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Responsiveness to Threat and Incentive in Bipolar Disorder: Relations of the BIS/BAS Scales With Symptoms

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Abstract

Over the past 10 years, theorists have suggested that bipolar disorder symptoms result from increases and decreases in the activity of the Behavioral Activation or Facilitation System (BAS or BFS) and the Behavioral Inhibition System (BIS). These neurobehavioral systems are thought to determine the intensity of affective and behavioral responses to incentives and threats. This study examined cross-sectional and prospective associations of self-reported BIS and BAS with mania and depression in a sample of 59 individuals diagnosed with Bipolar I disorder. Depression was tied to BIS, pointing to the importance of sensitivity to threats in depression. However, links between BIS and depression appeared state-dependent. BAS subscales did not correlate with manic symptoms in a state-dependent manner; however, BAS (total scale and reward responsiveness subscale) predicted relative intensification of manic symptoms over time. Thus, evidence suggests that BAS sensitivity may constitute a vulnerability to mania among persons diagnosed with bipolar disorder. Discussion focuses on the integrative potential of the BIS/BAS constructs for linking psychosocial and biological research on bipolar disorder.

Keywords

bipolar disorder; behavioral activation; behavioral inhibition; mania; depression

Considerable progress has been made over the past decade in research on the pathology, course, and etiology of bipolar disorder. Evidence is converging for genetic links (Craddock & Jones, 1999; Pekkarinen, Terwilliger, Bredbacka, Lonnqvist, & Peltonen, 1995) as well as neurochemical and neuroanatomic abnormalities (Goodnick, 1997; Goodwin & Jamison, 1990; Lenox & Watson, 1994; Norris, Krishnan, & Ahearn, 1997). Empirical support for the efficacy of medication in the treatment of bipolar disorder is incontrovertible, but pharmacotherapy remains imperfect in the management of symptoms (e.g., Meltzer et al., 1995). Despite the firmly established biological foundation of bipolar disorder, there is clear evidence that psychosocial factors, such as stressful life events (Johnson & Miller, 1997; Johnson & Roberts, 1995), cognitive styles (Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999), and expressed emotion (e.g., Simoneau, Miklowitz, & Saleem, 1998), also influence the course and timing of episodes.

To integrate biological and psychosocial influences in bipolar disorder, diathesis-stress or vulnerability models have inspired interest (Depue & Iacono, 1989; Johnson & Roberts, 1995). According to these views, psychosocial variables influencing course may be best understood in the context of biological vulnerabilities. For example, stressful events may trigger episodes by disrupting social and biological circadian rhythms (Ehlers, Frank, &

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Kupfer, 1988; Malkoff-Schwartz et al., 1998). The propensity of events to trigger symptoms may also be influenced by functional circuits of the central nervous system (Depue & Iacono, 1989; Depue & Zald, 1993; Gray, 1990, 1994; Johnson & Roberts, 1995).

For example, Depue and colleagues (e.g., Depue & Iacono, 1989; Depue & Zald, 1993) have conceptualized symptoms of bipolar disorder as the result of increased activity in the "Behavioral Facilitation System (BFS)," similar to Gray's Behavioral Activation System (BAS; Gray, 1990, 1994). The BAS is viewed as a system that regulates approach behavior and positive affect in response to signals of reward. Evidence from a number of sources suggests that the BAS is linked to dopaminergic (DA) pathways ascending from the ventral tegmental area (VTA; Depue & Zald, 1993; Winters, Scott, & Beevers, 2000). When confronted with an incentive stimulus, BAS activity is thought to trigger positive affect (e.g., hope, elation, excitement), approach motivation (e.g., a feeling of desire), and approach behavior. These "system outputs" serve to increase the probability of incentive acquisition. An underactive BAS is linked with unresponsiveness to incentives, low positive affect, and a lack of environmental engagement (Depue & Zald, 1993).

Depue has noted the direct correspondence between the behaviors regulated by the BAS and manic symptoms of euphoric mood, inflated self-esteem, decreased need for sleep, increased talkativeness, flight of ideas, increased goal-directed activity, and excessive involvement in pleasurable activities. The state of mania, then, may be the outcome of a highly sensitive or active BAS system, producing elated mood and activation in response to even minimal incentives in the environment (Depue & Zald, 1993). The state of depression, in contrast, may be the outcome of a highly unresponsive or inactive BAS, failing to produce positive affect or activity even when incentives are present (cf., Mineka, Watson, & Clark, 1998; Sutton & Davidson, 1997).

At a simple level, this model suggests that BAS strength should correlate with mania and depression. Indeed, among 63 undergraduates classified as at-risk for mood disorders—but not among normal controls—BAS self-report scales correlated robustly with mania and with depression (Meyer, Johnson, & Carver, 1999). Hedonic deficits have also been associated with unipolar depression. For example, depressed individuals demonstrate decreased reinforcement sensitivity (Henriques, Glowacki, & Davidson, 1994) and less activity in cortical regions related to BAS functioning (Henriques & Davidson, 1991). Comparable links have not been examined in a clinical sample of individuals with bipolar disorder.

BAS strength is often regarded as a temporally stable disposition (Carver & White, 1994), although one theory suggests that it may oscillate over time among people with bipolar disorder (Depue & Iacono, 1989; Depue, Krauss, & Spoont, 1987; Depue & Zald, 1993). Fluctuations in BAS are hypothesized to depend on regulatory strength, which may be influenced by serotonin systems and other moderators (Depue & Zald, 1993). Individuals with low regulatory strength—an easily dysregulated BAS—have been hypothesized to be especially reactive to environmental challenges such as life events (cf. Depue & Iacono, 1989; Depue & Zald, 1993). Congruent with this regulatory model, a series of studies have shown that individuals with bipolar-spectrum disorders display more profound reactions to laboratory and daily stressors (Depue et al., 1981; Goplerud & Depue, 1985).

Gray (1994) has delineated the components and functions of a second major motivational system: The Behavioral Inhibition System (BIS). The BIS is hypothesized to regulate behavioral inhibition, arousal, and attention in the face of signals of punishment, innate fear stimuli, and novel stimuli. Further, the BIS is hypothesized to regulate anxiety (e.g., Gray, 1994; Mineka et al., 1998). A strong BIS may produce anxiety and inhibition even when few threats are encountered, whereas a weak BIS may fail to produce anxiety and inhibition even

when intense threats are present. In Gray's theory (1990 (1994), the septal area and hippocampal formation play central roles in the BIS, but the precise neural components are still subject to debate (cf. LeDoux, 1995).

According to Gray (1990, 1994), the BIS can best be thought of as an anxiety system, but it has widespread implications for a range of psychiatric conditions (cf. Fowles, 1993). With regard to bipolar disorder, Gray (1991) noted that "low activity in the behavioral inhibition system accompanied by high activity in the approach system [BAS] might be thought of as underlying mania" (p. 300; cf. Fowles, 1993, for similar arguments). Indeed, the *DSM-IV* criteria for mania specify a disregard for "potential painful consequences" (American Psychiatric Association, 1994). According to this view, the disinhibition and risk-taking behavior evident in the manic state reflect a mixture of reduced responsiveness to threats and exaggerated responsiveness to incentives. Among undergraduates at-risk for mood disorders, BIS strength was unrelated to manic symptoms (Meyer et al., 1999). This null finding might be due to the absence of clinical mania in the student sample. The current study examines whether self-reports of low BIS strength relate to manic symptoms in a sample of persons with bipolar disorder.

Drawing on theory by Depue, Gray, and Fowles, it was hypothesized that BAS strength would be tied to symptoms of mania and depression among individuals diagnosed with bipolar I disorder. Two models of how BIS and BAS might relate to symptoms were examined. According to the first model, fluctuations in self-reported BAS strength over time might covary with increases in manic symptoms and decreases in depressive symptoms. This would support the idea that BIS and BAS strength vary over time among those with bipolar disorder, even though these systems may be more stable among normals (Carver & White, 1994).

Second, it was hypothesized that baseline levels of BAS during recovery would predict follow-up symptom levels. This hypothesis was based on the assumption that a high level of incentive responsiveness at baseline would place a person at risk for experiencing intense positive affect and manic activation over time, particularly when incentives are actually encountered. Conceptually, the BAS produces "output" of positive affect and approach behavior in response to "input" of incentive-related information. Thus, persons with a highly sensitive BAS might be vulnerable to manic symptom intensification if they encounter incentives. In support of this idea, research has demonstrated that individuals with bipolar disorder tend to experience more manic symptoms after attaining goal or incentive-related events, such as receiving a promotion (Johnson, Sandrow et al., 2000). In the present study, the hypothesis was tested that self-reported BAS sensitivity alone (rather than the perception of incentive) would place individuals with bipolar disorder at risk for manic symptom intensification. Similarly, the hypothesis was tested that low levels of incentive responsiveness at baseline might predict depressive symptom intensification over time, as the person fails to respond to environmental incentives and subsequently becomes more depressed. In each of these analyses, the BIS was also considered, to explore whether self-reports of sensitivity to threats would be tied to symptoms of mania and depression in bipolar disorder.

METHODS

Sample Description

The sample was composed of 59 individuals (30 men, 29 women; mean age = 43.7; *SD* = 10.02; range = 26–65) diagnosed with bipolar I disorder as assessed by the Structured Clinical Interview for *DSM-IV* (SCID). Data were drawn from an ongoing, longitudinal NIMH-supported investigation. Individuals between the ages of 18 and 65 were recruited

from hospitals, outpatient clinics, support groups, and community advertising in South Florida. Exclusion criteria were (1) mood symptoms secondary to a general medical condition, (2) alcohol abuse or dependence in the past year, (3) substance abuse or dependence in the past year, and (4) inability to speak English or independently complete self-report measures. Previous reports have described effects of social support (Johnson, Winett, Meyer, Greenhouse, & Miller, 1999), self-esteem (Johnson, Meyer, Winett, & Small, 2000), coping (Greenhouse, Meyer, & Johnson, 2000), and life events (Johnson & Miller, 1997) in this sample.

Eighteen percent of the participants were married, and 46% were divorced, widowed, or separated. Despite high educational attainment (50% of the sample had completed college or beyond), only 28% were employed full-time. Thirty-two percent were receiving disability payments, 8% were retired, and 16% were unemployed. Another 10% were employed part-time, 2% were homemakers, and 4% were students. The sample was ethnically diverse; 72% were Caucasian non-Hispanic, 18.5% were Hispanic, 5.6% were African American, and 4% categorized themselves as belonging to other groups.

Most participants had experienced a long and severe course of illness; that is, the mean age of illness onset was 19.98 (*SD* = 9.80) and the mean number of previous episodes was 24.40 (*SD* = 19.09), with 17.47 previous manic episodes (*SD* = 20). The mean number of previous hospitalizations for bipolar disorder was 3.03 (*SD* = 3.03). No one in the sample was experiencing a first episode. At the time of entrance into the study, 96% were experiencing an episode. Approximately 30% were experiencing a manic episode, 41% were experiencing a depressive episode, and 24% were experiencing cycling between manic and depressive poles or mixed episodes. Of the individuals experiencing an episode at study entry, approximately 20% were experiencing psychosis. Twenty-two percent of the sample had experienced rapid cycling during their lifetime.

Design and Measures

Symptoms were assessed monthly using structured interviews, and BIS/BAS scales (described later) were completed at intake as well as at 2-, 6-, 12-, 18-, and 24-month follow-ups. On average, participants completed 20 months of follow-up (*SD* = 6.97).

For cross-sectional analyses, the first completed BIS/BAS scales were selected for each participant. Forty-one participants completed their first questionnaires at intake, 14 two months later, and 4 six months after intake. Symptom measures were administered in the same month. The time-lag between BIS/BAS self-reports and interview symptom measures ranged from hours to a couple of weeks.

Diagnosis—The SCID-I/P, Version 2.0 (First, Spitzer, Gibbon, & Williams, 1996) was used to make *DSM-IV* diagnoses. Interrater reliability for the SCID has been shown to be strong; Williams et al. (1992) achieved a kappa for bipolar disorder of .84. In the authors' research team, a high rate of agreement has been achieved for the specific symptoms of mania, $r = .94$, $N = 74$, $p < .0001$. A psychologist oversaw all diagnostic decisions, and a psychiatrist was consulted to confirm any differential diagnosis of organic brain syndrome.

To measure the intensity of current symptoms of mania and depression, the Modified Hamilton Revised Scale for Depression (MHRSD) and the Bech–Rafaelsen Mania Scale (BRMS) were administered in monthly interviews.

MHRSD—The MHRSD (Miller, Bishop, Norman, & Maddever, 1985) was used as a measure of depressive symptom severity. This modification of the Hamilton Rating Scale for Depression includes a standardized interview format and behavioral references for each

item. The 17-item index, which was used in this study, achieves strong correlations with the traditional HRSD (Hamilton, 1960), and the authors' team has high interrater reliability (intraclass correlation = .95). The MHRSD has been used extensively to assess changes in depressive symptoms (e.g., Miller, Norman, & Keitner, 1989, 1999), and has been shown to be a valid measure of bipolar depression (Johnson, Meyer et al., 2000). The MHRSD targets a broad range of depressive symptoms (e.g., depressed mood, excessive guilt, suicidal ideation, sleep disturbance, appetite/weight changes, energy loss).

BRMS—The BRMS (Bech, Bolwig, Kramp, & Rafaelsen, 1979) was used as a measure of manic symptom severity. The BRMS is a 12-item measure with strong psychometric properties; interrater reliability as assessed using Spearman correlation coefficients ranged from .97 to .99. The scale is widely used for the detection of changes in clinical status. Interviewers completed extensive training, and specific probes were used for evaluating each type of symptom. Interrater reliability for this team of raters was high (intraclass correlations = .92). Previous analyses have suggested that the BRMS is sensitive to changes in clinical symptom levels and correlates well with sleep and other risk variables for mania (Johnson, Winett, & Mellman, 1998; Lozano & Johnson, 2001). The BRMS assesses a broad range of manic symptoms (e.g., euphoric mood, heightened self-esteem, irritability, increased motor activity, hypertalkativeness, racing thoughts, reduced need for sleep). Both the HRSD and the BRMS assessed symptoms during the most severe week within the month.

Recovery Criteria—Previous research has established standardized criteria for recovery on these symptom severity measures for unipolar depression (Frank et al., 1991), and these criteria have been validated for bipolar disorder (Rosenberg, Winett, & Johnson, 1998). These criteria for recovery include a MHRSD <17 and a BRMS <16. Forty individuals achieved recovery and were followed for 6 more months, allowing for the examination of the BIS/BAS scales as predictors of symptom changes following recovery.

BIS/BAS Scales—BIS and BAS sensitivities were measured with Carver and White's 20-item BIS/BAS scales (Carver & White, 1994). The BIS scale (seven items) measures the tendency to respond with negative affect, anxiety, or fear in response to threatening events (i.e., threat responsiveness). It contains items such as "If I think something unpleasant is going to happen I usually get pretty 'worked up'." The BAS scales focus on tendencies to respond with positive affect and motivation when faced with incentives or rewards. BAS is composed of three factorially distinct subscales: Reward Responsiveness, Drive, and Fun Seeking. People scoring high on Drive are strongly motivated to pursue desired goals and to "get what they want" (e.g., "If I see a chance to get something I want I move on it right away"). The Reward Responsiveness scale measures the tendency to respond with heightened energy and positive affect when desired events are experienced or anticipated (e.g., "When good things happen to me, it affects me strongly"). Conceptually, this scale demonstrates the strongest content overlap with models of Depue (e.g., Depue & Iacono, 1989; Depue, Luciana, Arbisi, Collins, & Leon, 1994), and with recent animal models of dopaminergic ventral tegmental neurons (e.g., Schultz, Dayan, & Montague, 1997; Schultz, Tremblay, & Hollerman, 2000). Fun Seeking captures the impulsive behavioral pursuit of pleasurable opportunities (e.g, "I will often do things for no other reason than that they might be fun").

Psychometric properties (internal consistency, factor structure, test-retest reliability) of the BIS/BAS scales have been found to be adequate (Carver & White, 1994; Jorm et al., 1999; Heubeck, Wilkinson, & Cologon, 1998). Convergent and discriminant validity has been demonstrated vis-à-vis measures of extraversion, trait anxiety, positive affect, and novelty seeking (Carver & White, 1994; Jorm et al., 1999; Heubeck et al., 1998). Normative data are

available from a major community sample (Jorm et al., 1999). In the present sample, internal consistencies were satisfactory (χ = .78 for BIS, .84 for total BAS, .67 for Reward Responsiveness, .81 for Drive, .76 for Fun Seeking). Test-retest reliability coefficients were high for BIS (r = .81, p < .001) and moderate for BAS (r = .50 for total BAS, .44 for Reward Responsiveness, .46 for Drive, .49 for Fun Seeking, p < .01). These stability coefficients were computed for a subset of 42 participants who completed the BIS/BAS scales both at intake and an average of 11.52 (*SD* = 6.44) months after the initial assessment.3

RESULTS

Preliminary analyses were conducted to examine descriptive statistics and potential confounds. Then, BIS/BAS levels were compared with normative samples and basic correlations were inspected between BIS/BAS levels and symptoms. All significance tests were two-tailed, with alpha set at .05 level.

Preliminary Analyses

Several variables that might influence both BIS/BAS levels and symptoms were examined first, including negative life events, social support at 2 months after intake, and medication adequacy at intake, age, and gender. Descriptions of the measures used to assess these variables are provided elsewhere (Johnson et al., 1999; Johnson & Miller, 1997; Johnson, Sandrow et al., 2000). Pearson's correlation coefficients were computed between these variables and the BIS/BAS scales. BIS and BAS scores were independent of life events, social support, medication adequacy, and age. An unexpected gender difference was observed for BAS Drive, with women (M = 12.52, SD = 2.31) scoring higher than men (M = 11.13, SD = 2.64), $t(57) = 2.14$, p < .04.

Descriptive statistics for the symptom measures BRMS and MHRSD are presented in Table I, and for the BIS/BAS scales in Table II. On average, symptom severity levels were in the mild-to-moderate ranges. MHRSD and BRMS did not correlate with each other at significant level, r = .22, p = .10. Correlations among the BIS/BAS scales were generally consistent with previous research (e.g., Carver & White, 1994;Heubeck et al., 1998;Jorm et al., 1999). BIS did not correlate with any of the BAS subscales, $r < |.11|$, $p > .43$., and all three BAS sub-scales were moderately intercorrelated. Specifically, Reward Responsiveness correlated .40, p < .01, with Drive and .53, p < .001, with Fun Seeking. The correlation between Drive and Fun Seeking was .43, p < .01.

In a large Australian community sample, Jorm et al. (1999) documented lower levels of BIS and BAS among older compared to younger cohorts. Compared to age-matched Australians (aged 40–49; Jorm et al., 1999), participants with bipolar disorder reported higher levels of

[3]Additional findings have been reported to support the validity of the BIS/BAS scales. For example, the BIS scale has been found to predict anxiety in response to impending punishment (even when statistically controlling for the effect of trait anxiety) and the BAS scales (Drive and Reward Responsiveness only) have been found to predict happiness in response to impending reward, even when statistically controlling for the effect of extraversion (Carver & White, 1994). BIS correlates moderately with manifest anxiety, negative affectivity, and neuroticism but is relatively distinct from positive affectivity and extraversion—the opposite pattern holds for BAS (Carver & White, 1994; Heubeck et al., 1998; Jorm et al., 1999). BAS is also correlated with sensitivity to cues of reward in a conditioning task, but not to cues of punishment (Zinbarg & Mohlman, 1998). Elevations on these scales are also associated with asymmetries in frontal cortical activation (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). BIS has been linked with greater right frontal activation, and BAS with greater left frontal activation. Evidence suggests that the three BAS scales are only moderately correlated (e.g., Carver & White, 1994; Heubeck et al., 1998; Jorm et al., 1999) and show a differential pattern of associations with symptoms. For example, the BAS Fun Seeking scale (but not Drive or Reward Responsiveness) appears to be involved in substance abuse disorders (Johnson, Turner, & Iwata, 2001) Similarly, Fun Seeking was involved in predicting distress among cancer patients, but Reward Responsiveness was linked with the tendency to give up or disengage effort (Carver, Meyer, & Antoni, 2000). Reward Responsiveness appears to capture the facets of BAS activity that have been most emphasized in theoretical models of mania.

BIS, Drive, and Fun Seeking. For these comparisons, the initial BIS/BAS records were selected (see Table II).

Primary analyses focused on the longitudinal relations between BIS/BAS scores and symptoms. Before conducting these more complicated analyses, however, the cross-sectional correlations of BIS/BAS scores with symptoms were computed. Table III presents correlations of the BIS/BAS scales with interview-based symptoms. None of the BAS subscales were associated with manic symptom severity. Similarly, BIS scores were not tied to BRMS. In regard to depression, unexpectedly, none of the BAS scales correlated with MHRSD scores. As hypothesized, a positive correlation was observed between BIS and the MHRSD scale.

Longitudinal Associations of BIS/BAS With Changes in Symptoms

To examine whether BIS/BAS and symptom scores fluctuated in tandem over time in a state-dependent manner, mixed-effects regression models were used. These models allow for missing data and differences in the number of assessments across individuals, as well as measurements at different time points. Because observations were clustered within individuals (i.e., multiple assessments of BIS/BAS and mania/depression for each participant), it would be inappropriate to use simple linear regression models in which symptoms are predicted from BIS/BAS levels. Mixed effects regression takes into account this clustering of data, yielding a more accurate estimate of symptoms by both cluster-level (person) and individual-level (BIS/BAS) predictors. Although the cross-sectional analyses provide an index of how individuals with higher and lower BIS and BAS levels score on symptom measures, the mixed-effects regression models provide a more dynamic index of how BIS/BAS scores and symptoms are associated across time. In these analyses, the conjoint influence of BIS and the three BAS subscales was examined. BAS total scores were not included because the subscales and the total scale encompass the same items.

Analyses were conducted with the MIXREG program (Hedeker & Gibons, 1996), which allows for the examination of autocorrelation. Models (one for depression, and one for mania) were first fit without including an autocorrelation term. These models included participant ID number as a second-order (cluster variable) and the month of assessment, BIS, BAS Reward, BAS Drive, and BAS Fun seeking as independent variables. Then, second models fit an autocorrelation term to the errors, using a first-order moving average process (AR1). For the second models, Fisher-scoring iterations were used, with model estimates from the uncorrelated error model used as starting values.

Maximum Marginal Likelihood (MML) Estimates for the addition of the AR1 parameter to the model were not significant for depression—Log Likelihood Difference $n(df = 1) < .01$, estimated stationary autocorrelation = 0.02, $p = .92$. For mania, the addition of the AR1 parameter to the model was significant by the likelihood ratio test—$\chi^2 = 2(2.18) = 4.36$, $df = 1$, $p < .05$, estimated stationary autocorrelation = .27. Given this, a model without an autocorrelation term was used for depression and a model with an autocorrelation term was used for mania. Neither BIS nor any of the BAS scales fluctuated in tandem with manic symptoms (see Table IV). As hypothesized, BIS fluctuated with changes in depressive symptoms, as measured by the MHRSD. None of the BAS scales fluctuated with depressive symptoms (see Table IV).

Prospective Prediction of Symptom Changes Following Recovery

Among 49 individuals who achieved recovery, BIS/BAS scales were examined as potential predictors of increased symptoms of mania and depression. For these analyses, the first BIS/BAS scales completed after the individual achieved recovery were selected. To examine

increases in depression, partial correlations were computed of BIS/BAS scales with the mean MHRSD scores over the next 6 months, controlling for baseline MHRSD scores at the time of the assessment. Parallel analyses were conducted next, controlling for baseline BRMS scores, to examine the partial correlation between BIS/BAS scales and the mean BRMS scores over the next 6 months. As can be seen in Table V, increases in BRMS scores were predicted by baseline levels of BAS Reward Responsiveness (as well as BAS total). Partial correlations for BAS Drive and Fun seeking with BRMS were in predicted directions but did not attain significance. BRMS changes were not predicted by the BIS scale. None of the BIS/BAS scales predicted increased depression over time.

DISCUSSION

The behavioral activation and inhibition systems have been theorized to explain both mania and depression (Depue & Zald, 1993; Gray, 1990, 1994). This study was the first direct examination of the relation between BIS/BAS levels and symptoms among individuals diagnosed with bipolar I disorder. It was hypothesized that BAS scores would associate positively with mania and inversely with depression. The opposite pattern was expected for BIS.

Support for these hypotheses was generally weak, although evidence was obtained for a hypothesized link between BAS and manic symptom intensification over time. Although the primary analyses focused on longitudinal patterns, cross-sectional correlations were considered first. These analyses generally failed to support state-dependent links between symptoms and BIS/BAS levels. The observed pattern was somewhat incongruent with results from a previous study of undergraduates at risk for mood disorders, in that BAS did not correlate with concurrently assessed symptom severity (Meyer et al., 1999).

Cross-sectional analyses did reveal one significant link—BIS self-reports correlated with higher depressive symptoms. This finding is consistent with a broad literature that links threat responsiveness or neuroticism with depression (Clark, Watson, & Mineka, 1994; Mineka, Watson, & Clark, 1993; Watson, Clark, & Harkness, 1994). In contrast to this scarcity of findings, stronger and more consistent links were observed for all three BAS scales with current levels of both mania and depression in an at-risk undergraduate sample (Meyer et al., 1999). Two obvious reasons may account for this difference: (1) The earlier findings relied on BIS/BAS self-report–mania and depression symptoms self-report correlations, whereas in the present study clinician-administered interviews were used as symptom measures; (2) In the earlier study, BIS/BAS and symptom measures were collected at exactly the same time, whereas time was not as closely matched in the present study.

In addition to examining cross-sectional associations, analyses were conducted that specifically disentangle the longitudinal links between BIS/BAS levels and symptoms: (1) the random effects regression models, indexing how BIS/BAS levels fluctuated with symptom changes within individuals and (2) the partial correlation coefficients, indexing how BIS/BAS levels at recovery predicted changes in symptoms over time.

As hypothesized, BIS levels fluctuated with depressive symptoms. However, BIS levels after recovery did not predict increased depression over time. In short, BIS reports appeared to function as a state-dependent characteristic of depression, but there was little evidence that BIS operated as a vulnerability characteristic. Although it seems plausible to assume that threat responsiveness (BIS) places people at risk for future distress or depression, no evidence was found in support of such a link. In preliminary work, however, the interaction of BIS at baseline and subsequent stressful experience was associated with relative intensification of depressive symptoms over time (Meyer, Johnson, & Blaney, 2000). Thus,

high levels of threat responsiveness may constitute a vulnerability in combination with aversive experience, but not as a “main effect” predictor of symptom intensification.

BAS appeared unrelated to depression in the present analyses. This was surprising, given that BAS inactivity appears to be a marker of unipolar depression (Gotlib, Ranganath, & Rosenfeld, 1998; Harmon-Jones & Allen, 1997; Henriques et al., 1994; Mineka et al., 1998). Indeed, a correlation of −.53($p < .01$) between the total BAS scale and a depression self-report was observed in an undergraduate at-risk sample (Meyer et al., 1999). In another recent study, low BAS appeared to be relevant specifically for the anhedonic but not the negative affect components of depression (Beevers & Meyer, in press). Comparisons among these studies are limited, however, by the differences in measurement and design, as well as the fact that many previous studies focused on unipolar depression. The role of the BAS in bipolar depression certainly seems worthy of further study, despite the null findings reported here.

Although some have suggested that low BIS strength might be associated with manic disinhibition (Fowles, 1993; Gray, 1994), self-reported BIS strength did not correlate with or predict manic symptoms. This lack of association was also observed in an at-risk student sample (Meyer et al., 1999). Further study is needed to test whether these null findings are limited to BIS self-reports but can perhaps be detected with biochemical or behavioral indexes of BIS strength.

Additionally, no evidence was obtained to support the hypothesis that BAS scales would relate to concurrently assessed symptoms of mania. This null finding was surprising, given the theoretical plausibility of a BAS–mania link (Depue & Iacono, 1989; Depue & Zald, 1993; Fowles, 1993) as well as earlier findings in an undergraduate sample (Meyer et al., 1999). Again, methodological differences between the present study and the earlier study (e.g., reliance on self-report, time interval differences between BIS/BAS and symptom measures) may account for some of these discrepancies. On the other hand, there may be other reasons for this null finding. For example, the BAS scales were developed in an undergraduate sample, and item content may not capture adequately the construct of incentive responsiveness in clinical populations. The BAS scales were also developed to assess trait-like differences in threat and incentive responsiveness, but it was hypothesized here that these tendencies might fluctuate in tandem with symptoms. Thus, the BIS/BAS scales may not be suited to capture variability in threat- and incentive-responsiveness over time among patients with bipolar disorder. New measure development efforts may therefore be appropriate. One other possibility is that BAS sensitivity levels remain fairly stable as manic symptoms fluctuate—a model that is congruent with conceptualizing BAS as a vulnerability factor for manic symptoms.

Indeed, a significant relation was observed between BAS (total and reward responsiveness scales) and manic symptom intensification over time. Reward Responsiveness—the only BAS scale that was significantly linked with manic symptom intensification—appears to be a particularly good conceptual fit with Depue’s (Depue & Zald, 1993) and Gray’s (Gray, 1990, 1994) models. Partial correlations for the other two BAS subscales were also in predicted directions but did not attain significance. Thus, there was some evidence suggesting that heightened responsiveness to incentives places bipolar disordered persons at risk for mania. This is consistent with other recent findings. For example, bipolar individuals who experienced incentive-related events (e.g., receiving a promotion) were more likely to develop manic symptoms in subsequent months (Johnson et al., 2000). Presumably, the perception of goal-attainment-related events constitutes an input to the BAS. Given such input, a “malfunctioning” BAS in bipolar disorder may produce the output of excessive positive affect and unrestrained activity.

Although no previous research has directly examined BAS sensitivity and clinical levels of mania, other literature is congruent with a role for BAS in manic symptoms. Several models of the neural pathways involved in bipolar disorder emphasize dysregulation of the dopaminergic projections from the ventral tegmentum, which are involved in sensitivity to reward and incentive (Depue & Iacono, 1989; Depue & Zald, 1993). High levels of achievement striving among individuals with bipolar disorder and their family members have been noted in clinical lore and empirical findings (Coryell et al., 1989), and personality theorists have suggested that such traits are risk factors for mania (Plutchik, Platman, & Fieve, 1970). In sum, an emergent empirical literature suggests that BAS functioning and related constructs have potentially important implications for understanding mania. It is also important to note that mania is predicted not only by BAS, but by a range of other, independent variables, including medication and sleep changes (Johnson, Winett, & Mellman, 1998; Wehr, 1990; Wehr, Sack, & Rosenthal, 1987).

Several important limitations must be kept in mind when interpreting these findings, including the naturalistic design, limited power, and sample heterogeneity. One additional caveat concerns the content overlap between the items on the BAS scale and manic symptoms. The overlap between BAS Fun Seeking items such as “I crave excitement and new sensations” and manic symptoms such as “excessive involvement in pleasurable activities” cannot be denied, and indeed, such items appear to be a state marker of symptoms. However, items from the Reward Responsiveness scale (e.g., “It would excite me to win a contest”) appear to have less content overlap. It should be noted, however, that the strong conceptual overlap was an original influence on the development of the BAS model of bipolar disorder (e.g., Depue & Zald, 1993).

These results may be viewed as a first step of model testing. A fundamental goal will be to test this bipolar model using BIS and BAS measures that have less direct overlap with symptoms, including indexes of cortical asymmetry (e.g., EEG measures), reinforcement sensitivity tasks, and extinction paradigms. Although these indices have received attention in the literature relating BIS/BAS to personality and to other psychopathologies (e.g., Milich, Hartung, Martin, & Haigler, 1994; Newman, Patterson, & Kosson, 1987), the field of bipolar disorder has not yet used such indices. The present findings point to the utility of the BIS and BAS constructs in the integration of psychosocial and biological research on bipolar disorder, but further empirical inquiry will certainly be needed.

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Table I

Initial Symptom Severity Measures (N = 59)

	***M* (*SD*)**	**Range**
MHRSD	11.68 (7.61)	0–29
BRMS	6.95 (9.22)	0–36

Table II

BIS/BAS Scales Means and Standard Deviations: Bipolar Disorder Vs. Normative Samples

	Bipolar disorder sample: *N* = 59	Jorm et al. (1998): *N* = 729 (40–49-year-old group)	Significance test: *t* (786)
BIS	23.00 (3.79)	20.75 (3.54)	4.67**
BAS total	40.24 (6.21)	35.64 (5.55)	6.07**
Reward responsiveness	16.85 (2.39)	16.48 (2.05)	1.32
Drive	11.81 (2.56)	10.03 (2.56)	5.14**
Fun Seeking	11.58 (2.83)	10.71 (2.25)	2.80**

Note. Values are means and standard deviations (the latter in parentheses).

** $p < .01$.

Table III

Cross-sectional Associations Between BIS/BAS Scales and Symptoms (N = 59)

	BIS	BAS total	Reward responsiveness	Fun seeking	Drive
BRMS	.03	.16	.22	.12	.05
MHRSD	.45**	.07	.09	.13	−.06

** $p < .01$ (two-tailed).

Table IV

Random Effects Regression Estimates: Fluctuations Between BIS/BAS Scales and Symptoms Over Time (*N* = 59 Individuals, 156 Assessments)

Variable	Estimate	*SE*	*z*	*p*
MHRSD: Depression (without autocorrelation)				
Month	−0.14	0.08	−1.72	0.09
BAS reward responsiveness	−0.13	0.29	−0.45	0.65
BAS fun seeking	0.25	0.27	0.92	0.36
BAS drive	−0.44	0.27	−1.62	0.11
BIS	0.54	0.16	3.39	0.0007
BRMS: Mania (with autocorrelation)				
Month	−.58	.40	−1.46	.14
BAS reward responsiveness	.02	.32	.08	.94
BAS fun seeking	.51	.30	1.70	.09
BAS drive	−.36	.30	−1.21	.22
BIS	−.07	.20	−.34	.73

Table V

Partial Correlations of BIS/BAS Scales With Follow-up Symptoms, Controlling for Baseline Symptoms (N = 49)

	BIS	BAS total	Reward responsiveness	Fun seeking	Drive
BRMS	.12	.35*	.35*	.26	.23
MHRSD	.01	.01	−.05	−.05	.13

* $p < .02$ (two-tailed).