Journal of Abnormal Psychology
2007, Vol. 116, No. 1, 95–104

0021-843X/07/$12.00 DOI: 10.1037/0021-843X.116.1.95

Reward Sensitivity in Depression: A Biobehavioral Study

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The approach–withdrawal model posits 2 neural systems of motivation and emotion and hypothesizes that these systems are responsible for individual differences in emotional reactivity, or *affective styles*. The model also proposes that depression is characterized by a deficit in reward-seeking behavior (i.e., approach motivation) and is associated with a relative decrease in left frontal brain activity. The authors tested aspects of this model by comparing the electroencephalogram alpha power of depressed and nondepressed individuals during a task that manipulated approach motivation. The study found that control participants and individuals with late-onset depression exhibited the hypothesized increase in left frontal activity during the approach task but individuals with early-onset depression did not. This suggests that early-onset depression may be associated with a deficit in the hypothesized approach motivation system.

Keywords: EEG asymmetry, early-onset depression, approach motivation, personality

There has long been a great deal of interest in individual differences in approach and avoidance motivation (Dollard & Miller, 1950). Recently, researchers have begun to examine the neuropsychological basis of these motivational tendencies. One of the more widely studied of these models has been Davidson's (1992, 1998) approach–withdrawal model. This model posits two separate systems of motivation and emotion—an approach system and a withdrawal system. The approach system is hypothesized to control appetitive and goal-directed behavior and is proposed to respond to incentive, reward, and other positive stimuli (Davidson, 1998; Depue & Collins, 1999; Fowles, 1994; Gray, 1994). As the organism moves closer to an appetitive goal, the approach system is also viewed as being responsible for the generation of certain positive affects.

Davidson (1992) also posited that the approach system is represented by neural circuits that involve different regions of the frontal cortex. Specifically, relatively greater activity in the left prefrontal cortex is hypothesized to be associated with activation of the approach system (Davidson, 1992, 1998). Several electroencephalogram (EEG) studies have tested this hypothesis by examining the relative alpha power of electrodes placed over anterior regions of the right and left hemispheres. Although the use of alpha power as a measure of brain activity in frontal regions is controversial (Tenke & Kayser, 2005),[1] several studies have found associations between frontal alpha asymmetries and these motivational states (Davidson, Marshall, Tomarken, & Henriques, 2000; Miller & Tomarken, 2001).

The model also proposes that abnormalities in the approach system play an etiological role in depression (Davidson, 1998; Fowles, 1994; Kring & Bachorowski, 1999). Depression is seen as a deficit in approach motivation (Davidson, 1998; Gray, 1994), and thus depressed people are viewed as being less responsive to rewards (Meehl, 1975). Hence, similar to other biobehavioral models of depression (Clark & Watson, 1991; Depue & Collins, 1999), this theory focuses on the anhedonic element of depression. In support of this theory, several experimental studies have reported that dysphoric college students and individuals with major depression exhibited decreased responsiveness to reward compared with control participants (Henriques & Davidson, 2000; Henriques, Glowacki, & Davidson, 1994; although see Layne, Gross, & Buckley, 1980, for counterevidence).

Because the left prefrontal region is hypothesized to be part of the approach system and depression is proposed to arise from an underactivated approach system, the approach–withdrawal

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This work is based on a dissertation submitted to the Graduate School of Stony Brook University by Stewart A. Shankman. This study was supported by the American Psychological Foundation and the Council of Graduate Departments of Psychology Clarence J. Rosecrans Scholarship as well as National Institute of Mental Health Grant F31 MH67309, both awarded to Stewart A. Shankman. We acknowledge the consultation and support of Jurgen Kayser and the assistance of Suzanne Rose, Kathryn Messineo, Jana Kramer, and Justine Caiaccia in data collection and coding.

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[1] The premise that alpha power is inversely related to brain activity equally throughout the brain is controversial (Allen et al., 2004; Tenke & Kayser, 2005). There are some data to suggest that alpha power is inversely correlated with other measures of brain activity such as positron emission tomography and functional magnetic resonance imaging (Goldman, Stern, Engel, & Cohen, 2002; Oakes et al., 2004). Moreover, during neuropsychological tasks that are known to tap specific cortical regions, alpha power recorded over those regions has been shown to be associated with decreased performance (see Allen et al., 2004, for a review). More research is needed, however, on this issue in order to fully conclude that alpha power is an inverse measure of brain activity. Nevertheless, for ease of presentation, the term *brain activity* is used as a heuristic for inverse alpha power throughout this article.

model hypothesizes that depression should be associated with decreased activity in left prefrontal regions of the brain, that is, a frontal asymmetry (Davidson, 1994, 1998). In support of the model, individuals with clinical depression (Debener et al., 2000; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991), individuals who have recovered from episodes of depression (Gotlib et al., 1998; Henriques & Davidson, 1990), and children of depressed mothers (Dawson et al., 1999; Field, Fox, Pickens, & Nawrocki, 1995; Tomarken, Dichter, Garber, & Simien, 2004) have exhibited the hypothesized frontal asymmetry relative to control participants (although see Henriques, 1998; Reid, Duke, & Allen, 1998, for contrary evidence). On the basis of these findings, approach–withdrawal theorists have suggested that frontal EEG asymmetry is a biological marker for an affective style that predisposes individuals to depression (Davidson, 1992).

There is an important question, however, concerning the validity of the approach–withdrawal model, particularly regarding its predictions for depression (Shankman & Klein, 2003). Despite behavioral and EEG evidence supporting the approach–withdrawal model, no study has directly examined whether an EEG frontal asymmetry is associated with a lack of responsiveness to reward in depressed individuals. The majority of the EEG studies with depressed participants have examined hemispheric activity only during rest (i.e., participants were not performing any particular task); the results of which are then related to the findings from behavioral studies of reward responsiveness in depression. In other words, the literatures on the EEG and behavioral correlates of depression have been almost entirely independent. The present study thus attempts to provide a more scrupulous test of the approach–withdrawal model by comparing the brain activity (operationalized as a decrease in EEG alpha power) of depressed and nondepressed individuals during the hypothesized motivational state (i.e., while anticipating the possibility of reward) instead of at rest.

Heterogeneity of Depression: Age of Onset and Chronicity

The model proposes that these brain asymmetries may not be evident for all individuals with depression but only for depressed individuals with the hypothesized *low approach affective style*. Several lines of evidence suggest that individuals with early-onset and/or chronic depressions may be more likely to have this affective style than those with late-onset and/or nonchronic depressions. First, an early onset and chronic course are consistent with the notion of a temperamentally based affective style. Second, early-onset and chronic depressions are associated with greater personality abnormalities (Klein, Durbin, Shankman, & Santiago, 2002). In particular, depressed individuals with these characteristics exhibit lower levels of extraversion (which is closely related to low approach) than do depressed individuals without these characteristics (Hirschfeld, 1990; Klein, Taylor, Dickstein, & Harding, 1988). Third, preschool age children of mothers with depressive disorders, and particularly of mothers with early-onset and/or chronic depression, exhibit less positive affect and approach behavior in home (Neff & Klein, 1992) and laboratory observations (Durbin, Klein, Hayden, Buckley, & Moerk, 2005). Finally, in a follow-up of a large British birth cohort, children rated as behaviorally apathetic had a significantly increased risk for adolescent-onset depression and chronic depression in adulthood (van Os, Jones, Lewis, Wadsworth, & Murray, 1997). Therefore, we hypothesize that individuals with early onset and/or chronic forms of depression will exhibit lower levels of the putative *approach affective style*, as reflected by a frontal asymmetry, than will individuals with late-onset and/or nonchronic forms of depression and nondepressed individuals.

Method

Participants

The sample consisted of 70 individuals with current major depression (MDD), as defined by the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994), and 37 control participants. The control group was required to have no lifetime diagnoses of MDD, dysthymia, anxiety disorder, hard drug or alcohol dependence, or anorexia or bulimia nervosa. The control group was also required to have a 24-item Hamilton Rating Scale of Depression (HRSD; Hamilton, 1960) score of less than 8. All participants were recruited through advertising in the community (e.g., newspapers, flyers, Internet postings) and psychiatric and psychological clinics. The demographics (e.g., gender, education, age) of the two groups were closely monitored during recruitment in order to ensure that the two groups were comparable on these variables. All participants gave informed consent and were paid for their participation.

For this study, chronic depression was defined as MDD in which the present episode was at least 2 years long or MDD superimposed on dysthymic disorder (i.e., *double depression*). Previous research has shown that participants with these two forms of chronic depression have similar demographic and clinical characteristics and treatment response (McCullough et al., 2003). Participants with nonchronic depression never had a period of chronic depression. For age of onset, we used the onset of the earliest affective disorder (dysthymia or major depression).

Participants were excluded from the study if they had a lifetime diagnosis of schizophrenia or other psychotic disorder, bipolar disorder, or dementia; were unable to read and write English; had a history of head trauma in which they lost consciousness; or were left-handed (as determined by a prescreen during recruitment and confirmed by the Edinburgh Handedness Inventory, Oldfield, 1971, range of laterality quotient = +20 to +100). Eight participants were also excluded from analyses because they did not yield enough artifact-free EEG data (5 depressed participants and 3 control participants). Thus, the final sample consisted of 99 participants—34 control participants and 65 individuals with current MDD (mean age of onset = 18.8 years, SD = 10.2; 34 with chronic depression and 31 with nonchronic depression). As shown in Table 1, depressed and control participants did not differ on demographic variables. As would be expected in comparing a psychiatric with a control group, the depressed sample had lower Global Assessment of Functioning (GAF) scores, $t(97) = 21.7$, $p < .001$; less state, $t(97) = 2.48$, $p < .05$, and trait, $t(97) = 6.65$, $p < .001$ positive affect; and more state, $t(97) = 2.50$, $p < .05$, and trait, $t(97) = 10.62$, $p < .05$, negative affect than did the control group. Table 1 also presents the clinical characteristics of the 65 individuals with current MDD.

Interview and Self-Report Measures

Diagnoses were made via the Structured Clinical Interview for *DSM–IV* (SCID; First, Spitzer, Gibbon, & Williams, 1996). Severity of depressive symptomatology was assessed using the HRSD. The assessments were conducted by Stewart A. Shankman and a master's level diagnostician. The diagnostician has demonstrated high levels of interrater reliability in the past and has trained numerous diagnosticians on the SCID and HRSD for 10 years (Keller et al., 1995; Klein, Schwartz, Rose, & Leader, 2000). She

Table 1
Characteristics of the Sample and Association With Age of Onset and Chronicity of Depression

Variable	Control ($n = 34$)	Major depression ($n = 65$)	Association with chronicity[a]	Association with age of onset of depression[b]
Demographic				
Gender			$\chi^2(1) = 1.71$, *ns*	$t(63) = 1.33$, *ns*
Female	70.6%	66.2%		
Male	29.4%	33.8%		
Age	33.8 years	33.6 years	$t(63) = 1.12$, *ns*	$r = .45$, $p < .001$
Caucasian	73.5%	76.9%	$\chi^2(1) = 0.25$, *ns*	$t(63) = 1.14$, *ns*
Employed	82.4%	78.5%	$\chi^2(1) = 4.93$, $p < .05$	$t(63) = 0.55$, *ns*
Laterality quotient[c]	+86.9	+93.9	$t(63) = 0.80$, *ns*	$r = .18$, *ns*
Questionnaire				
General Temperament Survey				
Positive Emotionality			$F(1, 62) = 11.1$, $p < .01$	$pr = .22$, $p < .09$
M	20.2[d]	11.7		
SD	5.0	6.5		
Negative Emotionality			$F(1, 62) = 1.89$, *ns*	$pr = -.36$, $p < .01$
M	4.4[d]	20.0		
SD	6.0	−7.4		
PANAS ratings				
Positive affect			$F(1, 62) = 0.01$, *ns*	$pr = .37$, $p < .01$
M	26.8[d]	22.9		
SD	8.2	5.7		
Negative affect			$F(1, 62) = 0.01$, *ns*	$pr = -.19$, *ns*
M	11.5[d]	14.5		
SD	2.8	5.5		
Clinical				
Global Assessment of Function	85.1[d]	53.4	$t(63) = 6.35$, $p < .001$	$r = .02$, *ns*
HRSD			$t(63) = 2.48$, $p < .05$	$r = .07$, *ns*
M	1.8[d]	26.1		
SD	1.8	7.5		
Currently taking medication		49.2%	$\chi^2(1) = 21.2$, $p < .001$	$t(63) = 0.15$, *ns*
Lifetime psychiatric hospitalization		10.8%	$\chi^2(1) = 1.15$, *ns*	$t(63) = 1.24$, *ns*
Duration of major depression			$t(63) = 2.97$, $p < .01$	$r = -.14$, *ns*
M		34.2 months		
SD		84.1 months		
Recurrent major depression		81.5%	$\chi^2(1) = 0.03$, *ns*	$t(63) = 0.06$, *ns*
Current anxiety disorder[e]		33.8%	$\chi^2(1) = 1.71$, *ns*	$t(63) = 1.58$, *ns*
Lifetime alcohol abuse/dependence disorder		38.5%	$\chi^2(1) = 0.96$, *ns*	$t(63) = 1.16$, *ns*
Lifetime drug abuse/dependence disorder		21.5%	$\chi^2(1) = 1.02$, *ns*	$t(63) = 0.11$, *ns*

Note. PANAS = Positive and Negative Affect Schedule.
[a] Chi-squares are reported for dichotomous characteristics (e.g., gender), and *t* tests are reported for continuous demographic and clinical characteristics (e.g., age). For the questionnaires, analyses of covariance control for Hamiltan Rating Scale of Depression (HRSD) scores. [b] *t* tests are reported for dichotomous characteristics (e.g., gender) and Pearson *r*s are reported for continuous characteristics (e.g., age). The partial correlations control for HRSD scores. [c] Laterality quotients can vary between −100 (completely left-handed) and +100, (completely right-handed). [d] Significantly different from depressed group, $p < .05$. [e] Panic disorder, agoraphobia, social phobia, specific phobia, obsessive–compulsive disorder, posttraumatic stress disorder, or generalized anxiety disorder.

trained Stewart A. Shankman to criterion, and diagnoses were regularly discussed in best estimate meetings (Klein, Ouimette, Kelly, Ferro, & Riso, 1994).

All participants completed the General Temperament Survey (Clark & Watson, 1990), a self-report measure of trait positive and negative emotionality. Cronbach's alpha for the positive and negative emotionality scales were high in the depressed and nondepressed groups (αs $> .82$). Before and after the experiment, participants completed the Positive and Negative Affect Schedule (with "right now" instructions; Watson, Clark, & Carey, 1988), so that we could assess their current, momentary positive affect and negative affect. The two administrations were highly correlated (Pearson *r*s $> .54$ for positive affect and negative affect in depressed and control participants); so the two administrations were averaged to provide a more stable measure of affect.

Procedure

We used a bogus computerized slot machine paradigm (see Figure 1 for an image of the game) that consisted of three reels of numbers and fruit spinning simultaneously for exactly 11 s. The game consisted of 36 spins, which were divided into two different payoff situations of 18 trials each—reward (R) and no incentive (NI).[2] The 36 trials were presented in a pseudo-random order in which there were never more than 2 consecutive trials of similar type or outcome. Participants began the game with $5.00

[2] The game also included 18 loss trials so that we could explore whether anticipation of losing money was related to withdrawal motivation. This manipulation was ineffective, and the results from this condition are thus not discussed in this study.

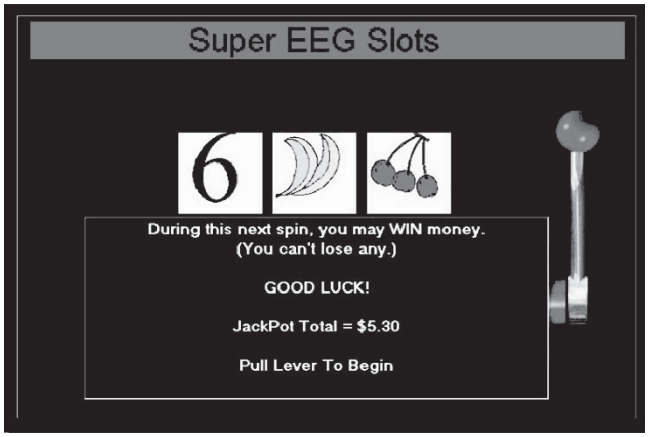

Figure 1. Picture of the electroencephalogram (EEG) slot machine game (reward condition).

in the bank and were notified of the specific payoff situations prior to each trial (i.e., whether it was an R or NI trial). To begin each trial, participants pressed a button with both thumbs to pull a lever on a computer screen that started the reels spinning. After the 11 s, the reels stopped spinning and a message appeared on the screen notifying the participant of the outcome. The amount of money won varied from $0.30 to $0.45.

The parameters for the R and NI conditions were similar in that a winning spin occurred when the reels landed on three pieces of fruit (e.g., three cherries or bananas). The difference between these conditions was that participants won money if this outcome occurred during the R condition but they did not win money if it occurred during the NI condition. The participant was not penalized during the R and NI conditions when this outcome did not occur.

The R condition was designed to elicit approach motivation. The NI condition served as a control for several aspects of the R condition. During both conditions, the participant was watching a slot machine game (i.e., the visual input was the same). In addition, the participant was anticipating an outcome, though in the NI condition the outcome did not have monetary implications.

Unbeknownst to the participant, the slot machine program was manipulated so that half of the two payoff situations landed on three fruits. There were thus four possible conditions–outcomes consisting of nine trials each: R–win money, R–not win money, NI–win, and NI–lose.

We told participants that the trials were in a random order to reduce the likelihood that they would perceive that they had control over the outcome of each trial. They were instructed to sit still and focus their gaze on the game. Participants were allowed to take breaks to rest their eyes between trials and were given longer breaks as needed. At the end of the 36 trials, all participants were given their winnings in cash. All procedures were approved by the Stony Brook University Institutional Review Board.

EEG Recording

EEGs were recorded in a sound attenuated booth. Electrodes were placed according to a modified version of the 10–20 system (Sharbrough et al., 1991). EEG was recorded from two homologous pairs of electrodes overlapping frontal (F3/F4; F7/F8), central (C3/C4; T7/T8), and posterior (P3/P4; P7/P8) brain regions over both left and right hemispheres and from one midline electrode (Cz) with a stretch-lycra electrode cap (Electro-Cap International, Inc., Eaton, OH; tin electrodes). The ground electrode was at the frontal pole (Fpz). The reference electrode was placed on the left earlobe (A1). Data were also recorded from the right earlobe (A2), enabling computation of an offline digitally derived "linked ears" reference. Electrodes were placed at right supra- and infra-orbital sites to monitor for eye blinks and vertical eye movements and right- and left-outer canthi to monitor horizontal eye movements. Electrode impedances were under 5,000 ohms, and homologous sites (e.g., F3/F4) were within 1,500 ohms of each other. The electrode cap was connected to a chest harness to reduce the likelihood of movement. Data were recorded through a Grass Neurodata acquisition system (Grass Technologies, West Warwick, RI) at a gain of 10 K (5 K for eye channels) with a bandpass of 1–30 Hz. A PC-based EEG acquisition system (Neuroscan, 2003) acquired and digitized the data continuously at a rate of 1000 Hz.

Continuous EEG from the 11-s spinning interval was segmented into consecutive 1.024-s epochs every 0.512 s (50% overlap). Data were then examined for evidence of amplifier saturation. After referencing to a linked ear reference offline and then applying a baseline correction, epochs contaminated by blinks, eye movements, and movement related artifacts were excluded from analyses by direct visual inspection of the raw data. This method of dealing with artifacts has been shown to preserve spectral power across bands better than the widely used method of electrooculogram correction (Somsen & van Beek, 1998). The EEG was tapered over the entire 1.024-s epoch by a Hanning window to suppress spectral side lobes. Artifact-free data, which were attenuated at the beginning and end of an epoch, were recovered in adjacent (overlapping) epochs. For the R and NI conditions, there were a mean of 218 ($SD = 61$) and 208 ($SD = 62$) epochs, respectively, and t tests revealed that depressed and control participants had comparable numbers of epochs in each condition.

Power spectra were computed offline from EEG data by using a fast Fourier transform. An examination of the topography of the power spectra for each condition of the slot machine task indicated a distinct alpha peak at approximately 10 Hz that was maximal in posterior and medial electrodes, relative to frontal and lateral electrodes, respectively. This was supported by significant main effects for region, $F(2, 194) = 94.1$, $p < .001$, Greenhouse–Geisser (G-G) $\varepsilon = .774$, and medial–lateral, $F(1, 97) = 779.8$, $p < .001$, in the analyses of variance (ANOVAs) described below. It is particularly important to verify the characteristics of alpha power for this task as it is not the typical paradigm for which alpha power is usually assessed (i.e., while the participant is at rest). The average absolute alpha power was computed for each electrode site and then natural log transformed in order to normalize the data. For consistency with previous research (Bruder et al., 1997; Henriques & Davidson, 1990), the alpha band was defined as 7.81–12.70 Hz and used as an inverse measure of regional brain activity. This range was validated by assuring that the alpha peak was approximately centered in this band for every participant.

Data Analyses

Group differences in asymmetries were tested using four-way ANOVAs in SPSS 13.0 that included three within-subject factors—hemisphere (right vs. left), condition (R vs. NI), and region (frontal vs. central vs. posterior)—and one between-subjects factor—group (e.g., depressed vs. control). When a medial–lateral repeated measures factor was added to this ANOVA model, there was no significant interaction with this factor and the pattern of results was nearly identical. Analyses were thus restricted to the less noisy medial electrodes (e.g., F3/4, C3/4, and P3/4). A G-G correction was used for repeated measures analyses involving factors with more than two levels (e.g., frontal vs. central vs. posterior region), and for these analyses, G-G epsilons are included. The condition main effect and Condition × Region, Condition × Group, and Condition × Region × Group interactions were not significant in any of the analyses below, suggesting that there was no overall bilateral difference in activity in the R versus NI conditions or any group differences in bilateral activity. We used partial eta squared (η_p^2) as a measure of effect size [$SS_{effect}/(SS_{effect} + SS_{error})$].

Because hemispheric asymmetry measures may differ depending on the reference scheme (Allen, Coan, & Nazarian, 2004; Bruder et al., 1997;

Hagemann, 2004; Tenke & Kayser, 2005) and can be affected by individual differences in scalp thickness (Pivik et al., 1993), the epoched data were also re-referenced to an average scalp electrode reference. These results were very similar to those found when we used the digitally derived linked ear reference. Thus, only the results with the linked ear reference are reported.

First, we examined the experimental manipulation in control participants only. Next, all participants with depression were compared with control participants. Last, we tested whether the hypothesized clinical characteristics (early onset and chronic depression) were related to EEG asymmetries. In order to include a continuous independent variable such as age of onset in the general linear model repeated measures ANOVA procedure in SPSS 13.0, we had to enter age of onset as a covariate.

Results

Control Participants Only

The first set of analyses tested whether the EEG asymmetries during the R and NI conditions differed. The initial group of analyses was limited to the control group so that we could test whether the experimental manipulation was effective at manipulating frontal EEG asymmetry. The three-way Condition × Hemisphere × Region interaction was not significant, but the two-way Condition × Hemisphere interaction was significant, $F(1, 33) = 4.19$, $p < .05$, $\eta_p^2 = .11$. These results indicate that the overall hemispheric asymmetry was different between the two conditions, but this effect did not vary by region. Because the hypotheses were for the frontal region, a two-way Condition × Hemisphere interaction was run for the frontal region alone, $F(1, 33) = 3.06$, $p < .09$, $\eta_p^2 = .09$ (see Figure 2). Although this interaction was only a trend, the effect was in the hypothesized direction (greater relative brain activity [less alpha] in the left frontal regions during the R condition compared with the NI condition), and the effect size was medium (Cohen, 1988). Given that there may be a ceiling on how much the asymmetry of a group free of lifetime psychopathology and high on positive emotionality could change, these results suggest that the reward manipulation was at least somewhat effective.

All Depressed Participants Versus Control Participants

The next set of analyses was similar to those above but compared control participants with the entire group of participants with depression. Neither the four-way Condition × Hemisphere × Region × Group interaction nor the three-way Condition × Hemisphere × Group interaction was significant ($ps > .1$). These results suggest that there was no condition-dependent difference in hemispheric asymmetry among groups and that the asymmetries did not vary by region. Because the hypotheses were for frontal regions, a three-way interaction (Condition × Hemisphere × Group) was run for this region but was not significant, $F(1, 97) = 1.81$, $p = .18$, $\eta_p^2 = .02$. However, the lack of an overall group effect could be masked by the heterogeneity of depression. Hence the next set of analyses distinguished between early- and late-onset depression and chronic and nonchronic forms of depression.

Early-Onset and Chronic Depression

As shown in Table 1, the only clinical or demographic variable that was associated with age of onset was age at EEG assessment. Those with chronic and nonchronic depression differed on employment status, GAF, HRSD, and medication status. Participants with chronic depression also had earlier onsets of depression than did those with nonchronic depression ($M = 15.8$, $SD = 11.0$, vs. $M = 22.2$, $SD = 8.2$), $t(63) = 2.62$, $p < .05$.

Next, we examined whether chronic or early-onset depressions were associated with the hypothesized pattern of reduced relative left frontal activity in participants while anticipating the possibility of a reward. These analyses were restricted to those with depression ($n = 65$), and age of onset and chronicity were examined as continuous and categorical independent variables, respectively.

Chronicity was not related to the hypothesized asymmetries during the reward condition: Condition × Hemisphere × Region × Chronicity interaction, $F(2, 126) = 0.32$, $p = .32$, G-G $\varepsilon = .85$; Condition × Hemisphere × Chronicity, $F(1, 63) = 0.04$, $p = .84$. Age of onset of depression, however, was related to the

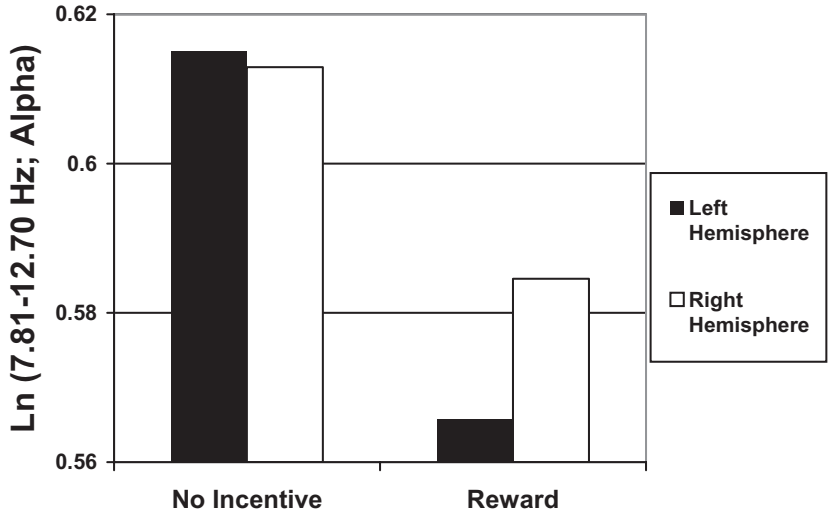

Figure 2. Frontal asymmetries of control participants ($n = 34$) during reward and no incentive conditions.

hypothesized asymmetries. The Condition × Hemisphere × Region × Age of Onset interaction yielded a trend, $F(2, 126) = 2.94$, $p = .06$, G-G $\varepsilon = .85$, $\eta_p^2 = .05$. Because of specific hypotheses for the frontal region, we followed this trend up with separate three-way interactions for each of the three regions (frontal, central, and posterior). Although the central and posterior three-way interactions were nonsignificant, $F(1, 63) = 0.21$, $p = .65$, and $F(1, 63) = 0.34$, $p = .56$, respectively, the three-way interaction for the frontal region was significant, $F(1, 63) = 6.52$, $p = .01$, $\eta_p^2 = .09$. In other words, the trend for the four-way interaction appeared to be due to differences in the frontal region and not to differences in any other region. The effects were even stronger when age of onset of major depression was used in the model instead of age of onset of earliest affective disorder: three-way interaction, $F(1, 63) = 10.84$, $p = .002$, $\eta_p^2 = .15$.

We next performed a median split on age of onset of depression and labeled depressed participants whose onset was before 17 *early onset* ($n = 31$) and depressed participants whose onset was 17 or older *late onset* ($n = 34$). Although dichotomizing continuous variables is rarely an optimal data analytic strategy (Maxwell & Delaney, 1993), it was required for the present analyses so that we could include the nondepressed control group in the analyses. We ran a Condition (R vs. NI) × Hemisphere (right vs. left) × Age of Onset (control vs. early onset vs. late onset) ANOVA for the frontal region, which yielded a significant three-way interaction, $F(2, 96) = 3.92$, $p = .02$, $\eta_p^2 = .08$. This interaction is displayed in Figure 3.

Figure 3 represents the full three-way interaction (Condition × Hemisphere × Age of Onset) for the frontal region. Under the assumption that alpha power is an inverse measure of brain activity (see Footnote 1), the *y*-axis represents the brain activity in the R condition minus the brain activity in the NI condition. That is, if there is more alpha power in the NI condition than in the R condition (i.e., NI − R is positive), then there is less activity in the NI condition than in the R condition. This is analogous to the use of a difference waveform in an event-related potential study in which a condition is controlled for (or subtracted out of) an active condition.

Figure 3 illustrates that the control participants and individuals with late-onset depression exhibited the hypothesized frontal asymmetry (i.e., relative increased left frontal brain activity while anticipating the possibility of reward) whereas the individuals with early-onset depression did not. To test this statistically, we followed up this three-way interaction by comparing asymmetries for the three diagnostic groups with each other (i.e., Condition × Hemisphere × Group [control vs. early onset]). Participants with early-onset depression had different frontal asymmetries from both those with late-onset depression, $F(1, 63) = 6.06$, $p = .02$, $\eta_p^2 = .09$, and control participants, $F(1, 63) = 5.38$, $p = .02$, $\eta_p^2 = .08$. Participants with late-onset depression and control participants did not differ, $F(1, 66) = 0.01$, $p = .99$.

To explore the independent and joint effects of chronicity and age of onset, we conducted an ANOVA using both variables as between-subjects factors. Age of onset remained a significant predictor of frontal asymmetry over and above chronicity, and the Age of Onset × Chronicity interaction was not significant.

Next, we considered potential confounding demographic and clinical variables. As shown in Table 1, the only clinical or demographic variable that was associated with age of onset was age at EEG assessment. We thus ran a Condition × Hemisphere × Age of Onset ANOVA with current age as an additional independent variable. This ANOVA continued to yield a significant Condition × Hemisphere × Age of Onset interaction, indicating that age of onset was related to frontal asymmetry over and above the

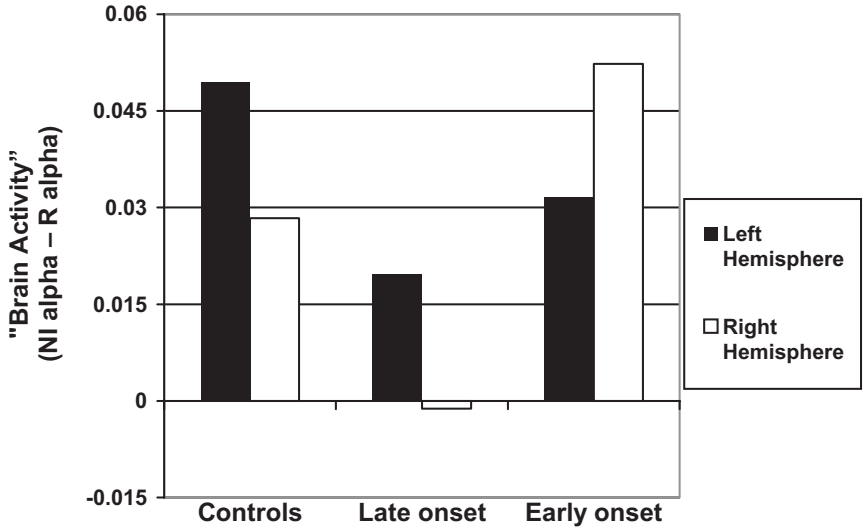

Figure 3. Frontal asymmetries of control participants ($n = 34$) and individuals with early-onset (<17 years old; $n = 31$) and late-onset (≥17 years old; $n = 34$) major depression during the approach task. Under the assumption that alpha power is an inverse measure of brain activity, the *y*-axis represents the brain activity in the reward (R) condition minus the brain activity in the no incentive (NI) condition (if greater alpha power in the NI condition than in the R condition [i.e., NI − R is positive], then there is less activity in the NI condition than in the R condition).

effects of age at the time of the assessment. The same pattern was found when medication status, gender, and depression severity (as assessed by the HRSD) were entered into similar ANOVAs.

Our hypothesis that early-onset (and chronic) depression would be associated with frontal asymmetries was based, in part, on evidence that persons with early onset and chronic forms of depression have greater personality disturbances, including lower levels of extraversion and/or positive emotionality. As shown in Table 1, age of onset of depression was associated with the self-report measures of affect and emotionality. Controlling for depression severity, we found that individuals with an earlier age of onset reported more negative emotionality and a trend for less positive emotionality. Additionally, an earlier age of onset was associated with a lower average rating of positive affect at the time of the EEG assessment. Chronic depression was associated with less positive emotionality.[3]

Discussion

This study tested hypotheses from the approach–withdrawal model, a widely studied neuropsychological model of emotion and emotional disorders. The model hypothesizes that individuals with a low motivation for appetitive and goal-directed behavior (i.e., low approach) are at risk for depression and exhibit an asymmetry of frontal brain activity due to reduced activity in left frontal regions. Whereas most of the EEG studies testing this model have recorded EEG while participants were at rest, this study recorded EEG during a task designed to elicit the hypothesized motivational states (i.e., while participants were anticipating the possibility of a reward).

To check the effectiveness of the reward manipulation, we examined whether control participants exhibited the hypothesized frontal asymmetry of greater relative left than right frontal brain activity during the R condition compared with the NI condition. Although this analysis only reached a trend level of significance ($p < .09$), it was in the hypothesized direction (see Figure 2), with a medium sized effect. There may be a ceiling as to how much an asymmetry could change in a high trait positive emotionality group with no history of psychopathology. We thus interpret these results as providing at least moderate support for the validity of the reward manipulation.

Early-Onset Depression and EEG Asymmetry During Anticipation of Reward

The hypothesis of this study was that participants with depression, and particularly those with early-onset and/or chronic forms of depression, would exhibit different frontal asymmetries than nondepressed control participants while anticipating the possibility of a reward. Depressed participants as a group, and the subgroup of chronically depressed participants, did not differ from nondepressed individuals. However, consistent with our hypothesis, we found that individuals with early-onset depression exhibited different frontal asymmetries than did control participants for the R condition. Individuals with early-onset depression also exhibited different frontal asymmetries than did individuals with late-onset depression, suggesting that the finding was not due to participants being depressed at the time of the assessment. Moreover, this finding was also not attributable to severity of depression or other clinical or demographic variables.

The results of this EEG study support the hypothesis that individuals with early-onset depression make up a distinct subgroup that exhibits a deficit in approach motivation, that is, a low approach affective style (Davidson, 1994, 1998). However, there are several possible interpretations of this finding. First, it is possible that individuals with a low approach affective style are more likely to develop depression early in life (i.e., the affective style is a predisposing or vulnerability factor). Consistent with this interpretation, young children who exhibit low levels of positive emotionality (including low positive affect and lack of approach behavior) in laboratory and home observations have an elevated rate of depression (particularly early onset and chronic forms) in their mothers (Durbin et al., 2005; Neff & Klein, 1992), exhibit neurophysiological characteristics that have been associated with risk for depression (Shankman et al., 2005), and display increased levels of depressotypic cognitive features at age 7 (Hayden, Klein, Durbin, & Olino, 2006).

A second interpretation of the age of onset finding is that having depression early in life caused the abnormal frontal asymmetry. Certain areas of the prefrontal cortex that are hypothesized to be related to the approach system (e.g., dorsolateral prefrontal cortex; Davidson, Pizzagalli, Nitschke, & Putnam, 2002) do not fully mature until adolescence or early adulthood; so perhaps an early onset of depression disrupts brain development. This interpretation would also be consistent with the finding that an early onset of depression leaves a "scar" on personality and psychosocial functioning (Rohde, Lewinsohn, & Seeley, 1994). This explanation, however, cannot account for other studies that reported the hypothesized frontal asymmetry in infants and never depressed adolescents with a familial risk for depression (Dawson et al., 1999; Field et al., 1995; Tomarken et al., 2004). Moreover, there is no evidence to suggest that the prefrontal cortex has to be fully developed in order to exhibit the hypothesized asymmetry.

A third interpretation of the finding is that a third variable caused both an early onset of depression and a low approach affective style. For example, it is possible that a psychosocial stressor (e.g., trauma) caused individuals to have a reduced anticipation of rewards and an early onset of depression (Ashman & Dawson, 2002). Alternatively, it is possible that the same gene or cluster of genes leads to both characteristics.

[3] We also recorded resting EEG (with eyes open and closed). It is interesting to note that the frontal asymmetry recorded at rest was not correlated with the frontal asymmetry recorded during the slot machine task in either the control or depressed groups ($ps > .4$). In addition, age of onset of depression was not related to asymmetry at rest, suggesting the importance of manipulating reward in examining EEG hemispheric asymmetries. We also examined whether the questionnaire data were related to EEG asymmetries during the slot machine task in the depressed sample. Positive emotionality, negative emotionality, and average positive affect during the slot machine task were not related to EEG asymmetries. Depressed participants who reported high negative affect during the task exhibited greater relative right frontal activity during the task ($p = .030$). When both age of onset and average negative affect were used as independent variables to predict frontal asymmetry during the task, age of onset remained significant, but negative affect became a trend.

Chronicity and the interaction of chronicity and age of onset were not associated with EEG asymmetry during the anticipation of reward condition. This is particularly interesting given that early onset and chronicity are often associated (Stewart, Bruder, McGrath, & Quitkin, 2003). The results of the present study, however, suggest that these clinical variables may be associated with different neurobehavioral processes. Other researchers have also found that age of onset has important diagnostic correlates even within individuals with chronic depression (Akiskal, 1983; Klein et al., 1999). It is thus possible that a low approach affective style relates to an early onset of a depressive condition and that different mechanisms maintain the depression at a chronic level (Joiner, 2000).

Interpreting the Difference Between the R and NI Conditions

There is some ambiguity in interpreting the difference between the R and NI conditions. For example, participants may have paid closer attention to the R than the NI condition; so it may be that the difference between the two conditions is not a reflection of affect or motivation but merely of attention. Attention, however, is a core feature of emotional experience (Zajonc, 1980), as participants are more likely to attend to emotional than nonemotional stimuli. Moreover, attention to reward has been shown to be associated with extraversion (Derryberry & Reed, 1994), a personality construct similar to approach motivation, and may also be regulated by frontal systems (see Depue & Collins, 1999, for a review).

It is also unclear whether the difference between the R and NI conditions is a measure of approach motivation or positive affect. Although approach motivation and positive affect are often related, they are distinguishable. Some researchers have argued that positive affect and approach motivation have different neural substrates (see Berridge & Robinson, 2003, for a review) and that motivational tendencies are more primary than the valence (positive or negative) of affect (Carver, 2001; Depue & Collins, 1999). Consistent with this view, several EEG studies found that frontal EEG asymmetries were more highly correlated with self-reports of motivational than affective constructs (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). Thus, it is possible that individuals with early-onset depression have a deficit in appetitive and goal-directed behavior rather than in positive affectivity per se.

Finally, even though participants were told that the slot machine game was random, it is possible that participants experienced the gambler's fallacy and perceived that they had control over or were able to predict the outcome. Additionally, it is possible that individuals with early-onset depression were less susceptible to the illusion that they had control over the outcome than were control participants or those with late-onset depression (i.e., depressive realism; Alloy & Abramson, 1988). Depressive realism, however, is not inconsistent with the low approach construct. It is important that future gambling studies explore this possibility.

Strengths and Limitations

The present study had several strengths. The sample ($N = 99$) was large by the standards of psychophysiological studies. The large number of depressed participants ($n = 65$) also afforded the opportunity to examine different subtypes of depression. Other strengths of the study included the use of semistructured diagnostic interviews and re-referencing the EEG data.

The study also had several limitations. First, as discussed in Footnote 1, alpha power has not been conclusively established as a valid proxy for brain activity in a particular region. More generally, it is unclear which regions of the brain contribute to alpha power at a given electrode site, particularly in frontal regions (Tenke & Kayser, 2005). To address this limitation, the slot machine task is presently being used with high resolution functional magnetic resonance imaging in order to provide converging evidence of regional activation. Second, we could not verify that participants experienced greater subjective positive affect or approach motivation during the R condition than during the NI condition. Participants could have been asked to report their mood during each trial; however, this would have been very susceptible to demand characteristics. Third, we did not assess the interrater reliability of the SCID and HRSD in this sample, although our lab has documented good reliability for these measures in the past (Keller et al., 1995; Klein et al., 2000).

Summary

The present study recorded EEG in depressed and nondepressed individuals during a task designed to elicit appetitive, approach motivation. Prior studies that have examined the relationship between EEG and depression have recorded EEG while participants were at rest. We extended the previous literature by recording EEG during the hypothesized motivational state. The results indicated that during the anticipation of reward, both nondepressed participants and participants with late-onset depression exhibited increased relative brain activity in the left frontal region. However, as hypothesized, participants with early-onset depression failed to show an increase in left frontal brain activity while anticipating a possible reward. These findings provide partial support for the approach–withdrawal model of depression (Davidson, 1994, 1998) and suggest that individuals with early-onset depression have a deficit in appetitive, incentive motivation that is consistent with a low approach affective style.

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Received September 12, 2005
Revision received August 2, 2006
Accepted August 11, 2006 ■