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Behavioral Activation and Inhibition Systems and the Severity and Course of Depression

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Theorists have proposed that depression is associated with abnormalities in the behavioral activation (BAS) and behavioral inhibition (BIS) systems. In particular, depressed individuals are hypothesized to exhibit deficient BAS and overactive BIS functioning. Self-reported levels of BAS and BIS were examined in 62 depressed participants and 27 nondepressed controls. Clinical functioning was assessed at intake and at 8-month follow-up. Relative to nondepressed controls, depressed participants reported lower BAS levels and higher BIS levels. Within the depressed group, lower BAS levels were associated with greater concurrent depression severity and predicted worse 8-month outcome. Levels of both BIS and BAS showed considerable stability over time and clinical state. Overall, results suggest that BAS dysregulation exacerbates the presentation and course of depressive illness.

In recent years there has been converging evidence from biological, neurological, and psychosocial investigations that several forms of psychopathology are characterized by specific patterns of deficits in approach- and withdrawal-related behavior (e.g., Arnett, Smith, & Newman, 1997; Rosenbaum et al., 2000). In general, research in this area has focused on the relation of Gray's (1973) behavioral activation system (BAS) and behavioral inhibition system (BIS) to different types of psychopathology. Whereas the BAS is an approach-related, positive-incentive motivation system, the BIS, in contrast, regulates sensitivity to threat and nonreward cues. Gray drew from the animal literature, as well as from work examining the mechanism of action of anxiolytic medications, to form his theory of the BIS. Although Gray's model was originally applied to anxiety, Depue and Iacono (1989) have postulated more recently that systems similar to those proposed by Gray are implicated in depressive illness. More specifically, Depue and Iacono proposed that major depression is characterized by deficits or dysfunctions in the behavioral activation system (which they labeled the *behavioral facilitation system*) and the behavioral inhibition system.

In support of this formulation, a wide range of empirical evidence indicates that depressed individuals are characterized by low BAS functioning and, to a lesser extent, by high BIS functioning. For example, Henriques and Davidson (2000) examined the behavioral responses of depressed and nondepressed participants to monetary reward, punishment, and neutral laboratory conditions. Participants were asked to identify previously presented words, and their responses were linked to monetary reward or punishment or to accuracy feedback in the neutral condition. Henriques and Davidson conceptualized (a) behavioral changes that occurred as a function of reward as an analogue of BAS responding and (b) behavioral changes that occurred as a function of punishment as an analogue of BIS responding. Consistent with the operation of a deficient BAS, depressed participants, unlike the normal controls, failed to change their pattern of response to the reward condition, whereas the punishment condition did not yield group differences in response.

Research in affective neuroscience has also implicated the BAS and BIS in biological vulnerabilities that are associated with depression (e.g., Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Henriques & Davidson, 1991). Perhaps most notably, Davidson (1992) has argued that tendencies toward approach and withdrawal are lateralized hemispherically in the brain, with the left hemisphere serving the approach system, or BAS, and the right hemisphere serving the withdrawal system, or BIS. Consistent with this formulation, studies using electroencephalography (EEG) have found that depressed individuals exhibit weaker activation in their left than in their right frontal lobes, a pattern of lateralization that is consistent with reduced approach and/or increased withdrawal in depression (e.g., Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991). In fact, Henriques, Glowacki, and Davidson (1994) have explicitly linked left frontal hypoactivation in depression to the hypothesized deficit in approach-related behavior in depression. Notably, left frontal hypoactivation has also been demonstrated with remitted depressives (Gotlib et al., 1998; Hen-

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riques & Davidson, 1990), suggesting that BAS-associated deficits persist beyond episodes of depression.

Consistent with the conceptualization of BIS and BAS as broad motivational constructs, self-report measures of BIS and BAS functioning have been found to converge with behavioral and biological indices of BIS and BAS activation (e.g., Sutton & Davidson, 1997). Indeed, a number of investigators have used self-report methodologies to examine BIS and BAS functioning in individuals with mood disorders. For example, Meyer, Johnson, and Carver (1999) found that BAS levels as measured by the BIS/BAS scales (Carver & White, 1994) were correlated significantly with the severity of manic symptoms in a sample of undergraduate students high in self-reported manic symptoms. In addition, Meyer, Johnson, and Winters (2001) found that BIS levels were associated with current levels of depression in a group of depressed bipolar patients admitted to an inpatient facility.

Finally, a number of theorists have argued that positive and negative affect can be conceptualized as expressions of BAS and BIS, respectively (e.g., Carver & White, 1994; Harmon-Jones & Allen, 1997). In this context, investigators have demonstrated that depressed individuals are characterized by low levels of positive affect and, presumably, by low BAS activity (see Mineka, Watson, & Clark, 1998, for a review of this literature). It is noteworthy that Mineka et al. postulated that whereas low positive affect may be specific to depression, high negative affect may characterize a broader range of psychopathology. Although there is not uniform agreement on the congruence of positive affect and the BAS (e.g., Harmon-Jones & Sigelman, 2001), the importance of low positive affect in depression may suggest a specific role for a deficient BAS in this disorder and a more diffuse role for elevated levels of BIS (e.g., Gable, Reis, & Elliot, 2000).

In sum, although there is substantial evidence to indicate that low BAS and, to a lesser extent, elevated BIS are implicated in depression, many questions remain unanswered. For example, although the theory implicating BIS and BAS in depression suggests that these constructs represent relatively stable risk factors for the persistence of this disorder (e.g., Depue & Collins, 1999), there is little empirical work documenting the role played by BIS and BAS in affecting the course of depression. Furthermore, it is also unclear whether BIS and BAS functioning are stable over time and over clinical state. Although the results of some studies suggest that these constructs may be stable (e.g., Sutton & Davidson, 1997), few longitudinal investigations have been conducted with clinical samples. Finally, the close relationship that Gray (1976) and others have proposed between anxiety states and BIS activation suggests that BIS and BAS functioning may be influenced by the symptoms of anxiety that often accompany depression. Again, however, few investigations have considered the effects that anxiety symptoms might have on levels of BIS and BAS in depression, and the studies that have examined this question have been limited by their use of relatively small sample sizes (e.g., Henriques & Davidson, 2000).

The present study was designed to address these issues. Diagnosed depressed and nondepressed participants were assessed with respect to levels of BIS and BAS, as well as a number of other areas of functioning, at two points in time: Time 1, when all of the participants in the depressed group met diagnostic criteria for major depressive disorder (MDD) according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition; *DSM–IV*; American Psychiatric Association, 1994); and Time 2, 8 months later, when many of the depressed participants had improved clinically and no longer met criteria for MDD.

We generated three predictions in this study. One cross-sectional prediction concerned the relation of Time 1 BIS/BAS scores with other measures taken at Time 1, and two longitudinal predictions concerned the relation of Time 1 BIS/BAS scores with constructs assessed 8 months later, at Time 2.

1. Participants with MDD would have higher scores on the BIS scale and lower scores on the BAS scales at Time 1 than would nondepressed participants. Furthermore, within the sample of depressed participants, scores on the BIS and BAS scales would be correlated positively and negatively, respectively, with severity of current depressive symptomatology and number of previous depressive episodes; and negatively and positively, respectively, with level of psychosocial functioning, as indexed by participants' Global Assessment of Functioning Scale scores (Axis V Disorders, DSM–IV; American Psychiatric Association, 1994).

2. BIS and BAS scores would be stable over time and, for the depressed participants, over clinical state.

3. Within the sample of depressed participants, higher scores on the BIS scale and lower scores on the BAS scales at Time 1 would be associated prospectively with poorer clinical outcome at Time 2.

Method

Participants

Participants were 62 individuals with MDD and 27 nondepressed controls (NCs). All participants were fluent English speakers between the ages of 18 and 60. Approximately half of the depressed participants were recruited from two outpatient university hospital psychiatry clinics, and the other half were self-referred from the community. Clinical participants had no reported lifetime history of brain injury or primary psychotic ideation, no current diagnoses of panic disorder or social phobia, and showed no behavioral indications of impaired mental status or mental retardation. Clinical participants were also excluded from the sample if they were alcohol or substance dependent or if they showed signs of substance or alcohol abuse within the past 6 months. Potential control participants were excluded from the study on the basis of the same general and medical criteria that were used for the clinical participants. In addition, control participants were interviewed with the Structured Clinical Interview for *DSM–IV* Axis I Disorders—Patient Edition (SCID–IP; First, Spitzer, Gibbons, & Williams, 1995) to exclude those with lifetime diagnoses of any Axis I disorder.

On average, the depressed participants (all of whom met *DSM–IV* diagnostic criteria for MDD) were moderately depressed, with an average of 6.3 symptoms ($SD = 1.1$) during their current episode and an average of 4.6 prior episodes among those who were able to enumerate them. Thirty-seven of the depressed participants (57.8%) were taking psychotropic medication at the time of the initial interview. One control participant (3.7%) was taking an anticonvulsant for a seizure disorder at the time of the first interview. Participants were predominantly female (69% of the MDD group, and 67% of the NC group), Caucasian (72% of the MDD group, and 56% of the NC group), and single (61% of the MDD group, and 63% of the NC group). The mean age of the depressed sample was 34.6 years. Both groups of participants were relatively highly educated, with a mean education level of 6.6 on our ordinal scale from 1 to 8 (equivalent to

completing some college education), and both had a mean income in the range of $25,000–$50,000 per year. All participants were paid $25 per hour for their participation.

Overview of Procedure

Participants were recruited through referrals from the Department of Psychiatry and Behavioral Sciences at Stanford University Hospital and through advertisements and flyers soliciting participation of individuals who thought they might be depressed. Nonpsychiatric controls were recruited from the community through advertisements and flyers posted in numerous locations (e.g., Internet bulletin boards, university kiosks) soliciting healthy individuals. All participants were initially screened by telephone. Those who were considered likely to be eligible for participation in the study (approximately 34% of those screened) were scheduled for a session composed of an interview and a questionnaire battery. Those who qualified for the study (approximately 65% of subjects who were interviewed) returned approximately 1 week later to complete a second battery of questionnaires.

Participants were then followed up 8 months later and were reinterviewed with a structured diagnostic interview to determine whether they still met *DSM–IV* diagnostic criteria for MDD. Participants who still met *DSM–IV* criteria for depression at this follow-up assessment were classified as not improved; those who no longer met *DSM–IV* criteria for a diagnosis of depression (i.e., who were partially or completely remitted during the 8 weeks prior to the follow-up interview, according to National Institute of Mental Health criteria for recovery) were classified as improved. Participants also completed a number of questionnaires in this follow-up session.

Initial Evaluation

At the initial evaluation, all subjects were interviewed using the Structured Clinical Interview for *DSM–IV* (SCID; First, Spitzer, Gibbon, & Williams, 1996). Interviewers were two advanced psychology graduate students and two postbaccalaureate research assistants, all of whom were trained in the use of the SCID. To assess interrater reliability, an independent, trained rater who was blind to group membership evaluated 15 randomly selected audiotapes of SCID interviews with depressed and nondepressed participants, and with nonparticipants who met diagnostic criteria for disorders other than depression (e.g., panic disorder); for each participant, a rater determined whether the they met *DSM–IV* diagnostic criteria for MDD. In all 15 cases, ratings matched the diagnosis made by the original interviewer ($\kappa = 1.00$).

The GAF was used to assess global functioning. This is a single rating scale used to evaluate an individual's overall level of psychological, social, and occupational functioning. Ratings are made on the basis of information obtained during the SCID–I interview. Values on the scale range from 1 (*lowest level of functioning*) to 100 (*highest level of functioning*) and are divided into ten 10-point intervals. Each interval is anchored with a detailed, behaviorally oriented descriptor of functioning. Validation studies conducted with both inpatients and outpatients have indicated that the *DSM–IV* GAF and its predecessors correlate highly with other previously validated measures of overall severity of illness and changes in severity (e.g., Mental Status Examination Record; Endicott, Spitzer, & Fleiss, 1975) as well as with therapists' and relatives' ratings of patient functioning (Endicott, Spitzer, Fleiss, & Cohen, 1976). The GAF has also been found to have good interrater reliability (Endicott et al., 1976). For reliability purposes in the present study, an independent, rater trained in SCID–I who was blind to group membership listened to the audiotaped SCID interviews of 14 randomly selected participants and made GAF ratings. Interrater reliability for the GAF was high ($r = .92$).

Self-Report Measures

Participants also completed a number of self-report instruments, including the Hamilton Depression Inventory (HDI; Reynolds & Kobak, 1995), the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988), and the Behavioral Inhibition and Activation Scales (BIS/BAS; Carver & White, 1994). The HDI is a 23-item self-report version of the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) that has been shown to correlate highly with structured clinical interviews and has demonstrated high reliability and validity (Kobak & Reynolds, 2000; Reynolds & Kobak, 1995). Because the HRSD has been found to include symptoms of anxiety as well as depression (e.g., Fleck, Poirierlittre, Guelfi, Bourdel, & Loo, 1995), we used the scoring method developed by Riskind, Beck, Brown, and Steer (1987) to reduce the overlap with anxiety symptoms and to yield a purer measure of self-reported depression severity.[1] The internal consistency of the HDI in the present study was .94.

The BAI is a 21-item self-report measure of anxiety symptoms. This measure has been demonstrated to have good reliability and validity (see Steer & Beck, 1997, for a review) and has been widely used in studies of depression and anxiety. We included the BAI in this study to provide a measure of severity of anxiety symptoms. It is important to note, however, that we excluded individuals from participation in this study if they met diagnostic criteria for either panic disorder or social phobia. Consequently, we expected that we might have a restricted range on the BAI in our participants. The internal consistency of the BAI in our sample was .90.

The BIS/BAS scale is a 20-item self-report questionnaire that assesses how people typically react to certain situations. This scale has four subscales: Behavioral Inhibition (BIS); and Behavioral Activation Reward Responsiveness (BAS–RR), Drive (BAS–Drive), and Fun-Seeking (BAS–Fun). Representative subscale items include: "If I think something unpleasant is going to happen, I usually get pretty 'worked up' " (BIS); "When I'm doing something well, I love to keep at it" (BAS–RR); "I'm always willing to try something new if I think it will be fun" (BAS–Fun); and "I go out of my way to get things I want" (BAS–Drive). Items were rated on a scale from 1 (*very true for me*) to 4 (*very false for me*). The internal consistencies of all of the BIS/BAS subscales in the present study were high (BIS = .78; BAS–RR = .80; BAS–Drive = .83; BAS–Fun = .69). The three BAS subscales were moderately intercorrelated (rs = .43–.60), and two of the BAS subscales were modestly inversely correlated with the BIS, BAS–Fun: $r(89) = -.29$; BAS–Drive: $r(89) = -.27$; both ps $< .05$. A number of investigators have found the BIS/BAS scales to yield a two-factor solution: behavioral inhibition and behavioral activation (e.g., Carver & White, 1994; Heubeck, Wilkinson, & Cologon, 1998; Jorm et al., 1999). Finally, the BIS/BAS scales have been found to possess good convergent and discriminant validity, with scores on the BIS scale being generally related to anxiety symptoms, negative affect, and neuroticism; and scores on the BAS scales typically related to positive affect and extraversion (e.g., Carver & White, 1994; Huebeck et al., 1998; Jorm et al., 1999).

Follow-Up Evaluation

All of the self-report questionnaires that were administered to participants at intake (Time 1) were readministered at Time 2, an average of 8 months after the Time 1 assessment (mode = 7.00 months, $SD = 2.55$ months). There were no differences between the depressed and nondepressed groups in the interval between the Time 1 and Time 2 assessments, $t(87) < 1$, $p > .05$.[2] Participants were reinterviewed at Time 2 using a modified form of the SCID designed to focus on and assess levels of depressive symptomatology in the past 8 weeks and to determine whether the participants who were diagnosed as depressed at Time 1 continued to meet diagnostic criteria for MDD at Time 2. At both time points, interviewers were blind to the participants' questionnaire data.

[1] Analyses using the reconstructed HDI and the original scoring method of the HDI yielded the same pattern of results.

[2] Results were unchanged when the longitudinal analyses were reconducted using the interval between the first and second sessions as a covariate.

Table 1
Demographic and Clinical Characteristics of Depressed and Nondepressed Participants

Variable	Depressed participants[a]		Nondepressed participants[b]	
	n (%)	*M* (*SD*)	*n* (%)	*M* (*SD*)
Gender, female	43 (69.4)		18 (66.7)	
Age		35.1 (11.5)		31.7 (11.9)
Race, White	45 (72.6)		15 (55.6)	
Years of education[c]		6.7 (1.4)		6.6 (1.4)
Annual income	$25,000–$50,000		$25,000–$50,000	
Recurrent MDD	51 (82.3)			
No. of prior episodes		4.6 (6.1)		
No. of MDD symptoms		6.3 (1.1)		
Duration of index MDE (months)		13.3 (23.1)		
GAF score at intake		54.3 (8.8)		87.0 (3.8)*
HDI score at intake		17.3 (8.6)		4.4 (4.2)*
Lifetime history of anxiety disorder	23 (37.1)			

Note. MDD = major depressive disorder; MDE = major depressive episode; GAF = Global Assessment of Functioning Scale; HDI = Hamilton Depression Inventory.
[a] $n = 61$. [b] $n = 27$. [c] Education was assessed on an 8-point scale, with higher numbers representing more education; scores of 6.6 and 6.7 reflect some college education.
* $p < .001$ for group differences; no other group differences were statistically significant.

Results

Demographic and Clinical Characteristics

Demographic characteristics for the MDD and NC participants are presented in Table 1. The two groups did not differ on any of the demographic variables assessed in this study: age, $t(87) = 1.35$; gender composition, $\chi^2(1, N = 89) < 1$; education level, $t(87) < 1$; income, $t(76) = 1.94$; percentage of Caucasian participants, $\chi^2(1, N = 89) = 2.48$, all ps $< .05$.[3] As expected, compared with the nondepressed controls, the depressed participants were significantly more depressed, as indexed by scores on the HDI, $t(86) = 16.00$, and were characterized by significantly poorer psychosocial functioning, as assessed by scores on the GAF, $t(85) = 16.37$, both ps $< .001$.

Cross-Sectional Analyses

Scores on the BAS and BIS subscales for the depressed and nondepressed participants are presented in Table 2. As predicted, compared to the nondepressed controls, at Time 1 the depressed participants had significantly higher scores on the BIS scale, $t(87) = 4.94$, $p < .001$, and significantly lower scores on all of the BAS scales: BAS–RR, $t(87) = 3.55$, $p < .001$; BAS–Drive, $t(87) = 3.23$, $p < .005$; BAS–Fun, $t(87) = 2.90$, $p = .005$.

We also hypothesized that, within the depressed group, scores on the BIS and BAS scales would be correlated positively and negatively, respectively, with severity of current depressive symptomatology and number of previous depressive episodes; and negatively and positively, respectively, with level of psychosocial functioning as indexed by GAF scores. Correlations of the BIS and BAS scales with measures of clinical functioning within the depressed group are presented in Table 3. Scores on the BIS scale were not correlated significantly with any of the clinical variables (HDI scores, number of depressive symptoms on the SCID, duration of the current episode, age at first onset of depression, total number of lifetime episodes of depression, BAI scores, or GAF scores).[4] In contrast, scores on all three of the BAS subscales were correlated significantly with measures of depression severity within the depressed group. As predicted, BAS–RR was correlated negatively with HDI, number of depressive symptoms on the SCID, and duration of current episode and was correlated positively with GAF scores, indicating that depressed participants with lower self-reported reward responsiveness were characterized by poorer global functioning. The BAS–Drive and, to a lesser extent, the BAS–Fun subscales showed a similar pattern of associations with these measures of depression severity.[5] Finally, none of the BAS scale scores was correlated significantly with scores on the BAI.

Longitudinal Analyses

Approximately 8 months after their initial interview, all participants were administered a follow-up structured diagnostic interview. Inadvertently, some of our participants were not administered the BIS/BAS scales at Time 2; Time 2 scores are available

[3] Some income data are missing because some participants did not respond to that question. Education level data are missing for 1 participant.

[4] Because we expected that BIS scores would be significantly related to level of anxiety, we also examined the correlations between BIS and BAI scores for the nondepressed controls and for the full sample. Although the BIS–BAI correlation was not significant within the sample of nondepressed controls, $r(26) = .05$, it was significant for the full sample, $r(88) = .23$, $p < .05$. Thus, with a larger sample and a bigger range of BAI scores, BIS was related to symptoms of anxiety.

[5] We also examined the relation of scores on the BIS and BAS scales to psychotropic medication status (use vs. nonuse). Only scores on the BAS–RR subscale were significantly related to use of psychotropic medication: Depressed participants who, at the initial interview, reported using psychotropic medications had lower BAS–RR scores than did depressed participants who reported not using medications, $t(60) = 2.28$, $p < .05$. None of the other Time 1 BIS/BAS subscales was associated with medication use. All significant effects reported in this study remained significant when we repeated the analyses using medication status as a covariate.

Table 2
Scores on the BIS and BAS Scales for the Depressed and Nondepressed Participants

Variable	Depressed participants[a]		Nondepressed participants[b]	
	Time 1 *M* (*SD*)	Time 2 *M* (*SD*)	Time 1 *M* (*SD*)	Time 2 *M* (*SD*)
BIS	24.0 (3.3)**	23.7 (3.5)	20.0 (3.8)**	19.0 (4.2)
BAS–RR	14.9 (3.5)**	15.9 (2.9)	17.7 (2.3)**	17.6 (2.1)
BAS–Drive	9.2 (2.5)*	10.3 (2.5)	11.9 (2.9)*	11.7 (2.7)
BAS–Fun-Seeking	10.7 (2.4)*	11.1 (2.4)	12.6 (2.0)*	12.3 (2.2)

Note. Sample sizes for the two time periods are the same for all variables within the depressed and nondepressed groups because we used listwise deletion of scores. Mean interval between Time 1 and Time 2 is 8 months. BIS = Behavioral Inhibition Scale; BAS = Behavioral Activation Scale; BAS–RR = BAS Reward Responsiveness.
[a] $n = 41$. [b] $n = 21$.
* $p < .005$. ** $p < .001$ for group differences at the Time 1 assessment.

for 66% (41 of 62) of the depressed participants and 78% (21 of 27) of the nondepressed control participants. With one exception, there were no significant differences on the demographic or clinical measures between those participants who were administered the Time 2 BIS/BAS questionnaires and those who were not. Within the depressed group, participants who were not administered the BIS/BAS questionnaire at Time 2 had significantly lower scores on the HDI at Time 1 than did participants who were administered the BIS/BAS questionnaire at Time 2, $t(59) = 2.13$, $p < .05$. Within the nondepressed control group, there were no significant demographic or clinical differences between participants who were administered the questionnaires and those who were not (all ps $> .10$). We used the smaller sample of participants who completed the BIS/BAS scales at both assessments in analyses that involved BIS/BAS scores at Time 2 and used the full sample of participants for all other analyses.

Stability of BIS and BAS. We predicted that scores on the BIS and BAS scales would be stable from Time 1 to Time 2. Consistent with this prediction, Time 1 and Time 2 BIS/BAS scale scores were strongly correlated within the depressed group (BIS = .75; BAS–RR = .70; BAS–Drive = .62; BAS–Fun = .73; all ps $<$ .001). Within the nondepressed group, Time 1 and Time 2 scores were also highly correlated (BIS = .84; BAS–RR = .81; BAS–Drive = .92; BAS–Fun = .74, all ps $< .001$). The only variable for which the test–retest correlation differed between depressed and nondepressed participants was BAS–Drive, which was found to be more stable among nondepressed than among depressed participants ($p < .01$). In sum, all analyses indicated that BIS and BAS scores were stable over time in both groups of participants.

To examine whether scores on the BIS and BAS scales were also stable over clinical state, we conducted a two-way (clinical status repeated over time) multivariate analysis of variance (MANOVA) on BIS/BAS scale scores within the depressed group. This analysis did not yield significant main effects for clinical status (improved vs. non-improved), $F(1, 39) = 2.72$, $\eta^2 = .07$, or time, $F(1, 39) = 4.02$, $\eta^2 = .09$, or a significant interaction of clinical status and time, $F(1, 39) < 1$, $\eta^2 = .01$, all ps $> .05$. To further assess whether changes in BIS and BAS scores were related to changes in clinical state, we computed correlations between changes in BIS and BAS scale scores and changes in HDI scores. In no case were changes in BIS and BAS scale scores associated with changes in self-reported depression severity: BIS, $r(41) = .26$; BAS–RR; $r(41) = .25$; BAS–Drive, $r(41) = -.13$; BAS–Fun, $r(41) = -.26$; all ps $> .10$, although it is important to acknowledge that change score analyses can attenuate observed correlations through increased measurement error (Cohen & Cohen, 1983). In sum, consistent with the high test–retest correlations for the BIS and BAS scales, depressed participants' scores on these scales at Time 1 did not different significantly from their scores at Time 2, regardless of their clinical status at Time 2. Moreover, changes in BIS and BAS scores from Time 1 to Time 2 were unrelated to changes in depressive symptoms.

Predictive utility of BIS and BAS. Finally, we hypothesized that depressed participants' scores on the BIS and BAS at Time 1 would predict their clinical status at Time 2 as well as symptom change from Time 1 to Time 2. BIS and BAS scores at Time 1 and Time 2 for initially depressed participants who were no longer diagnosed as depressed at Time 2, and for initially depressed participants who continued to meet diagnostic criteria for MDD at Time 2, are presented in Table 4. Because initial regression analyses using each predictor as a dependent variable indicated a significant shared variance among the predictors (e.g., Rs for BAS–RR and BAS–Drive = .76 and .74, respectively), we conducted separate regressions for the BIS and BAS scales predicting clinical status at Time 2. The results of these logistic regressions,

Table 3
Correlations Between the BIS and BAS Scales and Measures of Severity of Depression and Anxiety Within the Depressed Group at Time 1

Variable	BIS	BAS–RR	BAS–Fun	BAS–Drive
HDI score	.02	−.50**	−.34**	−.33*
Duration	−.02	−.28*	−.31*	−.32*
No. of episodes	−.06	−.16	.23	−.06
No. of symptoms	−.15	−.35*	−.23	−.23
BAI score	−.10	−.08	−.02	−.02
GAF	−.19	.44**	.23	.41**

Note. Listwise $N = 60$. BIS = Behavioral Inhibition Scale; BAS = Behavioral Activation Scale; BAS–RR = BAS Reward Responsiveness; BAS–Fun = BAS Fun-Seeking; HDI = Hamilton Depression Inventory; Duration = length of current major depressive episode; BAI = Beck Anxiety Inventory; GAF = Global Assessment of Functioning Scale.
* $p < .05$. ** $p < .01$.

Table 4
Mean BIS/BAS and HDI Scores of Depressed Participants Who Were and Who Were Not Still Diagnosed as Depressed at Follow-Up

	Participants no longer diagnosed as depressed[a]		Participants still diagnosed as depressed[b]	
Variable	Time 1 *M* (*SD*)	Time 2 *M* (*SD*)	Time 1 *M* (*SD*)	Time 2 *M* (*SD*)
BIS	24.2 (4.6)	23.3 (3.8)	24.0 (2.3)	23.9 (3.4)
BAS–RR	16.3 (2.3)	16.6 (2.1)	14.0 (3.9)	15.4 (3.2)
BAS–Drive	9.9 (2.0)	11.1 (2.0)	8.8 (2.8)	9.8 (2.6)
BAS–Fun	11.1 (2.2)	11.7 (2.1)	10.5 (2.5)	10.7 (2.5)
HDI score	17.2 (4.4)	6.8 (3.5)	18.7 (5.1)	16.6 (6.1)

Note. Total listwise $N = 41$ (due to missing Time 2 BIS/BAS data). Mean interval between Time 1 and Time 2 is 8 months. BIS = Behavioral Inhibition Scale; BAS = Behavioral Activation Scale; BAS–RR = BAS Reward Responsiveness; BAS–Fun = BAS Fun-Seeking; HDI = Hamilton Depression Inventory.
[a] $n = 15$. [b] $n = 26$.

presented in Table 5, indicated that higher scores at Time 1 on the BAS–RR and BAS–Drive subscales predicted clinical improvement at Time 2. Even after first entering Time 1 HDI scores to control for initial severity of depression, the BAS–Drive subscale continued to predict improvement at Time 2. Time 1 scores on the BIS scale and on the BAS–Fun subscale did not predict depression status at Time 2.

We also assessed the ability of BIS and BAS scores to predict change in depressive symptoms from Time 1 to Time 2. First, we assessed whether scores on the BIS and BAS scales at Time 1 predicted HDI score at Time 2, controlling for Time 1 HDI score (see Table 6). After entering Time 1 HDI score into the regression, BAS–RR scores at Time 1 significantly predicted depression severity at Time 2; BAS–Drive, BAS–Fun, and BIS scores at Time 1 did not predict change in HDI scores. Second, we assessed whether scores on the BIS and BAS subscales predicted number of interviewer-rated, SCID-based symptoms at Time 2, controlling for the number of symptoms at Time 1 (see Table 7). Both BAS–RR and BAS–Drive significantly predicted change in symptoms from Time 1 to Time 2, but BAS–Fun and BIS scores did not.

Table 5
The Relation of BIS and BAS to Depression Status Outcome at Time 2

Variable	Beta (raw)	Odds ratio	95% CI
BIS	.02	1.02	0.86–1.20
BAS–RR	.21*	1.24	1.03–1.48
BAS–Drive	.24*	1.27	1.03–1.55
BAS–Fun	.12	1.12	0.91–1.39
Controlling for Time 1 HDI score			
BAS–RR	.20	1.22	0.99–1.50
BAS–Drive	.22*	1.25	1.01–1.55

Note. Total listwise $N = 62$; still diagnosed depressed at Time 2, $n = 31$; no longer diagnosed depressed at Time 2, $n = 31$. BIS = Behavioral Inhibition Scale; BAS = Behavioral Activation Scale; CI = confidence interval; BAS–RR = BAS Reward Responsiveness; BAS–Fun = BAS Fun-Seeking; HDI = Hamilton Depression Inventory.
* $p < .05$.

Discussion

Recently researchers have demonstrated considerable interest in applying in Gray's (1973) conceptualization of behavioral activation and inhibition systems to understanding various forms of psychopathology (e.g., Depue & Collins, 1999; Meyer et al., 2001). In this study, we examined differences between clinically depressed and nondepressed individuals in behavioral activation and inhibition as well as the utility of these constructs in predicting recovery from depression. Consistent with Depue and Iacono's (1989) formulation, we found strong evidence of diminished BAS functioning in depression. Indeed, clinically depressed participants obtained lower scores on all three of the BAS subscales than did their nondepressed counterparts. Depressed participants also obtained significantly higher scores on the BIS scale than did the nondepressed controls. These data are consistent with findings reported by other investigators (e.g., Meyer et al., 1999, 2001) indicating that the depressed state is associated with low BAS and high BIS activation.

BIS and BAS are also hypothesized to be stable dispositions (Depue & Collins, 1999). Indeed, we found that levels of BIS and BAS exhibited impressive evidence of stability over time and over clinical state in both depressed and nondepressed participants. Our data replicate and extend work that has found high temporal stability for BIS/BAS in samples of unselected individuals (Sutton

Table 6
Predicting Time 2 HDI Scores From Time 1 BIS/BAS Scores Controlling for Time 1 HDI Scores

Variable	Beta (raw)	*t*	Model R^2	Partial *r*
BIS	.10	.41	.17	.05
BAS–RR*	−.73	−2.90	.28	−.36
BAS–Drive	−.35	−1.25	.19	−.16
BAS–Fun	−.21	−.64	.18	−.09

Note. Total listwise $N = 62$. HDI = Hamilton Depression Inventory; BIS = Behavioral Inhibition Scale; BAS = Behavioral Activation Scale; BAS–RR = BAS Reward Responsiveness; BAS–Fun = BAS Fun-Seeking; HDI = Hamilton Depression Inventory.
* $p < .01$.

Table 7
Predicting Time 2 SCID-Based Depressive Symptoms From Time 1 BIS/BAS Scores Controlling for Time 1 SCID-Based Depressive Symptoms

Variable	Beta (raw)	*t*	Model R^2	Partial *r*
BIS	.08	.66	.06	.09
BAS–RR*	−.35	−3.26	.20	−.40
BAS–Drive*	−.32	−2.59	.15	−.33
BAS–Fun	−.19	−1.26	.08	−.17

Note. Total listwise $N = 62$. SCID = Structured Clinical Interview for DSM–IV; BIS = Behavioral Inhibition Scale; BAS = Behavioral Activation Scale; BAS–RR = BAS Reward Responsiveness; BAS–Fun = BAS Fun-Seeking; HDI = Hamilton Depression Inventory.
* $p < .01$.

& Davidson, 1997) and in individuals with bipolar disorder (Meyer et al., 2001). Finally, the present data are also conceptually consistent with the results of studies using EEG demonstrating that both currently and formerly depressed subjects exhibit the same pattern of hemispheric laterality that differs reliably from that exhibited by nondepressed controls (e.g., Gotlib et al., 1998). The patterns of hemispheric asymmetry that have been found in these studies are consistent with the formulation that increased BIS and diminished BAS are stable characteristics of individuals who are predisposed to experience depression (see Henriques & Davidson, 2000). Considered collectively, therefore, these findings suggest that levels of BIS and BAS represent stable markers of a tendency to develop or experience depression.

In addition to examining the role of BIS and BAS as potential markers of a vulnerability to depression, we explored whether these constructs might also be useful in predicting the course of this disorder. Indeed, our assessment of the relation between levels of BIS and BAS and improvement in depression indicated that the BAS subscales significantly predicted the course of this disorder. More specifically, levels of the BAS Reward Responsiveness and Drive subscales assessed at Time 1 predicted both depression status at Time 2, after statistically controlling for initial severity of depression, and change in depressive symptoms from Time 1 to Time 2. These results suggest that levels of BIS and BAS may represent risk factors for the persistence of depression and are consistent with formulations positing that an underactive BAS, or lowered responsiveness to reward and decreased motivational drive to pursue rewarding stimuli, may cause and/or maintain depression (see Depue & Iacono, 1989). It is likely that depressed individuals who have particularly low levels of BAS are unresponsive to positive events or stimuli in their environment. This lack of responsiveness to the environment may serve to maintain a depressive episode by making it less likely that these depressed individuals will seek out positive stimulation or engage in pleasurable activities.

It is noteworthy that levels of behavioral inhibition were not found to predict the course of depression. In fact, levels of BIS were not related concurrently to levels of anxious or depressive symptomatology within the group of depressed participants in this study. Although our depressed sample was characterized by the presence of significant anxiety symptoms, our exclusion of individuals with diagnoses of current panic disorder or social phobia (because this study was part of a larger project using these exclusion criteria) led us to screen out a substantial number of cases with diagnosable anxiety conditions. It is possible, therefore, that scores on the BIS scale would have shown greater predictive utility in a sample of depressed participants in which there was a higher level of comorbid anxiety disorders. Interesting, however, and mitigating this concern, Meyer et al. (2001) did not screen out individuals diagnosed with anxiety disorders and obtained results virtually identical to those reported here: BIS scores did not predict the course of depressive symptoms in their sample, despite a concurrent association between BIS scores and depression severity. Considered together, the results of these investigations suggest that the BAS plays a more important role than the BIS in affecting the course of depression.

In extending these findings in future work, it will be important to administer other measures of temperament and personality to depressed individuals, such as the Eysenck Personality Inventory (Eysenck, 1964), the General Temperament Survey (Clark & Watson, 1999), and the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988). Administering these measures would permit an examination of the discriminant and convergent validity of the BIS and BAS scales relative to other major measures of temperament and personality. Perhaps more important, inclusion of these measures would also address the question of whether the BIS/BAS scales demonstrate incremental validity with respect to other measures that might also assess risk for depression.

Although we examined both concurrent and prospective associations between BIS/BAS functioning and depression in this study, it is important to note that there are a number of issues concerning these associations that we could not address. For example, we do not know whether the depressed and nondepressed participants would have differed with respect to levels of BIS and BAS premorbidly (i.e., before the onset of depression). Thus, it remains for future research to examine whether the depression-associated effects that we obtained in this study reflect the existence of premorbid differences between depressed and nondepressed individuals, are a function of current depressed mood, or are "scars" caused by from previous depressive episodes (e.g., Barnett & Gotlib, 1988; Rohde, Lewinsohn, & Seeley, 1990). Similarly, replication and extension of our results with larger samples would permit an examination of whether the factor structure of the BIS and BAS scales are comparable in clinical and nonclinical populations.

We also obtained relatively little information concerning the treatments received by participants over the course of follow-up. The results of analyses conducted using psychotropic medication status (taking vs. not taking medications) as a covariate indicated that medications did not alter the relation between BAS functioning and the course of depression (see Footnote 5). Nevertheless, we could not address whether various types of treatment differentially affected the association between BAS functioning and depression outcome. Furthermore, because this was in some ways a naturalistic study, patients were heterogeneous with respect to type and duration of treatment. Consequently, we could not assess the impact of type of treatment on levels of BIS and BAS. Future research would benefit from randomly assigning patients to treatment to examine whether BIS/BAS interacts with treatment type in predicting course of depression and outcome of treatment.

In closing, we would like to underscore a number of important strengths of this study. First, in contrast to other investigations of reward or inhibition in depression—which have typically used

nonclinical samples with elevated scores on a self-report measure of depressive symptoms (e.g., Henriques et al., 1994)—we studied a sample of individuals assessed with structured clinical interviews and diagnosed with MDD. Second, because our sample was obtained in part through local clinics and the outpatient psychiatry department at a large teaching hospital, a substantial proportion of our subjects were in treatment. This affords us excellent external validity to generalize to a treated outpatient population. The clinical relevance of the sample was underscored by the fact that most subjects were moderately impaired on the HDI and GAF measures, had recurrent major depression, and many had comorbid conditions. Finally, whereas most studies of BIS and BAS have been restricted to examining cross-sectional associations, we assessed the relation between these constructs and depression both concurrently and prospectively over a relatively long follow-up period.

The data from this study provide the best evidence to date that the BIS and BAS scales assess stable characteristics of depressed and nondepressed individuals. Moreover, BAS scores predicted the course of depression over an 8-month follow-up period, even after controlling for a number of potential confounding factors. Future research may profitably examine whether BIS and BAS are similarly stable in samples of participants who are at high risk for depression before they develop the disorder in order to determine the influence of mood state on levels of BIS and BAS. In addition, it will be important to extend our predictive findings and examine whether premorbid levels of BIS and BAS are related to the onset or the course of depression. These data could potentially provide an easily obtained and stable marker of a tendency to develop and maintain depression. In that case, BIS and BAS would be important constructs to assess in targeting people in greatest need for preventive services.

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