Frontal Alpha EEG Asymmetry Before and After Behavioral Activation Treatment for Depression

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Abstract

Background—Mid-frontal and mid-lateral (F3/F4 and F7/F8) EEG asymmetry has been associated with motivation and affect. We examined alpha EEG asymmetry in depressed and healthy participants before and after Behavioral Activation treatment for depression; examined the association between alpha EEG asymmetry and motivational systems and affect; and evaluated the utility of alpha EEG asymmetry in predicting remission.

Methods—Depressed ($n = 37$) and healthy participants ($n = 35$) were assessed before and after treatment using a clinical interview, a task to measure baseline EEG, and questionnaires of behavioral activation and inhibition, avoidance, and affect.

Results—Alpha EEG asymmetry was significantly higher in depressed than healthy participants at pre-treatment, positively correlated with negative affect and behavioral inhibition, and inversely correlated with lower behavioral activation sensitivity.

Conclusions—Heightened alpha EEG asymmetry in depressed participants was significantly associated with increased behavioral inhibition and negative emotion and was independent of clinical remission.

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Introduction

Available evidence from studies on biologic mechanisms suggests that identifying neurophysiologic markers of major depression may advance efforts to identify underlying factors that characterize depression (Allen, Iacono, Depue, & Arbisi, 1993; Sutton & Davidson, 1997). Much of the focus has been on the role of the frontal cortex, which has been linked with impairment of information processing and with potential markers of treatment response (Cook et al., 2013). Specifically, research has focused on resting electroencepholagraphic activity in the alpha band (8–12Hz), which reflects a reliable index of relaxed wakefulness in the brain when an individual has their eyes closed. Alpha activity may be suppressed or 'desynchronized' when individuals open their eyes, engage in mental activity, become alert, or conversely, when drowsy (Pizzagalli, 2007). Alpha activity in healthy samples has been associated with emotional experience (Allen, Urry, Hitt, & Coan, 2004; Maxwell & Davidson, 2007; Tenke & Kayser, 2005) and internalized attention during the practice of meditation (Aftanas & Golocheikine, 2001). Moreover, greater alpha activity has been inversely associated with cognitive function and attention (Dujardin et al., 1993; Gevins & Smith, 2000; Ray & Cole, 1985; Rugg & Dickens, 1982).

Increased alpha activity has been conceptualized a psychophysiological trait-like characteristic distinguishable in current and remitted depressed participants from healthy participants (Brenner et al., 1986; Pollock & Schneider, 1989). Notably, frontal alpha EEG asymmetry, defined as the difference in alpha activity over right vs. left hemisphere of the brain, has been observed such that higher scores indicate greater relative left activation (i.e., increased right alpha activity). More specifically, data has shown that current and remitted depression is associated with increased left (versus right) alpha activity, which corresponds to decreased left (versus right) hemispheric activation, though this pattern has not always been reported (Allen et al., 1993; Debener et al., 2000; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991; Mathersul, Williams, Hopkinson, & Kemp, 2008; Rosenfeld, Baehr, Baehr, Gotlib, & Ranganath, 1996; Stewart, Bismark, Towers, Coan, & Allen, 2010; Thibodeau, Jorgensen, & Kim, 2006). The magnitude of average effects associated with studies of depressed and healthy participants on resting frontal alpha EEG asymmetry indicates mean weighted effect sizes by Cz reference scheme of $\kappa = 16$, and mean weighted effects of $r = .29$ (95% CI = .24, .34). Also, mean weighted effects linked with the mid-frontal (F3/4) site were bigger than the lateral frontal (F7/8; Thibodeau et al., 2006). However, several studies have not been able to differentiate depressed from healthy participants in alpha EEG asymmetry (Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Reid, Duke, & Allen, 1998).

Such inconsistent results may be explained by different measures of depression, smaller patient samples with heterogeneous symptoms, and varied recruitment strategies.

Specifically, inadvertent enrolling of bipolar versus unipolar patients may have altered results (Lieber & Prichep, 1988) in which each diagnosis may have a unique neurophysiologic signature that is not necessarily observed in milder depression or sufficiently measured using a cursory assessment of depression symptoms (e.g., self-reports of depression). Also, consistent with the idea that alpha asymmetry is a reliable and stable psychophysiological marker of depression versus a state-specific factor (Coan & Allen, 2003; Sutton & Davidson, 1997), Allen and colleagues' careful research (Allen et al., 2004) found high internal consistency coefficients for alpha asymmetry at frontal regions (F34, F78, and FTC12) across reference schemes ranging from .86 to .89, with a median of .89 at baseline, and from .61 to .92, with a median of .86 across five assessment sessions. Further, Tomarken, Davidson, Wheeler, and Kinney (1992) evaluated the psychometric properties of frontal EEG asymmetry in 85 college students twice (separated by three weeks), and observed that EEG asymmetry assessed at mid-frontal and anterior temporal regions, under both Cz-referenced and computer averaged ears referenced data, showed high internal consistency (Cronbach's alphas ranging from .81 to .92) with acceptable test-retest stability, showing intraclass correlations ranging from .53 to .72 (Tomarken, Davidson, Wheeler, & Kinney, 1992). Finally, in support of the contention that frontal EEG asymmetry represents a stable neurophysiological marker, data has shown that frontal EEG asymmetry has 60% stable trait variance (Hagemann, Naumann, Thayer, & Bartussek, 2002), potentially serving as a vulnerability to first onset of depression (Nusslock et al., 2011) and predictive of reoccurrence (Allen, McKnight, Moreno, Demaree, & Delgado, 2009).

Alpha activity has been associated with approach- and withdrawal-related motivation measured by the Behavioral Activation System and Behavioral Inhibition System Scales (Gray, 1970). Specifically, less relative left frontal alpha activity (greater left activation) has been associated with heightened behavioral activation sensitivity, or an increased motivation to approach when goal-directed action is indicated (Coan & Allen, 2003; Sutton & Davidson, 1997). Also, stimuli intended to elicit approach-oriented responses, including reward cues and anger-evoking stimuli, have been associated with lower relative left frontal alpha activity (Coan & Allen, 2004; Harmon-Jones & Allen, 1998). These data support an association between behavioral activation sensitivity in the context of both pleasant and unpleasant (i.e., including anger-inducing) stimuli (Harmon-Jones & Allen, 1998). The link between alpha activity and BAS scores may also be explained by decreased right frontal activation, as opposed to increased left frontal activation, still indicating decreased relative left alpha activity (Coan & Allen, 2003). Conversely, less alpha activity over the right prefrontal regions (greater relative right activation) has been associated with measures of behavioral inhibition (Buss, Davidson, Kalin, & Goldsmith, 2004; Shackman, McMenamin, Maxwell, Greischar, & Davidson, 2009). The consistency and strength of association between EEG asymmetry and behavioral activation and inhibition sensitivity suggests that they strongly relate to an individual's tendency to approach or avoid. Minimal research, however, has linked frontal EEG asymmetry to avoidance behavior in depressed individuals before and after a treatment designed to modify behavioral avoidance.

Finally, as frontal EEG asymmetry has been linked with affect, data have indicated an association between greater relative left prefrontal activation (i.e., *less* alpha activity) with

positive affect measured by the Positive and Negative Affect Schedule (PANAS; Tomarken, Davidson, Wheeler, & Doss, 1992). Conversely, greater relative right prefrontal activation (i.e., *more* alpha activity) has been associated with negative personality factors (Sutton & Davidson, 1997) and more intense reactions to negative films (Tomarken, Davidson, & Henriques, 1990) in addition to trait anxiety and negative emotions (Coan & Allen, 2003). This differential association between frontal EEG asymmetry and positive and negative affect may indicate a biological substrate of affect (Sutton & Davidson, 1997).

Alpha activity over the occipital sites has predicted treatment response (Bruder et al., 2008). Also, there has been support for alpha power as a state-independent neurophysiologic characteristic in treatment-seeking samples early in treatment. Higher alpha-theta ratios derived from frontal QEEG assessments at pre-treatment and Week 1 of escitalopram treatment have predicted 8-week outcomes (Leuchter et al., 2009*a*), as well as differential response to antidepressants (Leuchter et al., 2009*b*). They have also predicted shorter time to remission during treatment with significantly more depressed patients attaining sustained remission (Cook et al., 2013). Additionally, significantly greater alpha amplitude in eyes closed versus eyes open conditions has predicted response to antidepressants (Tenke et al., 2011). However, frontal EEG asymmetry has been observed to change in response to interventions, such as Cognitive Behavioral Treatment (CBT) for Post-Traumatic Stress Disorder (PTSD; Rabe, Zoellner, Beauducel, Maercker, & Karl, 2008), which found a greater reduction in relative right anterior activation in the participants receiving CBT when compared to a Wait-list condition. Significant findings revealed a shift in hemispheric asymmetry, mainly due to an effect of a decreased alpha activity in the right [$t(16) = 2.33$, $p = .03$] but not the left anterior region [$t(16) = 0.69$, $p = .50$]. Conversely, participants in the Wait-list condition exhibited no shift in hemispheric activity over time. However, with an intervention using mindfulness meditation with suicidal adults, Barnhofer and colleagues (2007) showed significant pre to post-decreases in F4/F3 asymmetry scores in the treatment as usual group, whereas no significant pre to post changes were evident in the Mindfulness Behavioral Cognitive Treatment (MBCT) group (Barnhofer et al., 2007). In summary, there are inconsistent findings regarding the stability and independence of cortical activity with and without interventions for depression and no study of frontal EEG asymmetry before and after treatment for depression.

Behavioral Activation treatment (BA) is an evidence-based treatment for depression (Dimidjian, Barrera, Martell, Munoz, & Lewinsohn, 2011; Martell, Dimidjian, & Herman-Dunn, 2010) and aims to alter inhibitory and exploratory behavior and modify cognitive activity (Jacobson et al., 1996). BA for major depression proposes that symptom reduction occurs via monitoring daily activities to identify patterns of low reward and mood, assessing and assigning tasks that generate pleasure and competence, understanding and reducing unproductive avoidance, and improving skill deficits to promote action towards treatment goals (Cuijpers, van Straten, & Warmerdam, 2007; Daughters et al., 2008; Dimidjian et al., 2006; Jacobson et al., 1996). There has been minimal research to clarify the extent to which BA may influence frontal EEG asymmetry in depressed patients, and the extent to which frontal EEG asymmetry underlies the changes in behavioral inhibition and activation, cognitive and behavioral avoidance, and positive and negative affect that occur during BA.

This study aimed to test the extent to which depressed, relative to healthy participants, show greater alpha EEG asymmetry and whether there are unique associations between frontal alpha EEG asymmetry, motivation, and affect among depressed versus healthy participants. This study also aimed to test whether frontal alpha EEG asymmetry would not change with BA treatment in depressed participants in an attempt to clarify conflicting results from prior findings, but that pre-treatment frontal alpha EEG asymmetry, controlling for motivation and affect, would predict post-treatment depression severity. We hypothesized:

1. Depressed relative to healthy participants will exhibit significantly greater frontal alpha EEG asymmetry scores at pre-treatment.

2. Frontal alpha EEG asymmetry will not be correlated with depressive severity, but will be correlated with Behavioral Inhibition Sensitivity, negative affect, and self-reported avoidance in depressed participants at pre-treatment.

3. Given the research on the stability of frontal alpha EEG asymmetry, symptomatic and remitted depressed patients will differ from healthy (never-disordered) participants at post-treatment.

4. Based on findings from aforementioned treatment studies on predictive utility of frontal alpha EEG asymmetry, pre-treatment frontal alpha EEG asymmetry will predict remission status at post-treatment in depressed participants.

Methods and Materials

Participants

Participants were 72 males and females (aged 18–65, $M = 35.65y$, $SD = 13.05y$), including 37 participants diagnosed with current major depressive disorder per the Structured Clinical Interview for the DSM-IV Axis I Disorders, (SCID; First, Spitzer, Gibbon, & Williams, 2002) *and* a score 24 on the Inventory of Depressive Symptomatology-Clinician-Rated, (IDS-C; Rush et al., 2003; Rush et al., 1986). Thirty-five healthy participants were enrolled with no lifetime history or current presentation of psychiatric symptoms per the SCID *and* a score 11 on the IDS-C. To enroll, all participants had to be medically healthy, unmedicated with no recent medication wash out, and between ages 18 and 65 years. Depressed participants were excluded if they had current diagnoses of bipolar I or II, psychosis, obsessive-compulsive disorder, substance abuse/dependence, borderline, schizoid, schizotypal, or antisocial personality disorders. There was available EEG data at pre-treatment for 35 depressed and 37 healthy participants. At post-treatment, EEG data was available for 29 depressed and 26 healthy participants.

Procedure

Participants were initially screened via phone to ensure eligibility, and then invited to the laboratory for two occasions, separated by one week. In the first session, participants provided informed consent, passed a toxicology urine screen, and completed the clinical interview and self-report questionnaires. On the day of the psychophysiological assessment (second session), participants were asked to come with their hair washed and free of styling products. They were also asked to refrain from smoking or drinking caffeine within two

hours of testing. Upon arrival, participants were asked to sign a consent form and take a urine toxicology screen, and were then seated in a comfortable chair in a light and sound attenuated room. Participants completed a second toxicology urine screen and the EEG assessment, in which sensors were applied, high quality data was assured, and 8 1-minute periods of resting EEG data were collected, half with of eyes open (O) and half with eyes-closed (C) (order counterbalanced that coordinated with event sequence in raw EEG). For the EEG assessment, participants were seated in a sound-attenuated room in a recliner. Compensation was offered upon completion. Thereafter, depressed participants were scheduled for their first of 16 treatment sessions to start the following week, and healthy participants were evaluated prospectively for 16 weeks. Compensation and debriefing was offered upon study completion.

Measures

Trained clinical psychology masters and doctoral students conducted psychiatric evaluations. All evaluators were supervised to prevent rater drift.

Structured Clinical Interview for the DSM-IV Axis I Disorders, Outpatient Version (SCID-I, First et al., 2002)—The SCID is a semi-structured interview that collects demographic information (age, years of education, marital and employment status), as well as clinical data (severity of depression, lifetime and current DSM-IV Axis I diagnoses). The SCID has adequate inter-rater reliability with kappa values for modules reported to be between .7 and 1 (First, Spitzer, Gibbon, & Williams, 1995). Our evaluators underwent a training program with SCID training tapes (Spitzer, Williams, Gibbon, & First, 1989), formal training, observing and demonstrating SCID competency, and then co-rating and reviewing SCID interviews. Our reliability checks of five separate tapes yielded κ 0.8 for the Mood and Anxiety modules.

Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II; First, Gibbon, & Spitzer, 1997)—The SCID-II is a 47-item self-report measure in which participants give 'yes' or 'no' responses to items probing for symptoms of Axis II disorders. All 'yes' responses are further probed by clinical interview to exclude those meeting criteria for Antisocial, Borderline, Schizotypal, or Schizoid Personality Disorder.

Longitudinal Follow-up Evaluation (LIFE; Keller et al., 1987)—The LIFE is a semi-structured retrospective interview focused on psychiatric status (e.g., onset, offset, duration of symptoms). The LIFE captures weekly status of DSM-IV depressive symptoms, asking the rater to assign a Psychiatric Status Rating (PSR) for each week. Data is recorded using the PSRs to reflect minimal symptoms (*PSRs 1–2)* to moderate to severe symptoms (*PSRs 3–6*).

Inventory of Depressive Symptomatology – Clinician-Rated (IDS-C; Rush, Carmody, & Reimitz, 2000; Rush et al., 1986)—IDS-C is a validated 30-item clinician-rated measure that assesses DSM-IV symptom domains for MDD, including mood, cognitive, psychomotor, and vegetative, to diagnose a Major Depressive Episode and commonly associated symptoms (e.g., anxiety, somatization). Inter-rater reliability estimates

from this study's sample yielded a Krippendorff's alpha ratio of .87 for IDS-C total scores. Cronbach alpha values for our sample were .62 for the depressed group and .56 for the healthy group.

Inventory of Depressive Symptomatology – Self-Rated (IDS-SR; J. A. Rush et al., 1986—The IDS-SR is a 30-item patient self-rated measure that corresponds to the IDS-C, used for the purpose of comparing subjective and clinician-rated depression severity. Convergent validity with the IDS-C in our sample was strong, with correlations of .96 at pre-treatment and .91 at post-treatment. Cronbach alpha estimates for our sample were .73 for the depressed group and .59 for the healthy group.

Behavioral Inhibition System/Behavioral Activation System (BIS/BAS scales; Carver & White, 1994)—The BIS/BAS is a 20-item self-report form. Seven items measure the Behavioral Inhibition System (i.e., aversive motivation) and 13 items measure the Behavioral Activation System (i.e., appetitive motivation). Responses are recorded using a four-point Likert scale ranging from strong agreement (*1 = very true for me*) to strong disagreement (*4 = very false for me*). Behavioral Inhibition is measured using a single subscale relevant to punishment sensitivity. The BAS scale contains three subscales: (1) Drive, (2) Reward Responsiveness, and (3) Fun Seeking, representing behavioral, affective, and combined behavioral-affective motivational responding to reward. Cronbach alpha estimates in our sample were equivalent for healthy and depressed participants on BIS (.78 and .77, respectively), BAS Reward (.85 and .61, respectively), BAS Drive (.83 and .75, respectively), and BAS Fun Seeking (.75 and .53).

Positive Affect and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988)—The PANAS is a 20-item self-report measure, divided into two ten-item subscales and measures activation of positive and negative affect. The PANAS consists of a list of 20 emotion states. The rater is asked to indicate the extent to which the rater has experienced this emotion in the preceding seven days, using a five-item Likert scale ranging from '*Very slightly or Not at all*' to '*Extremely*'. The subscales of the PANAS have been shown to be reliable: Cronbach alphas were .89 for positive affect and .85 for negative affect in a normative sample (Crawford & Henry, 2004). In our sample, Cronbach alpha estimates were .86 for positive affect in the depressed group and .87 in the healthy group. For negative affect, internal consistency estimates were .85 in the depressed group and .92 in the healthy group.

Cognitive Behavioral Avoidance Scale (CBAS; Ottenbreit & Dobson, 2004)—The Cognitive Behavioral Avoidance Scale is a 31 item self-report measure that assesses four reliable factors reflecting combinations of cognitive/behavioral and social/nonsocial dimensions of avoidance. Participants are asked to identify the extent to which they use strategies to respond to situations and problems, using a rating system (*1 = not at all true* to *5 = extremely true)*. The cognitive social subscale items represent avoidance or passivity of addressing relationship problems, waiting for social tension to change, and failing to address tension in friendships. The behavioral social dimension refers to social withdrawal during social activities, avoiding opportunities to meet with the opposite sex, and not responding to

invitations to be social. The CBAS has been shown to be a valid and reliable measure of avoidance in an undergraduate student population (Ottenbreit & Dobson, 2004). Cronbach alphas were generally equivalent for the depressed and healthy participants on the behavioral social avoidance subscale (.81 and .51, respectively), on the behavioral nonsocial subscale (.74 and .67, respectively), on the cognitive social avoidance subscale (.78 and .79), and on the cognitive nonsocial avoidance subscale (.89 and .84, respectively).

EEG Recording, Reduction, and Analyses—EEG was recorded for 8 minutes while participants were awake and relaxed (Mindware Technologies, Gahanna, OH). Using a stretch-lycra cap with Ag/AgCl electrodes, continuous EEG was collected over 20 scalp sites using the International 10–20 system (AF3/AF4, F3/F4, F7/F8, FC1/FC2, FC5/FC6, C3/C4, T7/T8, P3/P4, P7/P8, O1/O2, referenced to M1 with M2 as an active channel and the area between Fz and Fpz as the participant ground; bandpass 0.01–100 Hz with 60 Hz notch; impedances < 5kΩ with homologs within ± 1kΩ; digitization rate 500 Hz). Offline, a digital average mastoid reference, (M1+M2)/2, was performed.

Following data recording, a technologist who was uninvolved in data collection and blind to participant identity visually inspected the EEG data for eye movements and muscle artifacts, and data with artifacts were rejected from all data channels. Then, we applied a principal-component-based spatial filter that was designed to remove ocular artifact components while sparing frontal EEG components (Ford, Sands & Lew, 2004; as implemented in EMSE 5.5.1, 2013). Power spectra were derived by means of a fast Fourier transform with a Hamming window (50% overlap) for each two second epoch within each baseline type (eyes open, eyes closed) for each channel for each participant, thus EEG finding are specific to both open and closed eyes. Thus, each one minute baseline type contained 59 overlapping two-second epochs. The median power spectrum, which is robust with respect to outliers, was obtained by rank ordering the 59 Fourier transformed magnitudes at each 0.5 Hz frequency bin and each channel and then selecting the median (middle) value. This processing was conducted using a 50% trimmed mean in EMSE version 5.5.1 (Source Signal Imaging, Inc., La Mesa, CA). Half Hertz frequency bins in the 8–13 Hz range were averaged to produce band average median alpha power.

Frontal EEG asymmetry scores were calculated over the midfrontal sites, subtracting the natural log of the alpha power of the electrode in the left hemisphere (F3 or F7) from that of the right frontal electrode (F4 or F8). A higher score thus reflected greater relative left versus right frontal activation (e.g., relatively higher right alpha activity; Coan & Allen, 2004; Lindsley & Wicke, 1974). However, we were interested in asymmetry scores in general, and therefore, the absolute value of this difference score was taken. Using the natural log transformation is commonly used in EEG asymmetry research as EEG power appears to be positively skewed (Tomarken, Davidson, Wheeler, & Doss, 1992). The choice of sites was based on previous research (i.e., F3/F4; Shackman et al., 2009). Mean Cronbach's alpha internal consistency estimates for resting alpha asymmetry as a function of length of recording (eight 1 minute recordings) were 0.96 and 0.95 for F4 and F3 at pre- and post-treatment in the depressed group and 0.95 and 0.94 in the healthy sample.

Treatment

Weekly sessions for 16 consecutive weeks and a BA patient workbook (Addis & Martell, 2004) were offered. Clinicians were offered two clinician handbooks (Martell, Addis, & Jacobson, 2001; Martell et al., 2010), training, supervision, and integrity reviews by off-site experts. Patients learned the BA model of depression, monitored daily activities, assessed pleasure and competence, assigned or scheduled activities that promoted mastery or pleasure, rehearsal of scheduled activities, clarified patterns of avoidance, identified goals, reduced rumination, and modified skill deficits. Clinicians included postdoctoral fellows in clinical psychology ($n = 2$) and licensed clinical psychologists ($n = 2$) trained with one pilot case and then were assessed for competency thereafter. Study clinicians underwent competency reviews by off-site BA experts, Drs. Christopher Martell and Ruth Herman-Dunn. These experts reviewed 10% of audiotaped treatment sessions and issued independent ratings using the Behavioral Activation Treatment Scale (BATS; Jacobson et al., 1996), a 16-item measure designed to assess core competencies of BA (Dimidjian et al., 2006; Jacobson et al., 1996), using ratings assigned on a scale of 1 to 6 (*1 = Poor*, *6 = Excellent*). Clinicians achieved competency ratings over the minimum requirement of 60 ($M = 68.26$, $SD = 5.21$, Range = 63–75). Finally, clinicians exhibited strong adherence to BA, per assessments conducted by trained evaluators (KHF, DH) using the BA items from the Collaborative Study Psychotherapy Rating Scale (CSPRS; Hollon et al., 1998). The adherence measure outlined 28 items, rated on a 0–7 scale. After establishing inter-rater reliability (.86), adherence scores on 20% of the completer sample were generated ($M = 4.96$, $SD = .37$).

Statistical Analyses

Analyses of demographic and clinical characteristics at pre-treatment were conducted using one sample *t*-tests or Analysis of Variance (ANOVA) for continuous variables and Chi-square tests of independence for categorical variables. In the presence of small or empty cells in the tests of categorical variables, the Chi-square test was replaced by Fisher's exact test. Analyses included partial and full completers using the definitions from prior BA trials (Dimidjian et al., 2006; Jacobson et al., 1996). Partial completers (attended 5 – 11 sessions, $n = 4$) and completers (attended 12 sessions, $n = 32$) completed an average of 13.43 ($SD = 1.68$), or 84%, of the 16 available sessions. Analyses differentiating change in BA treatment defined remission as IDS-C 11 at post-treatment *and* at PSR of 1 or 2 at post-treatment (Frank et al., 1991).

A one way ANOVA to examine differences in alpha EEG asymmetry between the depressed and healthy groups at pretreatment was conducted. Analyses were two-tailed at the .05 level of significance. Two separate repeated measures ANOVAs to test group effects (depressed versus healthy) over time (pre- and post-treatment status) focused on F4-F3 alpha asymmetry and F8-F7 alpha asymmetry. This was conducted to retain the intent to treat sample. Pearson correlations were conducted to test the total alpha power at each site (F3, F4, F7 and F8) at pre- and at post-treatment with the scores on the BIS, BAS subscales, CBAS, and PANAS. All correlations were computed within diagnostic groups (depressed and healthy). Finally, logistic regressions were computed to evaluate the predictive use of pre-treatment F4-F3 and F8-F7 alpha asymmetry scores to identify participants who remit

after a course of BA treatment. Alpha asymmetry scores were entered in separate logistic regressions as predictor variables for the categorical dependent variable of remission measured at post-treatment.

Results

Demographic and Clinical Characteristics

Demographics are presented in Table 1. Of the depressed participants (M_{age} = 36, SD_{age} = 12, $Range_{age}$ = 22–62), 70% were female (n = 26) and 41% had completed college (n = 15). Over half of the participants were Caucasian (n = 22; 59.5%), a third were African American (n = 11; 29.7%) and the rest were Hispanic (n = 4; 10.8%).

Chi-square analyses revealed no group differences on demographic characteristics between healthy and depressed groups (χ_{gender} = 1.9, ns; $\chi_{ethnicity}$ = 5.9, ns; $\chi_{employment}$ = 7.2, ns; $\chi_{marital\ status}$ = 3.5, ns). Group differences were found on severity of depression at pre-treatment, F_{IDS-R} (1, 71) = 366.1, $p < 0.01$; $M_{Healthy}$ = 3.1 (SE = 0.5, 95% CI: 2.1, 4.1), $M_{Depressed}$ = 33.2 (SE = 1.4, 95% CI: 30.2, 36.2), and at post-treatment, F_{IDS-C} (1, 71) = 529.7, $p < 0.01$, $M_{Healthy}$ = 2.2 (SE = 0.4, 95% CI: 1.3, 3.1); $M_{Depressed}$ = 33.7 (SE = 1.2, 95% CI: 31.1, 36.1). The average age of onset of depression was 30.73 years (SD = 23.3) and 67% reported recurrent depression. Slightly less than half of participants (n = 15, 40.5%) had received treatment prior to study enrollment. Within this sample, 8 participants received consultation for treatment or described participating in a brief period of treatment (21.6%), 3 participants received continuous treatment for six months or several brief periods (8.1%), and 4 participants received continuous treatment lasting one year or more or numerous brief periods (10.8%). All participants were unmedicated at the start of treatment.

The mean and variance of scores on self-reported scales in healthy and depressed participants are in Table 2. Depressed participants showed significantly less behavioral activation sensitivity, more behavioral inhibition sensitivity, greater negative affect, decreased positive affect.

Frontal Alpha EEG Asymmetry Differences

Table 3 outlines means and standard deviations of alpha power at F3, F4, F7, F8, and alpha asymmetry in depressed and healthy groups. A one way ANOVA to examine differences in alpha EEG asymmetry by two groups (depressed v. healthy) at pretreatment indicated that depressed participants showed a significantly higher F4-F3 alpha asymmetry (M = 0.43, SE = 0.12, 95% CI: 0.18, 0.67) than the healthy participants (M = 0.16, SE = 0.05, 95% CI: 0.18, 0.67). No differences were found for F8-F7 alpha asymmetry and for absolute alpha power at each site at pre-treatment. Figure 1 shows that depressed participants have a more diffuse pattern of alpha activation from the anterior to posterior part of the brain unlike healthy participants who retain focused frontal right alpha activity. The contrast between groups appears most strongly at the 9.5 Hz peak, although it remains evident via the contour lines after averaging across 8–12 Hz.

Two separate repeated measures analysis of variance (ANOVA) to test group effects (depressed versus healthy) over time (pre- and post-treatment status) focused on F4-F3 alpha

asymmetry and F8-F7 alpha asymmetry. Results indicated that there was no significant interaction effect between group and time in F4-F3 alpha asymmetry $F(1,53) = 0.88$, $p = 1.44$, $\eta^2 = 0.01$). Similarly, there was no main group effect, $F(1,53) = 3.64$, $p = 0.62$, $\eta^2 = 0.06$), and no main effect of time $F(1,53) = 2.23$, $p = 0.14$, $\eta^2 = 0.04$). Results for the F8-F7 alpha asymmetry showed no significant interaction effect between group and time $F(1,53) = 1.35$, $p = 0.24$, $\eta^2 = 0.02$), no main effect for group $F(1,53) = 3.80$, $p = 0.56$), but there was a significant main effect for time, $F(1, 53) = 6.67$, $p = .013$, $\eta^2 = 0.11$). Given that 67% of the depressed sample reported recurrent depression, we conducted GLM Repeated Measures with episode (single, recurrent) as between-subjects factor and F4-F3 alpha asymmetry as within-subjects factor. Results indicated that there was no significant interaction effect between group and time in F4-F3 alpha asymmetry $F(1,25) = 1.26$, $p = 0.27$, $\eta^2 = 0.04$). Similarly, there was no main group effect, $F(1,25) = 0.49$, $p = 0.48$, $\eta^2 = 0.02$), and no main effect of time $F(1,25) = 2.82$, $p = 0.10$, $\eta^2 = 0.10$).

Additionally, correlation between F4-F3 alpha asymmetry at pre and post-treatment was not significant in the depressed subgroup with recurrent depression ($r = 0.07$, $p = 0.71$) and also statistically not significant in the single depression subgroup ($r = 0.14$, $p = 0.37$).

Correlational Analyses Between EEG Asymmetry, Depression, and Behavioral Measures

The healthy group exhibited no significant correlations between F4-F3 and F8-F7 alpha asymmetry indices and depression severity and behavioral measures at pre- and at post-treatment. Table 4 shows bivariate correlations between frontal EEG activity and measures of depression, avoidance, and approach at pre- and end-treatment in the depressed sample. The depressed group exhibited significant positive correlations between F4-F3 alpha EEG asymmetry and BIS, $r(37) = 0.41$, $p = 0.05$ and negative affect, $r(37) = 0.39$, $p = 0.05$, as well a significant inverse correlation between F4-F3 alpha EEG asymmetry and BAS Fun Seeking, $r(37) = -0.41$, $p = 0.05$ at pre-treatment. No significant correlations were observed between F4-F3 alpha asymmetry, depressive severity, and behavioral measures at post-treatment. In the depressed group, no significant correlations were observed between F8-F7 alpha asymmetry and behavioral measures at pre-treatment. However, significant positive correlations were observed between F8-F7 alpha asymmetry and BIS $r(31) = 0.44$, $p = 0.05$ and CBAS behavioral nonsocial avoidance subscale, $r(31) = 0.44$, $p = 0.05$ at post-treatment. No significant correlation was observed with depressive severity.

Utility of Alpha Asymmetry in Predicting BA Remission

The predictive use of pre-treatment alpha asymmetry for determining remission status at post-treatment was ascertained using logistic regression analyses. Results from the two logistic regression analyses using F4-F3 and F8-F7 alpha asymmetry did not predict group status (remitter, non-remitter, healthy). Multiple regression analyses indicated pre-treatment F4-F3 alpha asymmetry predicted negative affect at post-treatment, $R = 0.43$, $R_{square} = 0.18$, $R_{adjusted} = .16$, $SE = 6.75$, $F(1, 28) = 6.61$, $p = 0.01$. We further examined this finding by using pre-treatment negative affect and F4-F3 alpha asymmetry as predictors of negative affect at end of treatment to test whether negative affect was stable over time in the depressed group and pre-treatment F4-F3 asymmetry remained a significant predictor. In this model, we obtained $R = 0.43$, $R_{square} = 0.18$, $R_{adjusted} = 0.13$, $SE = 6.90$ ($F(1, 28) =$

3.23, $p = 0.05$) with pre-treatment frontal alpha asymmetry as a significant predictor ($\beta = 0.44$, $p = 0.02$, 95% *CI*: 0.81, 9.29) whereas pre-treatment negative affect was not significant ($\beta = -0.04$, $p = 0.80$, 95% *CI*: −0.37, 0.29).

Discussion

The current study was designed to investigate (a) differences in frontal alpha EEG asymmetry in depressed participants relative to healthy controls, (b) the effect of BA treatment on frontal alpha EEG asymmetry after treatment, (c) the strength and direction of associations between frontal alpha EEG asymmetry and motivational systems, behavioral avoidance, and affective disposition, and (d) the predictive utility of alpha EEG asymmetry in determining remission status after 16 weeks of BA treatment. Results showed that at pre-treatment, depressed, relative to healthy participants, exhibited significantly greater frontal alpha EEG asymmetry. Across treatment, there was an absence of change in frontal alpha EEG asymmetry in healthy and depressed patients, even when patients reported a significant alpha reduction of depressive symptoms at post-treatment. In terms of correlations, among depressed participants at pre-treatment, alpha EEG asymmetry showed a significant positive correlation with behavioral inhibition and negative affect, and a significant inverse correlation with behavioral activation sensitivity (fun-seeking motivation). In comparison, at post-treatment, depressed participants with higher frontal alpha EEG asymmetry showed a positive correlation with behavioral inhibition and behavioral avoidance. No correlation was observed at pre- and at post-treatment between alpha EEG asymmetry and depression severity. Finally, alpha EEG asymmetry status at pre-treatment did not predict treatment response at post-treatment, though it was predictive of negative affect at post-treatment.

The performance of resting frontal alpha EEG asymmetry from this study suggests that this neurophysiologic characteristic may be a stable, trait-like characteristic independent of depression status. Many of the results from this study echo prior research showing that alpha asymmetry distinguishes depressed from healthy individuals, and that this dimension of cortical activity remains steady over time in healthy individuals and among depressed individuals who report symptom reduction. In addition to being a stable trait associated with behavioral avoidance and behavioral inhibition sensitivity in depressed participants, frontal alpha EEG asymmetry was evidently unalterable by behavioral activation treatment strategies (see also Allen et al., 2004), but it does not rule out the possibility that the presence of hemispheric asymmetry in remitters may change with stability of the remission.

The results of this study support the preferential expression of behavioral inhibition sensitivity and avoidance and negative affect with frontal alpha EEG asymmetry, consistent with previous research outlining neuropsychological function of approach- and withdrawal-related behavior in depression (Coan & Allen, 2003; Shackman et al., 2009). The results from this study also offer support to the concept of frontal alpha EEG asymmetry related to inhibition sensitivity (Kemp, Gordon, Rush, & Williams, 2008; Shankman, Klein, Tenke, & Bruder, 2007). Furthermore, frontal alpha EEG asymmetry and behavioral inhibition sensitivity were tightly linked before and after treatment.

In the current BA model, a functional analyses is often used to ascertain the context and consequence of depression symptoms (Ferster, 1973), as well as how to modify avoidance and context to generate treatment response. Reversing behavioral inhibition, as well as promoting behavioral activation systems, could potentially represent BA-specific mechanisms. Also, according to the cognitive model (Beck, 2008), behavioral strategies are designed to modify dysfunctional attitudes, and when cognitions are modified early in treatment, there is subsequent clinical improvement (DeRubeis et al., 1990; Kwon & Oei, 2003). BA treatment does not focus on tasks requiring patients to directly modify attentional performance, potentially helping patients to practice and subsequently increase alpha power, but rather patients are asked to focus on behavioral modification of avoidance, which may not sufficiently modify frontal alpha EEG asymmetry. The use of BA, which aims to alter behavioral and cognitive avoidance, and does not specifically target cognitive activity and motivational substrates of inhibition and activation sensitivity, therefore may have limited the predictive utility of alpha EEG asymmetry in BA.

Clinically, the findings of this study indicate that depressed individuals are likely to show frontal asymmetry, which increases the tendency for the individual to report higher negative affect and behavioral inhibition sensitivity. Moreover, the lack of change in cortical activity does not correspond to the individual's response to treatment. If BA were viewed as an opportunity to modify cortical activity, the individual's pre-treatment status would not necessarily limit the potential for achieving recovery, offering clinical information to patients who potentially may wish to know if pre-treatment alpha EEG asymmetry influences the propensity to remit with BA. Despite an interest in improving early prediction of response to BA, very few studies have yet examined the extent to which change in specific patient-related characteristics, separately and in combination, serve as putative mediators of treatment response, and none, thus far, have examined alpha EEG asymmetry as a predictor of outcome. Understanding how frontal alpha EEG asymmetry predicts negative affect at post-treatment may advance our knowledge of potential predictors of relapse among those fortunate enough to experience remission.

In terms of limitations of the study, inferences cannot be made about which hemisphere was influenced (i.e., reduced left or increased right) due to the use of asymmetry scores in addition to type of measurement (i.e., absolute value). Furthermore, frontal alpha EEG asymmetry was measured while the participant was resting, and it may differ during a cognitive task (Coan, Allen, & McKnight, 2006). Specifically, alpha EEG synchronization during mental relaxation may not offer a sufficient test of capability as might be observed during task performance (Coan et al., 2006) given that decreased alpha power during cognitive tasks represents the proportion of cortical neural activity involved in the task (Dujardin et al., 1993; Gevins & Smith, 2000; Gevins et al., 1998). Also, because the study did not include a group of untreated depressed participants, we cannot determine if untreated depressed participants over time would exhibit similar features with the alpha asymmetry as observed in the remitted and depressed subgroups. Also, the anterior asymmetries in the healthy participants may be generated by the reduction of alpha at the left central/temporal sites, aligning with an asymmetric mu rhythm in sensorimotor regions (e.g., contraction of right-handed muscles) (Pineda, 2005), Mu rhythm has frequencies in the 8–13Hz band, which have been associated with greater left than right frontal activity and increased self-

reported approach affect (Harmon-Jones, 2006). Because we did not use a formal measure of handedness, rather we asked the participants to confirm that they were dominant right-handed, it is possible that mu rhythms explain the alpha asymmetry difference. Also, though all participants did not use a substance (e.g., alcohol, nicotine, or caffeine) within three hours of the tasks, there is the potential that some of these individuals were smokers, which can influence EEG asymmetry and mood in depression (Knott et al., 2012). Finally, though frontal alpha EEG asymmetry represents a neurophysiologic indicator of cognitive activity, there is no accompanying measure to assess cognitive activity. The use of neural-event related potentials (ERP) to valenced stimuli may offer new data to clarify the neural substrates of altered activity in depressed participants relative to healthy controls. Strengths of this study include the reliance of treatment-seeking depressed patients who were unmedicated at the time of enrollment, and we ensured that depressed participants endorsed moderate severity of illness.

In conclusion, the data from this study has shown that depressed relative to healthy participants show a greater frontal alpha EEG asymmetry before they enrolled into BA treatment, demonstrating a more diffuse pattern of activation from the anterior to posterior part of the brain unlike healthy participants who retain focused frontal right activity. Also, higher frontal alpha EEG asymmetry does not appear to underlie clinical severity before and after treatment, rather higher behavioral inhibition and negative affect along with lower behavioral activation sensitivity (fun-seeking motivation) at pre-treatment. The specific functions of EEG asymmetry where we observed diffuse pattern of activation remains to be determined, though continued study of the frontal alpha EEG asymmetry index may advance efforts to identify neurophysiologic markers of major depression.

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Highlights

- We test for alpha EEG asymmetry differences in depressed and healthy adults, as well as for predictive utility of alpha EEG asymmetry of treatment response to Behavioral Activation for depression.

- Alpha EEG asymmetry was significantly higher in depressed than healthy participants at pre-treatment.

- Alpha EEG asymmetry was positively correlated with negative affect and behavioral inhibition, and inversely correlated with lower behavioral activation sensitivity.

- Heightened alpha EEG asymmetry in depressed participants may reflect a stable neurophysiologic characteristic independent of depressive severity.
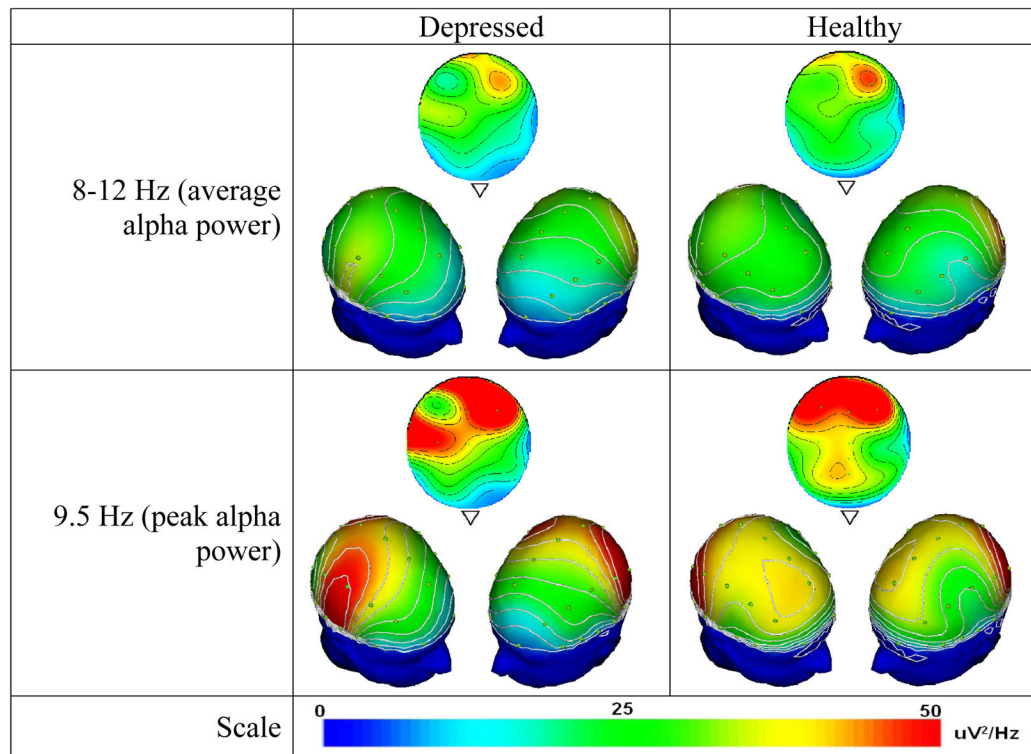
Figure 1.
Topographical distributions of 8–12 Hz band average alpha power (top) and 9.5 Hz peak alpha power (bottom) for depressed (left) and healthy (right) participants, showing a top view stretched across all recording sites, along with two 3D-rotated views to display frontal laterality. Depressed participants assessed at pretreatment show a more diffuse anterior-to-posterior pattern of alpha activation (note contour lines) unlike healthy participants who retain focused right frontal activity. Both groups have a peak frequency at 9.5 Hz, at which the noted contrast is particularly evident. Contour lines are spaced at intervals of 5 μV^2/Hz.

Table 1

Demographic and Clinical Characteristics

	MDD (n=37)		HV (n=35)		Relevant Statistic	p value
	n	(%)	n	(%)		
Sex						
Male	11	(29.7)	16	(45.7)	1.96	.16
Female	26	(70.3)	19	(54.3)		
Race						
Caucasian	22	(59.5)	19	(54.3)	5.97	.11
Black	11	(29.7)	11	(31.4)		
Hispanic	4	(10.8)	1	(2.9)		
Other	0	(0.0)	4	(11.4)		
Education						
Under 7 years of school	0	(0.0)	1	(2.9)	5.22	.39
Partial high school	1	(2.7)	0	(0.0)		
High school graduate	2	(5.4)	1	(2.9)		
Partial college	14	(37.8)	14	(40.0)		
College graduate	15	(40.5)	18	(51.4)		
Graduate training	5	(13.5)	1	(2.9)		
Marital Status						
Never married	27	(73.0)	24	(68.6)	3.52	.47
Married	2	(5.4)	6	(17.1)		
Separated	1	(2.7)	0	(0.0)		
Divorced	6	(16.2)	4	(11.4)		
Common Law	1	(2.7)	1	(2.9)		
Employment						
Unemployed	14	(37.8)	6	(17.1)	7.27	.06
Employed	18	(48.6)	19	(54.3)		
Full-time student	5	(13.5)	6	(17.1)		
Retired	0	(0.0)	4	(11.4)		
	M	(SD)	M	(SD)		

	MDD (*n*=37)		HV (*n*=35)		Relevant Statistic	*p* value
	n	(%)		(%)		
Age						
Years	36.2	(12.4)	35.1	(13.7)	.13	.72

Table 2

Clinical Measures at Pre- and Post-Treatment

| Measure | MDD ($n = 37$)[a] | | HV ($n = 35$)[b] | | | |
	M	SD	M	SD	F(df)	p value
IDS-C						
Pre-treatment	33.73	7.69	2.26	2.58	$F(1, 71) = 529.78$	<.001
Post-treatment	12.81	8.60	2.24	2.77	$F(1, 63) = 44.83$	<.001
IDS-SR						
Pre-treatment	33.19	8.82	3.17	2.97	$F(1, 71) = 366.12$	<.001
Post-treatment	15.26	10.69	3.03	4.64	$F(1, 63) = 35.98$	<.001
BIS						
Pre-treatment	20.97	3.57	15.8	3.55	$F(1, 71) = 38.00$	<.001
Post-treatment	20.06	4.33	15.55	3.76	$F(1, 63) = 19.96$	<.001
BAS Reward						
Pre-treatment	14.81	3.23	17.54	1.74	$F(1, 71) = 19.65$	<.001
Post-treatment	15.97	3.77	17.45	1.94	$F(1, 63) = 4.00$	.05
BAS Drive						
Pre-treatment	8.92	2.94	11.26	2.33	$F(1, 71) = 13.89$	<.001
Post-treatment	9.68	2.94	12.15	2.40	$F(1, 63) = 13.69$	<.001
BAS Fun						
Pre-treatment	9.89	2.32	12.00	1.91	$F(1, 71) = 17.62$	<.001
Post-treatment	11.32	2.80	12.33	2.34	$F(1, 63) = 2.47$	.12
PANAS-PA						
Pre-treatment	18.24	6.12	38.74	5.80	$F(1, 71) = 212.36$	<.001
Post-treatment	31.42	8.68	38.55	7.33	$F(1, 63) = 12.65$	<.001
PANAS-NA						
Pre-treatment	27.57	8.33	12.06	3.86	$F(1, 71) = 100.73$	<.001
Post-treatment	21.32	7.40	13.24	4.96	$F(1, 63) = 26.62$	<.001
CBAS-Cog-S						
Pre-treatment	15.92	5.60	9.63	3.53	$F(1, 71) = 32.04$	<.001
Post-treatment	12.81	4.51	9.70	3.29	$F(1, 63) = 10.00$	<.01

Measure	MDD ($n = 37$)[a]		HV ($n = 35$)[b]			
	M	SD	M	SD	F(df)	p value
CBAS Cog-NS						
Pre-treatment	22.89	9.19	14.20	5.71	$F(1, 71) = 22.94$	<.001
Post-treatment	20.00	8.33	14.09	5.60	$F(1, 63) = 11.22$	<.001
CBAS Beh-S						
Pre-treatment	19.41	6.87	11.49	2.96	$F(1, 71) = 39.57$	<.001
Post-treatment	16.16	6.70	10.85	3.02	$F(1, 63) = 17.07$	<.001
CBAS-Beh-NS						
Pre-treatment	14.16	4.72	8.43	2.74	$F(1, 71) = 39.14$	<.001
Post-treatment	11.87	4.46	8.15	2.21	$F(1, 63) = 18.24$	<.001

Note: IDS-C = Inventory of Depressive Symptomatology – Clinician; IDS – SR = Inventory of Depressive Symptomatology – Self-Report; BIS = Behavioral Inhibition System, BAS Reward = Behavioral Activation System Reward Subscale, BAS Drive = Behavioral Activation System Drive Subscale, BAS Fun = Behavioral Activation System Fun Seeking Subscale, PANAS-PA = Positive Affect Negative Affect Scale Positive Affect Subscale, PANAS-NA = Positive Affect Negative Affect Scale Negative Affect Subscale. CBAS-Cog-S = Cognitive Behavioral Avoidance Scale Cognitive Social Subscale, CBAS-Cog-NS = Cognitive Behavioral Avoidance Scale Cognitive

Nonsocial Subscale, CBAS-Beh-S = Cognitive Behavioral Avoidance Scale Behavioral Social Subscale, CBAS-Beh-NS = Cognitive Behavioral Avoidance Scale Behavioral Nonsocial Subscale.

[a]Post-treatment $n = 31$,

[b]Post-treatment $n = 33$

Table 3

EEG Estimates for Sites and Asymmetry Scores

EEG measure	MDD (n = 37)[a]				HV (n = 35)[b]					
	M	SD	SE	95% CI	M	SD	SE	95% CI	F(df)	p
F3										
Pre-treatment	−0.26	0.86	0.14	−0.54, 0.03	−0.37	0.91	0.15	−0.69, −0.06	$F(1, 71) = 0.31$	.58
Post-treatment	−0.02	1.38	0.26	−0.54, 0.51	−0.34	0.89	0.16	−0.68, 0.00	$F(1, 57) = 1.09$	.30
F4										
Pre-treatment	−0.36	0.98	0.16	−0.69, −0.04	−0.37	0.89	0.15	−0.67, −0.06	$F(1, 71) = 0.$	.98
Post-treatment	0.01	1.42	0.26	−0.53, 0.55	0.31	0.80	0.15	−0.62, 0.01	$F(1, 57) = 1.16$	.29
F7										
Pre-treatment	−0.39	1.08	0.18	−0.75, −0.03	−0.38	0.87	0.15	−0.68, −0.08	$F(1, 71) = 0.00$	.95
Post-treatment	0.06	1.07	0.20	−0.35, 0.47	−0.26	0.73	0.14	−0.53, 0.02	$F(1, 57) = 1.68$	0.20
F8										
Pre-treatment	−0.26	0.78	0.13	−0.52, −0.00	−0.35	0.83	0.14	−0.63, −0.06	$F(1, 71) = 0.19$	.66
Post-treatment	0.09	1.28	0.24	−0.40, 0.58	−0.27	0.74	0.14	−0.55, 0.01	$F(1, 57) = 1.78$	.19
F4 – F3[c]										
Pre-treatment	0.43	0.73	0.12	0.18, 0.67	0.16	0.27	0.05	0.06, 0.25	$F(1, 71) = 4.26$	.04*
Post-treatment	0.22	0.19	0.03	0.15, 0.29	0.13	0.10	0.02	0.09, 0.18	$F(1, 54) = 4.41$	.04*
F8 – F7[c]										
Pre-treatment	0.33	0.59	0.10	0.14, 0.53	0.17	0.18	0.03	0.11, 0.24	$F(1, 71) = 2.41$	.13
Post-treatment	0.39	0.41	0.08	0.23, 0.55	0.24	0.18	0.03	0.17, 0.31	$F(1, 56) = 3.22$	.08

Note:

* $p < .05$

[a] Post-treatment for F3/F4 n = 29, for F7/F8 n = 28

[b] Post-treatment for F3/F4 n = 26, for F7/F8 n = 29

[c] Absolute values

Table 4

Bivariate Correlations Between Frontal Activity, Depression, Approach, and Avoidance in the Depressed Group

	Pre F4-F3	Post F4-F3	Pre IDS-SR	Post IDS-SR	Pre BIS	Post BIS	Pre BAS-Reward	Post BAS-Reward	Pre BAS-Drive	Post BAS-Drive	Pre BAS-Fun	Post BAS-Fun
1	1	.07	.25	-.12	.41*	.34	.22	.16	.19	.06	-.41*	-.19
p		.72	.14	.54	.01	.06	.18	.38	.25	.74	.01	.31
2			.06	-.20	-.02	-.02	-.00	.11	.05	-.04	.07	-.03
p			.75	.29	.93	.94	.99	.58	.79	.85	.71	.89
3				.28	.14	.00	-.01	.11	.04	-.10	-.36*	-.04
p				.10	.38	.99	.95	.55	.81	.56	.02	.84
4					-.08	.14	-.21	-.18	-.16	.05	-.33	.14
p					.65	.45	.23	.31	.37	.77	.06	.44
5						.44**	.54**	.05	.20	-.23	.13	-.12
p						.01	.00	.80	.21	.19	.41	.51
6							.29	.10	-.11	-.22	-.08	.11
p							.10	.56	.54	.21	.64	.55
7								.16	.57**	-.13	.51**	-.06
p								.36	.00	.45	.00	.72
8									.44**	.55**	.14	.68**
p									.01	.00	.43	.00
9										.37*	.42**	.15
p										.03	.01	.40
10											.05	.47**
p											.78	.01
11												.12
p												.51

Note: Pre-tx= Pre-treatment; End-tx= End-Treatment; IDS-C = Inventory of Depressive Symptomatology – Clinician; IDS – SR = Inventory of Depressive Symptomatology –Self-Report; BIS = Behavioral Inhibition System, BAS Reward = Behavioral Activation System Reward Subscale, BAS Drive = Behavioral Activation System Drive Subscale, BAS Fun = Behavioral Activation System Fun Seeking Subscale, PANAS-PA = Positive Affect Negative Affect Scale Positive Affect Subscale, PANAS-NA = Positive Affect Negative Affect Scale Negative Affect Subscale. CBAS-Cog-S = Cognitive Behavioral Avoidance Scale Cognitive Social Subscale, CBAS-Cog-NS = Cognitive Behavioral Avoidance Scale Cognitive Nonsocial Subscale, CBAS-Beh-S = Cognitive Behavioral Avoidance Scale Behavioral Social Subscale, CBAS-Beh-NS = Cognitive Behavioral Avoidance Scale Behavioral Nonsocial Subscale.

*
$p < .05$,

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$p < .001$