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Shifts in frontal asymmetry underlying impulsive and controlled decision-making

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ABSTRACT

The frontal cortices are asymmetrically activated in impulsive and inhibitory action. However, no past work has examined shifts in frontal asymmetric activation during active impulse control or risk-taking behavior. The current study examined impulsive and controlled behavior in a behavioral risk-taking task (Balloon Analogue Risk Task) under alcohol or neutral cue exposure while EEG was recorded. Results revealed activity shifted towards greater relative left frontal activation on alcohol trials with impulsive behavior (balloon explosion) driven by reduced activation of the right inferior frontal gyrus. In contrast, activity from the first half to the second half of alcohol trials with successful impulse control (cash out) localized to reduced activation of the left inferior frontal gyrus. These findings suggest that shifting of right or left frontal asymmetry in inhibitory or impulsive behaviors stem from activation of the inferior frontal gyrus and reveal the importance of examining shifts in neural activity during behavioral processes.

1. Introduction

Over seventy five years research has revealed asymmetric activation of the frontal cortex relates to motivation and behavioral regulation (Davidson, 1992; Gainotti, 1972; Goldstein, 1939; Rossi & Rosadini, 1967). Patients with lesion to the left or right frontal cortices showed specific deficits in positive affect and inhibition, respectively (Gainotti, 1972; Goldstein, 1939). Later, examination of asymmetric frontal activity using electroencephalography (EEG) extended these earlier findings. Greater relative left frontal asymmetry as measured by EEG has been linked to approach motivation and appetitive responses (Davidson, 1992; Fox, 1991; Harmon-Jones & Allen, 1997; Harmon-Jones & Gable, 2018). While traditional theories of frontal asymmetry suggest that negative affect or withdrawal motivation is related to right frontal asymmetry, recent evidence and reviews have suggested that right frontal asymmetry may be more closely tied to motivational control and inhibition (Aron, Robbins, & Poldrack, 2014; Aron, Robbins, & Poldrack, 2004; Asahi, Okamoto, Okada, Yamawaki, & Yokota, 2004; Gable, Neal, & Threadgill, 2018; Garavan, Ross, & Stein, 1999). Evidence from multiple methodologies including EEG, fMRI, lesion, and brain stimulation has suggested that the right frontal cortex is involved with motivational and cognitive control. Many studies have found that higher trait levels of left frontal activity as measured by EEG relate to greater impulsive personality traits and behavior (Gable, Mechin, Hicks, & Adams, 2015; Gianotti et al., 2009; Knoch, Gianotti, Baumgartner, & Fehr, 2010; Neal & Gable, 2016, 2017; Santesso et al., 2008). However, no work has shown that EEG activity may actively shift from greater left frontal activity to greater right frontal activity during successful inhibition of impulsive behavior.

Converging evidence from fMRI, EEG source localization, and lesion studies suggest that the right and left inferior frontal gyrus (IFG) appears to be asymmetrically involved in inhibitory processes. The right IFG acts as a “brake”, inhibiting behavior or suppressing a response (Aron et al., 2004, 2014). For example, the rIFG appears to be essential for inhibition of predominant behaviors in a stop signal or go/no-go task (1999, Kawashima et al., 1996; Konishi, Nakajima, Uchida, Sekihara, & Miyashita, 1998; Whelan et al., 2012). In contrast, the left IFG may act as a“go” signal leading to less impulse control. For example, greater impulsivity is associated with greater gray matter volume of the lIFG in cocaine-addicted individuals (Moreno-López et al., 2012) and much EEG research links greater left frontal activation with approach motivation and impulsivity (Harmon-Jones & Gable, 2018; Neal & Gable, 2016; Santesso et al., 2008). However, the left and right IFG appear to interact to regulate impulse control. Greater trait impulsivity, or the tendency to rash action, has been linked with reduced activity in the rIFG (Gable, Mechin, Hicks et al., 2015), suggesting that reduced left or right IFG activity may drive asymmetric activation of the pre-frontal cortex (PFC) to enhance or reduce impulse control,

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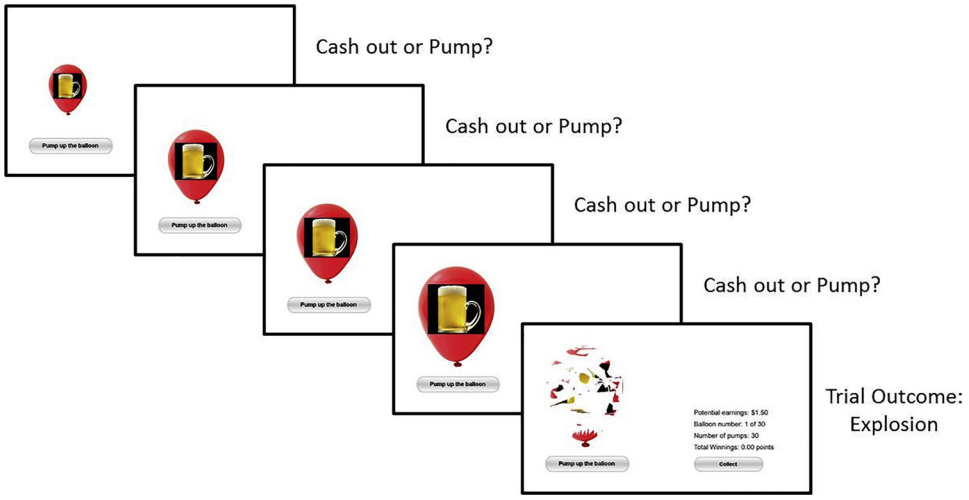

Fig. 1. Example Explosion Alcohol Trial from Modified BART.

respectively.

The current study sought to examine shifts in frontal asymmetric activation during active impulse control and whether shifts in frontal asymmetry related to activation of the IFG. No past work examining behavioral inhibition and impulsivity has examined shifts in frontal asymmetric activation during active impulse control or risk-taking behavior. The current study used a modified Balloon Analogue Risk Task (BART; Lejuez et al., 2002) while viewing alcohol and neutral cues to assess impulsive and controlled behavior during alcohol cue exposure. Consistent with much past work showing alcohol cues evoke strong approach motivation, trials with alcohol cues were hypothesized to enhance left frontal asymmetry relative to neutral cues (Gable, Mechin, Neal et al., 2015; Gordon, 2016; Mechin, Gable, & Hicks, 2016). Greater approach motivation brought on by alcohol cues should enhance inhibitory control required to override impulsive behavior. In the presence of alcohol cues, trials ending in successful inhibition of impulsive behavior should be accompanied by shifts to greater right (reduced left) frontal activity. In contrast, trials ending in impulsive behavior should be accompanied by shifts towards greater left (reduced right) frontal activity.

2. Materials and methods

2.1. Participants

Participants (N = 44; 31 female) consisted of Introduction to Psychology students participating for course credit. The average age of participants was 19.28 (SD = 1.35). Participants were excluded from participation if they were left handed or had any permanent obstruction on their scalp or hair that would prevent proper EEG recording. Based on past studies examining neural responses to appetitive stimuli (Neal & Gable, 2016; Ryerson, Neal, & Gable, 2017), we expected to achieve a medium effect size. Given this effect size and our within-subjects design, a power analysis suggested that 34 participants would be needed to achieve 0.80 power. The data collection stopping rule was to stop at the end of the semester, provided we had at least 40 participants.

2.2. Procedure

Participants entered the lab and provided informed consent. The experimenter applied a stretch-lycra EEG cap. Then, participants completed a modified Balloon Analogue Risk Task (BART). Following the BART, participants completed another unrelated task lasting 5 min before completing the study.

2.3. Measures

2.3.1. Balloon analogue risk task (BART)

Participants completed a modified BART (Lejuez et al., 2002) designed to manipulate alcohol cue exposure during risk taking. Similar to a traditional BART, participant chose to earn money by inflating a balloon on a computer screen. Each time the balloon was inflated by pressing a button on the keyboard, participants earned money. However, on each pump of the balloon, an integer from 1 to 128 would be selected and then not replaced in the list. A selection of the number 1 signaled that the balloon would explode. Thus, on the first pump the probability that the balloon would pop was 1/128 and on each subsequent pump the probability the balloon would explode would increase (1/127 on the second pump, 1/126 on the third pump, etc.). According to this algorithm, the optimal point to cash out would be at 64 balloons. After each pump, participants could choose to cash out by pressing a different key, where they would collect the money they earned and begin a new trial. Each trial resulted in either an explosion (e.g. the balloon pops on a selected pump) or a cash out where the participant opts to collect the earnings.

In order to enhance approach motivation on the BART, each balloon had a picture of an alcoholic beverage (alcohol) or a rock (neutral) superimposed onto it (see Fig. 1). There were 20 alcohol pictures and 20 matched neutral (rock) pictures. These images were taken from the internet and have been used extensively in previous research (Hicks, Fields, Davis, & Gable, 2015; Gable, Mechin, Hicks et al., 2015; Hicks, Friedman, Gable, & Davis, 2012; Ryerson et al., 2017). As the balloon increased in size after each pump, the alcohol or neutral image also increased in size. Thus, the influence of the alcohol cue was designed to increase with each pump, because the picture increased in size. Alcohol images included beer, wine, and liquor (mixed drinks) and are clearly identifiable as alcoholic. Alcohol and neutral trials were randomized. Additionally, because subjects participated for course credit rather than monetary incentive they were told they could accumulate points to be cashed out for prizes (candy). For each picture type, an adjusted total pump count and adjusted average pump count were calculated after excluding trials that ended in an explosion. Additionally, number of exploded balloons per trial type was calculated.

2.3.2. Electroencephalography (EEG) assessment

EEG activity was recorded using 64 tin electrodes in a stretch-lycra cap (Electro-Caps, Eaton, OH). A ground electrode was placed midway between FZ and FPZ. Data was referenced on-line to the left earlobe. All electrode impedances were kept under 5 kΩ and homologous sites were within 1 kΩ of one another. Data was amplified with NeuroScan

SynAmps RT amplifier units with AC gains of 2010 (El Paso, TX). Data were filtered online; data were low pass filtered at 100 Hz, high-pass filtered at 0.05 Hz, notch filtered at 60 Hz, and digitized at 500 Hz. Data were hand-inspected for artifacts. Channels were rejected during hand scoring if they had high impedances (> 5 kOhms) or were consistently crossing over other sensors. No participants had bad channels at F8 or F7, the sites examined in the current study. Data were then transformed with a regression-based eye blink correction in Neuroscan 4.3 Edit (Semlitsch, Anderer, Schuster, & Presslich, 1986). Blinks were detected using the FP1 electrode. Data were then visually inspected a second time to ensure proper correction.

Alpha activity was examined during alcohol and neutral trials from the time of balloon onset until the balloon pop or cash out. As the trial duration extended, the participant faced a higher risk of exploding the balloon on each subsequent pump. Therefore, for each participant, an average trial length was calculated. Based on the average trial length for each participant, alpha activity was aggregated for the first half (before the average midpoint) and second half (after the average midpoint) for trials resulting in an explosion or cash out. Due to individual differences in cash out behavior and the random probability of balloons exploding early, some participants did not have any trials ending in an explosion after their average trial length (7 participants for alcohol trials; 5 participants for neutral trials). Therefore, degrees of freedom for tests comparing first and last half activity differ accordingly.

All epochs 1.024 s in duration were extracted through a Hamming window, with consecutive epochs overlapping by 50%. Data were re-referenced to a common average reference. Power spectra were calculated using a fast Fourier transformation; power values from the traditional alpha band (8–13 Hz) were averaged across all epochs. For each picture type (alcohol vs. neutral) and trial type (explosion vs. cash out), an asymmetry difference score was created by subtracting log left from log right for homologous sites. Alpha activity was examined at lateral frontal sites F8 and F7 (Coan & Allen, 2003; Jacobs & Snyder, 1996). Because alpha activity is inversely related to cortical activation (Laufs et al., 2003), lower scores reflect greater relative right frontal activity and higher scores reflect greater relative left frontal activity. The mean number of epochs included for each trial type is presented in Table 1. The minimum number of epochs available for the last half of trials were 10.

2.4. Source localization

Source localization analyses were conducted using standardized low-resolution brain electromagnetic tomography (sLORETA; Fuchs, Kastner, Wagner, Hawes, & Ebersole, 2002; Jurcak, Tsuzuki, & Dan, 2007; Pascual-Marqui, 2002) software to investigate the intracerebral electrical source of alpha band frequency activity. Only participants for whom EEG data for all trial types (e.g. second half of alcohol explosion trials) was available were included in source localization analyses. Data from 37 participants were included in source localization analyses (7 participants were missing data).

Table 1
Mean Number of Epochs for each Trial Type.

	N	Mean	SD
Alcohol Cash Out First Half	43	214.35	78.73
Alcohol Explosion First Half	42	81.76	53.49
Alcohol Cash Out Last Half	43	65.16	45.81
Alcohol Explosion Last Half	36	25.06	24.20
Neutral Cash Out First Half	43	207.33	79.17
Neutral Explosion First Half	43	84.28	59.41
Neutral Cash Out Last Half	43	54.72	42.46
Neutral Explosion Last Half	38	32.13	28.11

2.5. Statistical analyses

First, behavioral outcomes of the BART task (e.g. adjusted pump count, number of explosions) were compared between alcohol and neutral trials. Then, EEG data was analyzed using a variety of repeated measures ANOVAs. In order to test whether trial type and trial outcome influenced frontal asymmetry, data from the first half of trials was compared using a 2 (trial outcome: cash out vs. pop) X 2 trial type (alcohol vs. neutral) repeated-measures ANOVA. Then, shifts in asymmetry from the first to the last half of the trial for each trial type were compared separately for alcohol and neutral trials. Following EEG analyses, source localization analyses were conducted using the *t*-test function to localize the source of the frontal asymmetry changes.

3. Results

3.1. Behavioral results

Adjusted total pump count did not reliably differ for alcohol (M = 35.23; SD = 15.43) and neutral (M = 33.43; SD = 15.01) balloons, $t(43) = 0.86$, $p = .39$, $d = 0.13$. Additionally, the number of exploded balloons did not differ between alcohol (M = 6.27; SD = 2.62) and neutral (M = 6.37; SD = 2.79) balloons, $t(43) = 0.24$, $p = .81$, $d = .04$.

3.2. Electroencephalography (EEG) results

Alpha asymmetry scores during the first half of alcohol and neutral trials were compared for trials ending in cash out versus an explosion. There was a main effect of greater left frontal activation for alcohol versus neutral trials, $F(1, 41) = 4.15$, $p = .04$, $\eta_p^2 = .09$. Thus, the alcohol trials successfully enhanced approach motivation.

For alcohol trials, there was a significant 2 (first half vs. second half) X 2 (trial ending: cash out vs. pop) interaction, $F(1, 35) = 4.24$, $p = .04$, $\eta_p^2 = .11$ (see Fig. 2). Post hoc tests revealed that participants demonstrated greater relative left frontal activation in the last half of trials ending in an explosion (M = -0.05; SD = .38) than the first half of explosion trials (M = -.14; SD = .28), the first half of cash out trials (M = -.17, SD = .30), and the last half of cash out trials (M = -.22, SD = .39). For neutral trials, the 2 (first half vs. second half) X 2 (trial ending: cash out vs. pop) interaction was not significant, $F(1, 37) = 0.18$, $p = .67$, $\eta_p^2 = .005$ (see Fig. 3).

3.3. Source localization

Alpha band power was analyzed by the sLORETA software package to estimate current source density. Alpha power was compared to examine differences between first half and last half of trials separately for

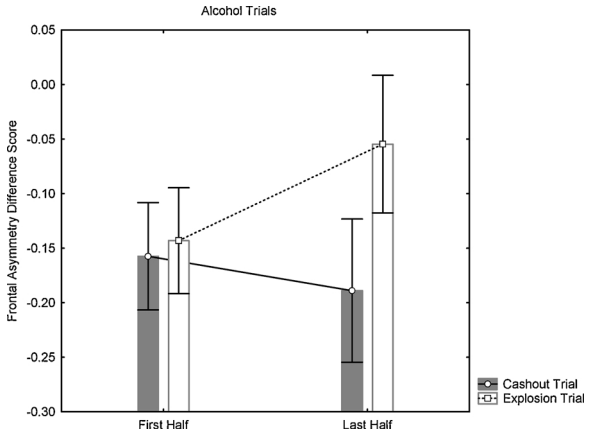

Fig. 2. Interaction of Trial Time and Trial Outcome for Alcohol Trials.

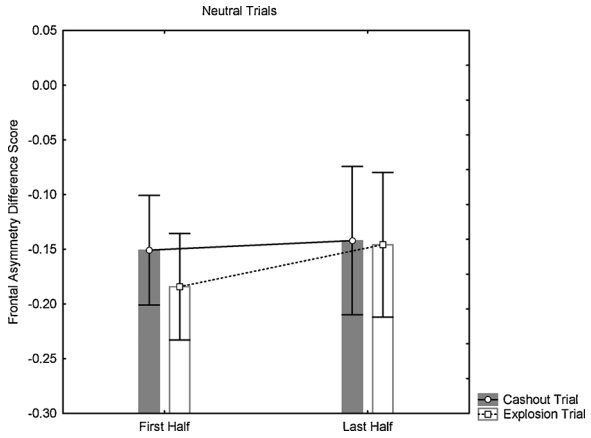

Fig. 3. Interaction of Trial Time and Trial Outcome for Neutral Trials.

the alcohol cash out and alcohol explosion trials. Significant differences between conditions were located with Montreal Neurological Institute (MNI) coordinates. For the alcohol cash out trials, differences were localized to less activation in the last half vs. first half of trials in the left inferior frontal gyrus (lIFG; x = −45, y = 40, z = 15; see Fig. 4a). For alcohol explosion trials, differences were localized to less activation in the last half vs. first half of trials in the right inferior frontal gyrus (rIFG; x = 45, y = 15, z = 25; see Fig. 4b).

4. Discussion

A shift in frontal asymmetry appears to occur when individuals engage in impulse control in the presence of alcohol cues. Participants demonstrated a non-significant shift towards greater relative right frontal activity when impulse control was successfully engaged. However, when impulsive behavior was exhibited (e.g. an exploded balloon), participants demonstrated a shift towards greater relative left frontal asymmetry. These effects were localized to reduced activity in the lIFG and rIFG, respectively. While previous studies have found right frontal activity to relate to personality traits and task performance assessing greater control, this study is the first to demonstrate that shifts in EEG alpha asymmetry throughout a single trial affect trial outcome (successful inhibition vs. impulsive behavior). Notably, these shifts in frontal asymmetry only occurred on trials containing alcohol cues. Neutral cues evoked less approach-motivation than alcohol cues. Therefore, participants likely did not need to engage significant impulse control or impulsive action to result in a cash out or explosion.

Source localization analyses of the current study revealed that shifts in relative frontal asymmetry were driven by activation in the inferior frontal gyrus, a brain region frequently implicated in cognitive control tasks in fMRI studies. These source localization analyses give key insights into why these shifts in relative frontal asymmetry occurred. On cash out trials, there was a reduction in activation of the lIFG. On trials that ended in an explosion, reduced activation of the rIFG created a relative left frontal asymmetry. These asymmetries are not frequently studied in fMRI paradigms, but have been linked in past EEG research to a variety of impulsive behaviors and personality traits (Knoch et al., 2010; Gable, Mechin, Hicks et al., 2015; Gable, Mechin, Neal et al., 2015; Gianotti et al., 2009; Neal & Gable, 2016, 2017). EEG asymmetry studies suggest that the cumulative relative activation of the left and right cortex underscore certain psychological states, rather than the activation of a certain hemisphere alone. The current study extends past asymmetry findings by implicating IFG as a potential source for EEG asymmetry during impulsive behavior. These results also suggest that reduced left hemisphere activation may be driving shifts in greater right frontal asymmetry and greater impulse control. In contrast, reduced right hemisphere activation may be driving shifts in greater left frontal asymmetry and greater impulsive behavior. While most fMRI studies

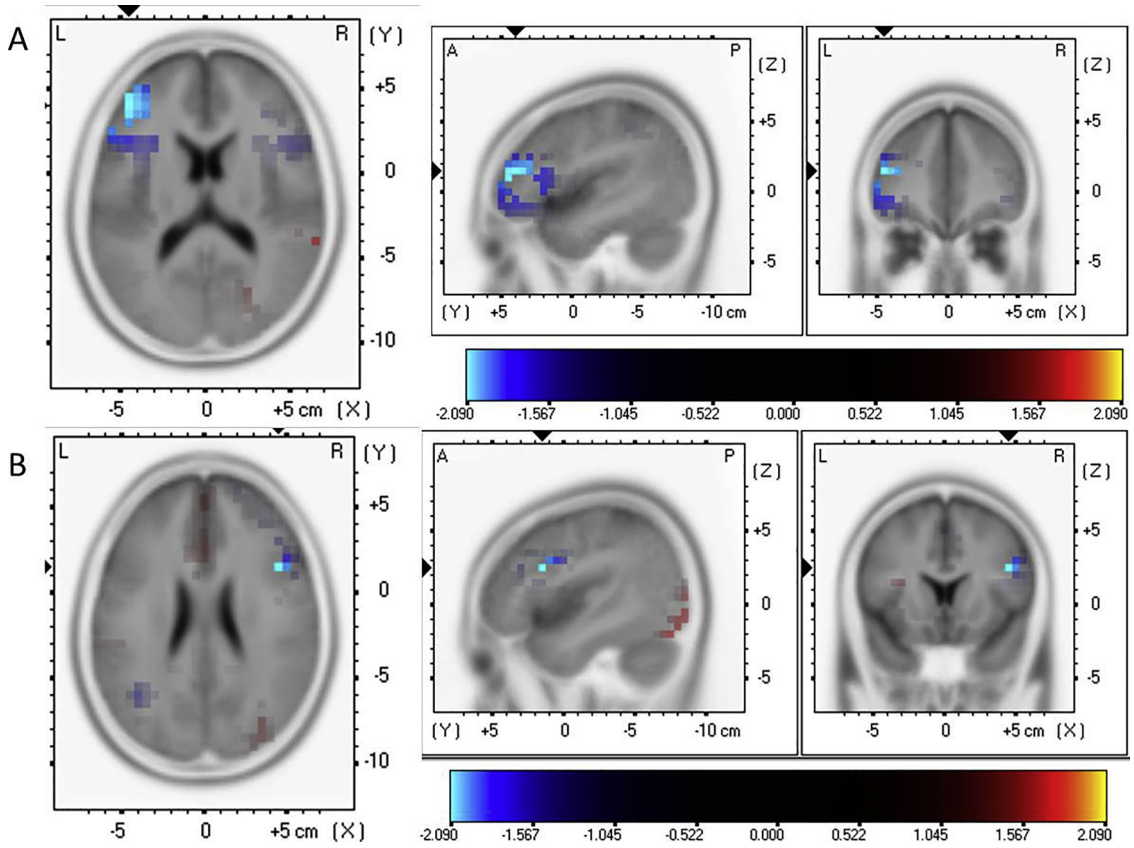

Fig. 4. Source Localization Analyses for Frontal Asymmetry to Cash Out Trials (a) and Explosion Trials (b). Arrows indicate coordinates of asymmetry localization.

focus on the increased activation of the rIFG as a source of inhibition and impulse control, our study highlights the importance of also considering reduced lIFG activation.

Past neuroimaging studies using the Balloon Analogue Risk Task (BART) have also demonstrated right frontal activation during cash outs. Rao, Korczykowski, Pluta, Hoang, and Detre, (2008) had participants complete two BART tasks in an fMRI paradigm: one where the participant was actively making decisions similar to a normal BART paradigm and another passive task where the computer pumped the balloon for participants. Decision making in the active condition activated the right DLPFC to a greater extent than the passive condition, suggesting that decisions to cash out trigger greater right frontal activity. Other fMRI research with the BART has found that the BOLD signal during the risky decision to inflate the balloon related to increased activation in the left ventral medial prefrontal cortex (vmPFC) (Fukunaga, Brown, & Bogg, 2012). This is consistent with the current findings that frontal asymmetry shifts to greater relative left frontal asymmetry on trials resulting in more impulsive behavior.

This study is the first to observe shifts in frontal asymmetric EEG activity during behavioral inhibition in a risk-taking paradigm. This work is in line with other studies using inhibition tasks (e.g. go/no-go) and impulsive behavior tasks. For example, much fMRI work has highlighted the role of the right inferior frontal gyrus (rIFG) in active inhibition (Aron et al., 2014; Garavan et al., 1999; Simmonds, Pekar, & Mostofsky, 2008; Whelan et al., 2012). Our source localization findings that shifts to left frontal asymmetry during failed impulse control localize to the rIFG are consistent with these past findings. Additionally, this work is supported by studies in which frontal asymmetry has been manipulated using stimulation techniques. Left hemisphere stimulation of the prefrontal cortex using transcranial direct current stimulation (tDCS) leads to more risky behavior (e.g. higher pump count) in the BART compared to right hemisphere or sham stimulation (Sela, Kilim, & Lavidor, 2012). Enhancing neuron excitability in the right frontal cortex using tDCS appears to lead to better motor inhibition and control of responses to motivationally-incongruent stimuli (Fecteau et al., 2007; Kelley & Schmeichel, 2016; Stramaccia et al., 2015). In contrast, temporarily lesioning rIFG (and therefore increasing left frontal asymmetry) leads to riskier decision making (Knoch et al., 2006). In our study, greater left frontal activity also underscored riskier trial outcomes.

Frontal asymmetry research has stemmed from decades of EEG and lesion studies. Together, this past work suggests that disruptions of the left and right prefrontal cortex differentially affect emotional, motivational, and executive control functions (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Gainotti, 1972; Goldstein, 1939; Robinson & Price, 1982; Tranel, Bechara, & Denburg, 2002;). One theory of frontal asymmetry is that the frontal cortices may interact through asymmetric inhibition (Grimshaw & Carmel, 2014). Increased activation in the left hemisphere inhibits activation of the right hemisphere, and vice versa. Based on the findings in the current study, asymmetric inhibition could be causing the enhanced right frontal activity associated with impulsive control. That is, left frontal activation could be inhibiting structures in the right hemisphere such as the right inferior frontal gyrus (Gable et al., 2018). Inhibition of the right hemisphere could result in greater impulsive behavior. Consistent with the asymmetric inhibition model, Cunillera, Fuentemilla, Brignani, Cucurell, and Miniussi, (2014) found that tDCS activation increasing right and decreasing left frontal activity resulted in greater response inhibition. These results suggest that deactivation of the left hemisphere may enhance impulse control. Additionally, increased right frontal and decreased left frontal activation using tDCS appears to decrease risk taking on a gambling task (Fecteau et al., 2007), suggesting asymmetric inhibition could be bi-directional. Activation of structures in one hemisphere may inhibit activation in the contralateral hemisphere.

Notably, the current study examined asymmetric activation of the PFC by examining relative differences in alpha activity. While fMRI and lesion studies sometimes report lateralized areas of activation in the prefrontal cortex, lesion research does not and fMRI research rarely explicitly examines asymmetric activation of the frontal cortex (Berkman & Lieberman, 2009). Typically these studies report absolute activity of each brain area rather than using a difference score. The temporal resolution in the BOLD signal does not allow clear differentiation of activation associated with decision-making to cash out and subsequent reward-related activation associated with receiving the cash out reward feedback (Kohno et al., 2015). In contrast, using EEG allows for precise temporal resolution, allowing results of the current study to isolate activity prior to the cash out for analysis.

The current study examines frontal alpha asymmetry shifts and their intracerebral sources, but does not speak to mechanisms facilitating these shifts such as neurotransmitters. What could be potential cellular substrates of frontal asymmetry? Gordon (2016) suggests that left-lateralized activation associated with craving may be associated with dopamine activity. Higher dopamine D2 receptor binding in the left hemisphere is associated with rewarding choices and trait behavioral approach sensitivity while higher D2 receptor in the right hemisphere was associated with avoiding punishment and trait behavioral inhibition sensitivity (Tomer et al., 2014). Asymmetric dopaminergic activation may be driving lateralized activation of the cortex associated with approach and inhibition. This activation is also likely influenced by reward-related dopamine release in the striatum and communication to the PFC through corticostriatal pathways. Kohno and colleagues (2015) have found that greater striatal dopamine D2/D3 dopamine receptor binding potential is associated with ventral striatal activation at cash out (e.g. reward) during the BART. Additionally, D2/D3 binding potential related negatively to activation of the DLPFC, suggesting a mechanism through which reward-related striatal dopamine release may affect subsequent inhibition facilitated by the PFC. Future research employing mixed methods may shed light on the role of dopamine in frontal asymmetry during impulsivity and approach behaviors.

The presence of alcohol cues did not lead to greater impulsivity as assessed by pump count or number of explosions. In the current sample, there was not a significant difference between average adjusted pump count or number of exploded balloons for the alcohol and neutral balloons. However, EEG results revealed that alcohol cues evoked greater relative left frontal activity than neutral cues. In addition to this main effect of left frontal asymmetry for alcohol trials, on trials where participants successfully inhibited impulsive behavior and cashed out, this activity shifted towards greater relative right frontal activity. Such shifts were not observed for neutral trials. It may be that the additional resources recruited from the right frontal hemisphere led to successful inhibition on alcohol trials, producing null findings on the behavioral measures. Because there was a lack of heightened left frontal activity to begin the neutral trials, shifts in frontal asymmetry were not used to successfully engage control on those trials. Individuals may have engaged in greater impulsive control on some trials including alcohol cues, thus reducing the overall pump count to alcohol cues These cognitive resources engaged to control impulsive behavior may be limited and depleted if used too frequently (Schmeichel, Baumeister, Baumeister, & Vohs, 2004; Robinson, Schmeichel, & Inzlicht, 2010; Vohs et al., 2014). Individuals who are exposed to alcohol cues more frequently may at first be successful at controlling impulsive urges, but over time may find that engaging right frontal activity to inhibit becomes more difficult. This work highlights the need to examine how repeated cue exposure and recruitment of cognitive control resources may eventually lead to unsuccessful inhibition of impulsive behavior.

It is well established that higher levels of impulsivity are associated with increased alcohol consumption, neural reactivity to alcohol cues, and alcohol-related problems (Clark, Vanyukov, & Cornelius, 2002; Dick et al., 2010; Mechin et al., 2016; Verdejo-García, Lawrence, & Clark, 2008). Specifically, the risk-taking component of impulsive behavior has been tied to increased alcohol use and alcohol-related problems (Lejuez et al., 2002; Skeel, Pilarski, Pytlak, & Neudecker, 2008).

Among college student social drinkers, risk-taking but not response inhibition or delay discounting predicted alcohol use (Fernie, Cole, Goudie, & Field, 2010). Thus, understanding the neural processes that accompany risk-taking in the context of alcohol cues could hold potential implications for the development and treatment of substance abuse. Potentially targeting responses to alcohol cues in the environment and developing strategies to recognize and avoid alcohol cues and triggers may prevent some of these neural responses and affect subsequent behavior.

The modified BART task incorporated into this study provided a novel way to examine the influence of alcohol cues on risk taking and frontal asymmetry. By including the picture on the balloon, participants continued to see the cue throughout the trial and the size of the picture increased with each balloon pump. Continual exposure to the pictures and the increasing size of the pictures may have revealed how alcohol cues alter neural activity related to impulsivity to a greater extent than simple prior exposure. However, this task also had some limitations. For example, due to the naturalistic design of the task, there were differences in the number of trial types for each person. In order to maintain the realistic nature of the study, participants could make decisions to cash out or keep going on each trial. Using a risk taking study with rigged feedback may help to create equal numbers of trials for each condition. Additionally, the current study used North American college students who likely differ from the general population. The current study did not screen for past psychiatric disorders or brain injuries such as concussion that could impact asymmetric function in the brain. A general community sample including individuals with alcohol use disorder could be used to determine if these shifts in frontal asymmetry occur in all segments of the population.

The current study sought to add to a growing body of literature linking asymmetric function of the frontal cortex to impulse control. Past studies have not examined shifts in frontal asymmetry across trials of a task, and the current findings point to the involvement of the lIFG and rIFG in producing impulsive or controlled behavior. While past literature has assumed a link between right frontal asymmetry and withdrawal, the current study supports a model of frontal asymmetry linking approach motivation and impulse control to the left and right frontal cortices, respectively. These results also occurred specifically in the context of alcohol cue exposure. The shifts towards left frontal asymmetry observed when impulse control was not engaged have implications for problem drinking in environments heavy with alcohol cues. Greater inhibitory control may be required for successful behavioral inhibition when individuals are exposed to alcohol cues, even when individuals have not consumed alcohol. The need for greater inhibitory control may lead to greater frequency of failed impulse control.

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