

# Frontal EEG Activation Asymmetry Reflects Cognitive Biases in Anxiety: Evidence from an Emotional Face Stroop Task

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**Abstract** Electroencephalography (EEG) has been extensively used in studies of the frontal asymmetry of emotion and motivation. This study investigated the mid-frontal EEG activation, heart rate and skin conductance during an emotional face analog of the Stroop task, in anxious and non-anxious participants. In this task, the participants were asked to identify the expression of calm, fearful and happy faces that had either a congruent or incongruent emotion name written across them. Anxious participants displayed a cognitive bias characterized by facilitated attentional engagement with fearful faces. Fearful face trials induced greater relative right frontal activation, whereas happy face trials induced greater relative left frontal activation. Moreover, anxiety specifically modulated the magnitude of the right frontal activation to fearful faces, which also correlated with the cognitive bias. Therefore, these results show that frontal EEG activation asymmetry reflects the bias toward facilitated processing of fearful faces in anxiety.

**Keywords** Frontal asymmetry · EEG · Cognitive bias · Anxiety

## Introduction

Since the first neuropsychological indications that affective symptoms are associated with lesions of the left frontal lobe (Gainotti 1972; Myers 1972; Robinson et al. 1984),

and the pioneering demonstration that electroencephalography (EEG) reflects the asymmetrical involvement of the frontal lobes in emotional experience (Davidson et al. 1979), the asymmetry of frontal EEG activity in emotion has become one of the most popular topics of investigation in affective neuroscience (Allen and Kline 2004; Davidson 2004).

In light of the inverse relationship between the engagement of cortical systems in information processing and EEG activity in the alpha range (8–13 Hz) (Larson et al. 1998; Lindsley and Wicke 1974), most of the studies have focused on how frontal EEG alpha power recorded during rest or information processing correlates with aspects of emotion and motivation (for reviews, see Coan and Allen 2004; Davidson 2004; Harmon-Jones 2004). Relatively greater left frontal activity has been associated with a general appetitive, approach, or behavioral activation system, in contrast with the relatively greater right frontal activity that has been related to an aversive, avoidance or withdrawal system (e.g., Coan and Allen 2003; Hagemann et al. 2002; Harmon-Jones et al. 2006; Tomarken et al. 1992). Moreover, there are individual differences in frontal EEG activity that index “affective styles” or trait-like propensities toward displaying certain emotions (i.e., reflected in the threshold or magnitude of emotions, emotional resilience, and efficiency of emotion regulation), and moderates the risk for anxious or affective psychopathology (Davidson 1998). Indeed, the asymmetry of frontal EEG activity is partly heritable (Coan et al. 2003; MacDhommhail et al. 1999), and active in emotional processing in newborns (e.g., Davidson and Fox 1982). According to the diathesis-stress hypothesis of frontal activation asymmetry, an affective style characterized by reduced relative right frontal activity is associated with altered aversive and withdrawal responses consistent with

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anxiety, whereas one characterized by reduced relative left frontal activity is associated with altered appetitive and approach responses consistent with depression (e.g., Coan and Allen 2004; Davidson 1992).

Following the suggestion that cortical systems that are involved in emotion regulation and emotion-cognition interactions may be specifically disturbed in anxiety and depression—in contrast to subcortical systems such as the amygdala, which contribute to both (Davidson 1998; see also Kentgen et al. 2000), several studies have investigated the asymmetry of frontal and parietal alpha EEG activity in subclinical and clinical anxiety. For instance, greater relative right frontal EEG activity has been reported in social phobia and panic disorder, which in addition correlated with state and trait self-report measures of anxiety (Davidson et al. 2000; Wiedemann et al. 1999). In contrast, other data indicated that high scorers on similar measures of anxiety showed greater relative left frontal activity (Heller et al. 1997). Consequently, it has been suggested (e.g., Heller et al. 1997) that self-reported trait anxiety (TA) indexes anxious apprehension or worry (i.e., verbal rumination about future events), which may be distinct from anxious arousal (i.e., symptoms of physiological arousal). In this and other subsequent studies, the former correlated with greater relative left frontal activity, whereas the latter induced right frontal or parietal activation (Heller et al. 1997; Mathersul et al. 2008; Metzger et al. 2004). However, this distinction of anxiety types and frontal-parietal EEG correlates needs to be further investigated. Based on the available literature, meta-analyses support only the moderate effect of anxiety on the right-sided asymmetry of frontal activity (Thibodeau et al. 2006).

The need to focus on the frontal EEG *activation* (i.e., EEG recorded during information processing) in conditions that are relevant to the pathogenesis of anxiety has been emphasized (Coan and Allen 2004; Coan et al. 2006; Davidson 1998). For instance, in a cognitive task that manipulated behavioral contingencies in order to produce approach and withdrawal states, rewards induced greater left frontal activation, whereas punishments induced greater right frontal activation (Sobotka et al. 1992). Using a directed facial action task, the simulation of facial fear and other emotions denoting withdrawal was associated with relatively less left frontal activation in comparison to approach states (Coan et al. 2001; see also van Honk and Schutter 2006). Based on this pattern of results, the authors concluded that: “withdrawal states produce relatively less left frontal activation rather than relatively greater right frontal activation” (Coan et al. 2001, p. 921). Clearly, further research would be useful in order to elucidate whether withdrawal-related emotions specifically modulate activity in the right or left frontal

lobe. The present study was designed to investigate frontal EEG activation in an emotional face analog of the Stroop task. Lexical and pictorial versions of the emotional Stroop task have long been used in cognitive psychology, in order to measure cognitive biases that are involved in anxiety psychopathology (for review see Eysenck et al. 2007; Mathews and MacLeod 2005; Miu and Visu-Petra 2010). However, the frontal EEG asymmetry has not been investigated in this clinically-relevant task.

We used an emotional face analog of the Stroop task, in which participants selected for extreme trait anxiety (TA) scores had to name the expression (e.g., calm, fear, happiness) that they were recognizing in pictures, while ignoring the congruent or incongruent name of an expression (e.g., the word “CALM”, “FEAR” or “HAPPY”) that was written across the picture. The monitoring and resolution of the emotional face-word conflict in incongruent trials relies on a prefrontal-amygdala circuit that has also been involved in emotion regulation (Egner et al. 2008; Etkin et al. 2006; Goldin et al. 2008). In light of the previous literature on cognitive biases in anxiety, we predicted that anxious participants would show a cognitive bias toward facilitated processing of fearful faces in this task, which would be reflected by increased relative right frontal activity.

## Methods

### Participants

Twenty-four right-handed college students (age between 19 and 22 years) were selected for extreme high ( $N = 15$ ; mean  $\pm$  SD. TA scores:  $55.13 \pm 5.59$ ) or low scores ( $N = 9$ ; mean  $\pm$  SD. TA scores:  $27 \pm 2.95$ ) of TA on the Romanian version of the State-Trait Anxiety Inventory (Pitariu and Peleasa 2007; Spielberger 1983). There was a significant difference between the TA scores of the two groups ( $t[23] = 13.88$ ,  $P < 0.0001$ , Cohen's  $d = 6.29$ ). Notably, the mean scores of the high TA participants to this study are above the mean TA scores reported for psychiatric patients (mean  $\pm$  SD  $52 \pm 12.4$ ; see de Visser et al. 2010). The trait portion of the form Y of STAI includes 20 items that evaluate feelings of apprehension, tension, nervousness, and worry, and discriminates anxiety from depression better than its previous form. No participant had antecedents of neuropsychiatric or cardiovascular disorders, and they were free of medication (e.g., beta-blockers, anxiolytics) that could interfere with the physiological recordings. In addition, they were asked to refrain from alcohol, caffeine and smoking at least 4 h before the experiment. They signed an informed consent to participate in this study, and all the experimental procedures were

non-invasive and complied to the recommendation of the WMA's Declaration of Helsinki.

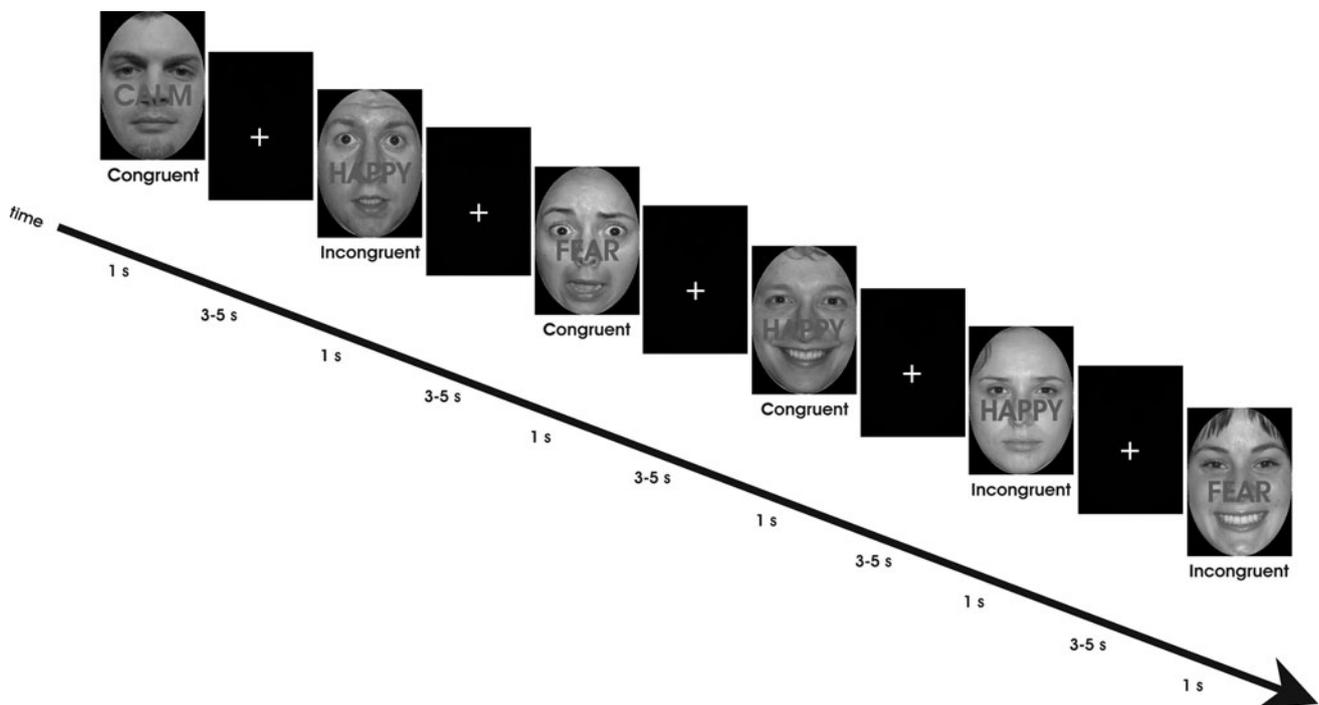
### Procedure

We adapted a computerized version of the emotional face Stroop analog, originally described by Etkin et al. (2006). Essentially, the task was run in SuperLab and included forty trials in which emotionally-neutral, happy or fearful faces appeared on the screen, with a congruent or incongruent name of each emotion (i.e., CALM, HAPPY, or FEAR) written across the face. The name was displayed in a region of the picture that did not occlude the parts of the face (e.g., eyes, corner of the lips) that are important in emotion recognition (see Fig. 1). The task was preceded by a similar 20-stimuli learning phase. The participants were instructed to ignore the word and name the facial expression that they recognize in the picture. Their vocal reaction times (RTs) were digitally recorded using a voice recognition software. Outlier RTs that were above or below the mean  $\pm 2$  SD of each participant were excluded (0.42% of the data), as recommended (see Williams et al. 1996). Cognitive biases were calculated by subtracting RTs in neutral trials from RTs in happy (appetitive bias) or fearful trials (aversive bias). Therefore, a decreased bias score indicates facilitation in processing that type of emotional stimulus.

### Psychophysiology

EEG, skin conductance (SCR) and electrocardiogram (ECG) were continuously recorded during the task. EEG was recorded using an ECI-Electrocap with electrodes positioned according to the International 10–20 method of electrode placement, including the earlobes. The ground electrode was placed between the frontal pole and the frontal site, whereas the reference electrode was placed on the left ear. Eye movements were also recorded in order to facilitate artifact scoring of the EEG. All electrode impedances were under 5 k $\Omega$  and homologous sites had impedances that were within close range relative to each other. EEG, electrooculogram, as well as SCR and ECG were amplified using a Biopac MP150 system, band-pass filtered (0.1–100 Hz), and digitized at 500 Hz onto a computer. Before each session, psychophysiological signals were recorded during rest in order to estimate the baseline.

Off-line, EEG was re-referenced to average earlobes and manually scored for movement artifact. Artifact-free epochs of 1,000 ms, which corresponded to intervals of interest starting 50 ms after stimulus onset, were extracted through a Hanning window (cosine window, tapered at the distal end 10% of each 1,000 ms epoch) to reduce leakage, and then submitted to the Fast Fourier Transform algorithm. Contiguous epochs were overlapped by 25%



**Fig. 1** Stimuli and timeline of the emotional face Stroop analog task. Participants were instructed to identify the expression that they recognize on the calm, fearful and happy faces, while ignoring the congruent or incongruent “CALM”, “FEAR”, “HAPPY” names written across them

avoid data loss due to tapering. Average alpha (8–13 Hz) power was extracted in 1 Hz bins and natural log transformed to normalize the distribution. Absolute band power values from midfrontal sites (F3/4) are reported here. Alpha power is inversely related to cortical activity so decreased power indicates cortical activation.

As previously described (Miu et al. 2009), ECG was recorded using Ag/AgCl electrodes placed in a bipolar precordial lead (sample rate of 500 Hz/s). After visual inspection of the recordings and editing to exclude artifacts in AcqKnowledge 3.7.1, all the recordings were analysed using Nevrokard 7.0.1 (Intellectual Services, Ljubljana, Slovenia). Heart rate (HR) was estimated in beats per minute (BPM) from the same 1,000 ms intervals of interest during which the stimuli were processed, and HR change was calculated by subtracting the individual baseline HR from the HR recorded during information processing. Baseline recordings were independently made, several days before the experiments.

SCR was recorded using TSD203 transducers filled with isotonic gel and attached to the volar surfaces of the index and medius fingers. The area under the curve ( $\mu\text{S}$ ) of SCRs in the intervals of interest was extracted, after the down-drift in the SCR waves was eliminated using the “difference” function of Acknowledge, as previously described (Miu et al. 2008).

#### Data Analysis

The data were analyzed using ANOVA, Student *t* and correlation tests, with the Bonferroni correction for multiple comparisons where required. The statistical analyses were run in Statview software.

## Results

#### Manipulation Checks

To check whether the task succeeded to induce emotional conflict, we inspected the raw RTs to congruent and incongruent faces. The RTs on incongruent trials were significantly higher than those in congruent trials ( $t[22] = 2.62$ ,  $P = 0.02$ , Cohen's  $d = 0.9$ ). In addition, the magnitude of HR deceleration was higher in incongruent than congruent stimuli, which indicated augmented orientation responses when the emotional face and word were incongruent ( $t[22] = 2.82$ ,  $P = 0.01$ , Cohen's  $d = 1.7$ ). The amplitude of SCRs was higher during the processing of fearful and happy faces compared to neutral faces ( $t[22] = 2.86$ ,  $P < 0.01$ , Cohen's  $d = 0.75$ ), which confirmed that the emotional stimuli induced physiological arousal.

#### Cognitive Bias

We analyzed the effects of anxiety on aversive and appetitive bias scores. Anxiety had a significant effect on the former ( $t[23] = 2.08$ ,  $P = 0.04$ , Cohen's  $d = 0.81$ ), but not the latter bias (Fig. 2). Post-hoc comparisons indicated that the processing of fearful faces was facilitated (i.e., decreased RTs) in anxious compared to non-anxious participants.

#### Frontal EEG Activation

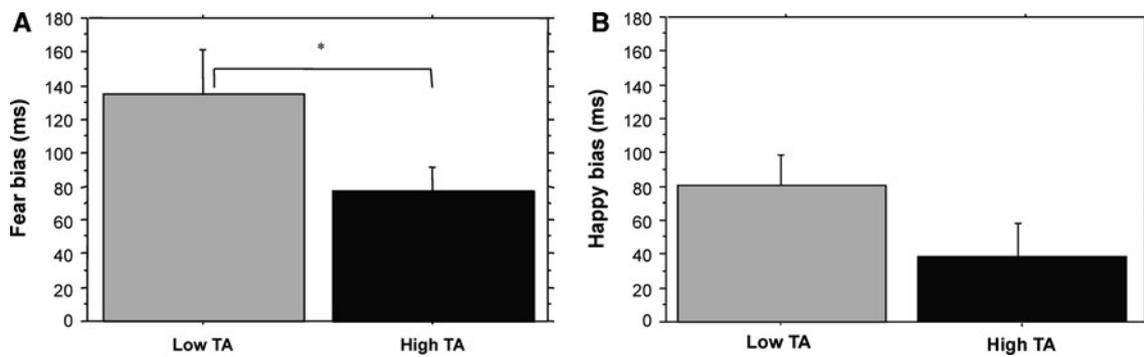
We used a  $2 \times 2 \times 3$  ANOVA to analyze the effects of anxiety (anxious vs. non-anxious), hemisphere (right vs. left), and type of stimulus (neutral vs. fearful vs. happy) on frontal EEG alpha power. The results indicated no significant main effects, but statistically significant interactions of hemisphere  $\times$  type of stimulus, and anxiety  $\times$  hemisphere  $\times$  type of stimulus. We isolated the sources of significance in follow-up ANOVA analyses. There was a significantly increased right compared to left frontal activation on trials with fearful faces ( $F[4, 20] = 5.95$ ,  $P = 0.05$ ,  $\eta^2 = 0.39$ ; Fig. 3a). In contrast, there was higher left compared to right frontal activation on trials with happy faces ( $F[4, 20] = 12.72$ ,  $P = 0.007$ ,  $\eta^2 = 0.18$ ).

#### Anxiety and Frontal EEG Activation

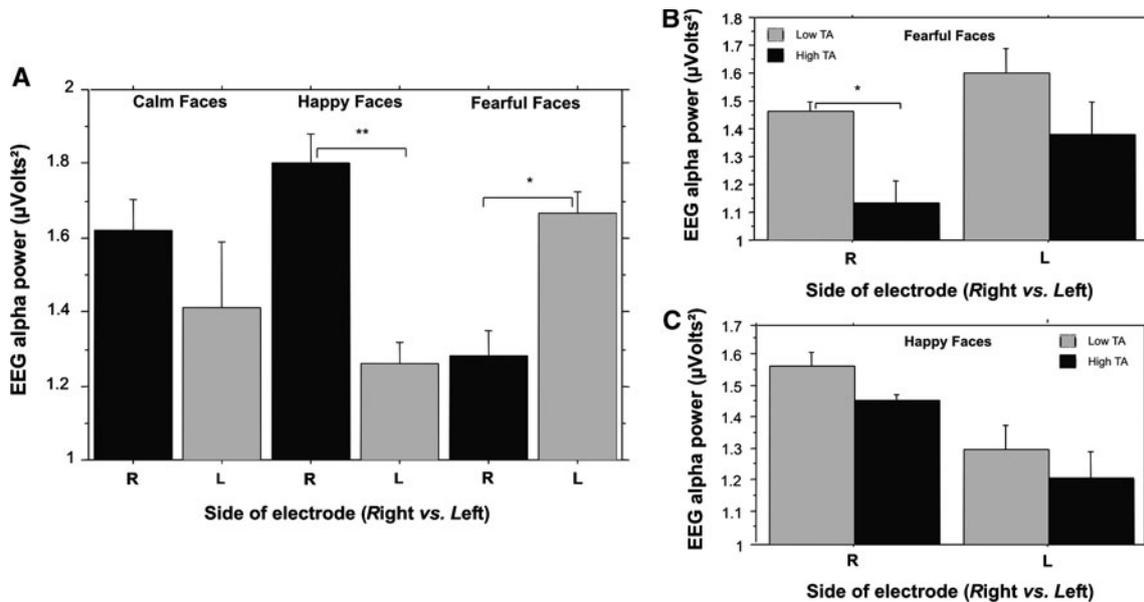
Anxiety had a significant effect on right frontal EEG activation on trials with fearful faces ( $F[4, 20] = 10.02$ ,  $P = 0.02$ ,  $\eta^2 = 0.13$ ; Fig. 3b). Anxious participants displayed higher right frontal activation on fearful trials, in comparison to non-anxious participants. This effect was specific to the right hemisphere and fearful trials (see Fig. 3b, c). Moreover, there was a significant correlation between the fearful face cognitive bias and right frontal EEG activation ( $r = 0.46$ ,  $P < 0.01$ ).

## Discussion

The present results supported our hypotheses that anxiety is associated with an affective style characterized by a bias toward facilitated processing of fearful faces, and increased right frontal activation to these stimuli. This study contributes to the psychophysiology of anxiety by showing for the first time that the asymmetry of frontal EEG activation reflects the type of cognitive bias that has been attributed a central role in cognitive theories of anxiety pathogenesis. In addition, this index of increased frontal cortical activation underlying the facilitated processing of aversive stimuli, may reflect the decreased cognitive efficiency that is characteristic of information processing in anxiety.



**Fig. 2** Comparisons of the aversive (fear—calm reaction times) (a) and appetitive (happy—calm reaction times) (b) cognitive biases in anxious and non-anxious participants. *Abbreviations:* TA, Trait anxiety (\* $P < 0.05$ )



**Fig. 3** Comparisons of the right and left frontal activations in calm, happy, and fearful face trials (a). The effects of anxiety on frontal activation asymmetry during trials with fearful (b) and happy faces (c) (\* $P < 0.05$ ; \*\* $P < 0.01$ )

Much of the literature on frontal EEG asymmetry is based on associations with self-report measures of emotion and motivation. Theories such as the diathesis-stress frontal activation asymmetry (Davidson 1998), and the “capability model” of frontal EEG asymmetry (Coan and Allen 2004; Coan et al. 2006) have emphasized that the effects of individual differences in frontal activity may develop particularly during controlled laboratory emotional challenges. For instance, an affective style characterized by relatively greater right frontal activity during a controlled fear-related challenge should display increased cognitive biases during that challenge. In the case of anxiety, these biases facilitate the processing of aversive stimuli either by facilitated attentional engagement with threat, or reduced attentional disengagement from threat (Mathews and MacLeod 2005; Miu and Visu-Petra 2010). Laboratory tasks such as the one we used in the present study are especially

sensitive to the first type of bias (Williams et al. 1996). Indeed, the present results offer direct evidence that participants selected for extremely high TA show a cognitive bias toward the processing of fearful faces. The increased skin conductance that we observed during the processing of emotional faces in comparison to neutral faces, supported our a priori expectation that emotional stimuli induced physiological arousal. This may be due to the increased socio-emotional saliency of faces in humans (Ashwin et al. 2006; Preston and Stansfield 2008; van Honk et al. 2000). Therefore, based on this emotional face analog of the Stroop task, the present study offer new evidence of facilitated engagement with threat in anxiety.

The processing of facial gestures of emotion also offers a good framework for studying the involvement of the frontal cortex in emotion and motivation. Previous studies have shown that the simulation of facial gestures of fear

and other withdrawal-related emotions in directed facial action tasks is associated with reduced left frontal EEG activation (Coan et al. 2001). More direct evidence for the involvement of the right frontal cortex in the processing of aversive information has come from studies in which the frontal cortex was temporarily inactivated by slow repetitive transcranial magnetic stimulation. The inactivation of the right frontal cortex biased attention toward the processing of angry faces in emotional analogs of the Stroop task (d'Alfonso et al. 2000; van Honk et al. 2002).

The present study used a different emotional face Stroop task that efficiently induces emotional conflict, and relies on a well-characterized neural circuit (Egner et al. 2008; Etkin et al. 2006). The generation and monitoring of emotional conflict in this task (i.e., naming the emotional face, while ignoring the incongruent name) involves the activation of the dorsal anterior cingulate cortex, and its successful resolution involves the specific activation of the rostral anterior cingulate cortex, as well as the amygdala (Egner et al. 2008). The present results indicate that the conflict in fearful face trials induced increased right frontal EEG activation, whereas that in happy face trials induced left frontal EEG activation. In addition, we show that anxiety specifically impacts the magnitude of right frontal EEG activation in trials with fearful faces. The right frontal EEG activation also correlated significantly with the fearful face cognitive bias. Overall, these results indicate that the right frontal activation asymmetry associated with the processing of fearful faces is modulated by anxiety, in a manner that quantitatively reflects the cognitive bias toward the selective engagement with fearful stimuli.

The attentional control theory argues that anxiety impairs goal-directed attention and increases the extent to which cognitive processing is influenced by stimulus-driven attention (Eysenck et al. 2007). This effect reduces cognitive efficiency (i.e., the relationship between the effectiveness of performance and the effort or resources spent in task performance, with efficiency decreasing as more resources are invested to attain a given performance level), but not necessarily cognitive effectiveness (i.e., quality of task performance). Two of the main candidate mechanisms underlying this effect are inhibition and shifting. In addition, anxiety also increases attention to threat-related stimuli. The task that we used in the present study is ideal for testing these hypotheses, for inhibition is necessary in order to name the emotion recognized on the face, while ignoring the incongruent name written on the face. If anxiety were to decrease goal-directed attention, then one would expect to find reduced cognitive efficiency and biased processing of threatening stimuli. Our results confirmed that anxiety biases attention toward the selective processing of fearful faces. In addition, we found that the right frontal EEG activation increases with this bias. This

pattern of results suggest that frontal EEG activation asymmetry reflects the influence of anxiety on (decreased) neural and cognitive efficiency.

This study integrated controlled laboratory measures of cognitive mechanisms that are involved in anxiety pathogenesis, and neurophysiological measures that are specifically related to emotion and motivation. Although we used only a global, self-report measure of anxiety, the observation that skin conductance increased during the processing of emotional, but not neutral stimuli indicated that fearful and happy faces succeeded to induce physiological arousal. However, one potential limit of this study is that due to our choice of the intervals of interest, it is impossible to say whether the variations of frontal EEG activation corresponded to the generation and monitoring, or resolution of the emotional conflict (see Egner et al. 2008). In addition to overcoming this limitation, future studies might focus on the modification of cognitive biases in anxiety (e.g., See et al. 2009), and its relationship with frontal EEG activation.

In conclusion, this study shows that anxiety is associated with facilitated attentional engagement with fearful faces in an emotional analog of the Stroop task. This cognitive bias is related to increased right frontal activation during the processing of fearful faces. The task used in this study might be particularly suitable for anxiety research, for its underlying neural circuits are well characterized, and the relationship with frontal EEG activation reported here may be relevant to the effects of anxiety on inhibition and cognitive efficiency.

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