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Caitlin Coyiuto
ccoyiuto@wellesley.edu

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Resting EEG Asymmetries and Levels of Irritability

Caitlin Coyiuto
PSYC 350
Wellesley College
Introduction

Although irritability is a common mood in everyone, it can be highly debilitating in its chronic and severe form. Irritability is a diagnostic criterion for multiple mood and anxiety disorders (Krieger, Leibenluft, Stringaris, & Polanczyk, 2013), and high levels of irritability in children or adolescents predict aggressive, anxious and depressive disorders in adulthood (Leibenluft & Stoddard, 2013; Stringaris, Cohen, Pine, & Leibenluft, 2009). Psychopathologies characterized by severe, persistent irritability, such as Severe Mood Dysregulation Disorder (SMD) or Disruptive Mood Dysregulation Disorder (DMDD), also possess high co-morbidity with mood disorders (Brotman et al., 2006; Copeland, Angold, Costello, & Egger, 2013; Krieger et al., 2013). Despite its prevalence in psychiatric disorders, a limited amount of research has been dedicated to understanding irritability. This has had clinical repercussions, such as the misdiagnoses of mental disorders. For example, chronic irritability was misdiagnosed as a developmental presentation of bipolar disorder, which lead to the administration of inappropriate treatments to children who did not have bipolar disorder (Krieger et al., 2013). To prevent future misdiagnoses and to better help identify populations at risk, the diagnostic and predictive capabilities of irritability should be elucidated.

Irritability can be viewed as a form of emotion dysregulation (Leibenluft & Stoddard, 2013). Usually, emotions are regulated by a series of processes that allow for an appraisal and modification of an individual’s affective state (Thompson, 1994). When emotions are dysregulated, maladaptive behaviors may arise, such as the production of emotional expressions that are inappropriate in both context and intensity (Thompson,
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1994). Irritability may serve as an example of emotion dysregulation, as persistent irritability produces a heightened reactivity to negative stimuli, often involving an angry mood state that may be accompanied by an aggressive behavioral response (Leibenluft & Stoddard, 2013).

Anger is a negatively valenced emotion that is typically elicited when a goal is blocked (Carver & Harmon-Jones, 2009). However, unlike other negative emotions, anger elicits appetitive behaviors that move one towards a stimulus rather than encourage withdrawal behaviors (Carver & Harmon-Jones, 2009). These appetitive behaviors are consequently directed towards the stimulus that blocked goal attainment (Harmon-Jones, Harmon-Jones, & Price, 2013).

In some cases, aggressive behaviors are also produced when goal blockage elicits anger (Harmon-Jones et al., 2013). Aggression is defined as a behavior intended to harm another, may manifest verbally or physically (Berkowitz, 1993) and may either be instrumental or reactive. Instrumental aggression is coercive and deliberate, often used in order to attain a goal (Price & Dodge, 1989). Such behaviors may manifest through social dominance, such as bullying, or damaging another’s reputation. In contrast, reactive aggression is more spontaneous and occurs in response to blocked goal attainment, manifesting as hostile or angry expressions (Price & Dodge, 1989). Reactive aggression is therefore closely tied to irritability.

Although research has identified irritability’s association with anger and aggression, along with its predictive capabilities for the development of depression and anxiety, the neural mechanisms of irritability are poorly understood. To date, limited work has been done on the neural correlates of irritability specifically; however, neural
models of related constructs such as depression, anxiety, anger and aggression have been proposed, and can be used to predict a neurophysiological profile for highly irritable individuals. Specifically, electroencephalography (EEG) asymmetries, which measure interhemispheric differences in activation levels, have been used to study depression, anxiety, anger and aggression. These models have focused on asymmetries in the frontal and parietal lobes, and will be discussed separately in the subsequent sections. The relationships proposed by these models of affect may help clarify a neurophysiological profile for irritability.

Frontal Asymmetry

Early studies on frontal asymmetries identified an association between frontal activity and the valence of emotions. Research using sodium amytal injections demonstrated how suppression of the left prefrontal cortex, yielding greater relative right activity, produced depression-like symptoms. Conversely, suppression of activity in the right prefrontal cortex, yielding greater relative left activity, produced euphoric behaviors (Terzian, 1964). These findings were corroborated by lesion studies, which also found depressive symptoms in patients with left hemisphere damage, while those with right impairments demonstrated symptoms of mania (Gainotti, 1972). The relationship between relative right prefrontal activation and depression was also supported by research in neurologically intact individuals with depression, as seen in research by Henriques and Davidson (1991), that demonstrated greater relative right frontal activation in depressed patients. Together these findings resulted in a valence model, which posits that greater relative right frontal activity is associated with increased negative affect and
right frontal activity, while positive affect is associated with left frontal activity (Harmon-Jones, 2003).

According to the valence model, anger may yield a relative right frontal asymmetry due to its negative valence. However, a competing model, the motivational model, makes an opposing prediction. Davidson (1983) argues how frontal activity may not only be driven by the valence of emotions, but also by the type of motivational behavior elicited by emotions. The motivational model claims that the right prefrontal cortex is associated with withdrawal-related emotions (i.e., sadness, anxiety), which motivates an individual to avoid harmful stimuli such as threats or punishments. Conversely, the left prefrontal cortex facilitates approach-related emotions (i.e., love, happiness), which drives an individual towards goal or reward-related stimuli. Because anger is associated with approach-related behaviors, greater relative left frontal activity may be associated with anger.

Studies reveal how anger inductions are capable of eliciting greater relative left frontal activity. Harmon-Jones and Sigelman (2001) induced anger by providing insulting feedback on participants’ essays, and recorded prefrontal EEG activity before and after the anger induction. Results demonstrated that participants in the insult condition reported more anger, and exhibited an increase in relative left prefrontal activation. A similar pattern of activity was found when inducing anger in individuals high in trait anger (Harmon-Jones, 2007). In the study, EEG was recorded while participants viewed anger-inducing images of racism and prejudice, followed by completion of the Aggression Questionnaire by Buss and Perry (1992). The anger subscale from the questionnaire was used as a measurement of trait anger, which correlated with greater
relative left frontal activity from the anger-inducing pictures. Findings from these studies therefore demonstrate how greater relative left frontal activity is associated with anger.

As anger may also be accompanied by aggressive behaviors, studies suggest that greater relative left frontal activity following an anger induction is also associated with aggressive behaviors. The study using insulting essay feedback by Harmon-Jones and Sigelman (2001) additionally measured levels of aggression after the anger induction. For those in the anger-inducing condition, a significant relationship was found between relative left prefrontal activity and aggression. These findings are corroborated by research using transcranial direct current stimulation (tDCS) (Hortensius, Schutter, & Harmon-Jones, 2012). Hortensius et al. (2012) first induced anger in participants, then applied tDCS to increase relative left frontal activity. Findings revealed how angered participants with increased left frontal activity exhibited greater behavioral aggression. Therefore, these studies indicate how higher relative left activity associated with anger may also indicate a propensity to express aggression.

A frontal asymmetry profile for irritability has yet to be identified; however, as irritability is defined as a tendency to produce aggressive expressions of anger, irritability may yield a similar frontal activation pattern as its emotional and behavioral components. Since anger and aggression are both associated with greater relative leftward frontal activity, the current study will investigate whether highly irritable individuals possess a leftward frontal asymmetry.
Parietal Asymmetry

Parietal asymmetries have been related to the regulation of physiological arousal. The arousal model proposed by Heller (1993) claims that greater right parietal activity may be associated with higher levels of arousal. This model has been corroborated by research on posttraumatic stress disorder (Metzger et al., 2004), depression (Moratti, Rubio, Campo, Keil, & Ortiz, 2008) and anxiety (Nitschke, Heller, Palmieri, & Miller, 1999).

Physiological arousal is especially pertinent to studies of anxiety, which is characterized by high arousal levels (Clark & Watson, 1991). However, research has differentiated two forms of anxiety, anxious apprehension and anxious arousal, which differ in their degree of arousal. Anxious apprehension is marked by worry, rumination and fear for the future, and may produce symptoms of fatigue and restlessness (Nitschke et al., 1999). Conversely, anxious arousal is characterized by panic, and symptoms reflective of autonomic arousal such as shortness in breath, dizziness or sweating. Metzger et al. (2004) demonstrated how arousal symptoms correlated with right parietal activity in nurse veterans diagnosed with post-traumatic stress disorder (PTSD), a condition that produces a symptomatology similar to that of anxious arousal. As supported by the arousal model, anxious arousal, marked by physiological hyperarousal, is therefore associated with greater relative right parietal activity.

In contrast, depressed patients experience hypoarousal, which is consequently related to lower relative right parietal activity (Heller & Nitschke, 1997). In investigations of low positive emotionality (PE), a risk factor for depression, children with lower PE levels have lower right parietal activity (Shankman et al., 2005). Similarly,
Depressed patients generated lower steady-state visual evoked potentials (ssVEPs) to arousing stimuli in the right temporoparietal cortex (Moratti et al., 2008). Depression therefore appears to be marked by lower relative right parietal activity, reflective of hypoarousal symptoms.

As irritability is capable of predicting anxiety and depression (Leibenluft & Stoddard, 2013; Stringaris et al., 2009), and is also co-morbid with both disorders (Brotman et al., 2006; Copeland et al., 2013; Krieger et al., 2013), anxiety and depression research may help elucidate a parietal asymmetry profile for irritability. However, these disorders are associated with contrasting levels of arousal, in that depression is associated with hypoarousal, while anxiety with hyperarousal. Although this could imply how irritability is associated with both hyper and hypoarousal, we hypothesize that irritability elicits hyperarousal. Hyperarousal may be elicited not only due to irritability’s comorbidity with anxiety, but also due to its relationship with anger, in which anger may elicit a high state of physiological arousal (Blair, 2012). Given the involvement of hyperarousal in irritability and predictions made by Heller’s (1993) arousal model, the current study will assess whether irritability is associated with greater relative right parietal activity.

**Objective of the Current Study**

The current study aims to identify a potential EEG asymmetry profile for irritability. Self-reported measures of irritability and baseline EEG levels were assessed in undergraduates. We hypothesized that individuals with greater self-reported levels of irritability would exhibit:
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a) Greater relative left frontal activity, reflective of the relationship between frontal asymmetry and anger, and,

b) Greater relative right parietal activity, reflective of the relationship between parietal asymmetry and hyperarousal.

Participants additionally performed an attention-based task under conditions of frustration and non-frustration. The task served as the frustration manipulation, which is capable of eliciting irritability experimentally (Leibenluft & Stoddard, 2013). Prior research demonstrated how frustration may impair attention shifting (Deveney et al., 2013), so it is additionally hypothesized that frontal and parietal asymmetry profiles of anger and hyperarousal may predict poorer performance on the attention-based task.

Methods

Procedure

Participants first completed self-reported measures of irritability, followed by baseline resting EEG recordings. Subjects then performed the Affective Posner task (see below) and reported their levels of arousal, frustration and valence using a 9-point Likert scale after each block of the task. Once the task was completed, participants were briefly interviewed and filled in a self-report questionnaire to test whether they were deceived by the rigged feedback or not.

Participants

Undergraduate students studying at a college in the Boston area were recruited for the study. Individuals first completed a prescreening questionnaire to screen for the
following eligibility criteria based on participant report: participants must be right-handed, reported no history of lost consciousness longer than 10 minutes, no brain injury, epilepsy, uncontrolled diseases (diabetes, thyroid), and cancer. Individuals were also ineligible if they participated in binge drinking (4 drinks or more in any given occasion), or used drugs in the past month. A total of forty-two students participated in the study.

On the testing day, participants received a description of the study and provided informed consent. The session lasted for approximately two hours, and all subjects were compensated $40 for their time.

**Self-reported Measures of Irritability**

Questionnaires were administered through an online survey system, Qualtrics. Participants completed self-reported measures of irritability, the Brief Irritability Test (BITe) (Holtzman, O'Connor, Barata, & Stewart, 2015), and the Affective Reactivity Index (ARI) (Stringaris et al., 2012).

**Baseline Electroencephalography**

Baseline electroencephalography (EEG) was collected while participants sat quietly in the testing room. Data was collected over eight 1-minute trials, in eyes open (O) or eyes closed (C) conditions in counterbalanced order (OCCOCOOC or COOCOCOOC).
Affective Posner Task

Subjects performed an adapted version of the Affective Posner task (Deveney et al., 2013). During the task, participants had to respond as quickly and accurately as possible to task stimuli. A single trial consisted of presentation of a fixation cross, followed by two white squares. The blue cue could appear in either of the squares. Valid trials occurred 75% of the time, in which the cue would predict target location (white cue). Invalid trials occurred 25% of the time, in which the cue was in the opposite square of the target. Participants received feedback after each trial by presentation of a coin image, with cumulative winnings at the bottom. Text included positive, negative or error feedback.

The task was performed as three games. In Game 1 (50 trials), participants received accurate feedback but did not win or lose money depending on task performance. Game 2 (100 trials) consisted of two blocks where participants received accurate feedback, and won or lost 50¢ depending on accuracy. Game 3 (100 trials) also consisted of 2 blocks, where participants were told to respond quickly and accurately in order to win money. Frustration was manipulated through rigged feedback (Figure 1). Participants received negative feedback on 60% of their correct responses, resulting in a loss of 50¢ per trial.

Data Analysis

EEG data acquisition. Baseline EEG was collected from 32 electrodes using the ActiChamp amplifier (Brain Products, Germany) and International 10-20 system for placement. Eye movements were recorded for potential artifact detection using
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electroculogram (EOG) channels positioned above and below the right eye, and at the temples. Recordings were referenced online to Cz and impedances were kept below 45 kΩ. Data were digitized at a 250 Hz sampling rate and filtered through a 0.01-100 Hz bandpass filter.

**EEG data reduction.** After acquisition, EEG data were filtered offline through a 30 Hz low-pass filter. Data then underwent an Independent Components Analysis to remove ocular artifacts, and were subsequently manually inspected for artifacts. Channels with large artifacts throughout the eight baseline trials were excluded from further analysis. Channel F4 had an abnormally large number of artifacts across multiple subjects (n=24). Artifact-free data were then segmented into 2.048s epochs and re-referenced to an Average reference. A Fast Fourier Transform (FFT) with 75% Hamming window overlap was applied to each epoch. Alpha power (8-13 Hz) was extracted for each electrode site and log-transformed (uV²). Asymmetry scores for homologous pairs were computed by subtracting Ln(Right)-Ln(Left). Higher alpha scores reflected less brain activity as alpha activity is inversely correlated to brain activity (Davidson, 1998).

Participants were divided into Left Frontal, Right Frontal, Left Parietal and Left Parietal subgroups using the following procedure. Overall frontal and parietal asymmetry scores were obtained by averaging alpha activity across all right or left sites for frontal (Ln(Fp2,F4,F8)-Ln(Fp1,F3,F7)) and parietal (Ln(P4,P8)-Ln(P3,P7)) regions. Scores greater or less than two standard deviations away from the mean were considered outliers (n=2, n=2, respectively) and excluded from further analysis. Frontal and parietal asymmetry scores were sorted in ascending order and divided into quartiles, so that those
in the highest quartile had the greatest asymmetry score and therefore greater relative left activity. Those in the highest quartile were classified as Left Frontal (n=9) or Left Parietal (n=8). Similarly, those in the bottom quartile had the lowest asymmetry score and therefore greater relative right activity. These individuals were classified as Right Frontal (n=9) or Right Parietal (n=8).

**Self-reported frustration ratings.** To assess whether the rigged feedback in Game 3 elicited frustration, self-reported frustration ratings were compared before and after the frustration condition using paired t-tests. Frustration difference scores were additionally computed by subtracting self-reported levels of frustration prior to the frustration condition from levels measured after the frustration condition. A higher difference score reflects a greater increase in frustration after the manipulation.

**Asymmetry scores, self-reported levels of irritability, and difference in frustration ratings.** Spearman correlations were performed between EEG asymmetry scores and self-reported trait irritability scores of ARI and BITe separately for each frontal and parietal homologous pair. Correlations were also computed between EEG asymmetry scores and frustration difference scores for each homologous pair. A positive correlation between EEG scores and self-reported levels of irritability indicate a relationship between greater relative left activity and greater self-reported irritability levels. Similarly, positive correlations between EEG scores and frustration difference scores indicate a relationship between greater relative left activity and greater reactivity to the frustration manipulation.
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**Parietal EEG scores and difference in arousal ratings.** Spearman correlations were performed between parietal EEG asymmetry scores and difference in arousal ratings. Arousal difference scores were computed by subtracting self-reported levels of arousal prior to the frustration condition from levels measured after the frustration condition. A higher difference score was indicative of a greater increase in arousal after the manipulation. A negative correlation between parietal EEG asymmetry scores and arousal difference scores suggests a relationship between greater relative right parietal activity and increase in arousal.

**Asymmetry groups and behavioral data.** If reaction times were too fast (< 150ms) and responses were inaccurate in 40% or more of trials, data were removed from further analysis, as this suggested that participants were responding randomly (n=6). Two separate mixed-design ANOVAs using condition (non-frustration, frustration) and validity (valid, invalid) as within-subjects factors and frontal asymmetry group (Left Frontal, Right Frontal) as between-subjects factor were computed for accuracy and response time. These ANOVAs were similarly performed for parietal asymmetry group to assess effects on accuracy and reaction time. An interaction effect between asymmetry group, condition, and validity on accuracy or reaction time is indicative of a relationship between EEG asymmetry and task performance.
Results

Participants were excluded from all analyses if they were not deceived by the rigged feedback in the frustration manipulation (n=10).

Effectiveness of Frustration Manipulation

Frustration ratings (Figure 2) were significantly greater after the frustration manipulation than after the non-frustration condition ($t(46)=8.78, p<0.001$).

Asymmetry Scores and Self-Reported Irritability

Frontal asymmetry scores. Alpha asymmetry scores correlated with ARI at F4-F3 ($\rho=-0.56, p=0.039$) and F8-F7 ($\rho=-0.37, p=0.029$) but not at Fp2-Fp1 ($\rho=-0.14, p=0.44$), such that higher ARI irritability scores were associated with greater right versus left prefrontal activity (Figure 3). No correlation was found between BITe and any of the frontal homologous pairs ($\rho=-0.06, p=0.31; \rho=-0.12, p=0.51; \rho=-0.19, p=0.31$) (Figure 4). Asymmetry scores did not correlate with difference in frustration ratings ($\rho=-0.34, p=0.25; \rho=0.20, p=0.27; \rho=0.12, p=0.51$) (Figure 5).

Parietal asymmetry scores. Alpha asymmetry scores at P4-P3 or P8-P7 did not correlate with ARI ($\rho=0.063, p=0.72; \rho=0.19, p=0.27$, respectively) (Figure 6), BITe ($\rho=-0.15, p=0.93; \rho=0.085, p=0.64$) (Figure 7) or difference in frustration ratings ($\rho=0.010, p=0.95; \rho=-0.30, p=0.079$) (Figure 8).
Parietal Asymmetry and Self-Reported Arousal

Alpha asymmetry scores did not correlate with difference in arousal ratings prior and after the frustration manipulation at P8-P7 ($\rho=-0.18, p=0.30$) or P4-P3 ($\rho=-0.95, p=0.58$) (Figure 9).

Asymmetry and Task Performance

Accuracy. Mixed ANOVAs of condition, validity and frontal asymmetry group (Figure 10A), $F(1,13)=20.76$, $p<0.01$, $\eta_p^2=0.63$, and condition, validity and parietal asymmetry group (Figure 10B), $F(1,11)=27.89$, $p<0.01$, $\eta_p^2=0.72$, on accuracy both revealed an interaction between condition and validity. Accuracy was poorer in invalid trials in the frustration condition than in invalid trials of the non-frustration condition ($p<0.01$). However, neither frontal asymmetry group nor parietal asymmetry group interacted with validity or condition on accuracy, $F(1,13)=0.13$, $p>0.05$, $\eta_p^2=0.01$, $F(1,11)=1.77$, $p>0.05$, $\eta_p^2=0.14$, respectively.

Reaction time. Mixed ANOVAs of condition, validity and frontal asymmetry group (Figure 11A), $F(1,13)=23.23$, $p<0.01$, $\eta_p^2=0.64$, and condition, validity and parietal asymmetry group (Figure 11B), $F(1,11)=10.14$, $p<0.01$, $\eta_p^2=0.48$, on reaction time both revealed a main effect for condition. Response times were faster in both invalid and valid trials in the frustration condition ($p<0.05$) versus the non-frustration condition. A main effect for validity was found for both the frontal asymmetry ANOVA, $F(1,13)=33.65$, $p<0.01$, $\eta_p^2=0.721$, and parietal asymmetry ANOVA, $F(1,11)=24.31$, $p<0.01$, $\eta_p^2=0.69$, where reaction times were faster in valid trials in both frustration and non-frustration
conditions \( p<0.01 \). Neither frontal asymmetry group nor parietal asymmetry group interacted with validity or condition on reaction time, \( F(1,13)=1.57, p>0.05, \eta^2_p=0.11, F(1,11)=0.16, p>0.05, \eta^2_p=0.02 \), respectively.

**Discussion**

Behavioral results from our study suggest that frustration was successfully induced, as participants reported a significantly higher level of frustration after the frustration condition. As predicted by previous research (Deveney et al., 2013), results also demonstrate that the frustration manipulation was capable of impairing attention, as accuracies were poorer in the invalid frustration trials than in invalid non-frustration trials. Overall, the current behavioral findings indicate that the task induced frustration, which consequently may have compromised performance on the attention task.

Given the association between irritability and anger, we hypothesized that greater relative left frontal asymmetry scores would correlate with greater self-reported levels of irritability. Contrary to our hypotheses, greater relative right frontal EEG asymmetry scores at F4-F3 and F8-F7 were associated with higher levels of irritability. This result is consistent with the valence model, in that emotions of negative valence, such as anger, are associated with relative right activity (Harmon-Jones, 2003). However, the findings contradict the motivational model (Davidson, 1983) and several prior studies of anger (Harmon-Jones & Sigelman, 2001; Harmon-Jones, 2007), where the approach-related emotion anger is associated with relative left prefrontal activity. The reason for the discrepancy may be that different types of anger elicit differing motivational behaviors. Hewig, Hagemann, Seifert, Naumann, and Bartussek (2004) suggest how anger-out,
marked by explicit aggressive behaviors, is associated with approach motivation, and accordingly demonstrated that greater relative left frontal activity was associated with anger-out. Conversely, anger-in involves inhibiting anger expressions, and is therefore associated with withdrawal motivation. It is possible that irritability elicits anger-in behaviors, given that irritability is described as “a preparation to anger” or a “less than violent” form of anger (Barata, Holtzman, Cunningham, O'Connor, & Stewart, 2015) which led to the greater relative right activation among individuals with higher irritability scores. However, this conclusion is speculative because the current study did not measure anger-in or anger-out behaviors.

We additionally hypothesized that irritability may be associated with hyperarousal, and consequently greater relative right parietal activity. No correlation was found between parietal asymmetry scores and self-reported irritability, frustration difference scores, or arousal difference scores. This may have to do with methodological issues, in that baseline EEG was only measured prior to the frustration condition. Baseline EEG was considered as a potential trait measurement for irritability; however, as irritability is described as a tendency to anger (Leibenluft and Stoddard, 2013), a provocation may be necessary to elicit an outburst. Consequently, any measurements made at rest may not adequately reflect an anger-related profile, such as a presentation of greater relative right parietal activity indicative of hyperarousal.

Failure to collect EEG measurements after the provocation may have also been a potential reason to a lack of association between higher self-reported measures of irritability and greater relative left frontal activity – rather, greater relative right frontal activity was related with higher levels of irritability in the current study. Previous
research by Harmon-Jones and Sigelman (2001) and Harmon-Jones (2007), which both suggested a relationship between anger and greater relative left frontal activity, collected EEG recordings prior and after the anger manipulation (Harmon-Jones & Sigelman, 2001; Harmon-Jones, 2007). Because the current study only collected EEG recordings prior to the provocation, a change in EEG asymmetries due to the provocation was not assessed. Failure to compare asymmetries before and after the frustration manipulation may account for our findings that are contradictory to that of previous research.

Neither frontal nor parietal asymmetry was associated with behavioral performance on the frustration task. This again suggests how baseline EEG measured prior to the provocation may not adequately predict any irritability-mediated effects on attention. Behavioral analyses also suffered from small sample sizes, as participant data were excluded if suggestive that they were performing randomly during the task. However, criteria for random performance included inaccurate responses on 40% or more of trials. It is possible that highly irritable individuals perform poorly in attention-based tasks, as demonstrated in previous research where frustration may impair attention shifting (Deveney et al., 2013). Therefore, highly irritable individuals were mistakenly excluded on the basis of performing randomly on the task, when their poor behavioral performance was effectively a result of a greater reactivity to the frustration manipulation. If this was the case, the lack of association between either frontal or parietal asymmetry on behavioral performance may be attributed to the exclusion of highly irritable individuals during data analyses.

In conclusion, future studies should look to assess the relationship between irritability and motivational tendencies similar to the subtypes of anger. In addition, EEG
measurements should be made prior and after a frustration manipulation to test whether irritability-related asymmetries exist in response to emotional challenges.

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Figure 1: Schematic of Affective Posner Task During the Frustration Condition (Game 3). The blue cue and white target could appear in either box. Valid trials involved both cue and target appearing in the same box, and occurred 75% of the time. Invalid trials involved the cue and target appearing in different boxes, and occurred 25% of the time. Participants had to press a button that corresponded to target location. In the frustration condition, participants could win or lose 50¢ depending on performance. Feedback was rigged so that participants received negative feedback and lost money on 60% of correct responses.
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*Figure 2: Frustration ratings after non-frustration and frustration conditions.* Greater scores indicate greater levels of frustration.
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![Chart showing average frustration rating for Non-Frustration and Frustration conditions. The Frustration condition has a higher average rating with more variability.]
Figure 3: Alpha asymmetry scores (8-13 Hz at Fp2-Fp1, F4-F3, F8-F7) against Affective Reactivity Index (ARI) scores. Higher ARI scores indicate greater levels of frustration. Greater alpha power asymmetry scores imply greater left activation.
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- $\rho = -0.14$, $p = 0.44$
- $\rho = -0.56$, $p = 0.039$
- $\rho = -0.37$, $p = 0.029$

Total Alpha Power ($\mu V^2$)
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*Figure 4: Alpha asymmetry scores (8-13 Hz at Fp2-Fp1, F4-F3, F8-F7) against Brief Irritability Test (BITe) scores.* Higher BITe scores indicate greater levels of frustration.

Greater alpha power asymmetry scores imply greater left activation.
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Total BITe Score

\[ \rho = -0.06 \]
\[ p = 0.31 \]

Total Alpha Power (uV^2)

\[ \rho = -0.12 \]
\[ p = 0.51 \]
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*Figure 5: Alpha asymmetry scores (8-13 Hz at Fp2-Fp1, F4-F3, F8-F7) against frustration difference scores.* Mood ratings were collected after the non-frustration and frustration conditions. Higher difference scores indicate greater reactivity to frustration manipulation. Greater alpha power asymmetry scores imply greater left activation.
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Total Alpha Power (uV²)
Figure 6: Alpha asymmetry scores (8-13 Hz at P4-P3, P8-P7) against Affective Reactivity Index (ARI) scores. Higher ARI scores indicate greater levels of frustration. Greater alpha power asymmetry scores imply greater left activation.
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Total ARI Score

$\ln(P4) - \ln(P3)$

$p = 0.063$

$p = 0.72$

Total Alpha Power ($\text{uV}^2$)

$\ln(P8) - \ln(P7)$

$p = 0.19$

$p = 0.27$
Figure 7: Alpha asymmetry scores (8-13 Hz at P4-P3, P8-P7) against Brief Irritability Test (BITe) scores. Higher BITe scores indicate greater levels of frustration. Greater alpha power asymmetry scores imply greater left activation.
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Total BIaTe Score

\[ \text{Ln}(P4) - \text{Ln}(P3) \]

\[ \rho = -0.15 \]
\[ p = 0.93 \]

\[ \text{Ln}(P8) - \text{Ln}(P7) \]

\[ \rho = 0.085 \]
\[ p = 0.64 \]

Total Alpha Power (uV^2)
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Figure 8: Alpha asymmetry scores (8-13 Hz at P4-P3, P8-P7) against frustration difference scores. Mood ratings were collected after the non-frustration and frustration conditions. Higher difference scores indicate greater reactivity to frustration manipulation. Greater alpha power asymmetry scores imply greater left activation.
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Total Alpha Power (uV²)
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*Figure 9: Alpha asymmetry scores (8-13 Hz at P4-P3, P8-P7) against arousal*

difference scores. Mood ratings were collected after the non-frustration and frustration conditions. Higher difference scores indicate a greater increase in arousal after the frustration manipulation. Greater alpha power asymmetry scores imply greater left activation.
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Total Alpha Power (uV^2)
Figure 10: Participants’ accuracy separated by condition (Non-Frustration and Frustration) and validity (Invalid and Valid). Average accuracies were also grouped according to participants’ average asymmetry scores for a) frontal and b) parietal sites.
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A) Left Frontal Activity

B) Left Parietal Activity
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Figure 11: Participants’ reaction time (RT) performance separated by condition (Non-Frustration and Frustration) and validity (Invalid and Valid). RTs were also grouped according to participants’ average asymmetry scores for a) frontal and b) parietal sites.
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A) Mean Reaction Time (msec)

B) Mean Reaction Time (msec)