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The Effect of Pre vs. Post-Reward Attainment on EEG Asymmetry in Melancholic Depression

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28 pages; 2 tables and 2 figures

KEYWORD: EEG asymmetry, melancholia, positive affect, anticipatory and consummatory

Abstract

Clinical investigators have long theorized about the role of reward processing and positive affect in depression. One theory posits that compared to nonmelancholic depressives, melancholic depressives experience less consummatory (i.e., post-reward), but comparably low anticipatory (prior to reward), positive affect. We tested whether frontal EEG asymmetry, a putative marker of the anticipatory reward system, is present only before an individual receives a reward or also after receiving a reward (i.e., during consummatory reward processing). We also examined whether melancholic depression, a condition characterized by a deficit in consummatory reward processing, is associated with abnormal EEG asymmetries in alpha band power. Effects in other frequency bands (delta, theta, or beta) were also explored. EEG was recorded in 34 controls, 48 nonmelancholic depressives, and 17 melancholic depressives during a slot machine task designed to elicit anticipatory and consummatory reward processing. Results indicated that, for alpha, the frontal EEG asymmetry of greater relative left activity was specific to anticipatory reward processing. During the consummatory phase, individuals with melancholic depression exhibited different posterior EEG asymmetries than individuals with nonmelancholic depression and controls at a trend level. This second finding was largely due to melancholics exhibiting relatively lower right posterior activity and nonmelancholics exhibiting relatively lower left activity. These results suggest that a posterior asymmetry may be a marker for melancholic depression and aberrant consummatory reward processing.

Researchers have long argued that the processing of rewards occurs in two phases – a pre-reward phase (i.e., anticipatory) and a post-reward phase (i.e., consummatory; D.F. Klein, 1974; 1987). While both are associated with positive affect (PA), numerous animal and human studies have found that pre-reward PA (e.g., enthusiasm, eagerness) is biologically and phenomenologically different than post-reward PA (e.g., contentment, pride; Berridge & Robinson, 2003; Gard et al., 2006; D.F. Klein, 1974; Knutson et al., 2001; Pfaus et al., 1999).

Clinical investigators have often theorized about the role of PA/pleasure deficits in individuals with depression (Davidson, 1998; 2004; Fowles, 1994; Gray, 1994). In his classic theoretical paper, D.F. Klein (1974) hypothesized that all depressed individuals have a deficit in anticipatory PA, but only individuals with endogenous depression have a deficit in consummatory pleasure. D.F. Klein stated that these individuals “enjoy nothing and their mood can not be improved by positive rewards” (p. 4). This conceptualization was later adopted by DSM-IV as one of the core features of major depression with melancholic features (i.e., criterion a2 – “lack of reactivity to usually pleasurable stimuli [does not feel much better, even temporarily, when something good happens.]”). Despite controversies with this subtype (Coryell, 2007; Rush & Weissenberger, 1994; Taylor & Fink, 2008), several taxometric studies have identified a melancholic subtype of depression for which anhedonia is a (if not the) core feature (Haslam & Beck, 1994; Leventhal & Rehm, 2005).

Depressed individuals’ deficit in anticipatory PA is purported to be reflected by an abnormal frontal asymmetry in cortical activity due to decreased activity in left prefrontal regions (Davidson, 1998; 2004). Consistent with D.F. Klein’s theory of anticipatory PA deficits in depression, EEG measurements of cortical activity have found that individuals with and at risk for MDD exhibit the hypothesized frontal asymmetry relative to controls (Debener et al., 2000; Gotlib et al., 1998; Henriques & Davidson, 1991; Tomarken et al., 2004; Thibodeau et al., 2006; although see Reid et al., 1998 for contrary evidence).

It has been purported that the frontal EEG asymmetry largely reflects activity in dorsolateral prefrontal cortex (DLPFC; Davidson, 2004), although other cortical and subcortical areas clearly play critical roles in reward processing (e.g., orbitofrontal [OFC] and ventromedial prefrontal cortex, ventral striatum; see

Haber & Knutson, 2010 for review). Interestingly, in one of the few fMRI studies to examine asymmetrical DLPFC activation in depression, Herrington et al. (2010) recently found that depressed and control participants exhibited different asymmetries in DLPFC activation during an emotional Stroop task. Although this study was not directly measuring reward processing or anticipatory PA, it suggests that the EEG asymmetry may be tapping an asymmetric localization of emotional processing in the DLPFC (although see Wager et al., 2003). The results of Herrington et al. also support other findings that individuals with and at risk for MDD exhibit abnormal activation in the DLPFC (Drevets, 1998; Hooley et al., 2005; Liotti & Mayberg, 2001; Steele et al., 2007).¹

There are several questions, however, concerning EEG asymmetry as a purported physiological marker for low anticipatory PA (Shankman & D.N. Klein, 2003). First, despite behavioral (Henriques & Davidson, 2000) and EEG evidence supporting the hypothesis, few studies have *directly* examined whether an EEG asymmetry is associated with low anticipatory PA in depressed individuals. The majority of the EEG studies with depressed participants have only examined hemispheric activity during rest (i.e., not during any specific task) which researchers then relate to the findings from behavioral and self-report studies of reward processing in depression (Clark et al., 1994; Henriques & Davidson, 2000; Pizzagalli et al., 2005). In other words, the literatures on the EEG and behavioral correlates of depression have been almost entirely independent. Recently, we published an attempt to combine these literatures by examining EEG asymmetries of depressed individuals while they anticipated the possibility of receiving a reward (Shankman et al., 2007).

A second question concerning the relation between EEG asymmetries and emotion is whether a frontal EEG asymmetry is specific to pre-reward states or whether it is evident during post-reward states as well (Davidson, 1998). If a frontal EEG asymmetry largely reflects DLPFC activation, (Davidson, 2004; Herrington et al., 2010), it is logical that an anticipatory state should be associated with a frontal EEG asymmetry, as the DLPFC has a long-established role in goal-directed and anticipatory processes (Davidson, 2004; Fuster, 2000; Wallis & Miller, 2003). However, no EEG study has examined whether frontal asymmetry is associated only with the anticipatory, and not the consummatory, phase of reward processing. Given the suggestion that an EEG asymmetry may be a possible endophenotype for depression (Allen, 2009),

it is important to examine what specific aspect of reward processing the marker may reflect. This is therefore the first aim of our study.

Additionally, D.F. Klein's (1974) theory suggests that melancholics and non-melancholics should respond differently during the consummatory phase of reward processing, and not the anticipatory phase. However, to our knowledge, this has never been tested experimentally. This is therefore the second goal of our study. The predicted pattern and region of this asymmetry is not entirely clear, although several findings suggest that melancholia may be associated with a reduced relative right posterior asymmetry during the consummatory phase. Specifically, although depression in general is associated with reduced right parietal processing (e.g., Deldin et al., 2000; Keller et al., 2000; Rabe et al., 2005), melancholia in particular has been associated with specific deficits in several right parietotemporal tasks, including processing of both positive emotional (Bruder et al., 2002) and non-emotional stimuli (Bruder et al., 1989). This hypothesis is also consistent with the finding that the right inferior parietal lobule is differentially activated by consummatory as opposed to anticipatory reward processes (Dillon et al., 2008), although other cortical areas also show differences. Taken together, these findings raise the possibility that melancholics and nonmelancholics may exhibit different posterior asymmetries during the consummatory phase of reward processing.

Method

Participants

The sample consisted of 70 individuals with current DSM-IV major depression (MDD) and 37 control participants. The control group was required to have no lifetime diagnoses of major Axis I disorders. All participants were recruited through advertising in the community and psychiatric and psychological clinics. This study was approved by the Stony Brook University and University of Illinois-Chicago IRBs. All participants gave informed consent and were compensated \$150US for their participation. See Shankman et al. (2007) for a more detailed description of the sample and inclusion and exclusion criteria.²

All participants were right handed (determined by the Edinburgh Inventory, Oldfield, 1971). Eight participants were also excluded from analyses because they did not yield enough artifact-free EEG data (5 depressed, 3 controls). Thus, the final sample consisted of 99 participants – 34 controls and 65 individuals

with current MDD. Of the 65 depressed participants, 17 (26.2%) met DSM-IV criteria for MDD with melancholic features.

 Insert Table 1 about here

Interview and self-report measures.

Diagnoses were made via the Structured Clinical Interview for DSM-IV (SCID; First et al., 1996). Current severity of depressive symptomatology was assessed using the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). The assessments were conducted by the first author and a Masters-level diagnostician. The latter diagnostician has demonstrated high levels of inter-rater reliability in the past and has trained numerous diagnosticians on the SCID and HRSD for 10 years (Keller et al., 1995; D.N. Klein et al., 2008). Diagnoses were regularly discussed in best estimate meetings (D.N. Klein et al., 1994). As our previous report found an effect for early-onset depression (see footnote 2), we examined whether melancholic and nonmelancholic depressives differed on age of onset and they did not (19.9 vs. 18.4, $p=.61$).

In order to validate the SCID diagnoses of melancholia, we used a subscale of the HRSD that has been shown to differentiate melancholic from nonmelancholic depression (Loas & Boyer, 1996; Thase et al., 1983). It includes the HRSD items for middle insomnia, late insomnia, work and activities, retardation, agitation, loss of weight, diurnal variation (AM), and hopelessness.

All participants completed the General Temperament Survey (GTS; Clark & Watson, 1990) and the Eysenck Personality Questionnaire (EPQ-R; Eysenck et al., 1985). The GTS is a self-report measure of trait positive and negative emotionality and the EPQ-R measures trait extraversion and neuroticism. Cronbach's alpha for the four subscales were high in the depressed and nondepressed groups (all α 's > .82).

Procedure

Slot machine game. See our previous report (Shankman et al., 2007) for a more detailed description of the task. Briefly, the task was a bogus computerized slot machine game that consisted of a pre-goal and a post-goal phase. During the pre-goal phase, three reels of numbers and fruit 'spun' simultaneously for

exactly 11-s and during the 11-s post-goal phase, the result was revealed (i.e., whether the participant won or did not win). The game consisted of 36 ‘spins’, which were divided into two different pay-off situations of 18 trials each – reward (R) and no incentive (NI). The amount of money won varied from \$0.30 to \$0.45 (see Table 2).

 Insert Table 2 about here

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. The difference between these conditions was that participants won money if this outcome occurred during the R condition but did not 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 anticipatory and consummatory PA. The NI condition served as a control for several aspects of the R condition (e.g., an outcome was anticipated in both) though in the NI condition, the outcome did not have monetary implications.

There were thus 4 possible conditions-outcomes consisting of 9 trials each; R-win money; R-not win money; NI-win; and NI-lose. Participants were told that the trial order was random so that they would not realize that the outcome of each trial was predetermined. At the end of the 36 trials, all participants were given their winnings in cash.

EEG Recording

Electroencephalograms 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 hemisphere, and from one midline electrode (Cz) using a stretch-lycra electrode cap (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 (VEOG), and right- and left-outer canthi to monitor horizontal eye movements (HEOG). Electrode impedances were under 5000 ohms. Data were recorded through a Grass Neurodata acquisition system at a gain of 10K (5K for eye channels) with a bandpass of 1-30 Hz. A PC-based EEG acquisition system (Neuroscan 4.2) acquired and digitized the data continuously at a rate of 1000 Hz.

Continuous EEG during the pre and post-goal phases were segmented into consecutive 1.024 s epochs every 0.512 s (50% overlap). 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 data. The EEG was tapered over the entire 1.024 s epoch by a Hanning window to suppress spectral side lobes. For the pre-goal R and NI conditions (i.e. anticipatory conditions), there were a mean of 218 ($SE=6.1$) and 208 ($SE=6.2$) epochs, respectively. For the R-win money and NI-win conditions (i.e., consummatory conditions), there were a mean of 96.15 ($SE=3.4$) and 96.41 ($SE=3.4$) epochs. Groups had comparable number of epochs in each condition.

Power spectra were computed offline from EEG data 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. 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 (that is, while at rest). Average absolute delta, theta, alpha, and beta power were computed for each electrode site and then natural log transformed in order to normalize the data. For consistency with previous research (Henriques & Davidson, 1990; Bruder et al., 1997) the alpha band was defined as 7.81-12.70 Hz and used as an inverse measure of regional brain activity.³ The delta, theta, and beta bands were defined as 0-3.90 Hz, 3.91-7.81 Hz, and 12.71-30.0 Hz, respectively.

Because hemispheric asymmetry measures may differ depending on the reference scheme (Allen et al., 2004; Bruder et al., 1997; Hagemann, 2004; Tenke & Kayser, 2005), the epoched data were also re-referenced to an average scalp electrode reference. These results were very similar to those using the digitally

derived linked ear reference. Thus, for parsimony of presentation, only the results with the linked ear reference will be reported.

Data Analyses

The first aim of the study was to determine whether frontal alpha asymmetry differs between pre- and post-goal phases of reward processing. This was tested in SPSS 16.0 using a 2 (Condition: R vs. NI) X 2 (Goal State: pre-goal vs. post-goal) X 2 (Hemisphere: left vs. right) repeated-measures analysis of variance (ANOVA) on alpha power over frontal regions. When a Medial-Lateral repeated measures factor was added to this model, there was no significant interaction with this factor and the pattern of results was nearly identical. We therefore averaged medial and lateral electrodes (e.g., F3/F7) to create regional values (e.g., average of F4 and F8 [right frontal]; average of F3 and F7 [left frontal]).

Of note is that in these analyses, any interaction with the Hemisphere factor (e.g., a Condition X Hemisphere or Condition X State X Hemisphere interaction) is equivalent to finding a main effect (or lower-order interaction) in an analysis where the dependent variable is alpha asymmetry rather than power. Including hemisphere as a repeated measures factor therefore allowed us to examine effects of asymmetry (which is consistent with previous studies; Gotlib et al., 1998; Tomarken et al., 2004), and to follow up these effects to determine whether any asymmetry findings were driven by one hemisphere in particular.

The second aim of the study was to test whether the groups showed different patterns of asymmetry in response to reward, particularly during the post-goal phase. We tested this using a 4-way mixed design ANOVA, consisting of the three within-subjects factors described above (Condition, Goal State, Hemisphere) as well as a three-level between-subjects Group factor (i.e., control vs. non-melancholic depression vs. melancholia). This model was run on frontal and posterior alpha power separately.

Finally, to determine whether there were effects in EEG rhythms other than alpha, exploratory analyses were conducted using the 4-way mixed design ANOVA described above on frontal and posterior delta, theta, and beta power.

Results

As shown in Table 1, control, nonmelancholic, and melancholic participants did not differ on

demographic variables, and the two depressed groups did not differ on percentage taking psychotropic medication. The melancholic MDD group had significantly higher scores on HRSD, Hamilton melancholic subscale, and lower self-reports of positive temperament (Extraversion and Positive Emotionality), but comparable self-reports of negative temperament (Neuroticism and Negative Emotionality) compared to the nonmelancholic MDD group. These findings thus validate the SCID diagnosis of melancholia.

Frontal asymmetries before and after reward

The first aim of this study was to test whether frontal EEG asymmetries were different while anticipating a reward than after receiving one. We tested this hypothesis using a 2 (Goal State: pre-goal vs. post-goal) X 2 (Condition: R vs. NI) X 2 (Hemisphere: left vs. right) ANOVA on frontal alpha power. We predicted a Goal State X Condition X Hemisphere interaction during the pregoal but not the postgoal condition, such that participants would demonstrate a greater relative left than right asymmetry during the reward condition compared to the no incentive condition, but only during the pre-goal phase.

This analysis revealed a main effect for Condition, $F[1, 98]=11.18, p<.05, \eta_p^2=.10$, reflecting more activity (less alpha) during the R condition, and a main effect for Hemisphere, $F[1, 98]=24.00, p<.05, \eta_p^2=.20$, reflecting more activity (less alpha) in left hemisphere. Consistent with the finding that asymmetries differed during pre- and post-goal states, a Goal State X Condition X Hemisphere interaction emerged, $F[1, 98]=4.29, p<.05, \eta_p^2=.04$. However, follow-up analyses within each hemisphere did not reveal significant Goal State X Condition interactions in either hemisphere (left, $F[1, 98]=0.08, ns$, and right, $F[1, 98]=2.21, ns$), suggesting that the significant overall change in asymmetry resulted from non-significant changes in activity in both left and right hemispheres, rather than being driven by one hemisphere alone.

To better understand the 3-way interaction, we graphed the change in frontal alpha asymmetry from the NI to the R condition, during both the pre-goal and post-goal phases (Figure 1). Specifically, the values for Y-axis were created by subtracting the asymmetry (Right minus Left) in the NI condition from the R condition, thus creating a metric where higher values indicate a greater relative left asymmetry of “activity” to the reward condition. Inspection of this figure indicates that the asymmetry was in the hypothesized direction during the pregoal phase (greater relative brain activity in left frontal regions) and in the reverse

direction during the postgoal period (greater relative brain activity in right frontal regions).

Because an admixture sample of depressed and healthy individuals may not be appropriate for testing a hypothesis relating to normal reward processing, we also performed these analyses in the control group only, and found a similar pattern of results (i.e., Condition X State X Hemisphere interaction, $F[1, 33]=5.07, p<.05, \eta_p^2=.13$).

 Insert Figure 1 about here

Group differences in asymmetries during reward processing

The second aim of this study was to examine whether melancholic depressives differed from non-melancholics and controls on measures of EEG asymmetry during consumption of reward. We therefore performed a 2 (Goal State: pre-goal vs. post-goal) X 2 (Condition: R vs. NI) X 2 (Hemisphere: left vs. right) X 3 (Group: control vs. non-melancholic depression vs. melancholia) mixed design ANOVA on frontal and posterior alpha power. We tentatively predicted a 4-way interaction for posterior (but not frontal) regions such that melancholics would show relatively greater left than right posterior asymmetry in response to reward compared to non-melancholics and controls during the post-goal phase.

For the posterior region, the analyses revealed a main effect of Condition, $F[1, 96]=10.05, p<.01, \eta_p^2=.10$, reflecting more activity (less alpha) during the R condition compared to the NI condition.⁴ A Group X Goal State X Condition X Hemisphere interaction also emerged, $F[2, 96]=3.60, p<.05, \eta_p^2=.07$. To follow up this interaction, we analyzed posterior alpha power during pre-goal and post-goal phases separately. These analyses indicated that a Group X Condition X Hemisphere interaction was present during the post-goal phase, $F[2, 96]=3.44, p<.05, \eta_p^2=.07$, but not during the pre-goal phase, $F[2, 96]=0.57, ns$. However, follow-up analyses within each hemisphere did not reveal simple Group X Condition interactions in either left, $F[2, 96]=1.64, ns$, or right hemisphere, $F[2, 96]=0.67, ns$, suggesting that significant group differences in posterior asymmetry during the post-goal phase resulted from non-significant differences in activity in both left and right hemispheres, rather than being driven by one hemisphere alone.

To better understand these differences in asymmetry during the post-goal phase, planned comparisons were conducted comparing the three groups' changes in posterior alpha asymmetry (Right minus Left) from the NI to the R condition. These comparisons indicated that melancholic depressives exhibited different post-goal posterior asymmetries from nonmelancholic depressives, $F[1, 63]=6.83, p<.01, \eta_p^2=.07$, and controls at a trend level, $F[1, 49]=2.78, p<.10, \eta_p^2=.03$. There was also a quadratic effect for Group from controls to nonmelancholics to melancholics, $F[1, 96]=22.38, p<.01, \eta_p^2=.19$. Follow-up simple effects indicated that after receiving a reward, melancholics exhibited relative less right posterior (greater relative left) activity while nonmelancholics exhibited relative greater right posterior activity and controls exhibited approximately symmetrical posterior activity (see Figure 2). Controls and nonmelancholic depressives had similar posterior asymmetries, $F[1, 80]=1.17, ns$.

Because melancholics and nonmelancholics differed on Hamilton severity, we examined whether group differences in posterior asymmetry were due to depression severity rather than the presence of melancholia. In contrast to the significant 4-way interaction previously described, when Hamilton severity was included as a between subjects predictor in the general linear model instead of group, the State X Condition X Hemisphere X Hamilton interaction was not significant, $F[1, 96]=0.99, ns$ (note: the State X Condition X Hemisphere X Group interaction remained significant when Hamilton was also included in the model, $F[2, 95]=3.42, p<.05, \eta_p^2=.07$). Therefore, it does not appear that these results are due to depression severity. Similar analyses with medication status indicated that use of medication did not affect EEG asymmetry or the pattern of results.

 Insert Figure 2 about here

We conducted similar analyses in the frontal region. The Group X Condition X Goal State X hemisphere interaction was not significant ($F[2, 96]=2.15, ns$), nor were the separate Group X Condition X Hemisphere in the two phases significant ($F[2, 96]=1.05, ns$, pre-goal; $F[2, 96]=1.14, ns$ post-goal). This suggests that the three groups did not differ on frontal asymmetry during the pre- or the post-goal phase and

the findings were therefore specific to posterior regions.

Other frequency bands

Frontal. To explore whether there were differences in frontal power or asymmetry of other frequency bands based on group or phase of reward processing, similar mixed design ANOVAs were run for delta, theta, and beta power over frontal regions. These analyses did not reveal any significant effects for frontal delta or beta power.

For frontal theta power, a main effect of Condition emerged, $F[1, 96]=10.70, p<.01, \eta_p^2=.10$, reflecting lower theta power during the R than during the NI condition. This model also revealed a State X Condition X Hemisphere X Group interaction, $F[2, 96]=3.53, p <.05, \eta_p^2=.07$. To follow up this interaction, similar to alpha, we analyzed frontal theta power during pre-goal and post-goal phases separately. These analyses revealed a Group X Condition X Hemisphere interaction during the post-goal phase $F[2, 96]=3.51, p <.05, \eta_p^2=.07$, but not during the pre-goal phase, $F[2, 96]=0.42, ns$. However, follow-up analyses within each hemisphere did not reveal simple Group X Condition interactions in either left, $F[2, 96]=1.30, ns$, or right hemisphere, $F[2, 96]=0.71, ns$, suggesting that significant group differences in frontal theta asymmetry during the post-goal phase resulted from non-significant differences in activity in both left and right hemispheres, rather than being driven by one hemisphere alone. To better understand these differences in asymmetry during the post-goal phase, as we did for the alpha results, planned comparisons were conducted comparing the three groups' changes in frontal theta asymmetry (Right minus Left) from the NI to the R condition. These comparisons indicated that non-melancholic depressives exhibited different post-goal frontal theta asymmetries from controls, $F[1, 80]=7.16, p<.01, \eta_p^2=.08$. Controls and melancholic depressives had similar post-goal frontal asymmetries, $F[1, 49]=0.12, ns$, and the difference between melancholic and non-melancholic depressives did not reach significance, $F[1, 63]=2.62, p=.11, \eta_p^2=.04$. After receiving a reward, both controls and melancholics exhibited relatively greater relative left theta activity, while non-melancholics exhibited relatively greater right theta (Figure 3).

Insert Figure 3 about here

Posterior. We ran a parallel set of Goal State X Condition X Hemisphere X Group ANOVAs for delta, theta, and beta power over posterior regions. Similar to the frontal results, these analyses did not reveal any significant effects for posterior delta or beta asymmetry.

However, for posterior theta asymmetry, a Condition X Hemisphere interaction emerged, $F[1, 96]=4.35, p<.05 \eta_p^2=.04$, indicating greater relative right theta power during the reward condition compared to the no incentive condition. Follow-up analyses within each hemisphere indicated that this effect was driven by a decrease in left posterior theta during the reward condition, $F[1, 96]=4.96, p<.05 \eta_p^2=.05$, with no significant change in right posterior theta power, $F[1, 96]=1.00, ns$.

Discussion

Over the last two decades, there has been a great deal of interest in examining hemispheric asymmetries as markers of emotional functioning in depression (Davidson, 2004; Thibodeau et al., 2006). The present study extended this literature by examining EEG asymmetries during a paradigm that elicited two phases of reward processing: anticipatory processing (i.e., before receiving a reward) and consummatory processing (i.e., after receiving a reward). We have previously reported results for the anticipatory/pre-goal phase of the paradigm (Shankman et al., 2007) and these results extend those findings by examining EEG asymmetries in multiple frequency bands during a consummatory/post-goal phase. There were two main findings.

First, while anticipating a reward, controls exhibited the EEG pattern hypothesized for anticipatory PA (i.e., relative increased activity in left frontal regions; Davidson, 1998) and after receiving a reward, they exhibited a different frontal asymmetry. This pattern was observed experimentally controlling for aspects of the pre-goal and post-goal phase (e.g., anticipation of an outcome, viewing three pieces of fruit, etc) suggesting that this effect was due to anticipatory and consummatory PA. This finding adds to the literature examining the time course of emotional processing or “affective chronometry” (Dichter & Tomarken, 2008; D.F. Klein, 1974) and supports other studies that have used other biological measures to differentiate anticipatory and consummatory PA (Berridge & Robinson, 2003; Haber & Knutson, 2010).

Although functional neuroimaging methods, which allow better spatial resolution than EEG, have begun to differentiate the neuroanatomical circuits that underlie anticipation and consumption of reward (e.g., Berridge & Robinson, 2003; Breiter et al., 2001; Dillon et al., 2008; Knutson et al., 2001; 2008), the current findings highlight the importance of testing for asymmetric activation in these structures. As Herrington et al. (2010) note, most functional imaging studies evaluate asymmetry only by observing whether regions of interest show significant activation in one hemisphere but not the other. This approach is not nearly as sensitive or reliable a test of functional asymmetry as directly comparing the level of activation in contralateral regions. High-resolution imaging studies employing this approach may find that asymmetric activation of structures plays an important role in reward processing. The current findings, along with other task-related and resting EEG studies, suggests that this may be particularly true for DLPFC, and possibly for some parietotemporal regions.

There may be a question as to whether the experimental paradigm truly taps D.F. Klein's (1974; 1987) constructs of anticipatory and consummatory PA. D.F. Klein defined consummatory pleasures as "pleasures in biological drive reduction such as pleasure in sexual orgasm, eating, and perhaps sleep" (p. 5; Klein, 1987). Thus, one may argue that the affective processes induced by playing a slot machine game may not fully engage an individual's more basic biological drives. However, previous studies utilizing analogous gambling paradigms have activated phylogenetically older brain structures such as the nucleus accumbens and insula (Knutson et al., 2001; Kirsch et al., 2003) suggesting that paradigms involving monetary rewards are adequate analogs for studying more basic drives.

The approach-withdrawal model of emotion (Davidson, 1998; 2004) proposes that the approach system becomes activated while an individual experiences anticipatory or pre-goal attainment PA and that this is associated with a relative increase in electrocortical activity in left frontal regions. Interestingly, "post-goal attainment PA is not expected to be associated with the [approach] circuit" as it is proposed that the "prefrontal cortex goes offline after a desired goal has been achieved" (p. 312; Davidson, 1998). Our results are generally consistent with this postulate. However, as the approach circuit is proposed to "go offline" during the consummatory phase, it is unclear what pattern should be expected from a frontal "consummatory

EEG asymmetry.” Thus, our finding of greater relative right frontal activity during this phase needs to be replicated.

Our second major finding was that melancholic depressives exhibited abnormal posterior asymmetries after receiving a reward. Moreover, this finding appeared to be due to the presence of melancholia and not medication or simply to the fact that the melancholic group had more severe depressive symptoms. This result is consistent with neuropsychological studies of reduced right hemispheric processing in melancholic patients (Bruder et al., 2002; Demaree et al., 2005). Additionally, this EEG result, along with our finding that those with melancholic depression reported lower positive temperament scores on the EPQ-R and GTS, is consistent with the conceptualization that melancholic depression is a distinct subtype of depression characterized by a deficit in consummatory pleasure (D.F. Klein, 1974; 1987; Loas & Boyer, 1996). As hypothesized by D.F. Klein (1974) and listed in DSM-IV, this hedonic deficit appears to be specific to consummatory pleasure as melancholics did not exhibit different asymmetries from nonmelancholics during the anticipatory phase. However, this interpretation is slightly qualified by the finding that the posterior asymmetries during the postgoal phase for melancholics were different from controls only at a trend level ($p < .10$). Nonetheless, this trend was likely due to a lack of statistical power.

An association between aberrant consummatory PA and right posterior regions is consistent with numerous studies that have implicated right posterior regions in trait positive emotionality (Shankman et al., 2005) and affective processing (Borod, 1992; Pell, 1999; Tucker et al., 1977), including evidence implicating the right inferior parietal lobule in consummatory reward processing (Dillon et al., 2008). Moreover, the finding of aberrant right posterior activity in depression is consistent with several resting EEG (Bruder et al., 1997; Bruder et al., in press; Heller et al., 1998; Stewart et al., in press) and neuroimaging (Moratti et al., 2008) studies as well as the finding that a right posterior lesion is associated with post-stroke depression (Shimoda & Robinson, 1999).

The current findings also support the utility of a capability model of EEG asymmetry in depression (Coan, et al., 2006; Wallace, 1966). This model proposes that rather than reflecting dispositional tendencies that predict behavior regardless of the situation, EEG asymmetry may better reflect “interactions between the

emotional demands of specific situations and the emotion-regulatory abilities individuals bring to those situations” (p. 198; Coan et al., 2006). This model therefore posits that individual differences in EEG asymmetry may be more robust in response to a specific behavioral or emotional challenge rather than across situations. Consistent with this, we only found group differences when examining specific phases of the task and there were no group differences across phases (i.e., there was no main effect for group – see footnote #4). This is not to say that EEG asymmetry only reflects ‘state’ effects. Indeed, test-retest studies of resting EEG suggest at least moderate stability (Hagemann et al., 2002; Stewart et al., 2010). Rather, our results and those of others (Coan et al., 2001; 2006) suggest that the best (or at least better) conditions under which to measure individual differences in frontal EEG asymmetry may be during various emotional states.

As an exploratory result, we also found that non-melancholic depressed individuals exhibited an abnormal frontal theta asymmetry during the post-goal phase of the task, in that controls exhibited greater relative left theta power while non-melancholics exhibited greater relative right theta. EEG studies of depression generally only examine activity in the alpha band and largely ignore other frequency bands. It is therefore noteworthy that we found group differences in frontal theta activity during the post-goal phase, particularly given recent findings that frontal theta power may reflect the evaluation of feedback as an organism learns and/or makes strategic adjustments (Cavanagh, Frank, T.J. Klein, & Allen, 2010; Cohen, Elger, & Ranganath, 2007). It is unclear, however, why the nonmelancholic group differed from controls while the melancholic group did not. Given the paucity of research on theta activity in depression, future investigations on this finding are needed.

As discussed in our previous report using this paradigm, there may be some ambiguity in interpreting the difference between the R and NI conditions in both the pregoal and postgoal phases (see Shankman et al., 2007 for a more detailed discussion). 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, as participants are more likely to attend to stimuli with affective significance (Zajonc, 1990).

The study had several limitations. First, the number of individuals with melancholic depression was small ($N = 17$). Second, as discussed in footnote 3, although alpha power has been shown to be inversely correlated with brain activity, it may not be related to brain activity equally throughout the brain, particularly in frontal regions (Hagemann, 2004). Additionally, although DLPFC activity (likely the primary source of the frontal EEG signal) is thought to be associated with anticipatory approach motivation, other cortical areas including ventromedial and orbital frontal cortex, as well as subcortical areas such as the ventral striatum, play centrally important roles in the reward circuit (Berridge & Robinson, 2003; Haber & Knutson, 2010; Knutson et al., 2001). To address these limitations, the slot machine task is presently being used with high resolution functional magnetic resonance imaging (fMRI) in order to provide converging evidence of regional activation and to perform a more fine-grained analysis of cortical and subcortical structures involved in reward processing. Second, we could not verify that participants experienced greater subjective PA during the R condition compared to the NI condition. Participants could have been asked to report their affect during each trial; however, this measure would have been very susceptible to demand characteristics. Additionally, more recent data that we are collecting with this task shows that participants robustly report more PA during the R compared to the NI condition. Third, the hedonic responses during the 11-s windows of the pre- and post-goal phases of the task are not likely to be constant (particularly in the post-goal phase). Nevertheless, this does not negate the comparison of the pre and post-goal conditions. Fourth, we did not assess the inter-rater reliability of the SCID and HRSD in this sample, although our group has documented good reliability for these measures in the past (Keller et al., 1995; D.N. Klein et al., 2008).

Conclusion

The present study recorded EEG in depressed and nondepressed individuals during a task designed to elicit anticipatory and consummatory PA. The majority of previous investigations examining the relationship between EEG and depression have recorded EEG while participants were at rest. We thus extended the literature by recording EEG during two aspects of reward processing proposed to relate to depression. There were two main findings. First, anticipatory and consummatory motivations were associated with different frontal EEG asymmetries, with the former associated with relative greater left activity and the

latter associated with relative greater right activity. Second, individuals with melancholic depression exhibited a different EEG asymmetry in the post-reward phase, and this effect appeared to be largely due to differences in posterior regions, with melancholics exhibiting reduced relative right (increased left) posterior activity and nonmelancholics exhibiting reduced relative left (increased right) posterior activity. This second finding suggests that hemisphere differences in posterior activity may be a marker of post-goal attainment PA and provides partial support for the hypothesis that melancholic depression is associated with a deficit in the consummatory pleasure system (D.F. Klein, 1974; 1987).

Footnotes

1. The exact role of the DLPFC in anticipatory reward processing is unclear. The DLPFC is important in integrating environmental and emotional information during emotional mood states (Liotti & Mayberg, 2001), and may be involved in using reward information received from OFC to guide behavior (Wallis & Miller, 2003). This region has been purported to exercise regulatory influence over mood and the reward circuit through projections to the limbic system and striatum, respectively (Haber & Knutson, 2010; Liotti & Mayberg, 2001). Future studies are needed, however, to fully test the exact role of the DLPFC in anticipatory reward processing.

2. The present sample is the same as that reported in our previous report (Shankman et al., 2007). However, Shankman et al. only reported data from the pre-goal phase of the task and not the post-goal phase and did not divide the depressed group into those with and without melancholia.

Given that Shankman et al. (2007) found that an early onset of depression was associated with an abnormal frontal asymmetry during the pre-goal phase, we explored whether an early onset was also associated with an abnormal asymmetry during the post-goal phase. These findings were nonsignificant. Taken together with the present findings, this suggests a double dissociation with an early onset associated with deficits in pre-goal (but not post-goal) PA and melancholia associated with deficits in post-goal (but not pre-goal) PA.

3. The premise that alpha power is inversely related to brain activity *equally* throughout the brain is somewhat controversial (Allen et al., 2004; Tenke & Kayser, 2005). There is some data to suggest that alpha power is inversely correlated with other measures of brain activity such as PET and fMRI (Oakes et al., 2004; Oishi et al., 2007). 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 (Allen et al., 2004). More research is needed, however, on this issue in order to definitively conclude that alpha power is an inverse measure of brain activity throughout the brain. Nevertheless, for ease of presentation and as is conventional in the literature, the term “brain activity” will be used as a heuristic for inverse alpha power throughout this paper.

4. For the posterior region, nonsignificant results were found for the Group X Condition X Hemisphere interaction: $F[2, 96]=1.81$, *ns*, the Group X Hemisphere interaction: $F[2, 96]=0.10$, *ns*, and the overall main effect for Group, $F[2, 96]=1.09$, *ns*. Similar non-significant results were also found for frontal regions (all F 's < 1.06).

References

- Allen, J.J.B., 2009. Frontal EEG asymmetry as an endophenotype for depression: A skeptic's journey. Paper presented at the meeting of the Society for Psychophysiological Research, Berlin, Germany.
- Allen, J.J.B., Coan, J.A., Nazarian, M., 2004. Issues and assumptions on the road from raw signals to metrics of frontal EEG asymmetry in emotion. *Biol Psychol*, 67, 183-218.
- Berridge, K.C., Robinson, T.E., 2003. Parsing reward. *Trends Neurosci*, 26, 507-513.
- Borod, J.C., 1992. Interhemispheric and intrahemispheric control of emotion: A focus on unilateral brain damage. *J Consult Clin Psych*, 60, 339-348.
- Breiter, H.C., Aharon, I., Kahneman, D., Dale, A., Shizgal, P., 2001. Functional imaging of neural responses to expectancy and experience of monetary gains and losses. *Neuron*, 30, 619-39.
- Bruder, G.E., Fong, R., Tenke, C.E., Leite, P., Towey, J.P., Stewart, J.E., et al., 1997. Regional brain asymmetries in major depression with or without an anxiety disorder: A quantitative electroencephalographic study. *Biol Psychiat*, 41, 939-948.
- Bruder, G.E., Kayser, J., Tenke, C.E., in press. Event-related brain potentials in depression: Clinical, cognitive and neurophysiologic implications. In Luck, S.J., Kappenman, E.S. (Eds.), *Event-related potential components: The ups and downs of brainwave recordings*. New York: Oxford University Press.
- Bruder, G.E., Quitkin, F.M., Stewart, J.W., Martin, C., Voglmaier, M.M., Harrison, W.M., 1989. Cerebral laterality and depression: Differences in perceptual asymmetry among diagnostic subtypes. *J Abnorm Psychol*, 98, 177-186.
- Bruder, G.E., Stewart, J.W., McGrath, P.J., Ma, G.J., Wexler, B.E., Quitkin, F.M., 2002. Atypical depression: Enhanced right hemispheric dominance for perceiving emotional chimeric faces. *J Abnorm Psychol*, 111, 446-454.
- Cavanagh, J.F., Frank, M.J., Klein, T.J., Allen, J.J.B., 2010. Frontal theta links prediction errors to behavioral adaptation in reinforcement learning. *NeuroImage*, 49, 3198-3209.
- Clark, L.A., Watson, D., 1990. *The General Temperament Survey*. Unpublished manuscript, Southern Methodist University, Dallas, TX.

Clark, L.A., Watson, D., Mineka, S., 1994. Temperament, personality, and the mood and anxiety disorders. *J Abnorm Psychol*, 103, 103-116.

Coan, J.A., Allen, J.J., Harmon-Jones, E., 2001. Voluntary facial expression and hemispheric asymmetry over the frontal cortex. *Psychophysiology*, 38, 912-25.

Coan, J.A., Allen, J.J., McKnight, P.E., 2006. A capability model of individual differences in frontal EEG asymmetry. *Biol Psychol*, 72, 198-207.

Cohen, M.X., Elger, C.E., Ranganath, C., 2007. Reward expectation modulates feedback-related negativity and EEG spectra. *Neuroimage*, 35, 968-978.

Coryell, W., 2007. The facets of melancholia. *Acta Psychiat Scand*, 115, 31-36.

Davidson, R.J., 1998. Affective style and affective disorders: Perspectives from affective neuroscience. *Cognition Emotion*, 12, 307-330.

Davidson, R.J., 2004. What does the prefrontal cortex "do" in affect: Perspectives on frontal EEG asymmetry research. *Biol Psychol*, 67, 219-233.

Debener, S., Beauducel, A., Nessler, D., Brocke, B., Heilemann, H., Kayser, J., 2000. Is resting anterior EEG alpha asymmetry a trait marker for depression? Findings for healthy adults and clinically depressed patients. *Neuropsychobiology*, 41, 31-37.

Deldin, P.J., Keller, J., Gergen, J.A., Miller, G.A., 2000. Right-posterior face processing anomaly in depression. *J Abnorm Psychol*, 109, 116-21.

Demaree, D.A., Everhart, D.E., Youngstrom, E.A., Harrison, D.W., 2005. Brain lateralization of emotional processing: Historical roots and a future incorporating dominance. *Behav Cogn Neurosci Revs*, 4, 3-20.

Dichter, G.S., Tomarken, A.J., 2008. The chronometry of affective startle modulation in unipolar depression. *J Abnorm Psychol*, 117, 1-15.

Dillon, D.G., Holmes, A.J., Jahn, A.L., Bogdan, R., Wald, L.L., Pizzagalli, D.A., 2008. Dissociation of neural regions associated with anticipatory versus consummatory phases of incentive processing. *Psychophysiology*, 45, 36-49.

Drevets, W.C., 1998. Functional neuroimaging studies of depression: The anatomy of melancholia. *Annu Rev Med*, 49, 341-61.

Eysenck, S.B.G., Eysenck. H.J., Barrett, P., 1985. A revised version of the psychoticism scale. *Pers Indiv Differ*, 6, 21-29.

First, M.B., Spitzer, R.L., Gibbon, M., Williams, J.B.W., 1996. Structured Clinical Interview for DSM-IV Axis I Disorders, Clinician Version (SCID-CV). Washington, D.C.: American Psychiatric Press, Inc.

Fowles, D.C., 1994. A motivational theory of psychopathology. In: Spalding, W.D. (Ed). *Nebraska Symposium on Motivation*. Lincoln, NB: University of Nebraska Press, pp. 181-238.

Fuster, J.M., 2000. The prefrontal cortex of the primate: A synopsis. *Psychobiology*, 28, 125-131.

Gard, D.E., Gard, M.G., Kring, A.M., John, O.P., 2006. Anticipatory and consummatory components of the experience of pleasure: A scale development study. *J Res Pers*, 40, 1086-1102.

Gotlib, I.H., Ranganath, C., Rosenfield, J.P., 1998. Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition Emotion*, 12, 449-478.

Gray, J.A., 1994. Framework for a taxonomy of psychiatric disorder. In: van Goozen, S.H.M., van de Poll, N.E., Sargeant, J.A. (Eds.), *Emotions: Essays on Emotion Theory*. Hillsdale, NJ: Lawrence Erlbaum, pp. 29-59.

Haber, S.N., Knutson, B., 2010. The reward circuit: linking primate anatomy and human imaging. *Neuropsychopharmacology*, 35, 4-26.

Hagemann, D., Naumann, E., Thayer, J.F., Bartussek, D., 2002. Does resting electroencephalograph asymmetry reflect a trait? An application of latent state-trait theory. *J Pers Soc Psychol*, 82, 619-41.

Hagemann, D., 2004. Individual differences in anterior EEG asymmetry: Methodological problems and solutions. *Biol Psychol*, 67, 157-182.

Hamilton, M., 1960. A rating scale for depression. *J Neurol Neurosurg Psychiatry*, 23, 56-62.

Haslam, N., Beck, A.T., 1994. Subtyping major depression: A taxometric analysis. *J Abnorm Psychol*, 103, 686-692.

Heller, W., Nitschke, J.B., Miller, G.A., 1998. Lateralization in emotion and emotional disorders. *Curr Dir Psychol Sci*, 7, 26-32.

Henriques, J.B., Davidson, R.J., 1990. Regional brain electrical asymmetries discriminate between previously depressed and healthy control subjects. *J Abnorm Psychol*, 99, 22-31.

Henriques, J.B., Davidson, R.J., 1991. Left frontal hypoactivation in depression. *J Abnorm Psychol*, 100, 535-545.

Henriques, J.B., Davidson, R.J., 2000. Decreased responsiveness to reward in depression. *Cognition Emotion*, 14, 711-724.

Herrington, J.D., Heller, W., Mohanty, A., Engels, A.S., Banich, M.T., Webb, A.G., Miller, G.A., 2010. Localization of asymmetric brain function in emotion and depression. *Psychophysiology*, 47, 442-454.

Hooley, J.M., Gruber, S.A., Scott, L.A., Hiller, J.B., Yurgelun-Todd, D.A., 2005. Activation in dorsolateral prefrontal cortex in response to maternal criticism and praise in recovered depressed and healthy control participants. *Biol Psychiatry*, 57, 809-812.

Keller, M.B., Klein, D.N., Hirschfeld, R.M.A., Kocsis, J.H., McCullough, J.P., Miller, L., et al., 1995. Results of the DSM-IV Mood Disorders Field Trial. *Am J Psychiat*, 152, 843-849.

Keller, J., Nitschke, J.B., Bhargava, T., Deldin, P.J., Gergen, J.A., Miller, G.A., Heller, W., 2000. Neuropsychological differentiation of depression and anxiety. *J Abnorm Psychol*, 109, 3-10.

Kirsch, P., Schienle, A., Stark, R., Sammer, G., Blecker, C., Walter, B., et al., 2003. Anticipation of reward in a nonaversive differential conditioning paradigm and the brain reward system: an event-related fMRI study. *Neuroimage*, 20, 1086-1095.

Klein, D.F., 1974. Endogenomorphic depression. A conceptual and terminological revision. *Arch Gen Psychiat*, 31, 447-454.

Klein, D.F., 1987. Depression and anhedonia. In: Clark, D.C., Fawcett, J. (Eds.), *Anhedonia and affect deficit states*. New York: PMA Publishing, pp. 1-14.

Klein, D.N., Ouimette, P.C., Kelly, H.S., Ferro, T., Riso, L.P., 1994. Test-retest reliability of team consensus best-estimate diagnoses of Axis I and II disorders in a family study. *Am J Psychiat*, 151, 1043-

1047.

Klein, D.N., Shankman, S.A., Rose, S., 2008. Dysthymic disorder and double depression: Baseline predictors of 10-year course and outcome. *J Psychiat Res*, 42, 408-415.

Knutson, B., Adams, C.M., Fong, G.W., Hommer, D., 2001. Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *J Neurosci*, 21, 159-163.

Knutson, B., Bhanji, J.P., Cooney, R.E., Atlas, L.Y., Gotlib, I.H., 2008. Neural responses to monetary incentives in major depression. *Biol Psychiatry*, 63, 686-92.

Leventhal, A.M., Rehm, L.P., 2005. The empirical status of melancholia: Implications for psychology. *Clin Psychol Rev*, 25, 25-44.

Liotti, M., Mayberg, H.S., 2001. The role of functional neuroimaging in the neuropsychology of depression. *J Clin Exp Neuropsychol*, 23, 121-136.

Loas, G., Boyer, P., 1996. Anhedonia in endogenomorphic depression. *Psychiat Res*, 60, 57-65.

Moratti, S., Rubio, G., Campo, P., Keil, A., Ortiz, T., 2008. Hypofunction of right temporoparietal cortex during emotional arousal in depression. *Arch Gen Psychiat*, 65, 532-541.

Oakes, T.R., Pizzagalli, D.A., Hendrick, A.M., Horras, K.A., Larson, C.L., et al., 2004. Functional coupling of simultaneous electrical and metabolic activity in the human brain. *Hum Brain Mapp*, 21, 257-270.

Oishi, N., Mima, T., Ishii, K., Bushara, K.O., Hiraoka, T., Ueki, Y., et al., 2007. Neural correlates of regional EEG power change. *Neuroimage*, 36, 1301-1312.

Oldfield, R.C., 1971. Assessment and analysis of handedness: Edinburgh Inventory. *Neuropsychologia*, 9, 97-113.

Pell, M.D., 1999. The temporal organization of affective and non-affective speech in patients with right-hemisphere infarcts. *Cortex*, 35, 455-477.

Pfaus, J.G., Smith, W.J., Coopersmith, C.B., 1999. Appetitive and consummatory sexual behaviors of female rats in bilevel chambers: I. A correlational and factor analysis and the effects of ovarian hormones. *Horm Behav*, 35, 224-240.

Pizzagalli, D.A., Jahn, A.L., O'Shea, J.P., 2005. Toward an objective characterization of an anhedonic phenotype: A signal-detection approach. *Biol Psychiat*, 57, 319-327.

Rabe, S., Debener, S., Brocke, B., Beauducel, A., 2005. Depression and its relation to posterior cortical activity during performance of neuropsychological verbal and spatial tasks. *Pers Individ Diff*, 39, 601-611.

Reid SA, Duke LM, Allen JJB. Resting frontal electroencephalographic asymmetry in depression: Inconsistencies suggest the need to identify mediating factors. *Psychophysiology*, 35, 389-404.

Rush, A.J., Weissenberger, J.E., 1994. Melancholic symptom features and DSM-IV. *Am J Psychiat*, 151, 489-498.

Shankman, S.A., Klein, D.N., 2003. The relation between depression and anxiety: An evaluation of the tripartite, approach-withdrawal, and valence-arousal models. *Clin Psychol Rev*, 23, 605-637.

Shankman, S.A., Klein, D.N., Tenke, C.E., Bruder, G.E., 2007. Reward sensitivity in depression: A biobehavioral study. *J Abnorm Psychol*, 116, 95-104.

Shankman, S.A., Tenke, C.E., Bruder, G.E., Durbin, C.E., Hayden, E.P., Klein, D.N., 2005. Low positive emotionality in young children: Association with EEG asymmetry. *Dev Psychopathol*, 17, 85-98.

Sharbrough, F., Chatrian, G.E., Lesser, R.P., Lüders, H., Nuwer, M., Picton, T.W., 1991. American Electroencephalographic Society guidelines for standard electrode position nomenclature. *J Clin Neurophysiol*, 8, 200-202.

Shimoda, K., Robinson, R.G., 1999. The relationship between poststroke depression and lesion location in long-term follow-up. *Biol Psychiat*, 45, 187-192.

Steele, J.D., Currie, J., Lawrie, S.M., Reid, I., 2007. Prefrontal cortical functional abnormality in major depressive disorder: A stereotactic meta-analysis. *J Affect Disord*, 101, 1-11.

Stewart, J.L., Bismark, A.W., Towers, D.N., Coan, J.A., Allen, J.J.B., 2010. Resting frontal EEG asymmetry as an endophenotype for depression risk: Sex-specific patterns of frontal brain asymmetry. *J Abnorm Psychol*, 119, 502-512.

Stewart, J.L., Towers, D.N., Coan, J.A., Allen, J.J.B., in press. The oft-neglected role of parietal EEG asymmetry and risk for major depressive disorder. *Psychophysiology*.

Taylor, M.A., Fink, M., 2008. Restoring melancholia in the classification of mood disorders. *J Affect Disorders*, 105, 1-14.

Tenke, C.E., Kayser, J., 2005. Reference-free quantification of EEG spectra: Combining current source density (CSD) and frequency principal components analysis (fPCA). *Clin Neurophysiol*, 116, 2826-2846.

Thase, M.E., Hersen, M., Bellack, A.S., Himmelhoch, J.M., Kupfer, D.J., 1983. Validation of a Hamilton subscale for endogenous depression. *J Affect Disorders*, 5, 267-278.

Thibodeau, R., Jorgensen, R.S., Kim, S., 2006. Depression, anxiety, and resting frontal EEG asymmetry: A meta-analytic review. *J Abnorm Psychol*, 115, 715-729.

Tomarken, A.J., Dichter, G.S., Garber, J., Simien, C., 2004. Resting frontal brain activity: Linkages to maternal depression and socio-economic status among adolescents. *Biol Psychol*, 67, 77-102.

Tucker, D.M., Watson, R.T., Heilman, K.M., 1977. Discrimination and evocation of affectively intoned speech in patients with right parietal disease. *Neurology*, 27, 947-958.

Wager, T.D., Phan, K.L., Liberzon, I., Taylor, S.F., 2003. Valence, gender, and lateralization of functional brain anatomy in emotion: A meta-analysis of findings from neuroimaging. *Neuroimage*, 19, 513-531.

Wallace, J., 1966. An abilities conception of personality: Some implications for personality measurement. *Am Psychol*, 21 132-138.

Wallis, J.D., Miller, E.K., 2003. Neuronal activity in primate dorsolateral and orbital prefrontal cortex during performance of a reward preference task. *Eur J Neurosci*, 18, 2069-2081.

Zajonc, R.B., 1990. Feeling and thinking: Preferences need no inferences. *Am Psychol*, 35, 151-175.

Author Note

This study was supported by the APF and COGDOP Clarence J. Rosecrans Scholarship as well as NIMH grant F31 MH67309, both awarded to Stewart A. Shankman. We would like to acknowledge the consultation and support of Craig Tenke, Jurgen Kayser and Gerard Bruder, and the assistance of Suzanne Rose, Brady Nelson, Kathryn Messineo, Jana Kramer, and Ana Genkova in data collection and coding.

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Table 1. Characteristics of sample¹

	Control (N=34)	Non-melancholic MDD (N = 48)	Melancholic MDD (N=17)
Demographic variables			
Female	70.6% _a	70.8% _a	52.9% _a
Age (yrs)	33.8 _a	33.2 _a	35.5 _a
Caucasian	73.5% _a	77.1% _a	76.5% _a
Employed	82.4% _a	79.2% _a	76.5% _a
Laterality Quotient ²	86.9 _a	94.1 _a	93.0 _a
Questionnaire Variables			
GTS			
Positive Emotionality (SE)	20.2 (0.9) _a	12.7 (0.9) _b	9.1 (1.5) _c
Negative Emotionality (SE)	4.4 (1.0) _a	19.0 (1.1) _b	22.9 (1.5) _b
EPQ-R			
Extraversion (SE)	16.2 (0.7) _a	11.6 (0.8) _b	7.9 (1.0) _c
Neuroticism (SE)	5.0 (0.8) _a	16.3 (0.8) _b	18.6 (1.0) _b
Clinical Variables			
Hamilton Rating Scale of Depression (HRSD; SE)	1.8 (0.3) _a	24.5 (1.0) _b	30.6 (1.9) _c
Hamilton Melancholic Subscale	0.5 (0.1) _a	7.3 (0.3) _b	8.8 (0.8) _c
Currently taking medication	-	50% _b	47.1% _b
Lifetime psychiatric hospitalization	-	8.3% _b	17.6% _b
Current anxiety disorder ³	-	27.1% _b	35.3% _b
Lifetime alcohol abuse/dependence disorder	-	41.7% _b	29.4% _b
Lifetime drug abuse/dependence disorder	-	25.0% _b	11.8% _b

¹ Means or percentages with different lettered subscripts across rows were significantly different in pairwise comparisons ($p < 0.05$).

² Laterality Quotients can vary between -100 (completely left-handed) to +100 (completely right-handed).

³ Panic disorder, agoraphobia, social phobia, specific phobia, OCD, PTSD, or GAD.

Table 2. Design of slot machine paradigm

Condition	Number of trials and time of each pre-result EEG recording	Result and outcome	Number of trials and time of each post-result EEG recording
Possible reward	18 trials, 11 seconds each	Win – win \$ 	9 trials, 11 seconds each
		Lose – no win \$ 	9 trials, 11 seconds each
No Incentive	18 trials, 11 seconds each	Win – no win \$ 	9 trials, 11 seconds each
		Lose – no lose \$ 	9 trials, 11 seconds each
Totals:	36 trials; 396 seconds in ‘pre-goal phase’		36 trials; 396 seconds in ‘post-goal phase’

Figure Captions

Figure 1. Frontal alpha asymmetries of full sample during pre-goal and post-goal phase. Using the assumption that alpha power is an inverse measure of brain activity, the Y-axis represents greater relative left brain “activity” for positive values (relative right alpha) or greater relative right brain “activity” for negative values (relative left alpha) during the R condition compared to the NI condition. Error bars represent standard errors.

Figure 2. Posterior alpha asymmetries of controls ($N=34$) and individuals with nonmelancholic ($N = 48$) and melancholic depression ($N=17$) during post-goal/consummatory phase of task. Using the assumption that alpha power is an inverse measure of brain activity, the Y-axis represents greater relative left brain “activity” for positive values (relative right alpha) or greater relative right brain “activity” for negative values (relative left alpha) during the R condition compared to the NI condition. Error bars represent standard errors.

Figure 3. Frontal theta asymmetries of controls ($N=34$) and individuals with nonmelancholic ($N = 48$) and melancholic depression ($N=17$) during post-goal/consummatory phase of task. The Y-axis represents the greater relative right theta power (positive values) or greater relative left theta power (negative values) during the R condition compared to the NI condition. Error bars represent standard errors.

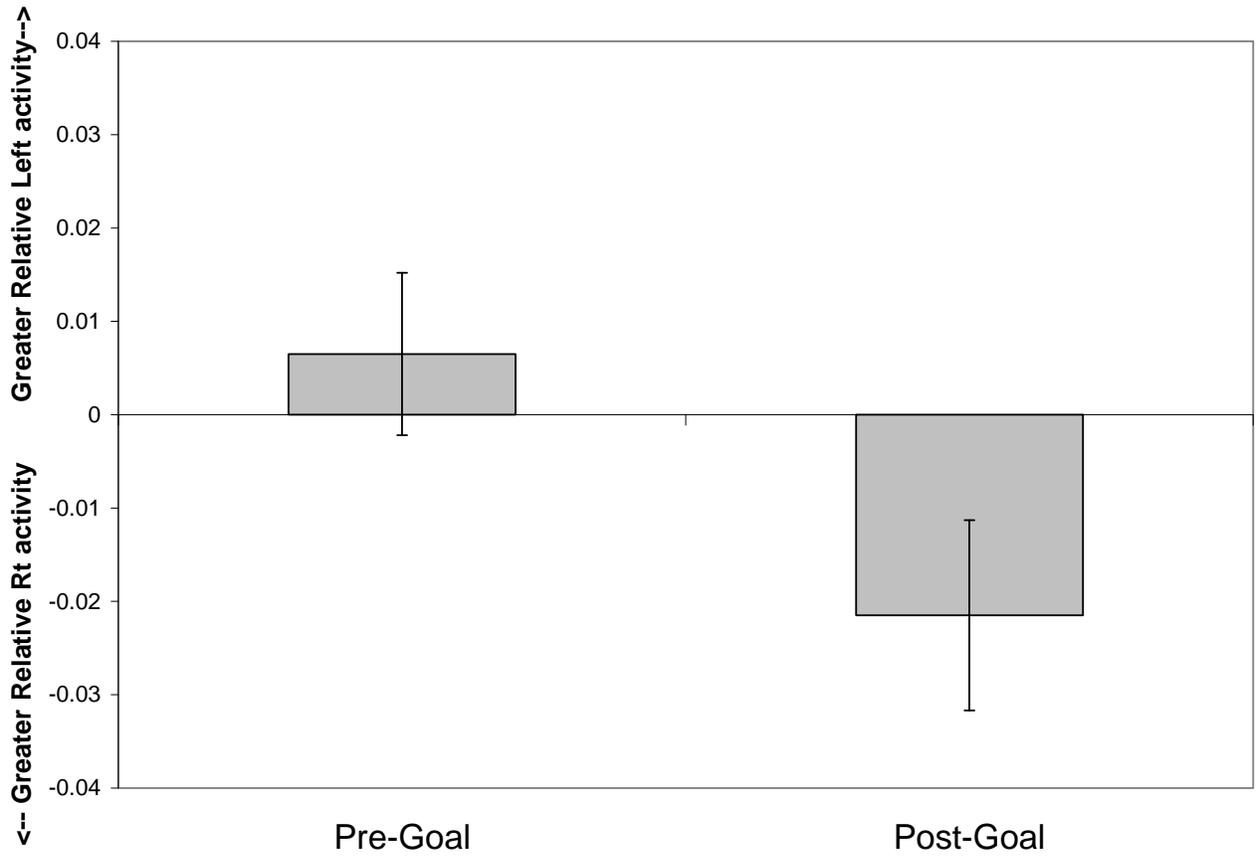


Figure 1

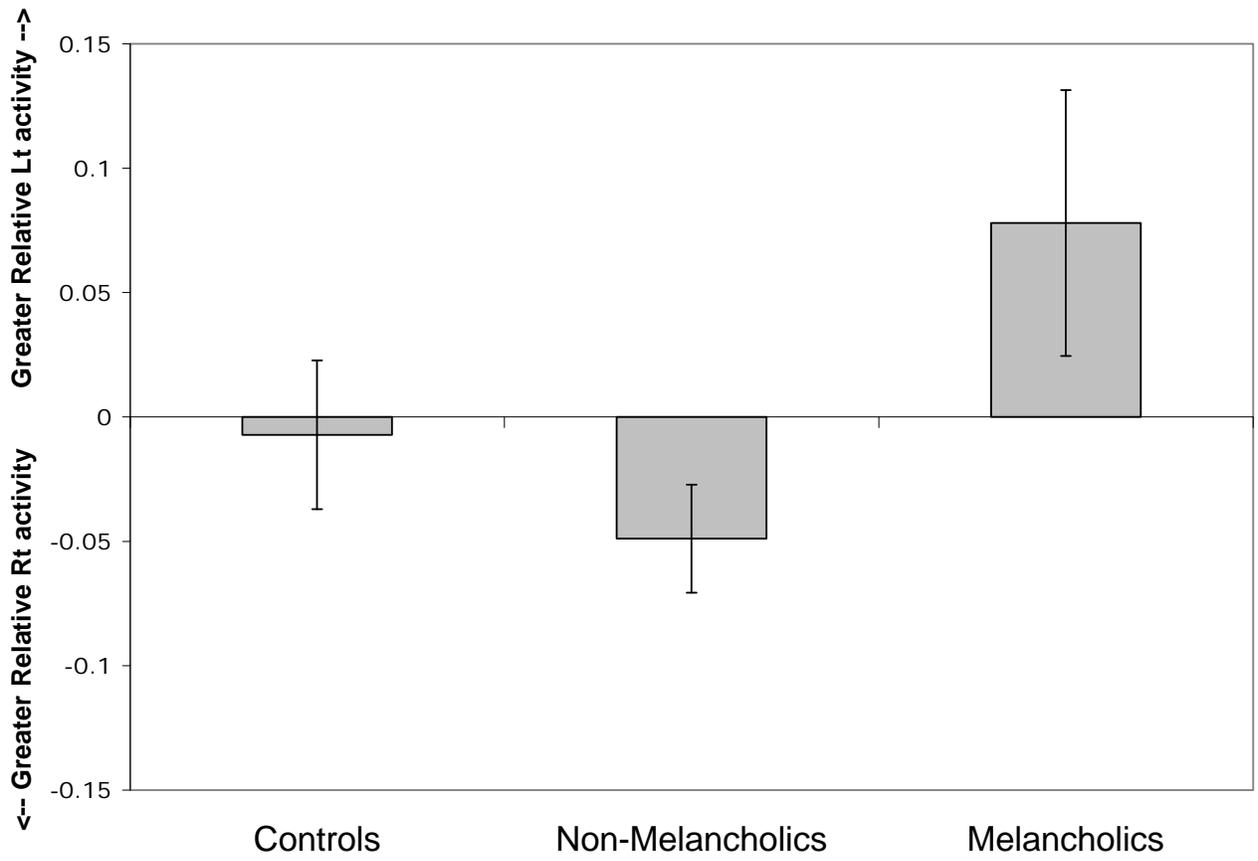


Figure 2

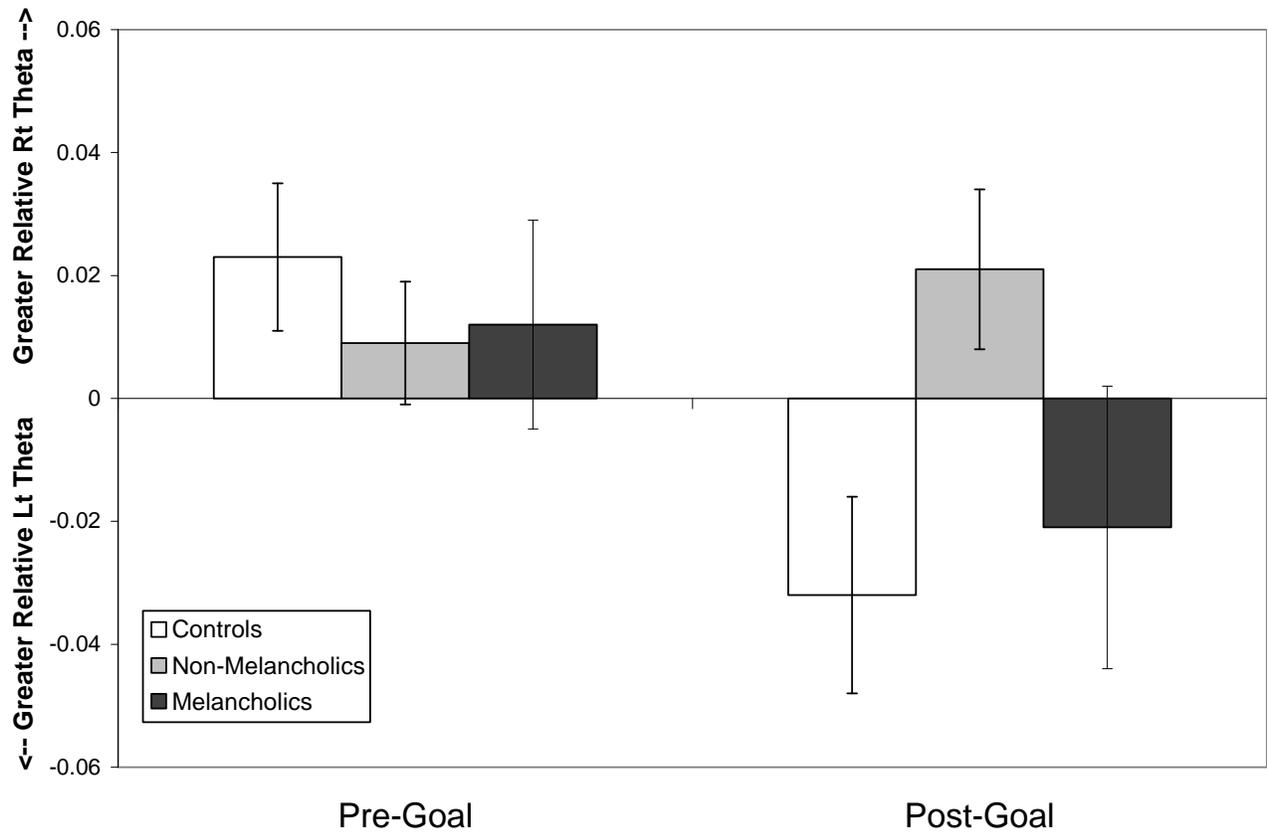


Figure 3