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# Approach-related left prefrontal EEG asymmetry predicts muted error-related negativity

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ABSTRACT

In two studies, we used electroencephalography (EEG) to test whether approach-motivation-related brain activity would predict reduced sensitivity to negative outcomes. In both studies, participants (Study 1, $N = 26$; Study 2, $N = 56$) were first recorded for baseline EEG to measure approach-related left frontal EEG activity. They then completed either the color-naming Stroop task (Study 1) or the Multi-Source Interference Task (Study 2) to measure error-related negativity (ERN), an event-related potential that has been associated with aversive motivation and distress. In both studies, higher leftward frontal EEG asymmetry predicted reduced ERN amplitude. Hierarchical regression analyses of the separate frontal nodes that comprised the asymmetry score further showed that left frontal activity predicted reduced ERN amplitude whereas right frontal activity predicted greater ERN amplitude. Results have implications for understanding emotion and motivation and for understanding the personal resilience associated with approach motivated states.



## 1. Introduction

Motivation and goal-regulation models generally recognize two basic systems. The approach system guides behavior toward rewards and positive outcomes whereas the avoidance system guides behavior away from punishment and negative outcomes (Elliot, 2006). Each system is thought to increase sensitivity to motivationally relevant stimuli and reduce sensitivity to irrelevant stimuli. For example, the approach system is thought to increase sensitivity to positive stimuli and decreases sensitivity to negative stimuli. Approach-mediated inhibition of negative stimuli is thought to be largely adaptive, as it promotes more efficient, unconflicted action toward rewarding goals (Harmon-Jones et al., 2008). Evidence that approach increases sensitivity to positive stimuli is abundant (Derryberry and Reed, 1994; Elliot, 2008; Lang, 1995). Evidence that approach antagonizes sensitivity to aversive stimuli is lacking, however. To address this empirical gap, in the present research we used electroencephalography (EEG) to examine whether approach motivation reduces sensitivity to negative stimuli and outcomes. Discovery of a basic, oppositional relationship between approach and avoidance systems would have broad implications for motivational and affective processes, including the reliable yet puzzling link between approach-related brain activity and resilience.

## 2. Approach motivated muting of negative stimuli

Several influential constructs have been formulated to describe approach motivation processes, including the behavioral approach/activation system (BAS; Gray and McNaughton, 2000; see also Fowles, 1980), the behavioral facilitation system (BFS; Depue and Collins, 1999), promotion focus (Higgins, 1997), and approach temperament (Elliot and Thrash, 2002; for similar approach constructs see also Carver and Scheier, 1998; Derryberry and Reed, 1994). Consistent across these models is the notion that approach motivation heightens sensitivity to positive, motivationally relevant stimuli—a notion that has been reliably demonstrated. For example, trait-levels of BAS predict increased positive affect to expected reward (Carver and White, 1994) and faster reaction times to signals of potential gains (Smillie and Jackson, 2005). Extraverts (the correspondent disposition of the BFS, Depue and Collins, 1999) are more sensitive to positive-mood inductions (Larsen and Ketelaar, 1989). Promotion focus predicts faster reactions to approach-related emotional words, reward-maximizing behavioral strategies, and more intense emotions during approach motivated goal pursuit (Higgins, 1997). Approach-positive emotions elicit a more focused breadth of attention (Gable and Harmon-Jones, 2008; Harmon-Jones and Gable, 2009), and approach-related arousal causes increased attentional

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'capture' by incentive-related spatial locations (Derryberry and Reed, 1994).

In order to promote unconflicted, efficient action aimed at rewarding outcomes, however, these same models also posit that approach systems antagonize, or directly mute avoidance- or inhibition-related systems and sensitivity to negative outcomes. The *joint subsystem hypothesis* (Corr, 2002) formalizes this idea in positing that the BAS can inhibit the behavioral inhibition system (BIS). The BIS is a neurobiological system activated by goal conflict that inhibits goal-pursuit, initiates anxiety, and involves septo-hippocampal circuitry, the amygdala, the ACC, and the PFC (Gray and McNaughton, 2000). Thus, according to the joint subsystems hypothesis (Corr, 2002) heightened BAS activation decreases BIS-mediated reactivity to aversive events. At the behavioral level, there is a hypothesized opponent process between approach reflexes and withdrawal reflexes, modulated at multiple stages ascending the central nervous system (Berntson and Cacioppo, 2008). At a more cognitive level, promotion focus and prevention focus (perceptual states related to approach and avoidance-motivation) are also assumed to be mutually inhibitory (Higgins, 1998) with promotion focus antagonizing sensitivity to avoidance-related stimuli. Thus, across several levels of analysis, approach states and traits are thought to mute avoidance systems or sensitivity to negative stimuli. Note that this contrasts with the opposing view that approach and avoidance systems are functionally orthogonal (Gable et al., 2003; Gray, 1970). For example, the BAS and BIS were originally thought to operate independently (Gray, 1970). That is, reward sensitivity was thought to be unrelated to aversive processes (Corr, 2002, 2008; Gable et al., 2003).

Despite these opposing views, psychological theorists have widely adopted the notion that approach mutes avoidance. For example, approach-related downregulation of anxiety and aversive stimuli is assumed in research on power reducing inhibition (Hirsh et al., 2011), approach motivated reactions to conflict (Harmon-Jones et al., 2008; McGregor et al., 2010), affect regulation through action-orientation (Gollwitzer, 1990; Koole and Coenen, 2007), disrupted-approach theories of depression (Haeffel et al., 2008) and action-based therapies for treating anxiety (Watson, 2005). However, this muting hypothesis has been supported by only preliminary evidence, despite its pervasive acceptance. For example, approach-related emotions mute the startle response (Corr, 2002; Lang, 1995). Appetitive stimuli can diminish aversive stimulus-response learning (Dickinson and Pearce, 1977). Activation of reward systems, even through viewing pictures of significant others, reduces the perception of pain (Younger et al., 2010). Additionally, impulsive people, who are presumably approach motivated, do not attempt to avoid punishment when in an aroused state (Corr, 2002).

Suggestive evidence can also be gleaned from research using the error-related negativity (ERN, Gehring et al., 1993)—an event-related scalp potential caused by error commission that has been source localized to the anterior cingulate cortex (ACC; Dehaene et al., 1994). The ERN is traditionally thought to index cognitive conflict, such as the conflict between competing responses (Yeung et al., 2004) or expectancy violations (Holroyd and Coles, 2002). Recent evidence, however, indicates the ERN is influenced by affect, prompting some to suggest this wave reflects a neural "distress signal" activated by aversive events (Bartholow et al., 2005; Luu et al., 2000). For example, ERN amplitude has been associated with anxiety-related personality traits, the defensive startle response, and is muted by anxiolytic agents (Hajcak and Foti, 2008; Hajcak et al., 2003; Johannes et al., 2001). Importantly, the ERN is also associated with dispositional BIS sensitivity (Amodio et al., 2008; Boksem et al., 2006). Moreover, in a recent meta-analysis the ACC appears to be a key neural structure for aversive motivation (Shackman et al., 2011). Thus, the ACC-localized ERN is a valid marker of distress and sensitivity to negative events.

Consistent with the idea that approach may downregulate reactivity to negative events, the ERN is muted in people sensitive to reward, particularly when errors are punished (i.e., an aversive context, Boksem et al., 2008). Similarly, participants with tendencies toward impulsivity and risk taking traits that are related to the BAS and approach motivation (Corr, 2002) also demonstrate decreased ERN after errors (Potts et al., 2006; Santesso and Segalowitz, 2008).

In the current research, we provide more direct neural evidence of an antagonistic relationship between approach motivation and sensitivity to negative outcomes. In two studies we assessed whether a neurophysiological marker of approach motivation (leftward PFC asymmetry) would predict lower amplitude ERNs (a neural marker of aversive motivation and distress). Relative left PFC activity has been reliably linked with a number of approach motivation-related phenomena in dozens of studies (many of which are reviewed in Elliot, 2008), including states of positive affect, anger, dispositional BAS, promotion focus orientation, social power, and reward sensitivity (Amodio et al., 2004; Boksem et al., 2009; Harmon-Jones, 2003; Harmon-Jones and Allen, 1997; Harmon-Jones and Sigelman, 2001; Pizzagalli et al., 2005; Tomarken et al., 1992). We thus predicted that greater leftward PFC asymmetry would predict a muted ERN.

Because frontal asymmetry is a relative measure, it raises the question of whether left (approach-related) or right (avoidance-related) PFC activity separately predict ERN amplitude. We used whole brain analyses (as recommended by Allen et al., 2004) to explore the relative contribution of left or right PFC activity to the predicted link between frontal asymmetry and the ERN. We hypothesized that both approach- and avoidance-related frontal activation would predict ERN amplitude, though in opposing directions.

## 3. Study 1

### 3.1. Method: Resting-state EEG and Stroop task ERN

Twenty-six right-handed participants (17 females; *median age* = 19) provided informed consent and were then recorded for eight 1-min intervals (4 eyes open, 4 eyes closed) of resting-state, baseline EEG. They then completed a standard color-naming Stroop task, in which they pressed a colored button that corresponded to the font-color of color words (e.g., 'green') that either matched or mismatched the word meaning. There were 240 trials presented, 160 of which matched and 80 of which mismatched. Each trial began with a center-screen fixation cross for 500 ms, and then the color word for 200 ms. Participants had 800 ms to respond. They were instructed to respond as quickly and accurately as possible. Error commission on the Stroop task reliably elicits the ERN distress signal that served as our criterion variable (e.g., Inzlicht et al., 2009).

### 3.2. EEG recording and processing

Baseline EEG and right-eye vertical electro-oculogram (VEOG) activity were recorded and digitized at 512 Hz with average ear reference and forehead ground. Recordings were collected from 32 tin electrode sites positioned according to the 10–20 system and all impedances were below 5000 Ω. EEG was bandpass filtered at .1–100 Hz and notch filtered at 60 Hz then corrected off-line for eye-blinks using the SOBI procedure (Tang et al., 2005). Movement artefacts were automatically detected with a −75 μV and +75 μV threshold. Contiguous artefact-free epochs of 2 s were extracted through a hamming window and overlapped by 75% to avoid

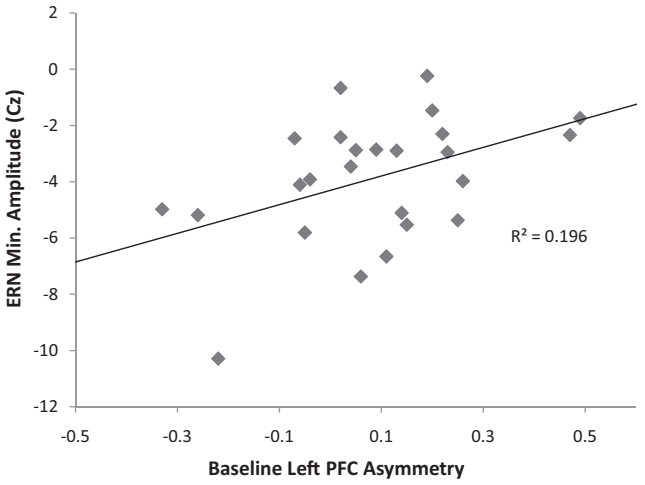


**Fig. 1.** Scatterplot of the relation between baseline left PFC asymmetry and ERN minimum amplitude in Study 1.

data loss. Power spectra were calculated via fast Fourier transform and power values (in $\mu V^2$) were averaged over the epochs from each 1 min interval of resting-state recording and total alpha band power (8–13 Hz), an inverse indication of cortical activity, was logarithmically transformed. Baseline left PFC asymmetry was calculated across the 8 min resting-state as F8 minus F7 electrode log alpha power. Higher scores indicate relatively greater left-than-right cortical activation (Coan and Allen, 2004). The 8 min intervals demonstrated satisfactory reliability, Cronbach $\alpha = .91$.

For computation of the ERN, EEG recorded during the Stroop task was digitally filtered between 1 and 15 Hz and baseline corrected between 300 and 200 ms before button press. Blink and artefact detection methods were the same as used for the baseline EEG. For each artefact-free trial, a 1000-ms epoch of EEG signal locked on the button press was averaged across incorrect trials for the ERN and correct trials for correct-related negativity (CRN); this window started 200 ms before and 800 ms after the response. The ERN was quantified as the peak negative amplitude between 50 ms before and 150 ms after response at the central midline electrode (Cz). A CRN peak amplitude score was also computed at Cz averaged across correct trials. To ensure the left PFC asymmetry predicted error-specific negativity rather than negativity to responding in general (see Luck, 2005), the ERN score was residualized on the CRN. All analyses thus used this residualized ERN variable.[1]

### 3.3. Results

Pearson correlations were conducted between baseline left PFC asymmetry, ERN scores, Stroop errors, and the Stroop incongruency effect (reaction time on correct mismatched trials minus reaction time on correct matched trials). Results showed that baseline left PFC asymmetry was significantly related to smaller ERN amplitudes, $r = .44$, $p < .05$ (see Figs. 1 and 2), whereas all other correlations with baseline left PFC asymmetry were non-significant (see Table 1).[2] Note that partial correlations between baseline left PFC asymmetry and the ERN peak amplitude remained significant when controlling for Stroop errors and the incongruency effect (both $p$'s $< .05$).

**Table 1**
Correlations between baseline left PFC asymmetry, error-related negativity (ERN), and Stroop behavior in Study 1.

| | 1. | 2. | 3. |
|---|---|---|---|
| 1. Baseline left PFC asymmetry (F8/F7) | | | |
| 2. ERN min. amplitude (Cz) | .49* | | |
| 3. Stroop errors | .32 | .20 | |
| 4. Stroop incongruency | .13 | −.04 | .33 |

* $p < .05$.

We next conducted exploratory hierarchical regression analyses, as recommended by Allen et al. (2004), to examine whether the separate left and right nodes that comprise the frontal asymmetry score themselves predict ERN amplitude (Allen et al., 2004). We regressed ERN on a logarithmically transformed whole brain alpha average of each node (to control for individual differences in EEG power), and the logarithmically transformed F7 (the left frontal node) and F8 (the right frontal node) alpha averages were entered together in the second step. Results demonstrate that both F7, $t = -2.06$, $p = .05$ and F8, $t = 2.64$, $p < .05$, significantly predicted ERN amplitude, but in opposite directions. More left PFC activity (i.e., less alpha) predicted reduced ERN amplitude, whereas more right PFC activity predicted increased ERN amplitude.

These results are the first to indicate that approach-related neural activity is associated with reduced ERN amplitude. This suggests that approach motivation may mute reactivity to aversive events. The single node analyses also indicate that this frontal asymmetry-ERN link may be due to both left and right frontal activity. These findings are consistent with the link between dispositional avoidance, the ERN, and right PFC activation and, more importantly, the idea that approach-related activity mutes avoidance motivation.

## 4. Study 2

In Study 2, we attempted to extend the findings from Study 1 and address certain limitations. It has been noted that ERN amplitude is often maximal at the FCz node, rather than the more central Cz or the more anterior Fz (e.g., Gehring et al., 1993), and asymmetry measures have used various homologous frontal nodes, including FC3 and FC4 (Coan and Allen, 2004). However, the EEG system used in Study 1 did not include the FCz, FC3, and FC4 nodes. Thus, in Study 2, we used an EEG system that included these nodes and employed a different reaction time task also known to elicit error-related ACC activation for multi-method replication (The Multi-Source Interference Task, MSIT; Bush and Shin, 2006).

### 4.1. Method: Resting-state EEG and the MSIT

Fifty-six right-handed participants (38 females; *median age* = 18 years), after providing informed consent, were first recorded for two 1-min intervals (1 eyes open, 1 eyes closed) of resting-state, baseline EEG. Participants then completed the MSIT. In this task, participants were instructed to press 1, 2, or 3 on a keyboard to correspond to a unique digit in a set of three (either 1, 2, 3, or 0) presented on the center of a computer screen. The unique digit either matched or mismatched its keyboard position. For example, a match trial could show the digits '122' and would require the keystroke '1', whereas a mismatch trial could show the digits '332' and require the keystroke '2'. Each MSIT session (pre- and post threat) involved 3 blocks of 45 trials, in which there were 30 'match' trials and 15 'mismatch' trials. Error commission on this task reliably activates the ACC (Bush and Shin, 2006).

[1] In both studies, processing parameters for frontal asymmetry scores and the ERN were selected based on precedent to allow comparison with prior research (Amodio et al., 2004, 2008; Harmon-Jones and Allen, 1997; Inzlicht and Al-Khindi, in press; see also Luck, 2005, for the recommendation that ERP results are best compared when using similar processing parameters). However, the 1 Hz high pass filter may contrast with the recommendation for more modest filters (Luck, 2005). We thus re-analyzed Study 1 using a 0.1–15 Hz window for filtering and found that all frontal asymmetry—ERN results remained significant (all $p$'s remained $< .05$).

[2] For comparison, Fig. 2 also includes the waveforms from the Fz node.

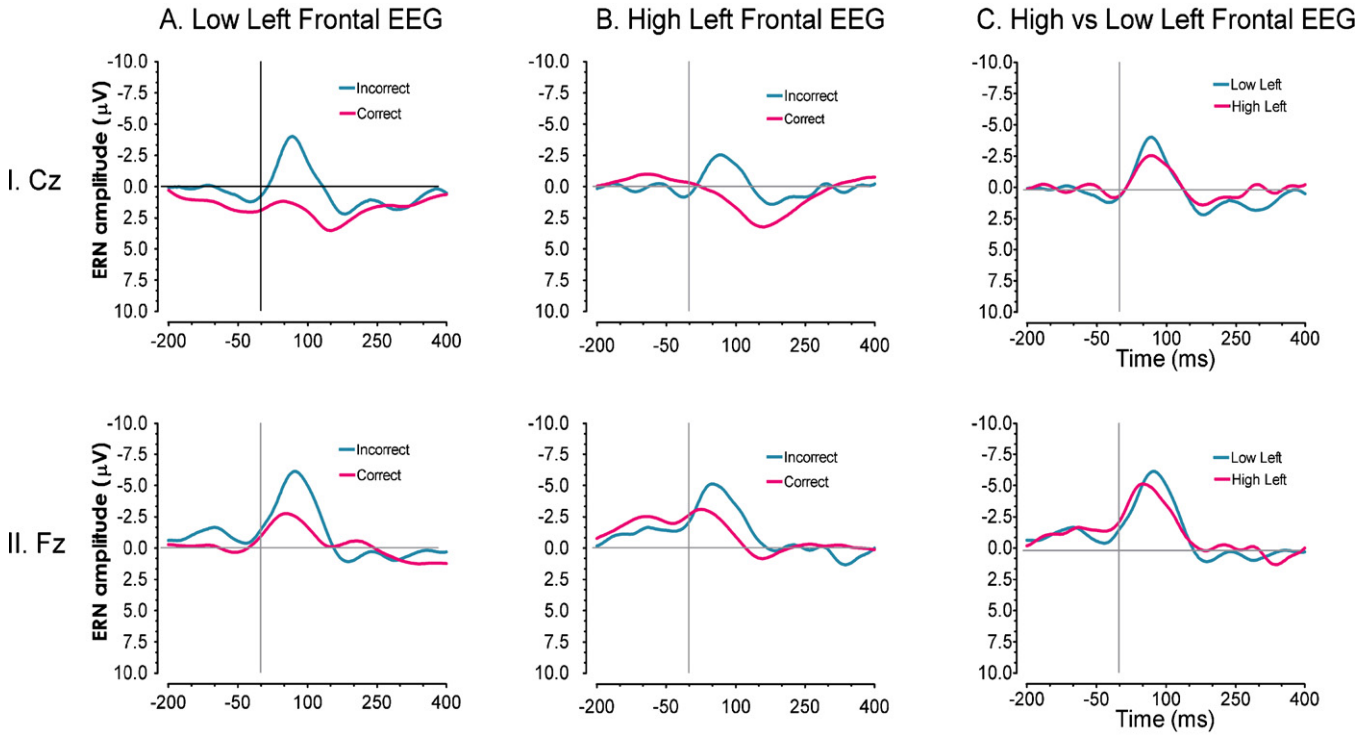


**Fig. 2.** Study 1 stroop response-related potentials at electrode (I) Cz and electrode, (II) Fz for (A) low left frontal EEG individuals; (B) high left frontal EEG individuals; and (C) the ERN for high vs. low left frontal EEG (groups determined on a median split).

### 4.2. EEG recording and processing

EEG and right-eye VEOG recording involved the same procedure as in Study 1. Baseline left PFC Asymmetry was calculated across the 2 min baseline as right site minus left site log alpha power at the following homologous pairs, F8/F7, F4/F3, and FC4/FC3. Again, higher scores indicate relatively greater left-than-right cortical activation. The 2 min intervals were strongly correlated at each node, all $r$'s > .63.

EEG processing and computation of the ERN was the same as in Study 1, though in relation to MSIT errors and quantified at Cz, FCz, and Fz (peak negative amplitude 50 ms before to 150 ms after error). Higher ERN amplitude is indicated by more negative values. A correct related negativity (CRN) peak amplitude score was also computed at Cz, FCz, and Fz, averaged across correct MSIT trials. As in Study 1, the ERN was residualized on the respective CRN score to ensure that left PFC asymmetry was related to error-specific processes (Luck, 2005).

### 4.3. Results

The baseline left PFC Asymmetry scores calculated at F8/F7, F4/F3, and FC4/FC3 were each entered into Pearson correlations with each ERN score (Cz, FCz, and Fz), Stroop errors, and the Stroop incongruency effect. As seen in Table 2 and Fig. 3, left PFC asymmetry was again related to reduced ERN amplitude, particularly at the FC4/FC3 node, all $r$'s > .31, $p$ < .05. Importantly, the F8/F7 correlation with ERN amplitude from Study 1 was replicated at Fz, $r$ = .27, $p$ < .05, and at FCz, though at $r$ = .24, $p$ = .07. The F4/F3 score was marginally related to the ERN at Cz, $r$ = .23, $p$ = .09. Additionally, as shown in Fig. 4, ERN amplitude was maximal at FCz (consistent with prior research, e.g., Gehring et al., 1993). Partial correlations between baseline left PFC Asymmetry and the ERN peak amplitude remained significant when controlling for the incongruency effect (all significant $p$'s remained at <.05). Interestingly, when controlling for number of errors committed, the left PFC asymmetry—ERN link became significant at F4/F3 and Cz, $r$ = .30, $p$ < .05, and at F8/F7 and both FCz and Fz, both $r$'s > .28, $p$'s < .05.[3]

**Table 2**
Baseline left PFC asymmetry scores and MSIT behavior correlations with error-related negativity (ERN) in Study 2.

| | ERN | | |
|---|---|---|---|
| | Fz | FCz | Cz |
| 1. F8/F7 | .27* | .24† | .15 |
| 2. F4/F3 | .19 | .21 | .23† |
| 3. FC4/FC3 | .32* | .32* | .36* |
| 4. MSIT errors | .33* | .35* | −.45* |
| 5. MSIT incongruency | .02 | −.08 | −.10 |

* $p$ < .05.
† $p$ < .10.

To explore the separate contribution of each frontal node to the relationship between frontal asymmetry and ERN amplitude, we conducted the same hierarchical regression analyses as in Study 1 (Allen et al., 2004), though with all three ERN scores. With log whole brain alpha entered in the first step and the F7 and F8 alpha averages entered in the second step, F7 predicted muted ERN amplitude at Cz, $t$ = −2.02, $p$ < .05; at FCz, $t$ = −1.98, $p$ = .05, and marginally at Fz, $t$ = −1.74, $p$ = .07. In contrast to Study 1, F8 did not predict ERN amplitude across all three ERN scores, all $p$'s > .21. With FC3 and FC4 entered into the second step, the left FC3 node pre-

[3] Errors in Study 2 were unrelated to frontal asymmetry; however, errors were related to ERN amplitude at all three nodes ($p$'s < .05) such that fewer errors predicted a heightened ERN. Thus, the reported partial correlations appear to control for conscientiousness. Prior research has indicated that conscientiousness is indeed associated with heightened ERN amplitude (Tops and Boksem, 2010). In this case, conscientiousness appears to not explain the frontal asymmetry → ERN link, consistent with our interpretation that approach motivation (and not heedless responding) mutes sensitivity to negative events.

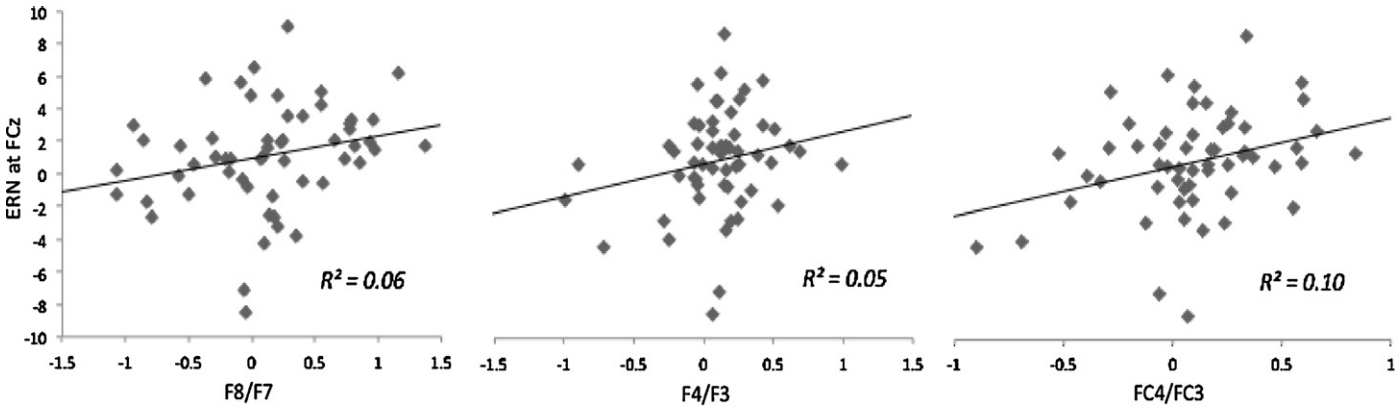


**Fig. 3.** Scatterplots of the relation between the baseline left PFC asymmetry scores at F8/F7, F4/F3, and FC4/FC3 and ERN minimum amplitude at the FCz node.

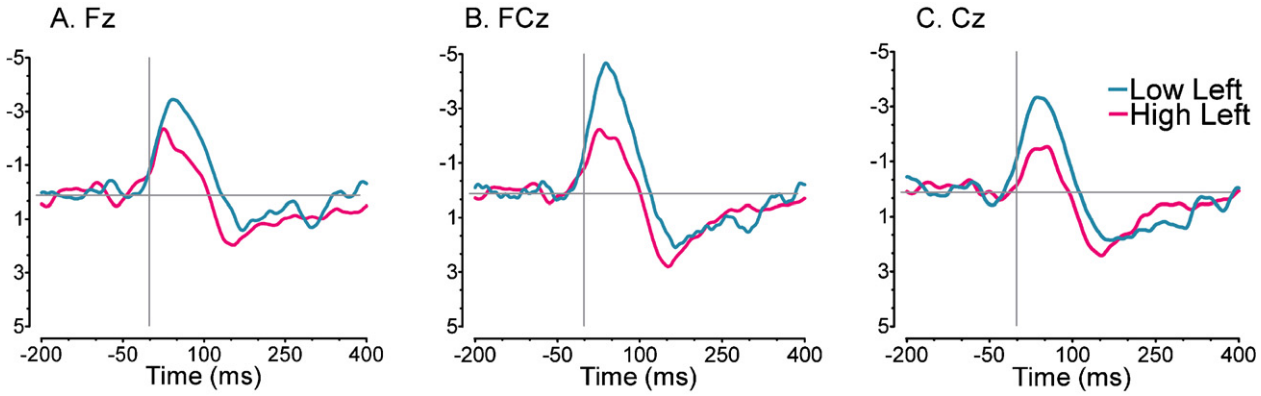


**Fig. 4.** ERNelicited by incorrect responsesfor high and low left PFC asymmetry (groups determined on a median split at FC4/FC3) for (A) Fz, (B) FCz, and (C) Cz in Study 2.

dicted muted ERN amplitude at Cz, $t = -2.24$, $p < .05$, Fz, $t = -2.49$, $p < .05$, and marginally at FCz, $t = -1.91$, $p = .06$, whereas the right FC4 node predicted amplified ERN scores at all three midline sites ($t$'s > 2.10, $p$'s < .05). Finally, the left F3 node was marginally related to muted ERN amplitude at Cz, $t = -1.74$, $p = .09$, and the right F4 node was marginally related to amplified ERN at Cz and FCz, $t = 1.72$ and $t = 1.85$, respectively, both $p$'s < .10 (see Table 3).

Broadly replicating Study 1, left PFC activity predicted muted ERN amplitude, whereas more right PFC activity predicted increased ERN amplitude. This relationship was demonstrated across three different ERN scores. However, there are important differences with Study 1. Specifically, in Study 1 the link between left PFC activation and muted ERN appeared stronger at F7, a more lateral node, whereas the link between right PFC activation and the ERN appeared more robust at FC4, a more medial node than in Study 1. Given the reduced spatial resolution of EEG, it would be premature to speculate as to why seemingly different PFC regions were involved. Future research using fMRI or high-density EEG is needed to resolve this question.[4]

**Table 3**
Beta coefficients between left and right frontal node activity and error-related negativity (ERN) from the hierarchical linear model analyses in Study 2.

| | ERN | | |
|---|---|---|---|
| | Fz | FCz | Cz |
| 1. F7 | −.48† | −.51* | −.52* |
| 2. F8 | .31 | .22 | −.03 |
| 3. F3 | −.74 | −.78 | −.84† |
| 4. F4 | .64 | .74† | .69† |
| 5. FC3 | −1.22* | −.94† | −1.09* |
| 6. FC4 | 1.09* | 1.04* | 1.14* |

* $p < .05$.
† $p < .10$.

## 5. General discussion

Although a number of motivational and goal-regulatory models postulate that approach and avoidance systems can sometimes operate in opposition (e.g., Corr, 2002, 2008), little evidence to date has explicitly examined such a relationship. Dispositional approach and avoidance/inhibition have been associated with left PFC asymmetry and the ERN, respectively (Amodio et al., 2008; Boksem et al., 2006; Harmon-Jones and Allen, 1997). The current study demonstrated that left PFC asymmetry predicts reduced ERN amplitude. The results provide the first direct neural support for what has been described as a 'joint subsystem hypothesis' (Corr, 2008)—independent electrophysiological markers of approach and avoidance were negatively correlated during aversive experiences.

Whole brain analyses also examined the distinct contribution of left and right PFC activity to the link between frontal asymmetry and ERN amplitude, with results indicating that left and right PFC are both involved. The link between right PFC activity and ERN amplitude is, perhaps, not surprising given that both neural measures have been separately related to avoidant/inhibited

[4] In both studies, the CRN was unrelated to all frontal asymmetry scores, except for a correlation between F8/F7 and CRN scores at FCz and Fz in Study 2. Importantly, hierarchical analyses revealed that left frontal activation (F7) was unrelated to the CRN, indicating that the CRN did not impact the left frontal → ERN relationship.

disposition (Amodio et al., 2008; Shackman et al., 2011). To our knowledge, this study is the first demonstration of such a link, however. Such findings corroborate research that indicates that the ACC and the right PFC may be integral components of a distributed inhibitory network (Goel, 2007; Kerns et al., 2004; Yan et al., 2009).

More intriguing is the link between left PFC activity and reduced ERN. Future research should examine if this relationship is mediated by dopaminergic systems. Consistent with the idea that approach-related states may regulate neural reactivity to distressing events, recent research in rats has demonstrated that left PFC dopaminergic afferents regulate the glutamatergic response in the right PFC to stressful events (Lupinsky et al., 2010). Dopamine has been strongly linked to approach motivation processes (Depue and Collins, 1999; Schultz, 1998) and frontal asymmetry is thought to arise due to different signalling strengths of the mesocortical dopaminergic pathways (Berridge et al., 2003). Indeed, induced left prefrontal activation (with repetitive transcranial magnetic stimulation) causes subcortical dopamine release in the ipsilateral caudate nucleus (Strafella et al., 2001). On the other hand, the ERN is thought to be initiated by a phasic dip in subcortical dopamine that disinhibits apical dendrites in the ACC (Holroyd and Coles, 2002). Future study could examine if approach-related neural processes increase subcortical dopamine levels, which then mute ERN amplitude.

These results also add to the growing literature supporting a motivational characterization of the ERN. Originally, computational models of ERN function emphasized this waveform as a neural index of cognitive conflict elicited by error commission, hypothesized as the conflict between predicted and actual outcomes (Holroyd and Coles, 2002) or conflict between simultaneous activation of correct and incorrect responses (Yeung et al., 2004). Purely cognitive models of the ERN, however, may be incomplete. Evidence shows that error commission elicits autonomic arousal and defensiveness (Critchley et al., 2005; Hajcak and Foti, 2008) and the ERN is bolstered by states and traits related to anxiety (Hajcak et al., 2003). This has led some to characterize the ERN as a neural "distress signal" (Bartholow et al., 2005) that reflects not only 'cold' error detection but also an emotional or motivational response to error (Luu et al., 2000). Consistent with this cognitive/motivational view of the ERN, our results showed that the ERN amplitude was linked to a neural index of motivation. Further research that experimentally induces motivational states (e.g., Hajcak et al., 2005; Legault and Inzlicht, submitted for publication) could help future research establish and determine the extent to which the ERN involves an emotion- or motivation-related component.

Finally, these findings hint at intriguing speculations about the reliable link between approach motivation and resilience or health (Davidson, 2004; Elliot, 2008). For example, relative left PFC activity has been associated with improved startle recovery, reduced cortisol, better immune functioning, positive emotions, and well-being (see Davidson, 2004). Additionally, inducing relative left prefrontal activity through transcranial magnetic stimulation can reduce symptoms of anxiety and depression (Ressler and Mayberg, 2007; Schutter et al., 2001). As suggested by the current findings, by muting ACC reactivity, approach motivation may inhibit the cascade of anxiety-related symptoms to stressful events that may be driven by the ACC (Critchley et al., 2005). If so, this would help make sense of experimental research showing that people react to anxiety-provoking experiences and motivational conflicts with a defensive surge in approach motivation (McGregor et al., 2010; Nash et al., 2011), behavioral activation (Schmeichel et al., 2010) and left PFC asymmetry (Harmon-Jones et al., 2008; McGregor et al., 2009; Nash et al., 2010). Reactive approach activation may be a rewarding response in frustrating and stressful circumstances because it dampens signals of distress. Future research should assess whether the appeal of compulsive gambling, risk-taking, sex or food addiction, power, anger, and ideology – all approach-motivation-related phenomena (Harmon-Jones and Sigelman, 2001; Keltner et al., 2003; McGregor et al., 2010) – may arise from their capacity to activate approach motivation and mute the ERN, thereby providing reliable relief in distressing circumstances.

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