 2: Symptom proneness and personality

Table 2 displays the means and SDs of all symptom and personality measures for the HBAS and MBAS groups. Hypothesis 2 was that the HBAS group would exhibit higher levels of proneness to depressive and hypomanic symptoms (GBI scores), higher hypomanic tendencies (HP scores), and higher impulsivity (IN scores) than would the MBAS group. We tested this hypothesis with a series of regression analyses in which BAS group was the predictor, and in the case of IN as the dependent variable, gender was included as a covariate. BAS group was not associated with GBI-D scores ($t(1, 50) = 0.78$, $B = 0.11$, $p < .50$); however, consistent with Hypothesis 2, BAS group did marginally predict GBI-HB scores ($t(1, 50) = 1.85$, $B = 0.25$, $p < .07$). The HBAS group had higher GBI-HB scores than the MBAS group (see Table 1). BAS group did not predict HP scores ($t(1, 50) = 1.30$, $B = 0.18$, $p < .20$); however, consistent with our hypothesis, BAS group was significantly associated with IN scores ($t(2, 49) = 2.61$, $B = 0.32$, $p < .01$). HBAS participants exhibited higher impulsivity than MBAS participants (see Table 1). Although we made no prediction regarding BAS group differences in current symptom levels, we also conducted regression analyses on BDI and HMI scores in an exploratory manner. BAS group was not significantly associated with either current depressive (BDI; $t(1, 50) = -0.12$, $B = -0.02$, $p < .91$) or hypomanic (HMI; $t(1, 50) = 1.31$, $B = 0.18$, $p < .20$) symptoms.

[2] For Bipolar II disorder, it was not possible to obtain Wald statistics and odds ratios because the logistic regression analysis could not be run, given that there were 0 participants in the MBAS group who had this diagnosis. In this case, we conducted χ^2 analyses instead (and these are presented in Table 2 instead of the Wald and Exp(B) statistics).

Exploratory question 1: Effects of BAS-reward responsiveness

To explore whether BAS-RR predicted variance in lifetime diagnoses or any of the symptom or personality measures over and beyond BAS group status (based on BAS-D, BAS-FS, and SR), we conducted hierarchical regression analyses in which BAS Group was entered on the first step and BAS-RR was entered on the second step. Controlling for BAS group status, BAS-RR did not significantly predict any diagnoses. However, BAS-RR did marginally predict HP scores ($t(2, 49) = 1.93$, $B = 0.27$, $p < .06$). Higher BAS-RR scores were associated with higher HP scores. BAS-RR was not predictive of GBI-D, GBI-HB, IN, BDI, or HMI scores.

Exploratory question 2: Effects of BIS

We also explored whether BIS sensitivity (BIS and SP scores) contributed to the prediction of lifetime diagnoses and concurrent symptom proneness and personality characteristics alone or in combination with BAS sensitivity. Consequently, we conducted hierarchical regression analyses with BAS Group entered on the first step, BIS or SP entered on the second step, and the BIS × BAS or SP × BAS interaction[3] entered on the third step. Neither BIS nor SP scores predicted any diagnoses either as main effects or in interaction with BAS. However, controlling for BAS group status, SP scores predicted higher proneness to both depressive (GBI-D; $t(2, 49) = 3.38$, $B = 0.45$, $p < .001$) and hypomanic (GBI-HB; $t(2, 49) = 3.44$, $B = 0.44$, $p < .001$) symptoms. In addition, controlling for BAS status, both BIS scores and SP scores predicted higher current depressive symptom levels on the BDI ($t(2, 49) = 3.21$, $B = 0.43$, $p < .002$ and $t(2, 49) = 2.74$, $B = 0.39$, $p < .009$, respectively).

There were two significant interactions of BIS and BAS sensitivity as well. HP scores were significantly predicted by the BIS × BAS interaction ($t(3, 48) = 2.81$, $B = 1.25$, $p < .007$), controlling for BAS group status and the main effect of BIS. To examine the pattern of this interaction, we regressed HP scores on BIS scores separately for the HBAS and MBAS groups. BIS scores were not significantly related to HP scores in either group, although they were more strongly associated with HP scores in the HBAS ($t(1, 26) = 1.19$, $B = .23$, $p < .25$) than in the MBAS group ($t(1, 22) = -0.19$, $B = -.04$, $p < .85$). The BIS × BAS interaction also significantly predicted Impulsivity (IN; $t(3, 48) = 2.17$, $B = 0.92$, $p < .04$), controlling for BAS group and the main effect of BIS. Separate regressions of IN scores on BIS for the two groups indicated that again, BIS scores were not significantly related to IN scores in either group, although they were negatively and more strongly related to IN scores in the HBAS ($t(1, 26) = -1.14$, $B = -.22$, $p < .27$) than in the MBAS group ($t(1, 22) = -0.06$, $B = -.01$, $p < .96$).

Discussion

According to the BAS hypersensitivity theory of bipolar disorders (Depue & Iacono, 1989; Depue et al., 1987; Urosevic et al., 2006a), a highly sensitive BAS is a vulnerability factor that leads individuals to be hyper-responsive to BAS activation-relevant and deactivation-relevant cues and, thus, increases their risk for developing bipolar spectrum disorders and concomitant features. Indeed, a trait-like, overly sensitive BAS may be part of the phenotypic representation of an underlying genetic predisposition to bipolar disorder. This study used the powerful, behavioral high-risk design (e.g., Alloy et al., 1992, 1999, 2000; Depue et al., 1981), in which participants are selected on the basis of higher vs. lower BAS sensitivity rather than on the basis of the presence vs. absence of bipolar symptoms or disorders, to test this BAS vulnerability to bipolar disorders hypothesis with respect to lifetime history of bipolar diagnosis.

BAS sensitivity and lifetime history of bipolar spectrum disorders

Consistent with the BAS vulnerability hypothesis, we found that based on semi-structured diagnostic interview and *DSM-IV* and RDC criteria, individuals with high BAS sensitivity were significantly more likely to have a lifetime bipolar spectrum disorder than individuals with moderate levels of BAS sensitivity. Indeed, the rate of bipolar spectrum disorders in the HBAS group was about 6 times greater than the rate in the MBAS group. In addition, this important finding is based on a conservative test of the BAS vulnerability hypothesis, inasmuch as high BAS sensitivity individuals were compared to individuals with moderate, rather than low, levels of BAS sensitivity. Our finding of an increased likelihood of bipolar disorders among high BAS sensitivity individuals is consistent with previous studies demonstrating that compared to relevant control groups, individuals exhibiting Bipolar I, Bipolar II, and Cyclothymic disorders (Meyer et al., 2001; Urosevic et al., 2006b) show elevated scores on self-reported BAS sensitivity. Moreover, HBAS participants exhibited specificity in their increased lifetime history of bipolar disorders; they did not differ from MBAS participants in their likelihood of a unipolar depressive disorder. These findings suggest that high BAS sensitivity may confer specific risk for clinically significant bipolar disorders.

[3] When forming the BIS × BAS and SP × BAS interactions, a BAS composite score (BAS-D + BAS-FS + SR) was used as the measure of BAS, rather than BAS group status (HBAS, MBAS).

Of course, the major conceptual limitation of these retrospective findings is that the causal direction of the association between high BAS sensitivity and increased lifetime rates of bipolar spectrum disorders is unclear. Did a highly sensitive BAS temporally precede and contribute to the onset of the bipolar disorder or did this highly sensitive BAS develop as a result of the past bipolarity? To more clearly test whether a hypersensitive BAS actually increases risk for bipolar disorder, a prospective test of the BAS vulnerability hypothesis is needed. Supportive of the possibility that high BAS sensitivity might also prospectively predict onset of bipolar spectrum disorders, Meyer et al. (2001) reported that high BAS sensitivity at the time of recovery predicted increased manic symptoms over 6 months in a Bipolar I sample.

Another limitation of the results is that they support the predictions derived from the BAS hypersensitivity theory for hypomanic/manic, but not depressive, episodes. Although HBAS individuals were more likely to exhibit hypomanic/manic episodes and, thus, were more likely to earn lifetime bipolar diagnoses, than MBAS individuals, the groups did not differ on lifetime unipolar depression. One possible interpretation is that bipolar disorder actually represents two comorbid but distinct disorders—hypomania/mania and depression—and that excessive BAS sensitivity provides vulnerability to the former but not the latter (see Joffe, Young, & MacQueen, 1999; Cuellar, Johnson, & Winters, 2005; Urosevic et al., 2006a for a general discussion of the "one-illness" vs. "two-illness" debate about bipolar disorder). An alternative explanation involves limitations of current measures of BAS sensitivity. Specifically, the measures of BAS sensitivity used in this study assess sensitivity to BAS activation-relevant cues (e.g., presence of rewards) but not to BAS deactivation-relevant cues (e.g., definite failures and losses). According to the BAS hypersensitivity theory, individuals vulnerable to both poles of bipolar disorder would exhibit excessive sensitivity to both kinds of cues. Thus, an important future direction for work on the BAS hypersensitivity model is development of measures of sensitivity to both BAS activation-relevant and BAS deactivation-relevant cues.

BAS sensitivity, symptom proneness, and personality

Based on the BAS vulnerability hypothesis, we also predicted that the HBAS group would exhibit higher levels of proneness to depressive and hypomanic symptoms (GBI scores), higher hypomanic tendencies (HP scores), and higher impulsivity (IN scores) than would the MBAS group. Our findings partially supported this hypothesis. High BAS sensitivity was significantly associated with greater impulsivity and marginally associated with greater proneness to hypomanic symptoms on the GBI. However, the groups did not differ on proneness to depressive symptoms or hypomanic tendencies on the HP scale. However, another measure of BAS sensitivity that was not a basis of the original selection of the groups, BAS-Reward Responsiveness, was marginally associated with HP scores, controlling for BAS group status.

Overall then, high BAS sensitivity was associated with proneness to hypomanic symptoms and hypomania-relevant personality characteristics (e.g., impulsivity), but was not associated with proneness to depressive symptoms. Again, measurement issues may account for the failure to observe an association between high BAS sensitivity and proneness to depressive symptoms. The GBI depression scale is nonspecific and assesses proneness to unipolar depression (major depression, dysthymia) as well as depression that is part of bipolar disorder. Given that we found that high BAS sensitivity was associated with increased lifetime rates of bipolar disorders specifically, but the HBAS and MBAS groups did not differ on rates of unipolar depression, the MBAS group could score as highly as the HBAS group on the GBI depression scale because they have equal vulnerability to unipolar depression.

The GBI results are consistent with our diagnostic findings, in that the HBAS group was more likely to receive a bipolar spectrum diagnosis than the MBAS group because they were more likely to have a history of hypomanic episodes, not depressive episodes (the two groups were equally likely to have a history of major depressive episodes). Our findings are also consistent with those of Meyer et al. (1999) and Urosevic et al. (2006b), who also found that self-reported BAS sensitivity was positively associated with proneness to hypomanic symptoms on the GBI in a normal sample and HP scores in a bipolar spectrum sample, respectively. In the present study, the relationship between high BAS sensitivity and the hypomania-relevant feature of impulsivity was particularly strong. The increased appetite for and seeking of rewards characteristic of high BAS sensitivity may be likely to make individuals especially prone to impulsive behaviors with the potential for pleasurable consequences. Thus, our findings suggest that a highly sensitive BAS may increase vulnerability to hypomanic/manic episodes and hypomania/mania-relevant symptoms and behaviors specifically, but may not raise risk for depressive episodes and symptoms above that of individuals with more moderate levels of BAS. If HBAS individuals are at equal risk for depressive episodes and symptoms and at greater risk for hypomanic/manic episodes and symptoms compared to MBAS individuals, then, this would lead them to be more vulnerable to bipolar spectrum disorders (both depressive and hypomanic/manic episodes) overall.

According to the BAS hypersensitivity model (Depue & Iacono, 1989; Depue et al., 1987; Urosevic et al., 2006a), a person's current depressive or hypomanic symptoms are determined by current level of BAS activation which, in turn,

is influenced not only by one's trait level of BAS sensitivity, but also by recent exposure to BAS deactivation or activation-relevant events. That is, the BAS hypersensitivity model is a vulnerability-stress theory. Given that we did not assess recent exposure to BAS-relevant life events in this study, we made no predictions regarding group differences on current depressive (BDI) and hypomanic (HMI) symptom levels. And, we did not obtain any group differences on current depressive or hypomanic symptoms. However, two recent studies found that life events involving goal-attainment (Johnson et al., 2000) and goal-striving (Nusslock et al., 2006), both BAS activation-relevant, predicted onset of hypomanic/manic symptoms or diagnosable hypomanic episodes in individuals with bipolar disorders. Consequently, it will be important in future studies to test whether individuals selected on the basis of high BAS sensitivity are more likely to exhibit current hypomanic/manic and depressive symptoms and to have onsets of hypomanic/manic and depressive episodes when they experience BAS activation- and deactivation-relevant life events, respectively.

The role of BIS

We also explored whether sensitivity of the BIS would be associated with lifetime mood disorder diagnoses or concurrent symptoms and personality, controlling for BAS sensitivity. Given that the BIS regulates withdrawal and/or inhibition of behavior in response to threat and punishment and frustrative non-reward (Fowles, 1988; Gray, 1991) and has been linked to anxiety and depression (Fowles, 1988; Gray, 1994; Gray & McNaughton, 2000), it is not surprising that higher self-reported BIS sensitivity was significantly associated with both current depressive symptom levels (BDI scores) and proneness to depressive symptoms (GBI-D scores). Individuals who are especially responsive to negative life events (threats and punishments) may be vulnerable to developing depressive symptoms.

More surprising, however, was our finding that the SP Scale from the SPSRQ was also positively associated with increased proneness to hypomanic symptoms (GBI-HB scores). Similarly, the combination of high BIS sensitivity (BIS scale from the BIS/BAS scales) and high BAS sensitivity (BIS $\times$ BAS interaction) was positively associated with hypomanic tendencies (HP scores). It is intriguing to consider the possibility that individuals who exhibit both highly sensitive BAS and BIS motivational systems are hyper-reactive to both rewards and goal incentives on the one hand, and threats and punishments on the other hand, thereby increasing their vulnerability to both positive (e.g., hypomania/mania) and negative (depression, anxiety) affective states and episodes. This line of reasoning is consistent with reviews of the life events and bipolar disorder literature (e.g., Alloy et al., 2005; Alloy, Abramson, Walshaw, & Neeren, in press; Johnson & Roberts, 1995) which document that both positive and negative life events appear to trigger affective episodes in people with bipolar spectrum disorders. In contrast, it was the combination of high BAS and low BIS sensitivity (BIS $\times$ BAS interaction) that was related to higher impulsivity (IN scores). It may be that, as some investigators (Fowles, 1988, 1993; Gray, 1991) hypothesized, a highly sensitive BAS coupled with low constraint of the BAS by an under-active BIS is associated particularly with impulsivity in pursuing activities with the potential for pleasurable, but potentially harmful, consequences. However, given that our findings in regard to BIS were exploratory and the breakdowns of our two significant BIS $\times$ BAS interactions did not yield significant relations, we await replications of these findings before drawing any firm conclusions regarding the role of BIS in bipolarity-relevant behaviors.

Study strengths and limitations

This study has several notable strengths, not the least of which is the use of a behavioral high-risk design, which provides a more powerful test of the BAS vulnerability hypothesis for bipolar disorders than offered by prior studies that select participants on the basis of bipolar symptoms or diagnosis (see Alloy et al., 1992, 1999, 2000; Just et al., 2001). Other strengths include participant selection blind to symptom and diagnostic status, the use of standardized diagnostic interviews and criteria, and interviewers blind to participants' BAS status.

However, it is also important to note the study's limitations. First, our sample size was relatively small and we may have had inadequate statistical power to detect some effects that were marginally significant. In addition, our sample consisted of undergraduates, which although ethnically diverse, may not be representative of a community sample. In addition, given that our sample was around 19-years-old on average, some of our participants may not have yet developed a lifetime history of a mood disorder. Thus, replication of our findings in a larger and more diverse community sample would be valuable.

As already discussed, another limitation of our study is that we employed a version of the behavioral high-risk design that was retrospective and concurrent, rather than prospective. Clearly, the strongest test of the BAS vulnerability hypothesis for bipolar disorder would involve selection of high vs. moderate or low BAS sensitivity individuals who are followed prospectively to determine whether they develop bipolar spectrum disorders. Such a prospective design would be even more valuable if it also assessed the prospective occurrence of BAS activation- and deactivation-relevant life events and their role in triggering hypomanic/manic and depressive episodes among high vs. moderate or low BAS sensitivity individuals. Our finding that BAS-Reward Responsiveness

may also contribute to the prediction of hypomania-relevant characteristics suggests that in future studies, it may be advisable to select participants on BAS sensitivity on the basis of all components of BAS, not just some.

Finally, participants were selected for our study based on self-reported BAS sensitivity. As noted above, currently available BAS questionnaires only assess sensitivity to BAS activation-relevant cues, but not BAS deactivation-relevant cues. Thus, further instrument development is needed to be able to more adequately test the BAS hypersensitivity theory of bipolar disorders. Moreover, although the self-report measures of BAS sensitivity have been validated against both behavioral (e.g., Heponiemi et al., 2003; Zinbarg & Mohlman, 1998) and neurobiological (e.g., Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997) indices of BAS activity, future studies would benefit from the use of such behavioral and neurobiological (e.g., EEG) indicators of BAS sensitivity as additional or alternative selection criteria in a behavioral high-risk design.

Conclusion

In summary, consistent with the vulnerability hypothesis of the BAS hypersensitivity theory of bipolar disorder (Depue & Iacono, 1989; Depue et al., 1987; Urosevic et al., 2006a), this study provides strong evidence that individuals selected on the basis of high self-reported BAS sensitivity are more likely to have a bipolar spectrum disorder than individuals with lower (moderate) levels of self-reported BAS sensitivity. In addition, our findings suggest that high BAS sensitivity individuals also exhibit higher impulsivity, hypomanic tendencies, and possibly higher proneness to hypomanic symptoms than moderate BAS sensitivity individuals. Thus, perhaps the hypomanic episode described by the young woman in the quote at the beginning of this article is explained by her highly sensitive BAS.

Acknowledgment The research reported in this article was supported by National Institute of Mental Health Grant MH 52617 to Lauren B. Alloy.

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