

Research report

Behavioral activation system deficits predict the six-month course of depression

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Abstract

Background: Behavioral activation system (BAS) deficits are hypothesized to increase risk for depression. This study tested the hypothesis that BAS deficits, measured with both self-report and electrophysiological methods, would predict the six-month course of depression.

Methods: 67 participants with major depressive disorder (MDD) with or without pre-existing dysthymia were assessed at baseline with Carver and White's [Carver, C.S., White, T.L., 1994. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS scales. *J. Pers. Soc. Psychol.* 67, 319–333.] BIS/BAS scales and resting EEG. The week-by-week course of their depressive symptoms was assessed six months later with the Longitudinal Interval Follow-up Evaluation (LIFE).

Results: Baseline self-reported BAS sensitivity predicted depression diagnosis (MDD or dysthymia) at follow-up, number of MDD symptoms at follow-up, average weekly level of depression, and time to recovery. These effects persisted after controlling for baseline clinical variables associated with a worse course. Baseline resting EEG alpha asymmetry did not significantly predict the course of depression.

Limitations: Although BAS sensitivity predicted the subsequent course of depression, we cannot determine whether it played a causal role in maintaining depression.

Conclusions: Lower self-reported BAS sensitivity predicts a worse course of depression but EEG asymmetries do not.

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1. Introduction

Several neuropsychological models of emotion and emotional disorders posit a central role for an approach system, or behavioral activation system (BAS), and a withdrawal system, or behavioral inhibition system (BIS) (Davidson, 1998a; Depue and Iacono, 1989; Fowles, 1988; Gray, 1994). Dispositional tendencies in

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BAS and BIS are manifest as affective styles, which may serve as risk factors for emotional disorders (Davidson, 1998a). An underactive BAS is hypothesized to be related to a reduction in positive affect (PA) and appetitive motivation (Davidson, 1992), increasing risk for depression (Fowles, 1988).

Considerable support for the role of BAS deficits in depression has been generated using both self-report and electrophysiological measures. Low PA, which can be viewed as a manifestation of diminished BAS activity, has been found to be a relatively specific characteristic of depression (Mineka et al., 1998). Additionally, some studies have demonstrated a direct link between depression and self-reported BAS sensitivity (Campbell-Sills et al., 2004; Kasch et al., 2002).

Electrophysiological research on the role of BAS deficits in depression is based on Davidson's (1992, 1998a) model hypothesizing that activity in the left prefrontal cortex is related to BAS, and activity in the right prefrontal cortex is related to BIS. Four studies using resting EEG alpha power as an inverse measure of cortical activity (Allen et al., 2004a; Shagass, 1972) have demonstrated a relationship between asymmetrical frontal activity and self-reported BAS sensitivity (Coan and Allen, 2003; Diego et al., 2001; Harmon-Jones and Allen, 1997; Sutton and Davidson, 1997). Davidson (1992, 1998a) also hypothesizes that a relative decrease in left frontal activity (i.e., diminished BAS) is a vulnerability marker for depression. There has been substantial support for this hypothesis, with several studies showing the predicted asymmetry in frontal regions among depressed participants compared to controls (e.g., Baehr et al., 1998; Gotlib et al., 1998; Henriques and Davidson, 1991; Schaffer et al., 1983; but not Reid et al., 1998). A few studies have also shown a reverse pattern of asymmetrical activity in posterior regions; that is, decreased right-relative-to-left posterior activity in depressed participants (Bruder et al., 1997; Kentgen et al., 2000; Schaffer et al., 1983).

The majority of studies in this area have focused on the association between BAS and a current or past history of depression. However, as BAS deficits are believed to reflect a trait-like affective style (Davidson, 1998a), it is reasonable to hypothesize that they would also be associated with a poorer course of depression.

To our knowledge, only two published studies have prospectively tested the relationship between BAS deficits and the course of unipolar depression. One found that self-reported BAS sensitivity predicted the presence, severity, and number of symptoms of major depressive disorder (MDD) at eight-month follow-up, controlling for baseline number of symptoms (Kasch et al., 2002). The other found that resting frontal EEG

alpha asymmetry did not significantly predict changes in MDD symptoms after eight weeks or MDD relapse after six months (Allen et al., 2004b). However, the latter findings may have been influenced by a relatively small ($N=29$) sample size.

The present study tested the hypothesis that both lower self-reported BAS scores and greater frontal EEG alpha asymmetry would predict a worse six-month course of depression. This study extends previous research in several important ways. First, the sample size was substantially larger than the previous electrophysiological study, and we examined posterior alpha asymmetry in addition to anterior. Second, the follow-up interviews included a more detailed assessment of the course of depression. In addition to assessing the number of MDD symptoms at follow-up, we obtained information on recovery, relapse, and average level of depressive symptoms during the follow-up period. Third, this is the first prospective study that has assessed the same depressed participants with both resting EEG and self-reported BIS/BAS.

2. Method

2.1. Participants

Seventy right-handed participants meeting DSM-IV (APA, 1994) criteria for MDD were assessed at baseline. Sixty-seven (95.7%) provided at least some data for the follow-up assessment. These 67 participants were aged 18–63 (M [SD]=34.64 [12.97]) and had completed 12–20 years of education (M [SD]=15.13 [2.44]); 44 (65.7%) were female; and 54 (80.6%) were Caucasian. Lifetime number of major depressive episodes (MDEs) ranged from 1 to 57 (M [SD]=5.28 [9.24]). Length of index MDE ranged from 1 to 581 months (M [SD]=33.63 [82.96]). Eighteen participants (26.9%) also met criteria for antecedent dysthymia. Twenty participants (29.9%) met criteria for a current comorbid Axis I anxiety disorder. Other current Axis I disorders were rare. Eighteen participants (26.9%) were recruited from an ongoing clinical trial, and the others were recruited with fliers posted in local outpatient treatment clinics and around the community. In all, 42 participants (62.7%) were receiving treatment at baseline. All participants provided informed consent at baseline and again at follow-up.

2.2. Measures

2.2.1. Baseline clinical assessment

Participants were interviewed with the Structured Clinical Interview for DSM-IV (SCID; First et al.,

1995). The diagnostic interviews were conducted by Masters-level clinicians who had completed a formal training course on the SCID and had several years of experience using it. In previous studies, our group has shown high levels of interrater reliability for the SCID (Keller et al., 1995).

Participants also completed the Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) scale (Carver and White, 1994). The BIS/BAS scale is a 20-item self-report questionnaire that consists of a Behavioral Inhibition (BIS) subscale and three Behavioral Activation subscales: Reward Responsiveness (BAS-RR), Drive (BAS-Drive), and Fun Seeking (BAS-Fun). The sum of the three BAS subscales (BAS Total) is often used as an indication of overall BAS sensitivity. Reliabilities (alphas) for this sample were .78, .80, .77, and .70 for BIS, BAS-RR, BAS-Drive, and BAS-Fun, respectively.

2.2.2. Resting EEG

EEG was collected from participants during six 1-min periods in which they were instructed to rest quietly in a comfortable chair with eyes open or closed, counter-balanced. EEG was recorded in a sound-attenuated booth. Electrodes were placed according to the International 10-20 system using a stretch-lycra electrode cap (Electro Cap International, Inc.; tin electrodes). Data were also recorded from the right earlobe (A2), enabling computation of an off-line digitally derived, 'linked ear' reference. Electrodes were placed at right supra- and infra-orbital sites to monitor for eye blinks and vertical eye movements (VEOG), and right- and left-outer canthi to monitor horizontal eye movements (HEOG). Electrode impedances were under 5000 Ω , and homologous sites (e.g., P3/P4) were within 1500 Ω of each other. The electrode cap was connected to a chest harness in order to reduce the likelihood of movement. Data were recorded through a Grass Neurodata acquisition system at a gain of 10K (5K for eye channels) with a bandpass of 1–30 Hz. A PC-based EEG acquisition system (Neuroscan version 4.2) acquired and digitized the data continuously at a rate of 1K Hz.

Continuous EEG was segmented into consecutive 1.024 s epochs every 0.512 s (50% overlap). This yielded approximately 180 epochs per each resting EEG condition. Data were then examined for evidence of amplifier saturation. After referencing to a linked ear reference offline and applying a baseline correction, epochs contaminated by blinks, eye movements, and movement-related artifacts were excluded from analyses. The EEG was tapered over the entire 1.024 s epoch by a Hanning window to suppress spectral side lobes.

Artifact-free data that have been attenuated at the beginning and end of an epoch are recovered in adjacent (overlapping) epochs.

Power spectra were computed off-line from EEG data using a Fast Fourier Transform. An examination of the topography of the power spectra indicated a distinct alpha peak at approximately 10 Hz that was maximal in posterior and medial electrodes, relative to anterior and lateral electrodes, respectively, and greater in the eyes closed than eyes open condition. The average absolute alpha power was computed for each electrode site and log transformed to normalize the data. The alpha band was defined as 8–13 Hz. This range was empirically validated by assuring that the alpha peak was approximately centered in this band for every subject.

Data from eyes open and closed conditions were averaged and asymmetry scores were calculated by subtracting left from right homologous electrodes (e.g., F4–F3). Additionally, pooled estimates were created by averaging ipsilateral electrodes within region and subtracting pooled left from pooled right scores (i.e., [pooled F4, F8]–[pooled F3, F7] for anterior, [pooled P4, P8]–[pooled P3, P7] for posterior). Analyses using an average reference produced identical results, as did analyses using a complete ANOVA model. Due to space limitations, only results using the linked ear reference and based on simple difference scores are reported here.

2.2.3. Follow-up assessments

Participants were interviewed by telephone approximately six months (M [SD]=27.87 [3.98] weeks) after completing the baseline assessment. Telephone interviews have been shown to be comparable to in-person interviews for assessment of Axis I disorders (Rohde et al., 1997). The follow-up assessments included an adaptation of the Longitudinal Interval Follow-up Evaluation (LIFE; Keller et al., 1987). The LIFE is a semistructured interview that assesses the longitudinal course of DSM-IV psychiatric disorders, including treatment information, on a week-by-week basis. The follow-up assessments were conducted by a Master's-level clinician who had been trained in the use of the LIFE. Our group has previously shown high levels of interrater reliability for the LIFE (Klein et al., 2000).

The course of MDD and dysthymia were recorded in weekly psychiatric status ratings (PSRs), which indicate whether a patient met full criteria for the disorder or was subthreshold or asymptomatic. The MDD and dysthymia PSRs were combined into weekly depression PSRs defined as follows: 4=meets MDD criteria, 3=meets dysthymia (but not MDD) criteria, 2=subthreshold MDD or dysthymia symptoms, and 1=absent or

Table 1
Correlations of baseline BIS/BAS scales with baseline resting EEG alpha asymmetry in anterior and posterior regions

	BIS	BAS-RR	BAS-Drive	BAS-Fun	BAS-total	BAS-BIS
F4–F3	.29*	.30*	.03	.12	.19	-.08
F8–F7	.14	.13	-.01	.03	.06	-.06
Pooled anterior	.22	.21	.01	.07	.12	-.08
P4–P3	-.01	.11	.03	.28*	.18	.14
P8–P7	.01	-.04	-.02	.23	.07	.05
Pooled posterior	.01	.01	-.01	.27*	.11	.08

Total listwise $N=62$. BIS = Behavioral Inhibition System; BAS-RR = Behavioral Activation System Reward Responsiveness; BAS-Fun = Fun-seeking. BAS-BIS = z -transformed BAS Total minus z -transformed BIS; Pooled Anterior=(pooled F4, F8)–(pooled F3, F7); Pooled Posterior=(pooled P4, P8)–(pooled P3, P7).

* $p < .05$.

minimal depressive symptoms. Our group has used this scale previously (see Klein et al., 2000). Per LIFE convention, recovery was defined as at least eight consecutive weeks with absent or minimal symptoms.

3. Results

Table 1 shows the correlations between the BIS/BAS scales and resting EEG alpha asymmetry. Higher scores on BIS and BAS-RR were associated with greater relative left frontal activity (F4–F3), and higher BAS-Fun was associated with greater relative left posterior activity (P4–P3 and pooled posterior asymmetry).

Table 2
Predictive utility of baseline clinical variables

Baseline variables	r			HR (95% CI)
	Depression diagnosis at follow-up	Number MDD symptoms at follow-up	Average weekly PSR	Time to recovery
Chronicity	.32**	.16	.09	0.52 (0.23–1.15)
Comorbid anxiety	.24*	.23	.35**	0.36 (0.12–1.04)
Number of MDD symptoms	.13	.25*	.13	0.80 (0.57–1.13)
Lifetime number of MDEs	-.10	-.04	-.11	1.02 (0.98–1.05)
Age of onset of depression	-.01	-.03	-.03	1.01 (0.98–1.05)

Total listwise $N=65$. MDD = Major Depressive Disorder; PSR = Psychiatric Status Rating; HR = Hazard Ratio; CI = Confidence Interval; MDE = Major Depressive Episode.

* $p < .05$, ** $p < .01$.

At 6-month follow-up, participants' number of MDD symptoms ranged from 0 to 9 (M [SD]=2.71 [2.42]). Average of weekly PSRs over the follow-up period ranged from 1.23 to 4.00 (M [SD]=2.72 [0.83]). Twenty-five participants (37.3%) recovered at some point during the follow-up period, only one of whom later had another MDE within the follow-up period. Psychotherapy use was associated with a worse course, consistent with previous work showing that patients with chronic forms of depression use mental health services at higher rates (McFarland and Klein, 2005). Pharmacotherapy use was not related to course. Results of analyses involving baseline predictors are reported without considering treatment use because the results did not change when treatment use was controlled statistically.

Table 2 summarizes the predictive utility of a number of baseline clinical variables. Chronicity was defined dichotomously, as previous research has shown that subtypes of chronic depression do not differ meaningfully

Table 3
Predictive utility of BIS/BAS scale

	r			HR (95% CI)
	Depression diagnosis at follow-up	Number MDD symptoms at follow-up	Average weekly PSR	Time to recovery
BIS	.02	.09	.19	0.94 (0.85–1.04)
BAS-RR	-.35**	-.31*	-.29*	1.12 (0.95–1.31)
BAS-Drive	-.33**	-.19	-.26*	1.16 (0.98–1.37)
BAS-Fun	-.22	-.18	-.30*	1.15 (0.98–1.36)
BAS total	-.38**	-.29*	-.36**	1.08* (1.00–1.17)

Controlling for baseline clinical variables^a

	Partial r		
	Depression diagnosis at follow-up	Number MDD symptoms at follow-up	Average weekly PSR
BAS-RR	-.31*	-.28*	-.29*
BAS-Drive	-.28*	-.16	-.23
BAS-Fun	-.21	-.14	-.32**
BAS total	-.34**	-.25*	-.36**

Total listwise $N=65$. MDD = Major Depressive Disorder; PSR = Psychiatric Status Rating; HR = Hazard Ratio; CI = Confidence Interval; BIS = Behavioral Inhibition System; BAS-RR = Behavioral Activation System Reward Responsiveness; BAS-Fun = Fun-seeking.

* $p < .05$, ** $p < .01$.

^a Controlling for those baseline clinical variables with significant bivariate correlations with the outcome measure of interest.

Table 4
Predictive utility of resting EEG alpha asymmetry in anterior and posterior regions

	<i>r</i>		HR (95% CI)	
	Depression diagnosis at follow-up	Number MDD symptoms at follow-up	Average weekly PSR	Time to recovery
F4–F3	.01	.01	–.14	2.26 (0.02–275.93)
F8–F7	.16	.10	.10	0.19 (0.01–2.91)
Pooled anterior	.12	.07	.01	0.22 (0.01–10.92)
P4–P3	.13	.02	–.06	1.08 (0.14–8.64)
P8–P7	.19	.18	.15	0.37 (0.11–1.22)
Pooled posterior	.18	.13	.09	0.38 (0.07–1.99)

Total listwise $N=60$. MDD = Major Depressive Disorder; PSR = Psychiatric Status Rating; HR = Hazard Ratio; CI = Confidence Interval; Pooled Anterior=(pooled F4, F8)–(pooled F3, F7); Pooled Posterior=(pooled P4, P8)–(pooled P3, P7).

(McCullough et al., 2003). Participants with chronic forms of depression at baseline were more likely to meet criteria for a depression diagnosis at follow-up. Participants with a current comorbid anxiety disorder at baseline were more likely to meet criteria for a depression diagnosis at follow-up and have higher average PSRs. Baseline number of MDD symptoms predicted number of MDD symptoms at follow-up. None of the baseline clinical variables predicted time to recovery in Cox proportional hazards models.

Table 3 summarizes the predictive utility of the BIS/BAS subscales. BIS did not significantly predict any outcome variable. BAS-RR predicted diagnosis and number of MDD symptoms at follow-up and average PSR. BAS-Drive predicted diagnosis at follow-up and average PSR. BAS-Fun predicted average PSR. BAS Total predicted each of the four outcome variables.

To assess the “incremental” predictive utility of BAS, we computed partial correlations between BAS subscales and outcome variables, controlling for the baseline clinical variables that had predicted each outcome variable. As shown in the bottom half of Table 3, BAS-RR continued to predict diagnosis and number of MDD symptoms at follow-up and average PSR. BAS-Drive continued to predict diagnosis at follow-up but no longer predicted average PSR. BAS-Fun continued to predict average PSR. BAS Total continued to predict diagnosis and number of MDD symptoms at follow-up and average PSR. Because none of the baseline clinical variable significantly predicted time to recovery, it was not necessary to run additional Cox proportional hazards models.

Table 4 summarizes the predictive utility of resting EEG alpha asymmetry. None of the asymmetry values predicted any outcome measure.

4. Discussion

Our results partially supported the hypothesis that BAS deficits predict a worse six-month course of depression. Self-reported BAS sensitivity predicted likelihood of depression diagnosis at follow-up, number of MDD symptoms at follow-up, average weekly PSR, and time to recovery. These results replicate and extend the only other published prospective study of self-reported BAS sensitivity and the course of unipolar depression (Kasch et al., 2002). We found that BAS predicted not only the presence of depression and number of MDD symptoms at follow-up, but also the average weekly level of depression and the time to recovery from depression. Additionally, we found that these results persisted after controlling not only for baseline number of MDD symptoms, but for baseline chronicity and anxiety comorbidity as well. Our results also replicate and extend previous findings that self-reported BIS does not significantly predict the course of depressive symptoms (Kasch et al., 2002).

We also found that resting EEG alpha asymmetry did not significantly predict the six-month course of depression. These results replicate and extend the only other published prospective study of EEG alpha asymmetry and the course of depression (Allen et al., 2004b). We found, in a much larger sample, that neither anterior nor posterior asymmetry was significantly correlated with the presence of depression or number of MDD symptoms at follow-up, average weekly level of depression, or time to recovery from depression.

We also failed to find a strong association between self-report and EEG measures of BAS, which have been correlated in previous studies. However, the three studies reporting the strongest associations all used nondepressed participants (Coan and Allen, 2003; Harmon-Jones and Allen, 1997; Sutton and Davidson, 1997). The only previous study that used depressed participants (Diego et al., 2001) reported the same pattern of correlations that we did; namely, midfrontal asymmetry was modestly correlated with BAS-RR but not significantly correlated with BAS-Drive or BAS-Fun. Thus, it appears the correlation between self-report and electrophysiological measures of BAS is weaker in depressed samples, perhaps due to reduced variability in both measures among depressed participants.

This study had several important strengths, including a prospective design, consideration of relevant clinical

variables, and assessment with both self-report and electrophysiological measures. It also had some important limitations. First, although we found that self-reported BAS deficits predicted the subsequent course of depression, we cannot determine whether these deficits played a causal role in the maintenance of depression. If BAS deficits do contribute to the maintenance of depressive episodes, it will be important to determine the neurobiological and psychosocial processes that mediate this association. Second, although much of the variance in frontal asymmetries in resting EEG reflect stable trait variance, state influences also play a role (Hagemann et al., 2002). Averaging over several assessments would provide a more reliable measure (Davidson, 1998b). Third, the follow-up was naturalistic and treatment was uncontrolled. Finally, longer term follow-ups are needed to examine the relationship between BAS deficits and relapse and recurrence.

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