

Neurocognitive components of the behavioral inhibition and activation systems: Implications for theories of self-regulation

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Abstract

We examined the neurocognitive correlates of the Behavioral Inhibition and Behavioral Activation Systems (BIS/BAS) in an effort to clarify ambiguities concerning interpretations of BIS as reflecting inhibition versus avoidance. We hypothesized that self-reported BIS should relate to neural mechanisms associated with conflict monitoring, whereas self-reported BAS should be associated with neural correlates of approach motivation. Consistent with these predictions, higher self-reported BIS was uniquely related to the N2 event-related potential on No-Go trials of a Go/No-Go task, linking BIS with conflict monitoring and sensitivity to No-Go cues. Higher BAS was uniquely related to greater left-sided baseline frontal cortical asymmetry associated with approach orientation. Implications for theories of self-regulation involving conflict monitoring, cognitive control, and approach/avoidance motivation are discussed.

Descriptors: Motivation, Behavioral inhibition system, Behavioral activation system, Approach, Inhibition, ERP, EEG, Error-related negativity, Conflict monitoring, Frontal asymmetry

A central question in psychophysiology research concerns how individual differences in neurobiological processes are manifested in motivation and personality. One dominant view suggests that the building blocks of these psychological constructs correspond to two general systems for orchestrating adaptive behavior (Carver & White, 1994; Fowles, 1980). The first system functions to halt ongoing behavior while processing potential threat cues and is referred to as the Behavioral Inhibition System (BIS; e.g., Gray, 1975, 1990; see also the aversive/defensive system; Lang, Bradley, & Cuthbert, 1990). A second system is believed to govern the engagement of action and has been referred to as the Behavioral Activation System (BAS; Fowles, 1980, 1988) or, alternatively, the Behavioral Approach System (Gray, 1982) or the Behavior Facilitation System (Depue & Collins, 1999; Depue & Iacono, 1989). Although conceptions of the BIS and BAS were originally developed from behavioral neuroscience research conducted primarily on nonhuman animals (Gray, 1972), the BIS/BAS model has proven to be a useful framework for understanding motivation and personality as they apply to a range of normal and abnormal human behaviors. More recently, the development of reliable self-report measures

of BIS and BAS sensitivity has facilitated the translation of BIS/BAS from animal models to the study of human social behavior. Yet despite enthusiasm for the BIS/BAS model, there is a notable lack of consensus in the way that measures of these constructs—particularly BIS—are interpreted in the literature. The purpose of the present research is to clarify the constructs of BIS and BAS in humans by discerning aspects of their underlying neurocognitive mechanisms.

The BIS|BAS Model of Personality

The BIS and BAS were proposed as a neuropsychological framework for understanding how mechanisms for behavioral regulation relate to personality and, by extension, psychological dysfunction (e.g., Fowles, 1980; Gray, 1972, 1982, 1987; Gray & McNaughton, 2000; Pickering & Gray, 1999). The BIS is conceptualized as an attentional system that is sensitive to cues of punishment, nonreward, and novelty and that functions to interrupt ongoing behavior in order to facilitate the processing of these cues in preparation for a response. “Inhibition” in the BIS framework refers to the abrogation of behavior in reaction to an expected or unexpected stimulus (Fowles, 2000; Yu & Dayan, 2005), rather than the deliberative process of inhibitory control (Aron, Robbins, & Poldrack, 2004). High BIS activation is associated with enhanced attention, arousal, vigilance, and anxiety, and very strong BIS corresponds to anxiety-related disorders (e.g., Fowles, 1988; Quay, 1988) whereas very weak BIS relates to primary psychopathy (Newman, MacCoun, Vaughn, & Sadeh, 2005).

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The complementary system to the BIS is the BAS. The BAS is conceptualized as a motivational system that is sensitive to signals of reward, nonpunishment, and escape from punishment and that is important for engaging behavior toward a reward or away from a threat. BAS has been associated with feelings of optimism, joy, and aggression (Gable, Reis, & Elliot, 2000; Gray & McNaughton, 2000; Wingrove & Bond, 1998). Extreme levels of BAS have been linked to impulsivity disorders (Wallace, Newman, & Bachorowski, 1991), bipolar disorder (Depue & Iacono, 1989), attention-deficit/hyperactivity disorder (Mitchell & Nelson-Gray, 2006), and secondary psychopathy (Newman et al., 2005). These psychological correlates are generally associated with the goal-driven activation of behavior, as compared with the more reflexive fight/flight system that is believed to engage behavioral responses to immediate threats (Gray & McNaughton, 2000).

Taken together, the original conceptions of BIS and BAS correspond to systems for stopping and going: *stopping* when a potential threat or reward is detected and *going* once a plan for action is devised (Demaree, Everhart, Youngstrom, & Harrison, 2005). Although the BIS/BAS model concerns behavioral regulation, researchers have become interested in how these constructs are manifested in individual differences, emotions, and psychological dysfunction. However, as we highlight in the next section, the translation of BIS and BAS sensitivity to self-reportable states and traits has created new ambiguities regarding the meaning of the core constructs.

Ambiguities in Measurement and Interpretation of BIS and BAS

Several efforts have been made to develop self-report scales to assess BIS and BAS sensitivity (MacAndrew & Steele, 1991; Torrubia & Tobena, 1984; Wilson, Gray, & Barrett, 1990) and related constructs (e.g., Cloninger, 1987; Gable, 2006). The most popular and well-validated measure of BIS and BAS sensitivity was developed by Carver and White (1994). Following closely from Gray's model (1982), items included in the BIS scale focus on anxiety in response to a threatening situation, such as "I worry about making mistakes" and "I feel pretty worried or upset when I think or know somebody is angry at me." Items included on the BAS scale emphasize activated approach toward a goal, such as "When I want something, I usually go all-out to get it" and "I crave excitement and new sensations." It is notable that items assessing BAS sensitivity focus on reward-related tendencies and do not include items associated with escape from punishment or threat. In this way, Carver and White's (1994) BAS scale focuses only on the approach aspects of BAS.

Although the constructs of BIS and BAS were clearly articulated in behavioral and physiological terms by Gray, Fowles, and their colleagues, the experiential meaning of these constructs in terms of self-reported states and traits has been less clear. The primary source of ambiguity concerns whether BIS is associated with the tendency to halt ongoing behavior or to engage in active avoidance behavior in response to a potential threat. On one hand, much research has operationalized BIS in terms of behavioral inhibition, as originally suggested by Gray (e.g., Arnett & Newman, 2000; Casada & Roache, 2005; Cools et al., 2005; Fowles, 1980, 1988; Gomez & Gomez, 2002; Harmon-Jones & Allen, 1997; Hewig, Hagemann, Seifert, Naumann, & Bartussek, 2006; Keltner, Gruenfeld, & Anderson, 2003; Mitchell & Nelson-Gray, 2006; Monteith, 1993; Monteith, Ashburn-Nardo, Voils, & Czopp, 2002; Newman et al., 2005; Patterson, Kosson, & Newman, 1987; Patterson & Newman, 1993). On the other

hand, many researchers have described BIS in terms of behavioral avoidance (e.g., Beer, 2002; Blair, Peters, & Granger, 2004; Cooper, Shapiro, & Powers, 1998; Elliot, Gable, & Mapes, 2006; Elliot & Thrash, 2002; Gable, 2006; Gable et al., 2000; Heimpel, Elliot, & Wood, 2006; Mann, Sherman, & Updegraff, 2004; Sherman, Mann, & Updegraff, 2006; Sutton & Davidson, 1997; Thrash & Elliot, 2003; Updegraff, Gable, & Taylor, 2004; Updegraff, Sherman, Luyster, & Mann, 2007). Thus, the literature appears to be in disagreement regarding the conceptual meaning of BIS. As a result, ambiguities exist in the literature regarding the roles of inhibition and avoidance tendencies in emotion, motivation, and behavior.

Clarifying BIS and BAS Constructs with Psychophysiology

BAS and frontal cortical asymmetry. Theoretical distinctions between BIS and BAS in humans have been addressed to some extent by the neurophysiological substrates proposed to underlie each process. For instance, BAS is organized primarily by the dopaminergic neurotransmitter system, which is believed to promote goal-related behavior in response to anticipated reward or punishment (Gray & McNaughton, 2000; Schultz, Dayan, & Montague, 1997). In humans, this function is associated with striatal dopamine projections primarily to areas of lateral and orbital regions of the prefrontal cortex (PFC; Alexander, DeLong, & Strick, 1986; Lehericy et al., 2004; Rolls, 2000). Activity in the dorsolateral PFC, as measured using electroencephalography (EEG; Pizzagalli, Sherwood, Henriques, & Davidson, 2005), has been linked to constructs such as approach versus avoidance (or withdrawal) motivational orientation (Harmon-Jones & Allen, 1998), promotion versus prevention regulatory focus (Amodio, Shah, Sigelman, Brazy, & Harmon-Jones, 2004), and positive versus negative emotion states (e.g., Davidson, 1992). Specifically, greater left-sided frontal asymmetry has been associated with approach-related motivation and emotions, whereas greater right-sided activity has been associated with avoidance-related motivation and emotions (Harmon-Jones, 2003a), although the link between frontal asymmetry and avoidance is somewhat ambiguous (Coan & Allen, 2004). These patterns of frontal asymmetry are believed to reflect asymmetric dopamine signaling from the striatum (Berridge, España, & Stalnaker, 2003). In bipolar patients, increased left (vs. right) PFC activity has been linked to mania, whereas decreased left-sided PFC asymmetry has been linked to depression in bipolar patients (Harmon-Jones et al., 2002). In other research, abnormalities in this frontal dopaminergic system have been linked with attention-deficit/hyperactivity disorder, which may be characterized by problems in initiating and sustaining goal-directed behavior (Aron & Poldrack, 2005). Hence, there is a strong precedence relating BAS to patterns of PFC activity associated with approach-motivated behavior in the anatomical and functional neuroscience literatures.

Building on this link between frontal EEG asymmetry and approach/avoidance motivation, several researchers have used frontal EEG as a physiological method for examining the correspondence of BAS and BIS with approach versus avoidance orientations (Harmon-Jones & Allen, 1997; Hewig et al., 2006; Sutton & Davidson, 1997; see also Fowles, 1988). This body of research provides general support for the view that greater left- (vs. right-) sided frontal cortical activity is associated with high BAS scores, at both the state and trait levels of analysis, suggesting that BAS is related to the approach/avoidance dimension

of motivation (Harmon-Jones, 2003b; for a review, see Coan & Allen, 2003). This finding is consistent with theorizing by Gray (1982) and Fowles (1980). By contrast, consensus in this literature is that BIS is not directly associated with approach/avoidance motivational orientation or with frontal EEG asymmetry (Coan & Allen, 2003; Hewig et al., 2006; but see Sutton & Davidson, 1997).

Proposed neurocognitive correlate of BIS. To date, research has not identified a neurocognitive correlate of BIS in humans. According to Gray (1982; Gray & McNaughton, 2000), BIS is associated with a neural circuit organized by monoamine neurotransmitter systems, including noradrenergic and serotonergic networks and their associated neural structures. The primary source of norepinephrine is the locus coeruleus, located in the brain stem. In turn, the locus coeruleus has modulatory noradrenergic effects on neural structures that function to detect potential threats and expectancy violations, one of which is the anterior cingulate cortex (ACC; Aston-Jones & Cohen, 2005; Aston-Jones, Foote, & Segal, 1985).

In the cognitive neuroscience literature, the ACC is believed to serve a *conflict-monitoring* function, whereby it monitors for conflict among cognitions and action tendencies and recruits additional mechanisms for top-down control to resolve such conflicts (Botvinick, Braver, Barch, Carter, & Cohen, 2001; see also Amodio & Frith, 2006). Consistent with this interpretation, ACC activity has been associated with sensitivity to expectancy violations, competing behavioral tendencies, and belief-discrepant behavior (Amodio, Harmon-Jones, et al., 2004; Carter et al., 1998; Hajcak, Moser, Yeung, & Simons, 2005; for a review, see Botvinick, Cohen, & Carter, 2004). The conflict-monitoring function of the ACC is conceptually similar to Gray's (1987; Gray & McNaughton, 2000) description of BIS as being sensitive to unexpected stimuli and conflicts between competing responses (e.g., competing approach vs. avoidance tendencies), and conflict-related ACC activity has been shown to be amplified for individuals high in forms of negative affectivity, a characteristic of high dispositional BIS sensitivity (Luu, Collins, & Tucker, 2000). Furthermore, fMRI research has dissociated the conflict-monitoring function linked to the ACC from a separate, but coordinated, PFC-related mechanism for engaging regulatory control in response to conflict (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 1998; Kerns et al., 2004). This dissociation between ACC and PFC mechanisms for self-regulation is consistent with theoretical distinctions between BIS and BAS.

When the evidence from multiple literatures is considered together, theory and past research suggest that the BIS should be associated with the process of detecting response conflicts in humans, vis-à-vis its ACC substrate, which in turn is associated with the interruption of action. Although research has not yet examined the relation between BIS and conflict monitoring, evidence of such a relationship would link BIS to more recent neurocognitive models of self-regulation and would provide evidence that BIS is appropriately conceived of as a system for evaluating a potential response conflict or threat rather than a predisposition for avoidance.

Overview and Hypotheses

We sought to clarify interpretations of BIS and BAS by designing a double-dissociation study to disentangle the underlying mechanisms of inhibition and of motivational orientation (i.e., ap-

proach vs. avoidance). Based on theory and past research, we identified separate neural substrates associated with inhibition and motivational orientation and examined the unique relationships between the activity of these systems and self-reported levels of BIS and BAS. We predicted that BAS would be uniquely associated with neural correlates of approach/avoidance tendencies, whereas BIS would be uniquely associated with the neural index of response-conflict sensitivity. However, if researchers who have conceptualized BIS as an avoidance system are correct, we would expect both BIS and BAS to be associated with the neural correlates of approach/avoidance orientation and neither to be linked with the neural index of response-conflict sensitivity.

To test our hypotheses, we chose two assessments that provided the clearest theoretical links to behavioral activation and behavioral inhibition, based on past research. We expected that BAS would be associated with baseline frontal cortical asymmetry, as measured using EEG. To test our hypotheses regarding the role of BIS in behavioral inhibition, it was important to use a behavioral task that required the abrogation of a prepotent response tendency. The Go/No-Go task was selected because successful performance requires sensitivity to infrequent cues that a response must be withheld—a process that should rely on the BIS. We predicted that BIS would be associated with conflict-related activity of the ACC, as assessed using event-related potential (ERP) measures during the Go/No-Go task. Successful inhibition on No-Go trials in the Go/No-Go task has been shown to elicit a negative voltage shift at the frontocentral scalp region (Falkenstein, Hoormann, & Hohnsbein, 1999; Kok, 1986). This voltage shift, referred to as the *No-Go N2* component, typically peaks approximately 250 ms following a No-Go stimulus. The No-Go N2 has been shown to arise from a neural generator located in the dorsal ACC (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; van Veen & Carter, 2002) and has been interpreted as reflecting conflict-related response monitoring (Yeung, Botvinick, & Cohen, 2004). In this context, the No-Go N2 reflects a reaction to the No-Go stimulus as a cue to change one's prepotent behavioral response, and therefore we expected that No-Go N2 amplitudes would be associated with BIS sensitivity.

A second ERP component, the error-related negativity (ERN), is typically observed in conjunction with a response error (Gehring, Goss, Coles, Meyer, & Donchin, 1993). This component has also been associated with the conflict-monitoring mechanism, as it represents a strong conflict between an intended response and the commission of the conflicting behavior (Yeung et al., 2004). Additionally, the ERN has been shown to arise from a dorsal ACC neural generator in several studies, much the same as the N2 (e.g., Dehaene, Posner, & Tucker, 1994; Nieuwenhuis et al., 2003; van Veen & Carter, 2002). In the context of the Go/No-Go task, the ERN reflects a reaction to the execution of an unintended behavioral response. Although the cause of conflict underlying the ERN and N2 responses are somewhat different, the underlying mechanism is believed to be the same, and thus the ERN is also expected to be associated with individual differences in BIS sensitivity.

Method

Participants and Procedure

Forty-eight undergraduate students or recent graduates (32 women, 16 men) participated individually in exchange for \$30 or for extra course credit. Upon arrival at the laboratory, partic-

Table 1. Zero-Order Correlations among Key Variables

	1	2	3	4	5
1. BIS					
2. BAS	-.03				
3. Frontal asymmetry	-.11	.36*			
4. No-Go N2	-.41**	.09	-.22		
5. ERN	-.35*	.03	-.11	.45**	
6. No-Go error rate	-.14	.10	.11	.08	.66**

Note. BIS: Behavioral Inhibition System; BAS: Behavioral Activation System; ERN: error-related negativity.

* $p < .05$; ** $p < .01$.

Participants provided their informed consent and were prepared for physiological recording. The participant was seated in a dimly lit, soundproofed room in a comfortable chair, approximately 1 m from a computer monitor. The experimenter explained that baseline EEG recordings would be made, after which the participant would complete a computer task while EEG was recorded. Each session lasted approximately 2 h. Data from eight participants were excluded because of excessive EEG artifact (4), failure to follow task instructions (2), or because scores on one or more measures were considered an outlier ($SD > 3$; 2).

BIS/BAS questionnaire. BIS and BAS were assessed using Carver and White's (1994) questionnaire, with BIS items (7) and BAS items (13) intermixed. Average scores were computed separately for the BIS and BAS scales. It is notable that Carver and White's (1994) factor analysis of BAS items revealed three first-order factors, labeled *Drive*, *Reward Responsiveness*, and *Fun Seeking*. However, these factors loaded strongly onto a single second-order factor. As in past research (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997), we included all responses to BAS items in a single BAS index. In this sample, BIS and BAS were uncorrelated (Table 1), as in previous research (e.g., Carver & White, 1994).

EEG recording. Participants were fitted with a stretch-lycra cap with embedded Ag/AgCl electrodes. EEG was collected from 28 scalp sites corresponding to midline (Fz, Fcz, Cz, Cpz, Pz, Oz), frontal (Fp1, Fp2, F3, F4, F7, F8, Ft7, Ft8), central (C3, C4, Fc3, Fc4), temporal (T7, T8, Tp7, Tp8), parietal (P5, P6, Cp3, Cp4), and occipital (O1, O2) locations of the 10-10 system. The active reference electrode was placed on the left earlobe, and a ground electrode was placed on the forehead. EEG was also recorded from the right earlobe. Vertical and horizontal electrooculogram (EOG) was collected to permit the removal of artifact due to eye movements. EEG was recorded using Electro-Gel (Eaton, OH), and impedances were below 5 k Ω at each scalp

site and below 10 k Ω at EOG sites. EEG was recorded with a 0.1–100-Hz bandpass filter and digitized at 1000 Hz using a Synamps amplifier (Neuroscan Labs, El Paso, TX). Off-line, EEG was manually scored for movement artifact and rereferenced to average earlobe activity.

Baseline EEG assessment. Eight 1-min intervals of EEG were recorded as participants sat in a comfortable chair. Participants were instructed to keep their eyes open for four intervals and closed for four intervals, in an order that was counterbalanced across participants. Instructions were given via intercom by the experimenter from the adjacent room.

Go/No-Go task. On each trial of the Go/No-Go task, either the letter “M” or “W” was presented in the center of a computer monitor screen (Figure 1), following the procedure of Nieuwenhuis et al. (2003). Half of the participants were instructed to make a “Go” response (button press on keyboard) when they saw “M” but to make no response when they saw “W”; the remaining participants completed a version in which “W” was the Go stimulus and “M” was the No-Go stimulus. Assignment to task version was random. Responses were registered on a computer keyboard placed in the participant's lap. Each trial began with a fixation point that was presented for 500 ms in the center of the screen. The target then appeared for 100 ms, followed by a blank screen. Participants were instructed to respond within 500 ms of target onset. This deadline was used to preclude deliberative response control and to elicit response errors. Although responses were recorded up to 1 s, a “Too slow!” warning message appeared following responses that exceeded the 500-ms deadline. The intertrial interval was 1 s. Feedback was also given following error responses. Feedback on correct responses was provided during practice trials, but not critical trials.

The task included 500 trials: 80% Go trials and 20% No-Go trials. As in past research, the high frequency of Go stimuli was designed to induce a habitual “Go” response, to establish the expectancy of Go stimuli, and to enhance the difficulty of successfully inhibiting a response on No-Go trials. Participants received a 2-min break halfway through the task, and the entire task took approximately 15 min to complete. Following task completion, participants were debriefed, paid, thanked, and dismissed.

EEG and ERP Processing

Frontal asymmetry assessment. For analyses of spectral power, an eyeblink-rejection algorithm was applied in which EOG deflections exceeding $\pm 75 \mu\text{V}$ were removed. Additional movement artifacts were removed manually. All artifact-free 2048-ms epochs were extracted through a Hamming window to prevent

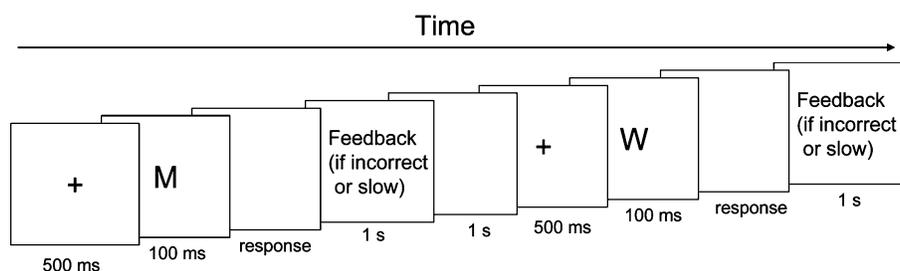


Figure 1. Schematic diagram illustrating sample stimuli in the Go/No-Go task.

spurious estimates of spectral power. Contiguous epochs were overlapped by 75% to minimize loss of data due to Hamming window extraction, and power spectra were calculated via fast Fourier transform (Davidson, Jackson, & Larson, 2000). These power values (in μV^2) were averaged across epochs within each 1-min resting trial. Because alpha power is inversely related to cortical activity (Lindsley & Wicke, 1974), total power within the alpha frequency range (8–13 Hz) was obtained for analysis. The power values at each site were submitted to a natural log transformation (to reduce skew) and averaged. Alpha asymmetry was calculated as right log-alpha power minus left log-alpha power, such that higher scores indicated greater left-sided cortical activity. Our measure of frontal asymmetry was quantified as the difference between activity at F4 and F3, positioned over the right and left dorsolateral regions of the prefrontal cortex, respectively. We also computed asymmetry scores corresponding to parietal (P6–P5), temporal (T8–T7), and occipital (O2–O1) regions to obtain discriminant validity in analyses of cortical asymmetry.

No-Go N2 and ERN. For ERP analyses, a regression-based eyeblink-correction procedure was applied (Semlitsch, Anderer, Schuster, & Presslich, 1986). Artifacts associated with movement were removed manually. Frequencies below 1 Hz and above 15 Hz were digitally filtered (48 dB, zero-phase shift). A 1000-ms stimulus-locked epoch of EEG signal, beginning 200 ms prior to target onset, was selected for each artifact-free trial. Baseline correction procedures subtracted the average prestimulus voltage within each epoch from the entire epoch. Epochs associated with correct and incorrect trials were averaged separately as a function of trial type. Following previous work (Falkenstein et al., 1999) and based on visual inspection of the waves, the N2 was scored as the peak negative deflection (in μV) occurring between 200 and 400 ms after target onset at the vertex (Cz), although identical results were obtained when the N2 was scored at the frontocentral site (Fcz). The *No-Go N2* component refers to the average N2 amplitude of the ERP associated with correct “No-Go” responses.

To quantify the ERN, an 800-ms response-locked epoch was selected for each artifact-free trial. Baseline correction procedures subtracted the average voltage prior to ERN onset (from –150 to –50 ms relative to response), and epochs associated with correct and incorrect responses were averaged separately as a function of trial type. The ERN was scored as the peak negative deflection occurring between –50 and 150 ms, relative to response, at the frontocentral scalp site (Fcz), as in previous research (e.g., Amodio, Harmon-Jones, et al., 2004). Although the ERN refers specifically to error-related activity, the corresponding activity for correct responses was also scored.

Results

Behavioral Analyses

Participants made significantly more response errors on No-Go trials ($M = 0.38$, $SD = 0.17$) than on Go trials ($M = 0.01$, $SD = 0.02$), $t(39) = 13.82$, $p < .001$. Go and No-Go error rates were not significantly correlated with BIS, BAS, No-Go N2, or frontal asymmetry. However, larger ERN amplitudes were strongly associated with lower error rates, replicating past research (Amodio, Harmon-Jones, et al., 2004; Gehring et al., 1993; Morris, Yee, & Nuechterlein, 2006; Nieuwenhuis et al., 2003; Table 1).

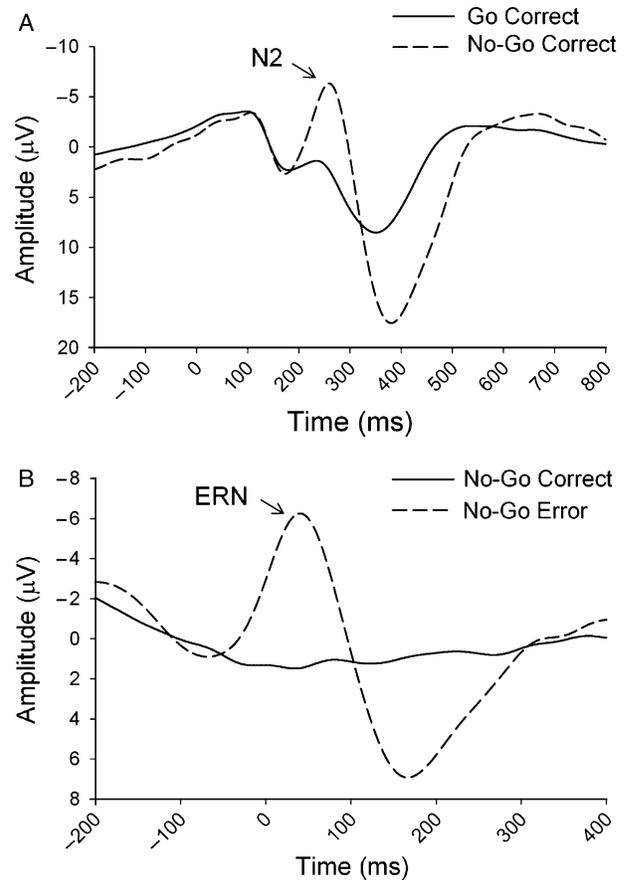


Figure 2. A: Averaged stimulus-locked ERP waveforms from correct responses on Go and No-Go trials illustrating the No-Go N2 effect (at Cz). B: Average response-locked ERP waveforms for correct and incorrect responses on No-Go trials illustrating the ERN effect (at Fcz).

Preliminary Analyses of Resting Frontal Asymmetry, No-Go N2, and ERN

On average, a significant left-sided EEG asymmetry was observed in the frontal region at baseline ($M = 0.26$, $SD = 0.10$), $t(39) = 16.47$, $p < .001$. Baseline alpha power asymmetry at temporal and parietal scalp sites also revealed significant left-sided asymmetries across participants, $t(39)s > 7.13$, $ps < .001$. The asymmetry score at the occipital sites did not differ from zero, $t(39) = 1.03$, $p = .31$.

As expected, N2 amplitudes associated with successful “No-Go” responses ($M = -8.74$, $SD = 4.80$) were significantly larger than those associated with Go responses ($M = -0.20$, $SD = 2.06$), $t(39) = 10.46$, $p < .001$ (Figure 2a). A single equivalent current dipole model of the N2 peak (270 ms poststimulus) placed the neural generator of the No-Go N2 component in the ACC (Figure 3a; $x = 4.1$ mm, $y = 38.8$ mm, $z = 88.7$ mm; dipole strength = 83.22 nAm; $R^2 = 91.4\%$). The result corroborated our interpretation of the No-Go N2 as indexing conflict monitoring that has been associated with ACC activity across several studies (Botvinick et al., 2004). However, it should be noted that the 28-site electrode array was not optimal for localization, and although results replicate past work (e.g., van Veen & Carter, 2002), they should be interpreted with some caution.

ERN amplitudes were larger for incorrect responses ($M = -12.55$, $SD = 6.07$) than correct responses ($M = -0.73$,

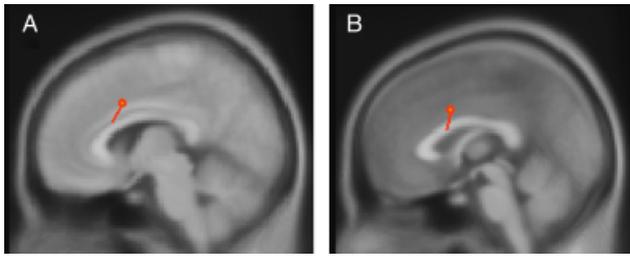


Figure 3. Dipole modeling of the No-Go N2 (A) and ERN (B), which placed both components in a similar region of dorsal ACC.

$SD = 1.99$) on No-Go trials, $t(39) = 10.97$, $p < .001$ (Figure 2b), replicating the typical pattern. Like the N2, dipole modeling of the ERN peak (44 ms after the response) placed its source in the dorsal ACC (Figure 3b; $x = -1.0$ mm, $y = 35.7$ mm, $z = 88.2$ mm; dipole strength = 167.8 nAm; $R^2 = .90$).

Primary Analyses: Neurocognitive Correlates of BIS and BAS

Our primary hypotheses concerned the unique associations of No-Go N2 amplitudes and frontal asymmetry with individual differences in BIS and BAS. Zero-order correlations among key variables are presented in Table 1. We predicted that BIS would be uniquely related to No-Go N2 amplitudes, whereas BAS would be uniquely related to frontal EEG asymmetry. We tested these predictions using hierarchical regression analyses.

Neurocognitive correlates of BIS. To examine the relationship between No-Go N2 amplitude and BIS, we tested a regression in which BAS and frontal asymmetry scores were entered in Step 1 as covariates, followed by No-Go N2 scores in Step 2, with BIS as the criterion. Effects were not significant for BAS, $\beta = .01$, $t(37) = 0.05$, $p = .96$, or for frontal asymmetry, $\beta = -.11$, $t(37) = -0.67$, $p = .51$. However, in support of our main hypothesis, the No-Go N2 effect was significant, $\beta = -.47$, $t(36) = 3.04$, $p < .005$. An additional analysis showed that N2 amplitudes from correct responses on Go trials were not related to BIS, $\beta = -.01$, $t(36) = 0.03$, $p = .98$, after covarying BAS and frontal asymmetry scores, thereby providing discriminant validity for the observed relation between the No-Go N2 and BIS scores.

Results for the ERN were similar to those of the N2 (see Table 1). To examine the relationship between ERN amplitude and BIS, BAS and frontal asymmetry scores were entered in Step 1 as covariates, followed by ERN scores in Step 2. As expected, larger ERN amplitudes were significantly associated with higher BIS scores, $\beta = -.37$, $t(36) = 2.38$, $p = .02$. By contrast, BIS scores were not associated with BAS, $\beta = .04$, $t(37) = 9.23$, $p = .82$, or frontal asymmetry, $\beta = -.17$, $t(37) = 1.01$, $p = .32$. An additional analysis showed that the ERN amplitudes scored from correct responses on No-Go trials were not related to BIS, $\beta = .09$, $t(36) = 0.56$, $p = .58$, after covarying BAS and frontal asymmetry.

Neurocognitive correlates of BAS. To examine the relationship between frontal asymmetry and BAS, we entered BIS and No-Go N2 scores in Step 1 of a multiple regression model, followed by frontal asymmetry scores in Step 2. Effects were not significant for BIS, $\beta = .00$, $t(37) = .01$, $p = .99$, or for No-Go N2 amplitudes, $\beta = .09$, $t(37) = 0.49$, $p = .63$. However, the effect of frontal asymmetry was significant, $\beta = .42$, $t(36) = 2.65$,

$p < .02$, such that greater left-sided asymmetry was associated with higher BAS scores, as in past research (Coan & Allen, 2003; Harmon-Jones & Allen, 1997). The effect of frontal asymmetry remained significant when the average alpha asymmetries at temporal, parietal, and occipital sites were included as covariates in Step 1 of the regression model, $\beta = .41$, $t(34) = 2.48$, $p < .02$. An additional analysis, in which ERN scores were substituted for the No-Go N2 scores in Step 1, produced a similar pattern. The effect for frontal asymmetry was a significant predictor of BAS, $\beta = .38$, $t(36) = 2.39$, $p = .02$, but the effects of ERN and BIS were not significant, $ps > .62$.¹

Discussion

The present research was designed to integrate the BIS/BAS model with recent research in cognitive neuroscience on the neural correlates of conflict monitoring and motivation and, in doing so, to address a critical theoretical ambiguity regarding the interpretation of BIS as reflecting the tendency to inhibit behavior or to engage in avoidance behavior. In a set of analyses designed to identify double dissociations, we found that higher BIS was uniquely associated with larger No-Go N2 and ERN amplitudes, which reflect greater conflict-related ACC activity (Botvinick et al., 2001; Yeung et al., 2004). By contrast, higher BAS was uniquely associated with greater left-sided frontal cortical asymmetry, which has been associated with approach orientation in past work (Harmon-Jones, 2003a, 2003b; Pizzagalli et al., 2005). These findings extend previous models of BIS and BAS to suggest that BIS corresponds to an attentional system for monitoring response conflicts, whereas BAS corresponds to a motivational system for coordinating approach/avoidance responses. In addition, the unique associations between BIS and BAS and neural mechanisms of conflict monitoring versus approach motivation suggest that BIS is associated with the tendency to halt ongoing behavior rather than to engage avoidance-related behaviors, thus clarifying recent ambiguities in the application of the BIS/BAS model to research on individual differences and psychopathology.

Relation of the BIS/BAS Framework to the Conflict-Monitoring Model of Control

Our findings highlight a parallel between the BIS/BAS model and more recent theorizing on mechanisms of self-regulation in the cognitive neuroscience literature. Our results suggest that BIS corresponds to a conflict-monitoring mechanism and its associated activity in the ACC, whereas BAS corresponds to a regulative mechanism and its associated activity in the PFC. Although the BIS/BAS and conflict-monitoring models developed from rather disparate fields of animal and human neuroscience research, respectively, a consideration of the parallels between these models provides insight into their broader applications. That is, whereas early BIS/BAS focused on behavioral outcomes, the present work suggests these systems correspond to a broader range of cognitive and self-regulatory processes. Similarly, research on conflict-monitoring has focused

¹We conducted additional analyses to examine the relationship between total frontal alpha power and BAS, given that some past research has reported this association (Hewig et al., 2006; Harmon-Jones & Allen, 1997). However, we did not observe an association between total frontal alpha power with either BAS or BIS ($rs < .11$, $ps > .50$).

on cognitive control and information processing, but our findings suggest that this model relates to broader motivational, emotional, and behavioral processes.

Implications for Previous Research using BIS as a Measure of Avoidance

Potential problems arise when usage of the BIS/BAS constructs is ambiguous. One issue concerns construct validity, such as when the BIS scale is selected as an index of avoidance orientation. For example, a theory regarding avoidance motivation may receive an inappropriate test if the variable of interest is measured using the BIS scale. A related issue concerns internal validity, such that significant or null effects observed using the BIS scale may be misinterpreted as evidence in support of a particular hypothesis. Indeed, in some research using the BIS scale as a measure of avoidance orientation, BIS was not related to avoidance-related behaviors or emotion, and this finding was interpreted as evidence that dispositional avoidance was not related to avoidance behavior or negative affect (e.g., Updegraff et al., 2004; Watson, Wiese, Vaidya, & Tellegen, 1999). It is possible that the observed null effects resulted because the measure did not capture the intended theoretical construct and, instead, reflected sensitivity to response conflict and inhibition of behavior. Given our findings that BIS was associated with conflict monitoring, but not with brain activity associated with approach/avoidance orientation, some previous interpretations of studies in which the BIS scale was used as a measure of avoidance motivation may benefit from reevaluation. It is important to note that although the present findings suggest that BIS may be inappropriately used as a measure of avoidance motivation, they are not inconsistent with the proposal that approach and avoidance responses reflect distinct motivational processes (e.g., Elliot et al., 2006; Gable et al., 2000).

Relation between BIS and Avoidance Motivation

Although the present findings suggest that BIS relates to the conflict-monitoring process rather than to approach or avoidance motivation, it is worth considering why BIS has been used as an individual difference measure of avoidance. One possibility is that, in practice, BIS activation precedes avoidance more often than approach. As suggested by Gray (1975), BIS is more sensitive to cues for punishment than to cues of reward, presumably because a threat (e.g., presence of a predator) has more imminent implications for survival than the presence of a reward (e.g., food or mates). Several other converging lines of research corroborate this idea (Ito, Cacioppo, & Lang, 1998; Kahneman & Tversky, 1984; Miller, 1959; Öhman, Lundqvist, & Esteves, 2001; Rozin & Royzman, 2001; Taylor, 1991). Thus, the activation of BIS may transition to avoidance behavior more often than to approach behavior. In line with this reasoning, individuals with strong BIS sensitivity may also possess a stronger dispositional avoidance (vs. approach) orientation.

When might the activation of BIS transition to approach behavior? Consider the emotion of surprise. Surprise is characterized by a highly aroused reaction to an unexpected event. In the moment of surprise, the BIS should be highly activated, leading to the momentary inhibition of ongoing behavior and enhanced vigilance (consistent with functional interpretations of the facial expression for surprise; Darwin, 1872; Reisenzein, Bördgen, Holtbernd, & Matz, 2006). Depending on whether the surprising stimulus turns out to be aversive or appetitive, one may quickly respond with either avoidance or approach. If the surprising

event turns out to be rewarding—such as friends appearing with a birthday cake at a surprise party—BIS activation should be linked with subsequent approach behavior. Our findings highlight the importance of carefully distinguishing between the process of behavioral inhibition and the engagement of active avoidance behavior, despite the fact that these two processes may often be closely related in nature.

More broadly, the present research raises questions about the complexity of the construct of “avoidance” (see also Coan & Allen, 2004). Although it may be convenient to conceive of avoidance as the opposite of approach, mounting evidence suggests that approach and avoidance may refer to very different sets of psychological processes (e.g., Scholer & Higgins, in press). For example, active avoidance involves simultaneous vigilance toward a threat and active regulation of ongoing behavior (e.g., approach toward safety). In this way, avoidance may represent a unique combination of attentional vigilance and behavioral activation that involves the coactivation of BIS and BAS.

Clarifying Inhibition in the BIS/BAS Framework

The term “inhibition” has been used to refer to different psychological and behavioral processes in different research literatures, which may contribute to ambiguities in theoretical interpretations of BIS. In the BIS/BAS framework, inhibition is a behavioral manifestation of attentional engagement to a cue for a potential threat. This form of inhibition corresponds to anxiety and neuroticism in personality research (e.g., Fowles, 2000). By contrast, in the human cognitive neuroscience literature, inhibition typically refers to a controlled process through which a person actively inhibits a response (Aron et al., 2004). Thus, the term “inhibition” is used to refer to different processes through which ongoing behavior is stopped. These include an initial bottom-up alerting to a response-relevant cue that is associated with a slowing or stopping of ongoing action, which may be followed by an intentional top-down withholding of a response. Although these two processes cannot be distinguished from behavior on tasks such as the Go/No-Go task, they may be distinguished using measures of brain activity, as in the present work and much previous research (Botvinick et al., 2004). Our results link BIS to the bottom-up process of inhibition, associated with conflict-monitoring and activity in the ACC. We speculate that BAS corresponds to the top-down form of inhibition. It will be important for future research on the BIS/BAS model to carefully distinguish these types of inhibition.

Conclusions

The BIS/BAS model has had a significant influence on research across several areas of psychology for over 25 years. In that time, cognitive neuroscience research has advanced our understanding of neural systems involved in the orchestration of behavior. The present research demonstrates a relationship between the BIS/BAS framework and recently identified neurocognitive mechanisms for detecting response conflicts (e.g., in response to threat) versus orchestrating regulatory behaviors associated with approach/avoidance motivation. Results of the present study also suggest that BIS corresponds to conflict monitoring and behavioral abatement rather than to avoidance, thereby addressing a critical ambiguity in the interpretation of BIS in the recent literature. Together, these findings serve to clarify the theoretical constructs of BIS and BAS and suggest new conceptual links between theories of behavior regulation and cognitive control.

REFERENCES

- Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, *9*, 357–381.
- Amodio, D. M., & Frith, C. D. (2006). Meeting of minds: The medial frontal cortex and social cognition. *Nature Reviews Neuroscience*, *7*, 268–277.
- Amodio, D. M., Harmon-Jones, E., Devine, P. G., Curtin, J. J., Hartley, S. L., & Covert, A. E. (2004). Neural signals for the detection of unintentional race bias. *Psychological Science*, *15*, 88–93.
- Amodio, D. M., Shah, J. Y., Sigelman, J., Brazy, P. C., & Harmon-Jones, E. (2004). Implicit regulatory focus associated with asymmetrical frontal cortical activity. *Journal of Experimental Social Psychology*, *40*, 225–232.
- Arnett, P. A., & Newman, J. P. (2000). Gray's three-arousal model: An empirical investigation. *Personality and Individual Differences*, *28*, 1171–1189.
- Aron, A. R., & Poldrack, R. A. (2005). The cognitive neuroscience of response inhibition: Relevance for genetic research in ADHD. *Biological Psychiatry*, *57*, 1285–1292.
- Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences*, *8*, 170–177.
- Aston-Jones, G., & Cohen, J. D. (2005). An integrative theory of locus coeruleus-norepinephrine function: Adaptive gain and optimal performance. *Annual Review of Neuroscience*, *28*, 403–450.
- Aston-Jones, G., Foote, S. L., & Segal, M. (1985). Impulse conduction properties of noradrenergic locus coeruleus axons projecting to monkey cerebrocortex. *Neuroscience*, *15*, 765–777.
- Beer, J. S. (2002). Implicit self-theories of shyness. *Journal of Personality and Social Psychology*, *83*, 1009–1024.
- Berridge, C. W., España, R. A., & Stalnaker, T. A. (2003). Stress and coping: Asymmetry of dopamine efferents within the prefrontal cortex. In K. Hugdahl & R. J. Davidson (Eds.), *The asymmetrical brain*. Cambridge, MA: MIT Press.
- Blair, C., Peters, R., & Granger, D. (2004). Physiological and neuropsychological correlates of approach/withdrawal tendencies in preschool: Further examination of the Behavioral Inhibition System/Behavioral Activation System scales for young children. *Developmental Psychobiology*, *45*, 113–124.
- Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*, 624–652.
- Botvinick, M., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences*, *8*, 539–546.
- Botvinick, M. M., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature*, *402*, 179–181.
- Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, *280*, 747–749.
- Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scales. *Journal of Personality and Social Psychology*, *67*, 319–333.
- Casada, J. H., & Roache, J. D. (2005). Behavioral inhibition and activation in posttraumatic stress disorder. *Journal of Nervous and Mental Disease*, *193*, 102–109.
- Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants: A proposal. *Archives of General Psychiatry*, *44*, 573–588.
- Coan, J. A., & Allen, J. J. B. (2003). Frontal EEG asymmetry and the behavioral activation and inhibition systems. *Psychophysiology*, *40*, 106–114.
- Coan, J. A., & Allen, J. J. B. (2004). Frontal EEG asymmetry as a moderator and mediator of emotion. *Biological Psychology*, *67*, 7–49.
- Cools, R., Calder, A. J., Lawrence, A. D., Clark, L., Bullmore, E., & Robbins, T. W. (2005). Individual differences in threat sensitivity predict serotonergic modulation of amygdala response to fearful faces. *Psychopharmacology*, *180*, 670–679.
- Cooper, M. L., Shapiro, C. M., & Powers, A. M. (1998). Motivations for sex and risky sexual behavior among adolescents and young adults: A functional perspective. *Journal of Personality and Social Psychology*, *75*, 1528–1558.
- Darwin, C. (1872, 1965). *The expression of the emotions in man and animals*. Chicago: University of Chicago Press.
- Davidson, R. J. (1992). Emotion and affective style: Hemispheric substrates. *Psychological Science*, *3*, 39–43.
- Davidson, R. J., Jackson, D. C., & Larson, C. L. (2000). Human electroencephalography. In J. T. Cacioppo, G. G. Bernston, & L. G. Tassinary (Eds.), *Handbook of psychophysiology* (2nd ed., pp. 27–52). New York: Cambridge University Press.
- Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science*, *5*, 303–305.
- Demaree, H. A., Everhart, D. E., Youngstrom, E. A., & Harrison, D. W. (2005). Brain lateralization of emotional processing: Historical roots and a future incorporating “dominance”. *Behavioral and Cognitive Neuroscience Reviews*, *4*, 3–20.
- Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Sciences*, *22*, 491–569.
- Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology*, *40*, 457–492.
- Elliot, A. J., Gable, S. L., & Mapes, R. R. (2006). Approach and avoidance motivation in the social domain. *Personality and Social Psychology Bulletin*, *32*, 378–391.
- Elliot, A. J., & Thrash, T. M. (2002). Approach-avoidance motivation in personality: Approach and avoidance temperaments and goals. *Journal of Personality and Social Psychology*, *82*, 804–818.
- Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in Go/Nogo tasks and their relation to inhibition. *Acta Psychologica*, *101*, 267–291.
- Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology*, *17*, 87–104.
- Fowles, D. C. (1988). Psychophysiology and psychopathology: A motivational approach. *Psychophysiology*, *25*, 373–391.
- Fowles, D. C. (2000). Electrodermal hyporeactivity and antisocial behavior: Does anxiety mediate the relationship? *Journal of Affective Disorders*, *61*, 177–189.
- Gable, S. L. (2006). Approach and avoidance social motives and goals. *Journal of Personality*, *74*, 175–222.
- Gable, S. L., Reis, H. T., & Elliot, A. J. (2000). Behavioral activation and inhibition in everyday life. *Journal of Personality and Social Psychology*, *78*, 1135–1149.
- Gehring, W. J., Goss, B., Coles, M. G., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science*, *4*, 385–390.
- Gomez, A., & Gomez, R. (2002). Personality traits of the behavioural approach and inhibition systems: Associations with processing of emotional stimuli. *Personality and Individual Differences*, *32*, 1299–1316.
- Gray, J. A. (1972). The structure of the emotions and the limbic system. In R. Porter & J. Knight (Eds.), *Physiology, emotion, and psychosomatic illness* (pp. 87–130). Amsterdam: Associated Scientific.
- Gray, J. A. (1975). *Elements of a two-process theory of learning*. London: Academic Press.
- Gray, J. A. (1982). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system*. London: Oxford University Press.
- Gray, J. A. (1987). *The psychology of fear and stress* (2nd ed.). London: Cambridge University Press.
- Gray, J. A. (1990). Brain systems that mediate both emotion and cognition. *Cognition & Emotion*, *4*, 269–288.
- Gray, J. A., & McNaughton, N. (2000). *The neuropsychology of anxiety*. London: Oxford University Press.
- Hajcak, G., Moser, J. S., Yeung, N., & Simons, R. F. (2005). On the ERN and the significance of errors. *Psychophysiology*, *42*, 151–160.
- Harmon-Jones, E. (2003a). Anger and the behavioral approach system. *Personality and Individual Differences*, *35*, 995–1005.
- Harmon-Jones, E. (2003b). Clarifying the emotive functions of asymmetrical frontal cortical activity. *Psychophysiology*, *40*, 838–848.
- Harmon-Jones, E., Abramson, L. Y., Sigelman, J., Bohlig, A., Hogan, M. E., & Harmon-Jones, C. (2002). Proneness to hypomania/mania symptoms or depression symptoms and asymmetrical frontal cortical responses to an anger-evoking event. *Journal of Personality and Social Psychology*, *82*, 610–618.

- Harmon-Jones, E., & Allen, J. J. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology, 106*, 159–163.
- Harmon-Jones, E., & Allen, J. J. B. (1998). Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology, 74*, 1310–1316.
- Heimpel, S. A., Elliot, A. J., & Wood, J. V. (2006). Basic personality dispositions, self-esteem, and personal goals: An approach-avoidance analysis. *Journal of Personality, 74*, 1293–1319.
- Hewig, J., Hagemann, D., Seifert, J., Naumann, E., & Bartussek, D. (2006). The relation of cortical activity and BIS/BAS on the trait level. *Biological Psychology, 71*, 42–53.
- Ito, T. A., Cacioppo, J. T., & Lang, P. J. (1998). Eliciting affect using the International Affective Picture System: Trajectories through evaluative space. *Personality and Social Psychology Bulletin, 24*, 855–879.
- Kahneman, D., & Tversky, A. (1984). Choices, values, and frames. *American Psychologist, 39*, 341–350.
- Keltner, D., Gruenfeld, D. H., & Anderson, C. (2003). Power, approach, and inhibition. *Psychological Review, 110*, 265–284.
- Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science, 303*, 1023–1026.
- Kok, A. (1986). Effects of degradation of visual stimulation on components of the event-related potential (ERP) in Go/No-Go reaction tasks. *Biological Psychology, 23*, 21–38.
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review, 97*, 377–395.
- Lehéricy, S., Ducros, M., Van De Moortele, P. F., Francois, C., Thivard, L., Poupon, C., et al. (2004). Diffusion tensor fiber tracking shows distinct corticostriatal circuits in humans. *Annals of Neurology, 55*, 522–529.
- Lindsley, D. B., & Wicke, J. D. (1974). The electroencephalogram: Autonomous electrical activity in man and animals. In *Bioelectric Recording Techniques* (pp. 3–83). New York: Academic Press.
- Luu, P., Collins, P., & Tucker, D. M. (2000). Mood, personality, and self-monitoring: Negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring. *Journal of Experimental Psychology: General, 129*, 43–60.
- MacAndrew, C., & Steele, T. (1991). Gray's behavioral inhibition system: A psychometric examination. *Personality and Individual Differences, 12*, 157–171.
- Mann, T., Sherman, D., & Updegraff, J. (2004). Dispositional motivations and message framing: A test of the congruency hypothesis in college students. *Health Psychology, 23*, 330–334.
- Miller, N. E. (1959). Liberalization of the basic S-R concepts: Extensions to conflict behavior, motivation, and social learning. In S. Kock (Ed.), *Psychology: A study of science* (pp. 198–292). New York: McGraw-Hill.
- Mitchell, J. T., & Nelson-Gray, R. O. (2006). Attention-Deficit/Hyperactivity Disorder symptoms in adults: Relationship to Gray's Behavioral Approach System. *Personality and Individual Differences, 40*, 749–760.
- Monteith, M. J. (1993). Self-regulation of prejudiced responses: Implications for progress in prejudice-reduction efforts. *Journal of Personality and Social Psychology, 65*, 469–485.
- Monteith, M. J., Ashburn-Nardo, L., Voils, C. I., & Czopp, A. M. (2002). Putting the brakes on prejudice: On the development and operation of cues for control. *Journal of Personality and Social Psychology, 83*, 1029–1050.
- Morris, S. E., Yee, C. M., & Nuechterlein, K. H. (2006). Electrophysiological analysis of error monitoring in schizophrenia. *Journal of Abnormal Psychology, 115*, 239–250.
- Newman, J. P., MacCoon, D. G., Vaughn, L. J., & Sadeh, N. (2005). Validating a distinction between primary and secondary psychopathy with measures of Gray's BIS and BAS constructs. *Journal of Abnormal Psychology, 114*, 319–323.
- Nieuwenhuis, S., Yeung, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. *Cognitive, Affective and Behavioral Neuroscience, 3*, 17–26.
- Öhman, A., Lundqvist, D., & Esteves, F. (2001). The face in the crowd revisited: A threat advantage with schematic stimuli. *Journal of Personality and Social Psychology, 80*, 381–396.
- Patterson, C. M., Kosson, D. S., & Newman, J. P. (1987). Reaction to punishment, reflectivity, and passive avoidance learning in extraverts. *Journal of Personality and Social Psychology, 52*, 565–575.
- Patterson, C. M., & Newman, J. P. (1993). Reflectivity and learning from aversive events: Toward a psychological mechanism for the syndromes of disinhibition. *Psychological Review, 100*, 716–736.
- Pickering, A. D., & Gray, J. A. (Eds.). (1999). *The neuroscience of personality*. New York: Guilford Press.
- Pizzagalli, D. A., Sherwood, R. J., Henriques, J. B., & Davidson, R. J. (2005). Frontal brain asymmetry and reward responsiveness: A source-localization study. *Psychological Science, 16*, 805–813.
- Quay, H. C. (Ed.). (1988). *The behavioral reward and inhibition system in childhood behavior disorder*. Elmsford, NY: Pergamon Press.
- Reisenzein, R., Bördgen, S., Holtbernd, T., & Matz, D. (2006). Evidence for strong dissociation between emotion and facial displays: The case of surprise. *Journal of Personality and Social Psychology, 91*, 295–315.
- Rolls, E. T. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex, 10*, 284–294.
- Rozin, P., & Royzman, E. B. (2001). Negativity bias, negativity dominance, and contagion. *Personality and Social Psychology Review, 5*, 296–320.
- Scholer, A. A., & Higgins, E. T. (in press). Distinguishing levels of approach and avoidance: An exploration using regulatory focus theory. In A. J. Elliot (Ed.), *Handbook of approach and avoidance motivation*.
- Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science, 275*, 1593–1599.
- Semlitsch, H. V., Anderer, P., Schuster, P., & Presslich, O. (1986). A solution for reliable and valid reduction of ocular artifacts, applied to the P300 ERP. *Psychophysiology, 23*, 695–703.
- Sherman, D. K., Mann, T., & Updegraff, J. A. (2006). Approach/avoidance orientation, message framing, and health behavior: Understanding the congruency effect. *Motivation and Emotion, 30*, 165–169.
- Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science, 8*, 204–210.
- Taylor, S. E. (1991). Asymmetrical effects of positive and negative events: The mobilization-minimization hypothesis. *Psychological Bulletin, 110*, 67–85.
- Thrash, T. M., & Elliot, A. J. (2003). Inspiration as a psychological construct. *Journal of Personality and Social Psychology, 84*, 871–889.
- Torrubia, R., & Tobena, A. (1984). A scale for the assessment of "susceptibility to punishment" as a measure of anxiety: Preliminary results. *Personality and Individual Differences, 5*, 371–375.
- Updegraff, J. A., Gable, S. L., & Taylor, S. E. (2004). What makes experiences satisfying? The interaction of approach-avoidance motivations and emotions in well-being. *Journal of Personality and Social Psychology, 86*, 496–504.
- Updegraff, J. A., Sherman, D. K., Luyster, F. S., & Mann, T. L. (2007). The effects of message quality and congruency on perceptions of tailored health communications. *Journal of Experimental Social Psychology, 43*, 249–257.
- Van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 14*, 593–602.
- Wallace, J. F., Newman, J. P., & Bachorowski, J.-A. (1991). Failures of response modulation: Impulsive behavior in anxious and impulsive individuals. *Journal of Research in Personality, 25*, 23–44.
- Watson, D., Wiese, D., Vaidya, J., & Tellegen, A. (1999). The two general activation systems of affect: Structural findings, evolutionary considerations, and psychobiological evidence. *Journal of Personality and Social Psychology, 76*, 820–838.
- Wilson, G. D., Gray, J. A., & Barrett, P. T. (1990). A factor analysis of the Gray-Wilson Personality Questionnaire. *Personality and Individual Differences, 11*, 1037–1045.
- Wingrove, J., & Bond, A. J. (1998). Angry reactions to failure on a cooperative computer game: The effect of trait hostility, behavioural inhibition, and behavioural activation. *Aggressive Behavior, 24*, 27–36.
- Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111*, 931–959.
- Yu, A. J., & Dayan, P. (2005). Uncertainty, neuromodulation, and attention. *Neuron, 46*, 681–692.