Proneness to Hypomania/Mania Symptoms or Depression Symptoms and Asymmetrical Frontal Cortical Responses to an Anger-Evoking Event

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The behavioral approach system (BAS) reflects the propensity to respond to signals of reward, including stimuli associated with safety and goal-oriented attack (e.g., anger). Hypomania/mania has been posited to involve increased BAS activity. In contrast, depression has been posited to involve decreased BAS activity. Building on past research, which suggests that increased left frontal cortical activity is a neurophysiological index of BAS activity, the present research tested the hypotheses that proneness toward hypomania/mania symptoms would be related to increased relative left frontal activity and that proneness toward depression symptoms would be related to decreased relative left frontal activity in response to an anger-evoking event. Results from 67 individuals who had completed a measure of proneness toward these affective symptoms and were exposed to an anger-evoking event supported the hypotheses.

Prominent motivation theories share the assumption that two systems underlie much behavior. One system manages appetitive, incentive motivation and approach behavior. It has been called a behavioral activation system (Fowles, 1980, 1988), behavioral approach system (BAS; Gray, 1982, 1987, 1990, 1994a, 1994b), and behavioral facilitation system (Depue & Collins, 1999; Depue & Iacono, 1989; Depue, Krauss, & Spoont, 1987). The other system, the behavioral inhibition system (BIS), inhibits ongoing behavior (Gray, 1982, 1987, 1990, 1994b).

Gray's (1982, 1987, 1994a, 1994b) theory has served as the basis for most of the other theories. Hence, we focus on it. Gray has posited that these fundamental motivation systems are present in the mammalian central nervous system and that each responds to separate classes of reinforcing events with particular types of behavior. Moreover, each is mediated by a separate set of interacting brain structures that process particular types of information. The BIS responds to conditioned stimuli associated with punishment, conditioned stimuli associated with the omission or termination of reward (frustrative nonreward), and novel stimuli. It

causes the interruption of ongoing behavior, increases arousal, which prepares the organism for the next behavior, and increases attention toward stimuli, which would increase information gain. The neural structures posited to be involved in the BIS are the septohippocampal system (composed of the septal area, entorhinal cortex, dentate gyrus, hippocampus, and subicular area), the prefrontal cortex, and the monoaminergic pathways that ascend from the mesencephalon to innervate the septohippocampal system.

The BAS is posited to respond to stimuli associated with reward and relieving nonpunishment (safety). It also responds to stimuli associated with skilled escape and predatory aggression. The neural structures involved in the BAS are the basal ganglia (the dorsal and ventral striatum, and dorsal and ventral pallidum), the dopaminergic fibers that ascend from the mesencephalon (substantia nigra and nucleus A 10 in the ventral tegmental area) to innervate the basal ganglia, thalamic nuclei linked to the basal ganglia, and neocortical areas (motor, sensorimotor, and prefrontal cortex) linked to the basal ganglia.

In addition to these two systems involving different neural substrates and exerting distinct influences on action, these motive systems are posited to be involved in the generation of emotions that are relevant to approach behavior and the inhibition of behavior. For example, the BAS has been posited to be involved in the generation of euphoria and anger (Depue & Iacono, 1989), whereas the BIS has been posited to be involved in the generation of anxiety (Gray, 1982). In addition, theories of psychopathology have posited that depression involves a hypoactive BAS (Fowles, 1988, 1993) and that mania/hypomania involves a hyperactive BAS (Depue & Iacono, 1989; Depue et al., 1987).

BAS and Left Frontal Cortical Activity

Recent research with humans has found that scores on a subjective measure of BAS sensitivity, Carver and White's (1994) BIS–BAS scale, are associated with increased left frontal cortical activity during resting baseline (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). These findings suggest that left frontal

cortical activity is a neurophysiological index of BAS activity. In addition, these results fit well with conceptualizing depression as involving a hypoactive BAS, because other research has indicated that depression is associated with decreased left frontal cortical activity (Debener, Beauducel, Nessler, Brocke, Heilemann, & Kayser, 2000; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1990, 1991).

Consistent with the prediction that hypomania/mania involves a hyperactive BAS, research has found that hypomania/mania is associated with increased approach-related emotional experiences such as euphoria and anger (Cassidy, Forest, Murry, & Carroll, 1998; Depue & Iacono, 1989; Tyrer & Shopsin, 1982). Furthermore, lithium carbonate, a treatment for bipolar disorder, reduces aggression (Malone, Delaney, Luebbert, Cater, & Campbell, 2000), further suggesting that anger and aggression cohere with the other symptoms of bipolar disorder such as excessive reward seeking. Also consistent with these ideas is research demonstrating that left frontal cortical activity is associated with anger (Harmon-Jones, 2001; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001).

Other research supports the hypotheses that depression involves decreased BAS activity and that mania involves increased BAS activity as indexed by asymmetrical frontal cortical activity. In this research, it has been found that individuals who have suffered damage to right prefrontal cortex are more likely to evidence mania, and individuals who have suffered damage to the left prefrontal cortex are more likely to evidence depression (see review by Robinson & Downhill, 1995). Thus, this research is consistent with the view that mania may be associated with increased left prefrontal activity and increased approach tendencies, because the approach motivation functions of the left prefrontal cortex are released and not restrained by the withdrawal system in the right prefrontal cortex. In contrast, damage to the left prefrontal cortex may cause depression because the decreased left prefrontal cortical activity reduces approach motivation, an observed deficit in depression.

Hypomania/Mania and Self-Reported BAS

Research that has examined relationships between indices of self-reported BAS activity and indices of self-reported depression and hypomania/mania symptoms among unselected undergraduate students also supports the hypotheses that hypomania/mania is related to increased BAS activity and that depression is related to decreased BAS activity. In these studies, the General Behavior Inventory (GBI; Depue, Krauss, Spoont, & Arbisi, 1989) was used to assess depression and hypomania/mania symptoms. In one study, when depression was statistically controlled, hypomania/mania symptoms were positively related to positive affect and not related to negative affect, whereas depressive symptoms were negatively related to positive affect and positively related to negative affect (Lovejoy & Steuerwald, 1992), as assessed by the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). It is important to note that positive affect, as measured by the PANAS, has been posited to reflect the activation of the BAS, and negative affect has been posited to reflect the activation of the BIS (Watson, 2000). In another study, results revealed that hypomania/mania symptoms were positively related to self-reported BAS activity and not self-reported BIS activity

(Meyer, Johnson, & Carver, 1999), as assessed by the BIS–BAS questionnaire (Carver & White, 1994). These studies support the idea that hypomania/mania symptoms are related to aspects of the BAS, even in samples unselected for extreme scores on depression and hypomania/mania indices. In this regard, it is important to note research suggesting that the subsyndromal bipolar conditions identified by the GBI (as in Lovejoy & Steuerwald, 1992, and Meyer et al., 1999) appear to be on a continuum with full-blown syndromal bipolar disorder (e.g., Depue et al., 1989; Depue, Slater, Wolfstetter-Kausch, Klein, Goplerud, & Farr, 1981). Specifically, individuals from university populations identified by the GBI as exhibiting subsyndromal bipolar symptoms show diagnostic, family history, clinical course, behavioral, and biological characteristics similar to those of patients with bipolar disorder (Depue et al., 1989, 1981).

Of course, because these studies involved only self-report assessments, it is difficult to know whether affective disorder symptoms actually related to BAS activity or whether the semantic overlap of the items on each self-report questionnaire caused the relationship between affective disorder symptoms and BAS activity. Thus, one purpose of the present research was to assess whether affective disorder symptoms would relate to a non-self-report assessment of BAS activity—left frontal cortical activity.

Anger and Approach Motivation

Although research suggests that hypomania/mania symptoms are associated with BAS tendencies such as fun seeking and positive affect, no previous research has tested the prediction that hypomania/mania symptoms relate to neurophysiological correlates of anger, an emotion also thought to be associated with BAS functioning (Depue & Iacono, 1989). At first glance, the idea that anger and positive affect are both associated with BAS functioning may seem perplexing, as anger is considered a negative emotion. However, it is important to remember that the BAS functions to motivate the organism to actively engage with the environment. When an aversive situation is encountered, the BAS may motivate angry and aggressive behaviors aimed at removing the stimulus associated with the aversive situation. Consistent with the idea that anger is associated with BAS functioning, research has demonstrated that both trait and state anger are associated with relative left frontal activity. This research has revealed that trait anger is associated with increased relative left frontal activity (Harmon-Jones, 2001; Harmon-Jones & Allen, 1998) and that this relationship is not due to anger being regarded as a positive feeling (Harmon-Jones, 2001). Moreover, controlling for positive and negative affect (as measured by the PANAS) does not alter the magnitude of the relationship between anger and asymmetrical frontal activity (Harmon-Jones, 2001; Harmon-Jones & Allen, 1998).

Other research has revealed that individuals who are insulted respond with increased reported anger and relative left frontal activity (Harmon-Jones & Sigelman, 2001; Harmon-Jones, Vaughn, Mohr, Sigelman, & Harmon-Jones, 2001). In addition, individuals who respond with relative left frontal activity after the insult report feeling angrier and evidence increased aggression (Harmon-Jones & Sigelman, 2001). Additional support for the idea that anger is associated with BAS functioning comes from research testing the conceptual model that integrated reactance theory with

learned helplessness theory (Wortman & Brehm, 1975). According to this model, how individuals respond to uncontrollable outcomes depends on their expectation of being able to control the outcome and the importance of the outcome. When an individual expects to be able to control outcomes that are important, and those outcomes are found to be uncontrollable, psychological reactance should be aroused. Thus, for individuals who initially expect control, the first few bouts of uncontrollable outcomes should arouse reactance, a motivational state aimed at restoring control. After several exposures to uncontrollable outcomes, these individuals should become convinced that they cannot control the outcomes and should show decreased motivation (i.e., learned helplessness). In other words, reactance will precede helplessness for individuals who initially expect control. In one study that tested this model, individuals who exhibited anger in response to one unsolvable problem had better performance on a subsequent cognitive task than did participants who exhibited less anger (Mikulincer, 1988). Other research has found that infants who expressed anger during extinction maintained interest during subsequent relearning and were more able to learn a new task (Lewis, Sullivan, Ramsay, & Alessandri, 1992). Thus, subsequent to frustrating events, anger may maintain and increase task engagement and approach motivation, behaviors that are part of BAS functioning.

Also consistent with the view that anger is associated with BAS functioning is research indicating that trait anger is associated with trait assertiveness and competitiveness (Buss & Perry, 1992). Research has also revealed that the neurotransmitter dopamine is involved in BAS activity (Gray, 1991) and irritable aggression (Depue & Spoont, 1986). Finally, animal behavior research on offensive aggression suggests that anger is an emotion with approach behavioral tendencies (Blanchard & Blanchard, 1984; Moyer, 1976).

The Present Study

The present study was designed to examine the relationship between proneness to symptoms of depression and hypomania/mania and frontal cortical asymmetrical activity in response to an anger-evoking event. Drawing on the hypothesized link between hypomania/mania and increased reactivity of the BAS to anger-evoking stimuli (Depue & Iacono, 1989), we predicted that proneness toward hypomania/mania symptoms would be related to increased relative left frontal activity in response to an anger-evoking event, as these individuals may have approach-related action tendencies rather than withdrawal-related action tendencies in response to anger-evoking events. In contrast, we predicted that proneness toward depression symptoms would be related to decreased relative left frontal activity in response to an anger-evoking event, as these individuals may have withdrawal-related action tendencies rather than approach-related action tendencies in response to anger-evoking events.

To assess these individual differences characteristics, we used the GBI, which was developed to identify individuals who are at risk for developing bipolar or depressive disorders (Depue & Klein, 1988; Depue et al., 1989). To induce anger, we exposed university students who were opposed to a tuition increase to an editorial that argued for a tuition increase at their university. To assess relative left frontal activity, we measured electroencepha-lographic (EEG) activity during a baseline resting session and immediately after exposure to the editorial.

Method

Participants

Participants were 72 (37 women, 35 men) introductory psychology students who participated in exchange for extra credit in their psychology course. They all paid at least 33% of their tuition by themselves or with student loans, and they moderately or strongly disagreed with a statement indicating that tuition should be increased by 10% at the university. Percentage of tuition paid, attitude toward the tuition increase, and the GBI were assessed in a mass survey session at the beginning of the semester. Participants were right-handed (score ≤17 on the Chapman & Chapman, 1987, handedness questionnaire), and reported no history of psychiatric disorder, neurologic disorder, or brain trauma. Three women and 1 man expressed doubts about the cover story, and their data were removed prior to all analyses, leaving 68 participants (34 women, 34 men) for final analyses. One woman had excessive movement artifact in the EEG, and her EEG data were not analyzed, leaving 67 participants for the EEG analyses.

Procedure

Prior to the experiment, participants were phoned and told that the study concerned EEG reactions to pilot radio broadcasts. During the phone call, handedness was assessed, using Chapman and Chapman's (1987) questionnaire. Participants were also asked whether they were currently taking any psychoactive medications and whether they had seen a health care professional for emotional or psychiatric problems; those who gave an affirmative reply were not invited to participate.

At the experiment lab, participants sat in a comfortable chair in a sound-attenuated room. The experimenter explained that a professor was conducting the study as a service for a local public radio station, WERN, whose broadcasting was targeted toward University of Wisconsin—Madison (UW). He then explained that WERN was considering the introduction of new programs and that the professor, an expert on responses to mass media, had pilot-tested new programming ideas for WERN. He finished the introduction by explaining that participants would listen to a brief pilot broadcast and that their emotional responses to the broadcast would be assessed by using questionnaires and brain wave activity. After hearing this introduction, participants read an introduction written on WERN letterhead that reiterated this information, and then they read and signed a consent form.

Participants were prepared for the EEG recording. Following the attachment of the electrodes, they completed a questionnaire that assessed current emotional state. Eight 1-min epochs of baseline EEG were then recorded.

Next, participants received written instructions that explained that they would be listening to an editorial concerning a tuition increase at UW and that the Board of Regents had approved a 9.6% tuition increase for the upcoming academic year. The instructions indicated that UW administrators and the state legislature were considering an additional increase of 10% for the following years.[1] Finally, the instructions informed participants to tell the researcher when they finished reading the instructions and that the researcher would start the tape-recorded editorial.

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[1] The research design included three experimental conditions that manipulated when the tuition increase would occur and whether action could be taken to alter the increase. All three conditions evoked equal levels of anger. Because depression and hypomania-plus-biphasia did not interact with condition in predicting the asymmetrical frontal activity or anger in response to the editorial, the discussion of these conditions is not presented in this article.

Participants then listened to the Bulletin Board pilot broadcast in which a man made persuasive arguments in favor of a 10% tuition increase. EEG was collected for 3 min following the completion of the broadcast. During this period of time, participants reflected on how the broadcast made them feel, as the experimenter had instructed them to do. Then, participants completed an emotion questionnaire that asked them to rate how they felt while they listened to the broadcast.

After collecting the questionnaire, the experimenter questioned participants about whether they thought the editorial was veridical and explained the study's purpose.

Questionnaires

The baseline and posteditorial emotion questionnaires asked participants to indicate how much they felt certain emotions, on a scale of 0 to 8, (0 = *none*, 8 = *the most in my life*). Items were included to assess anger (mad, agitated, angry, annoyed, bothered, disgusted, irritated, frustrated; Cronbach's $\alpha = .90$ for baseline and .95 for editorial), sadness (sad, down, low; Cronbach's $\alpha = .92$ for baseline and .86 for editorial), fear (tense, fear; $r = .29$, $p < .02$ for baseline and $r = .53$, $p < .001$ for editorial), and happiness (happy, good, relaxed, relief, content, amused; Cronbach's $\alpha = .77$ for baseline and .83 for editorial). All emotion scales were created by averaging the reported intensities for the emotion words within the particular emotion type (e.g., anger word intensities were averaged to create the anger index).

To assess symptomatic behaviors associated with hypomania and depression, we used the GBI, a 73-item self-report measure. From the GBI, depression (D) and hypomania-plus-biphasia (HB) scores were calculated using the method recommended by Depue et al. (1989). The internal consistency of both scales was acceptable (for D, Cronbach's $\alpha = .96$; for HB, Cronbach's $\alpha = .93$). The means and standard deviations for the D scale were 5.04 and 7.41, respectively, and they were 3.86 and 5.06, respectively, for the HB scale.

EEG Recording and Analyses

To record EEG, we placed 27 (22 homologous and 5 midline) electrodes mounted in a stretch-lycra electrode cap (Electro-Cap, Eaton, OH) on the participant's head, using known anatomical landmarks (Blom & Anneveldt, 1982). EEG was recorded from the frontal, central, temporal, parietal, and occipital regions of the scalp (and regions in between), using the 10% electrode system (Chatrian, Lettich, & Nelson, 1985), which was based on the 10–20 international system (Jasper, 1958). The ground electrode was mounted in the cap on the midline between the frontal pole and the frontal site. The reference electrode was placed on the left ear (A1), and data were also acquired from an electrode placed on the right ear (A2), so that an off-line digitally derived, averaged ears' reference could be computed. Eye movements (EOG) were also recorded to facilitate artifact scoring of the EEG. Eye movements were recorded from the supra- and suborbit of the left eye, to assess vertical eye movements, and from the left and right outer canthus, to assess horizontal eye movements. All electrode impedances were under 5,000 ohms, and homologous sites (e.g., F3 and F4) were within 1,000 ohms of each other. Electro-Gel (Eaton, OH) was used as the conducting medium. EEG and EOG were amplified with Neuroscan Synamps (Herndon, VA), bandpass filtered (0.1 to 100 Hz; 60-Hz notch filter enabled), digitized at 500 Hz, and stored onto the hard drive of a Pentium 200 MMX computer. The amplifier gain was set at 500 (11-mV range, 0.168-uV/bit resolution), and a 16-bit A-D converter board was used. Prior to each participant, to assess the technical integrity of the recording system, we ran and inspected 400 microvolts 20-Hz calibration signals.

The EEG and EOG signals were visually scored on a high-resolution computer monitor, and portions of the data that contained eye movements, muscle movements, or other sources of artifact were removed. When

artifact occurred in one channel at a point in time, data from all channels were removed at that point in time. Derived averaged-ears reference was used for further data reduction, so that each channel reflected the voltage between the relatively inactive ears and an active scalp site. All artifact-free epochs that were 2.048 s in duration were extracted through a Hamming window, which was used to prevent spurious estimates of spectral power. Contiguous epochs were overlapped by 75%, to minimize loss of data due to Hamming window extraction. A fast Fourier transform was used to calculate the power spectra. These power values were averaged across the 2.048-s epochs. Total power within the alpha (8–13 Hz) frequency range was obtained. The power values were log transformed for all sites, to normalize the distributions. Asymmetry indices (log-right minus log-left alpha power) were computed for each scalp region. By computing asymmetry scores for regions other than the frontal ones, we were able to test whether the predicted effects were specific to the frontal regions. Because alpha power is inversely related to cortical activity (Cook, O'Hara, Uijt-dehaage, Mandelkern, & Leuchter, 1998; Davidson, Chapman, Chapman, & Henriques, 1990; Lindsley & Wicke, 1974), higher scores on the indices indicate greater relative left-hemisphere activity.

In the baseline EEG recording, eight 1-min epochs were recorded, alternating eyes-open (O) with eyes-closed (C) trials, in one of two orders (i.e., C, O, O, C, O, C, C, O or O, C, C, O, C, O, O, C), as in previous research (Tomarken, Davidson, Wheeler, & Kinney, 1992). Data were averaged across eyes-open and eyes-closed minutes. An average of 695.22 (72% of possible) artifact-free epochs ($SD = 165.18$) composed the resting baseline data, and all participants had greater than 267 artifact-free epochs (28% of possible). Cronbach's coefficient alphas computed by using each minute of the 8-min resting period as "items" were greater than .90 for all asymmetry indices.

Immediately after the tuition-increase editorial, 3 min of EEG were recorded. An average of 276.30 (77% of possible) artifact-free epochs ($SD = 62.70$) composed these data, and all participants had greater than 124 artifact-free epochs (34% of possible). Cronbach's coefficient alphas across the 3 min were greater than .85 for all asymmetry indices.

Results

Overall Responses to the Anger-Evoking Event

Asymmetrical activity. Relative left-midfrontal activity increased from baseline ($M = 0.018$, $SD = 0.0520$) to after the tuition-increase editorial ($M = 0.025$, $SD = 0.049$), $F(1, 66) = 4.14$, $p < .05$. No other asymmetry indices changed from baseline to after the editorial ($ps > .14$). These results suggest that the tuition-increase editorial engaged the approach motivational system as assessed by relative left frontal activity.

As a follow-up analysis, a 2 (hemisphere) × 2 (time: resting baseline vs. anger) analysis of variance (ANOVA) was performed with alpha power (the inverse of cortical activity) as the dependent variable. A main effect of time, $F(1, 66) = 25.40$, $p < .001$, and a main effect of hemisphere, $F(1, 66) = 13.40$, $p < .001$, occurred. The main effect of time indicated that alpha power increased from baseline to after the editorial, as would be expected as individuals habituate to the laboratory situation. The main effect of hemisphere indicated that alpha power was greater in the right- than left-midfrontal hemisphere. In addition, the interaction was significant, $F(1, 66) = 4.63$, $p < .04$, and it revealed that left-midfrontal alpha power after the editorial message ($M = 0.078$, $SD = 0.332$) was less than right-midfrontal alpha power after the editorial message ($M = 0.103$, $SD = 0.331$), whereas the difference between right and left alpha power was not as great at baseline (left $M = -0.015$, $SD = 0.302$; right $M = 0.002$, $SD = 0.304$). These

results indicate that relative left-midfrontal cortical activity (inverse of alpha) was greater after the editorial than at baseline.

Reported emotions. To examine the experienced emotional responses to the tuition-increase editorial, we performed a repeated measures ANOVA with emotional responses (anger, fear, sad, happy) to the tuition-increase editorial serving as dependent variables (i.e., repeated measures). There was a significant effect of emotion, $F(3, 201) = 28.38$, $p < .001$, with anger being reported as more intense ($M = 3.27$, $SD = 1.89$) than any other emotion (happy: $M = 2.41$, $SD = 1.27$; sad: $M = 1.35$, $SD = 1.28$; fear: $M = 1.64$, $SD = 1.67$; all $ps < .001$ in planned comparisons). In addition, separate repeated measures ANOVAs with emotion at baseline and emotion following the editorial serving as dependent variables (repeated measures) revealed that anger increased from baseline ($M = 1.13$, $SD = 1.17$) to after the tuition-increase editorial, $F(1, 67) = 113.04$, $p < .001$. Happiness decreased from baseline ($M = 3.75$, $SD = 1.22$) to after the tuition-increase editorial, $F(1, 67) = 92.96$, $p < .001$. Fear and sadness did not change from baseline (fear: $M = 1.38$, $SD = 1.14$; sad: $M = 1.21$, $SD = 1.33$) to after the tuition-increase editorial, $Fs < 2.0$, $ps > .17$. These effects indicate that the tuition-increase editorial increased anger and that anger was the major emotional response to the tuition-increase editorial.

Relationships of Proneness to Affective Symptoms With Frontal Activity and Experienced Emotions in Response to the Anger-Evoking Event

Asymmetrical activity. To test the prediction that D symptoms would relate to decreased left frontal activity and that HB symptoms would relate to increased left frontal activity in response to an anger-provoking event, we performed a regression analysis in which relative left frontal activity in the minutes following the anger-provoking event was predicted by resting baseline relative left frontal activity, D, and HB. As shown in Table 1, the results of this analysis provided support for predictions, as D related negatively to relative left-midfrontal, lateral-frontal, and frontal-temporal activity, and HB related positively to relative left-midfrontal, lateral-frontal, and frontal-temporal activity.[2,3]

To explore the regional specificity of the effects, we examined relationships between D and HB with all other asymmetry indices. As shown in Table 1, nonsignificant effects were observed at all other sites. These latter effects suggested that the relationships of D and HB to asymmetrical activity were restricted to the frontal regions.

Independent assessments of left and right frontal activity. Our primary predictions involved relative activation of the left as compared to the right hemisphere, and, thus, we computed an asymmetry index. The asymmetry index controls for individual differences in skull thickness and volume conduction, which could produce differences in alpha power. In an effort to assess the effects of left and right hemispheres separately, individual differences in skull thickness and volume conduction need to be controlled. In an effort to accomplish this control, regression analyses can be performed in which alpha power at one site is regressed onto the average of alpha power at all sites, alpha power at the homologous site, and individual differences of interest (e.g., Wheeler, Davidson, & Tomarken, 1993). Such analyses were performed in this study to assess the relationships between D, HB,

Table 1

Regression of Hypomania Plus Biphasia (HB) and Depression (D) on Cortical Asymmetry Indices

Asymmetry Index	β	Partial r	t value
Fp1/Fp2			
D	−.10	−.08	<1.0
HB	.05	.04	<1.0
F3/F4			
D	−.22	−.25	−2.02*
HB	.22	.26	2.11*
F7/F8			
D	−.24	−.29	−2.43*
HB	.24	.30	2.48*
FT7/FT8			
D	−.29	−.32	−2.71*
HB	.23	.26	2.18*
FC3/FC4			
D	−.16	−.16	−1.26
HB	.16	.16	1.30
T3/T4			
D	−.19	−.20	−1.65
HB	.18	.20	1.62
T5/T6			
D	−.008	−.01	<−1.0
HB	−.05	−.07	<−1.0
C3/C4			
D	−.13	−.16	−1.31
HB	.10	.13	1.04
CP3/CP4			
D	−.10	−.12	<−1.0
HB	.02	.02	<1.0
P3/P4			
D	−.07	−.10	<−1.0
HB	−.002	−.003	<−1.0
O1/O2			
D	−.03	−.05	<−1.0
HB	−.05	−.08	<−1.0

Note. In EEG research, Fp1/Fp2 = frontal pole sites, with odd number (1, 3, etc.) indicating left hemisphere and even number (2, 4, etc.) indicating right hemisphere (the same is true for all electrodes); F3/F4 = midfrontal; F7/F8 = lateral frontal; FT7/FT8 = frontal-temporal; FC3/FC4 = frontal central; T3/T4 = anterior temporal; T5/T6 = posterior temporal; C3/C4 = central; CP3/CP4 = central-parietal; P3/P4 = parietal; O1/O2 = occipital.
* $p < .05$.

and left and right frontal activity after the anger-evoking event. For these analyses, alpha power at each frontal site served as the criterion; thus, when interpreting these results, it is important to recall that increased alpha power reflects decreased cortical activ-

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[2] Resting frontal asymmetry did not relate to either HB or D. The lack of a relationship between depression and frontal asymmetrical activity is consistent with some past research (Nitschke, Heller, Palmieri, & Miller, 1999; Reid, Duke, & Allen, 1998; Tomarken & Davidson, 1994) but inconsistent with other research (Gotlib et al., 1998; Henriques & Davidson, 1990, 1991; Schaffer, Davidson, & Saron, 1983). Relationships between depression and resting frontal asymmetrical activity are not inevitable, and the asymmetry may only contribute to depression, with not all depressed persons showing relative right frontal activity because of the multiple complex causes of the disorder (Davidson, 1998).

[3] Regression analyses with each minute of EEG after the tuition editorial used as a criterion variable revealed similar relationships of HB and D with relative left frontal activity.

ity. For these analyses, we first created left and right frontal resting baseline and editorial indices by averaging the three scalp sites that produced significant asymmetry effects (F3/4, F7/8, FT7/8). Thus, we averaged these three sites for each hemisphere (right, left) and time (resting baseline, editorial). The sites were highly interrelated (Cronbach's α for each index was >.84). Results indicated that after the anger-evoking event, HB was associated with increased left frontal activity and decreased right frontal activity, whereas D was associated with increased right frontal activity and decreased left frontal activity. These results were revealed in a regression analysis in which left frontal alpha power after the editorial was predicted by D ($\beta = .29$, partial $r = .36$), $t(63) = 3.04$, $p < .004$; HB ($\beta = -.26$, partial $r = -.34$), $t(63) = -2.85$, $p < .006$; and left frontal alpha power at resting baseline ($\beta = .93$, partial $r = .89$), $t(63) = 15.46$, $p < .001$; and a regression analysis in which right frontal alpha power after the editorial was predicted by D ($\beta = -.29$, partial $r = -.35$), $t(63) = -2.93$, $p < .005$; HB ($\beta = .24$, partial $r = .30$), $t(63) = 2.52$, $p < .02$; and right frontal alpha power at resting baseline ($\beta = .89$, partial $r = .87$), $t(63) = 14.24$, $p < .001$.[4]

Reported emotions. For reported anger in response to the anger-provoking event, we performed a regression analysis in which baseline anger, D, and HB predicted anger in response to the tuition-increase editorial. The results of this analysis did not demonstrate significant relationships ($ps > .40$).[5]

Discussion

As predicted, individuals with proneness toward hypomania/mania symptoms evidenced stronger approach motivation, as measured by relative left frontal activation, when confronted with an anger-evoking situation, whereas individuals with proneness toward depression symptoms evidenced stronger withdrawal motivation, as measured by relative right frontal activation, when confronted with the same anger-evoking situation. These results are the first to provide support for the predicted relationships between proneness toward hypomania/mania symptoms and approach motivation by using an index of asymmetrical frontal cortical activity. Moreover, the results support predictions derived from models relating hypomania/mania to BAS activity (Depue & Iacono, 1989; Fowles, 1988, 1993), models of the motivational functions of asymmetrical frontal activity (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997), and models that consider anger as part of the BAS (Depue & Iacono, 1989; Harmon-Jones, 2001; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001; Harmon-Jones et al., 2001).

Experienced anger did not relate positively with HB. Our results suggest that the neurophysiological and reported evidence of approach motivational tendencies (e.g., anger) were dissociated in their relationships with the proneness to affective disorder symptoms (HB and D) indices. On the basis of past research, it is fair to assume that relative left frontal activity may serve as an index of approach motivation. However, it is unlikely that left frontal activity is singularly related to anger and instead probably provides an index of the approach motivational characteristics of anger. The subjective feelings identified by participants as anger may include feelings associated with approach motivation as well as feelings not associated with approach motivation. More sensitive measures of the subjective state of anger might be developed to discriminate

these two distinct anger-related emotional states. Emotions are complex phenomena, involve several processes, and serve multiple functions. As such, the physiology underlying and/or associated with an emotion is complex and probably depends more on the nature of the particular emotion—its cause, motivational output, and so forth—than on the emotion label per se (e.g., anger). As such, there are probably varieties of anger and a variety of factors (e.g., antecedents) that may determine the particular anger evoked. Each variety may be associated with particular physiological response patterns. In short, the common language term "anger" may lump together distinct states. Such a perspective is consistent with research by Stemmler (1989) that has demonstrated that the emotion-eliciting context (real life vs. imagery) plays a significant role in the pattern of autonomic and somatic responses evoked, even when self-reported emotional intensity is equivalent between the different contexts.

It is also possible that HB and D related to the frontal asymmetry and not reported anger because reported anger had a more restricted range (range of standardized scores = 3.97) than did the frontal asymmetry (range of standardized scores = 5.45). This more restricted range for reported anger may have reduced the likelihood of observing a significant relationship between reported anger and HB.

Individual Differences in Responses to Anger-Inducing Situations

Recent evidence has suggested that anger is associated with increased left frontal cortical activity (Harmon-Jones, 2001; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001; Harmon-Jones et al., 2001). The present research extends the past

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[4] Because of past suggestions that right parietal activity relates to emotional arousal (see review by Heller, 1990), analyses were also performed to assess whether right parietal alpha power differed from baseline to after the tuition-increase editorial: It did not ($p > .30$). In addition, regression analyses were performed to assess whether HB and D related to right parietal alpha power: They did not ($ps > .30$). Although some past research has supported the hypothesis that right parietal activity relates to emotional arousal, other research has not supported this hypothesis (e.g., Carretie, Mercado, & Tapia, 2000; Dawson, Panagiotides, Klinger, & Hill, 1992).

[5] Because of research that suggests depression and anxiety are often highly correlated (e.g., Katon & Roy-Byrne, 1991) and research that suggests that anxious apprehension is associated with decreased right frontal activity (e.g., Heller, Nitschke, Etienne, & Miller, 1997), whereas anxious arousal is associated with increased right frontal activity (Nitschke et al., 1999), we examined the effects of HB and D on frontal asymmetry indices (and we examined left and right activity separately) while statistically controlling for baseline reported fear, a proxy of anxiety. Statistically adjusting for fear in these regression analyses did not alter the size or significance of any of the reported effects. Moreover, the results of Heller et al. (1997) suggest that individuals with anxious apprehension show decreased right frontal activity but not increased left frontal activity. In order for anxious apprehension to explain our results, HB should relate to anxious apprehension, decreased right frontal activity, and not increased left frontal activity. Our results are not in accord with this interpretation because (a) HB related to increased left frontal activity, and (b) HB is not positively associated with anxious apprehension. In fact, Lovejoy and Steuerwald (1992) found that HB was negatively associated with anxious apprehension when controlling for D.

research on anger and frontal brain activity by revealing individual difference characteristics that predict who is more likely, as well as less likely, to respond with increased left frontal activity in anger-inducing situations. That is, individuals with proneness toward hypomania/mania symptoms evidence greater relative left frontal activity, whereas individuals with proneness toward depressive symptoms evidence lesser relative left frontal activity when confronted with an anger-evoking event. From these results, it seems plausible to predict that proneness toward hypomania/mania symptoms may predispose persons toward responding with increased approach (and decreased withdrawal) motivational tendencies given challenging or frustrating situations, whereas proneness toward depressive symptoms may predispose persons toward responding with decreased approach (and increased withdrawal) motivational tendencies given these same situations. In other words, proneness toward hypomania/mania symptoms may lead to reactance-like responses, and proneness toward depressive symptoms may lead to helpless responses in the face of challenges (e.g., Abramson, Metalsky, & Alloy, 1989; Mikulincer, 1988; Wortman & Brehm, 1975).

Future Directions

The present results suggest several lines of inquiry that would further develop the theoretical understanding of the involvement of the BAS in proneness to affective disorders. For instance, does relative left frontal activity in response to anger-evoking events characterize individuals who are prone to hypomania/mania regardless of their current state, or is it a characteristic of the hypomanic/manic phase itself that changes when individuals go into a different phase? Although our results cannot definitively address this question, our regression analyses in which HB related to relative left frontal activity in response to the anger challenge while controlling for D suggest that individuals who are prone to hypomania and frequently in the state, regardless of their proneness to depression, show relative left frontal activation when exposed to anger cues. Future research should address this question by examining such individuals when they are in a hypomanic/manic state compared with when they are not.

Future research will also need to test whether hypomanic symptoms relate to relative left frontal activity in other situations that would engage the BAS. On the basis of the present research as well as past and present theorizing, we would expect that hypomanic symptoms would relate to relative left frontal activation in other appetitive situations.

It is also important to note that the present evidence supported theoretically derived predictions, even in a sample of subsyndromal individuals who merely self-reported behavioral tendencies that may indicate proneness toward hypomania/mania or depression. This research suggests that subsyndromal scores on the GBI predict neurophysiological responses in anger-producing situations, as would be expected on the basis of theory. Future research will need to examine whether individuals who respond with exaggerated approach motivational responses are more likely to develop psychopathological syndromes, and whether individuals with clinical syndromes show the same pattern of results as those with subclinical tendencies.

Finally, given past research, which suggested that increased cardiovascular responsiveness (particularly heart rate) is an index of BAS activation (e.g., Arnett & Newman, 2000; Fowles, 1980, 1988; Tomaka & Palacios-Esquivel, 1997), it would be potentially fruitful to examine cardiovascular responses of individuals with tendencies toward mania and depression in approach motivational situations. In addition to more fully elucidating theoretical models integrating the BAS, psychopathology, and autonomic responses, such research might shed light on the oft-observed relationship between trait anger and cardiovascular disease and morbidity (e.g., Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Williams, Paton, Seigler, Eigenbrodt, Nieto, & Tyroler, 2000), and other research linking anger with cardiovascular reactivity (e.g., Smith, 1992). It has been suggested that the elevated cardiovascular reactivity associated with anger may lead to cardiovascular disease and death (e.g., Suarez & Williams, 1989), which may suggest that hypomania/mania may predispose individuals toward exaggerated cardiovascular reactivity in approach-related situations and ultimately may lead to cardiovascular problems.

Conclusion

The present research extends recent research on the relationship between relative left frontal cortical activity and anger by further suggesting that BAS functioning is not exclusively related to positive affect. Important to add, the present research builds on the past research on anger and left frontal cortical activity by providing individual difference characteristics that predict BAS-related responses in anger-producing situations.

In addition to furthering our understanding of anger, the BAS, and the functions of asymmetrical frontal cortical activity, the present research integrated theories concerned with motivation, emotion, brain function, and psychopathology to generate predictions regarding the relationships between proneness toward affective disorder symptoms and responses to anger-producing situations. This integrative theoretical approach holds promise for increasing our understanding of the motivational and neurophysiological underpinnings of individual difference characteristics that may predispose individuals toward certain affective disorders.

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