

## Enhanced perceptual responses during visual processing of facial stimuli in young socially anxious individuals<sup>☆</sup>

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### HIGHLIGHTS

- ▶ We use an emotional oddball paradigm in young individuals reporting social anxiety.
- ▶ We examine P1, N170 and P3b to consider different stages of cognitive processing.
- ▶ Social anxiety individuals produce enhanced P1 in response to facial cues.
- ▶ N170, P3b and behavioural responses were not modulated by social anxiety.

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### ABSTRACT

The present study investigated whether social anxiety modulates the processing of facial expressions. Event-related potentials were recorded during an oddball task in young adults reporting high or low levels of social anxiety as evaluated by the Liebowitz Social Anxiety Scale. Repeated pictures of faces with a neutral expression were infrequently replaced by pictures of the same face displaying happiness, anger, fear or disgust. For all participants, response latencies were shorter in detecting faces expressing disgust and happiness as compared to fear or anger. Low social anxiety individuals evoked enhanced P1 in response to angry faces as compared to other stimuli while high socially anxious participants displayed enlarged P1 for all emotional stimuli as compared to neutral ones, and general higher amplitudes as compared to non-anxious individuals. Conversely, the face-specific N170 and the task-related decision P3b were not influenced by social anxiety. These results suggest increased pre-attentive detection of facial cues in socially anxious individuals and are discussed within the framework of recent models of anxiety.

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### 1. Introduction

Emotional facial expressions (EFE) are of particular relevance for the regulation of social behaviour. Behavioural and neuroimaging studies have provided a wealth of evidence that faces capture attention to be rapidly and efficiently processed [22]. Due to its high temporal resolution, event-related potentials (ERP) technique has been widely used to explore the temporal dimension of the processes involved in EFE perception in humans [22]. The influence of

the emotional facial features begins as soon as the P1 generated by extrastriate visual areas in a perceptual stage of information processing [6,22]. The hypothesis of an enhanced sensory processing of EFE associated with a representation of threat is sustained by congruent reports of an increased P1 wave in response to fearful faces [1]. The emotional charge could therefore serve as a guide in the early stages of information processing [22]. The emotional impact is less clear on the N170 component, a posterior occipitotemporal negative deflection linked to structural encoding of facial stimuli [6]. Larger N170 amplitudes have been reported in response to anger, disgust, and fear EFE as compared to neutral facial expressions [1,17]. However, some authors did not observe such emotional effects and postulated an independency of structural analysis and emotional expression analysis [6]. Finally, top-down manipulation of stimulus significance is also demonstrated on the P3b component [13], a positive parietal wave that arises when an infrequent stimulus requires a response. That component appeared

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enhanced and faster for fearful as compared to happy faces [17] which is thought to reflect emotional salience processing [13].

These three components are of particular interest in studying the modulation of emotional processing in psychopathological states disturbing the perception and production of emotions. Reading others' EFE is particularly challenging in social anxiety disorder (SAD) which is characterized by biased social perceptions and expectations, together with a tendency to detect negative social responses rather than positive ones [21]. Increased amygdala activations have been reported during EFE processing in SAD compared to non-anxious participants, for anger and fear but also for disgust and neutral faces [20,21]. However, electrophysiological studies exploring the ability to decode EFE in SAD remain rare and focused either on early or late components (for a review, see [20]). In this context, it is not surprising to find mixed evidences of higher amplitudes for the P1 [7–9,11,15], the N170 [9] and/or the P3 [11,18].

This lack of congruency about the timing of occurrence of biased emotional processes encompasses several questions. First, the specificity of the enhanced perceptual processing of threatening faces in SAD is still a matter of debate [20,21], since increased P1 amplitudes have been recorded specifically for negative EFE such as anger [11,18] but also for happy faces [7–9,12] and for both neutral and emotional faces [15]. Consequently, studies are needed to investigate the extent of biased emotional process by comparing neural responses to negative, positive, and neutral faces. Second, higher right occipitotemporal N170 amplitudes have been recorded in response to angry faces when SAD individuals had to identify EFE, with a positive correlation between N170 amplitude and self-reported measures of social anxiety [9]. However, other studies using similar tasks with schematic faces [7,8] or passive viewing of natural and artificial faces [12] did not reproduce this effect. Hence, it is crucial to further assess the role of social anxiety on configural processes of facial information, since SAD might be characterized by an abnormal visual scanning of human faces [20,21]. Third, SAD has been reported to potentiate P3b amplitude during threatening faces processing [11]. Moreover, a positive relation between the P3b peak voltage and the level of social anxiety for angry but not for happy faces suggests that social anxious individuals pay special attention to threat-related faces [18]. However, other studies did not report modulation of late positive ERP during EFE processing in SAD [14], and the current literature does not offer convincing evidence of P3b modulation in SAD [20].

In this context, the present study aimed to evaluate the influence of social anxiety on positive and negative EFE processing by examining the whole stream of information, ranging from P1 to P3b including the N170 component. To this end, we used an oddball paradigm [5,16,17], which is especially sensitive to early stages of face processing during which the discriminative cues are processed, and ERPs were recorded while participants watched photographs of neutral faces on a computer screen. They were instructed to signal changes in facial expression by pressing a key. In order to investigate the role of emotion on facial processing, deviant stimuli displayed different expressions: anger and fear, since these expressions have been shown to be particularly relevant in the studies listed above; happiness, to provide a positive control; and disgust, since some data suggest a particular role for that emotion in the aetiology and persistence of anxiety disorders and specially in SAD [14]. We expected in SAD an enhanced processing of negative emotions as compared to neutral and happy ones, observable through larger amplitudes of the P1 component as compared to non-anxious participants, as attended stimuli have shown to generate larger P1 due to attentional top-down processes [22]. However, as the social anxiety influence on the N170 may depend on the explicit nature of EFE processing [9], we did not expect N170 modulations by social anxiety in the present oddball task. Rather, emotional variations between emotions may be shown as in previous studies [1,17].

Finally, if social anxious participants voluntarily attend to threatening faces, an enhancement of the P3b component should appear [13], together with faster behavioural responses to negative stimuli.

## 2. Methods

Twenty-four right-handed participants were selected from a pool of 250 University of Louvain first-year students screened using the Liebowitz Social Anxiety Scale (LSAS) [10]. High social anxiety (HSA) individuals ( $N = 12$ ; 8 females) were defined as those scoring 65 or more on the LSAS (range 65–123) and the low-anxiety (LSA) individuals ( $N = 12$ ; 5 females; the ratio of male and female participants was not significantly different between groups,  $\chi^2 = 2.253$ ,  $p = .133$ ) were defined as those scoring 50 or below (range 12–48) on that scale [10]. After the experiment, participants also completed the State-Trait Anxiety Inventory (STAI) [19] and the 13-items Beck Depression Inventory (BDI) [3] in order to control for a possible comorbidity. LSA and HSA were significantly different in LSAS (LSA: 29.1,  $SD = 10.5$ ; HSA: 79.8,  $SD = 18.2$ ,  $t(22) = 8.662$ ,  $p < .001$ ). However, they did not differ on the basis of mean-trait anxiety (LSA, 55.6,  $SD = 2.6$ ; HSA: 53.9,  $SD = 2.0$ ,  $t(22) = 1.895$ , NS), state-anxiety (LSA, 64.3,  $SD = 3.3$ ; HSA: 65.5,  $SD = 2.6$ ,  $t(22) = .996$ , NS) or depression (LSA: 5.1,  $SD = 3.1$ ; HSA: 3.0,  $SD = 2.9$ ,  $t(22) = 1.751$ , NS). Age did not differ between groups (LSA: 19.9,  $SD = 1.6$ ; HSA: 19.7,  $SD = 1.5$ ,  $t(22) = .304$ , NS).

### 2.1. Stimuli and task

The stimuli set comprised 30 grey pictures of six individuals (three males) each posing neutrality, anger, disgust, happiness and fear [2]. After being trimmed to exclude non-facial contours and hair, each facial stimulus was enclosed within a rectangular frame measuring 4 cm  $\times$  6 cm. The experimental procedure used an 'emotional oddball paradigm' [5,17]: each block started with the presentation of a white cross (1 cm  $\times$  1 cm). Stimuli were then presented one by one for 500 ms on a black background, with a black screen displayed as an inter-trial interval lasting randomly between 800 and 1300 ms. Six blocks were composed, each defined by 108 stimuli (e.g. 84 frequent neutral faces and 24 deviant faces with the same identity but happy, angry, fearful and disgusted expressions). Each block was repeated twice. The order of the twelve blocks varied across participants. Participants were instructed to detect as quickly as possible the occurrence of a stimulus differing from the frequent one by pressing a mouse button with their right index finger. They sat in a chair in a dark room with their head placed 1 m from the screen and restrained in a chin rest.

### 2.2. ERP recording and data analysis

Electroencephalogram (EEG) was recorded (EEprobe software, A.N.T., The Netherlands) with 32 electrodes mounted in an electrode Quick-Cap with the standard 10–20 International System and intermediate positions. Recordings were made with a linked mastoid physical reference and re-referenced by using a common average. The EEG was amplified by battery-operated A.N.T.® amplifiers with a gain of 30,000 and a band-pass of 0.01–100 Hz and sampled at 512 Hz. The impedance of all electrodes was kept below 5 k $\Omega$ . The EEG was continuously recorded and the vertical electrooculogram (VEOG) was recorded from electrodes placed on the supraorbital and infraorbital ridges of the left eye. Trials contaminated by EOG artefacts were eliminated off-line by computing an average artefact response based on the percentage of the maximum eye movement potential. The EEG was band-pass filtered offline using cut-off frequencies of 0.16–30 Hz. Codes synchronized with stimulus delivery were used to average selectively the

epochs associated with the different categories of stimuli. A baseline correction was computed using a 100 ms interval and epochs beginning 100 ms prior to stimulus onset and continuing for 800 ms were created.

The overall averaged ERPs were examined to first, define temporal windows on interest electrodes kept constant for all conditions and participants. Second, an algorithm was used to identify the maximum positive or negative value within the specified time window on these interest electrodes, and that point was identified as the peak latency. Third, mean amplitudes were calculated for each defined window. Three ERPs usually described on the literature focusing of face processing in SAD [20] were selected for the analyses: The P1, the first positive deflection occurring on occipital sites between 120 and 150 ms after stimulus occurrence and measured on O1 and O2; the N170 recorded on lateral parietal sites between 150 and 220 ms after face presentation and measured on P7 and P8; and the P3b component, peaking on parietal sites and averaged on P3 and P4 between 450 and 500 ms after stimulus occurrence.

Statistical analyses were computed using the IBM® SPSS® Statistics Release 20.0.0. Response latencies, mean amplitudes and latencies of the ERPs were subjected to repeated measures analysis of variance (ANOVA) with Group (LSA and HSA) as the between-subjects factor, and Emotion (neutral, happiness, fear, anger and disgust) and Hemisphere of recording (Left, Right) as within-subject factors. The reported *p*-levels of all the other ANOVAs were corrected for violations of the sphericity assumption using the Greenhouse–Geisser epsilon correction. Simple effects were explored throughout, and a Bonferroni correction for multiple comparisons was applied to all the *t*-tests. For reasons of conciseness, only significant effects (level of significance at .05) are reported.

### 3. Results

#### 3.1. Behavioural data

Analyses on correct response latencies disclosed a main effect of Emotion,  $F(3,66)=85.941$ ,  $p<.001$ ,  $\eta^2=.790$ : Angry and fearful faces were detected slower than disgusted and happy faces (Bonferroni post hoc tests: all *p*-values  $p<.001$ ). Anger gave rise to the longest response latencies, and disgust to the fastest (comparison disgust–happiness:  $p=.024$ ).

#### 3.2. ERP results<sup>1</sup>

##### 3.2.1. P1 component at occipital locations (120–150 ms post-stimulus temporal windows)

Latencies: A significant effect of Hemisphere,  $F(1,22)=7.227$ ,  $p=.013$ ,  $\eta^2=.247$  indicated shorter latencies on the right electrode (O1: 151 ms; O2: 147 ms).

Amplitudes: First, a main effect of Group,  $F(1,22)=9.127$ ,  $p=.006$ ,  $\eta^2=.293$ , demonstrated larger P1 in HSA (6.044  $\mu$ V) as compared to LSA (3.752  $\mu$ V). Follow-up independent *t*-tests showed that HSA produced enhanced P1 in all conditions on both electrodes (all  $p<.05$ , except for Anger on O2,  $p=.094$ ). The enhancement of P1 amplitude with LSAS score was confirmed by a positive correlation ( $r=.423$ ,  $p=.04$ ).

Second, a main effect of Emotion was observed,  $F(4,88)=5.077$ ,  $p=.003$ ,  $\eta^2=.187$ : Angry and happy EFE evoked larger P1 amplitudes as compared to neutral faces ( $p>.01$ ). Conversely, there were

no significant differences between P1 amplitudes in response to neutral faces or deviant disgust ( $p=.265$ ) or fearful faces ( $p=.065$ ).

A main effect of Hemisphere demonstrated larger P1 on right electrode,  $F(1,22)=6.747$ ,  $p=.016$ ,  $\eta^2=.235$  (O1: 4.6  $\mu$ V; O2: 5.16  $\mu$ V).

Finally, a Group  $\times$  Emotion  $\times$  Hemisphere interaction,  $F(4,88)=2.919$ ,  $p=.026$ ,  $\eta^2=.117$ , was broken down further by examining LSA and HSA separately. Analysis showed that Hemisphere effect was significant in LSA ( $F(1,11)=6.742$ ,  $p=.025$ ) but not in HSA ( $F(1,10)=2.376$ ,  $p=.151$ ). Moreover, P1 amplitudes in LSA and HSA were differently modulated by Emotion. On the one hand, LSA produced enhanced P1 in response to angry faces exclusively ( $F(4,44)=3.440$ ,  $p=.028$ ; follow-up comparisons were significant when comparing anger to disgust,  $p=.04$ ; happiness,  $p=.001$  and fear,  $p=.017$ ). On the other hand, HSA produced enhanced responses for all deviant faces as compared to frequent neutral faces ( $F(4,44)=3.420$ ,  $p=.047$ ; comparison with angry,  $p=.08$ ; disgusted,  $p=.005$ ; happy,  $p<.001$ ; fearful:  $p=.015$ ) (Fig. 1 and Table 1).

##### 3.2.2. N170 at lateral-parietal locations (160–220 ms post-stimulus temporal windows)

Latencies: A main effect of Emotion,  $F(4,88)=3.337$ ,  $p=.020$ ,  $\eta^2=.132$  indicated early latency for neutral faces as compared to fearful ones ( $p=.009$ ).

Amplitudes: A main Emotion effect,  $F(4,88)=16.02$ ,  $p<.001$ ,  $\eta^2=.421$  indicated that disgusted and happy faces evoked the largest N170, while fearful, neutral and finally angry faces evoked smaller N170 amplitudes (paired comparisons were significant for anger–disgust and anger–happiness: both  $p<.000$ ; disgust–fear and disgust–neutrality: both  $p<.000$ ; neutrality–happiness:  $p=.002$ ; fear–happiness:  $p=.001$ ).

##### 3.2.3. P3b (450–500 ms post-stimulus temporal windows)

Latencies: Results showed a main effect of Emotion,  $F(6,66)=11.740$ ,  $p<.001$  with earlier P3b for disgust as compared to anger ( $p<.001$ ), and fear ( $p=.018$ ). Anger gave rise to later P3b responses, as compared to disgust, fear ( $p=.002$ ), and happiness ( $p=.05$ ).

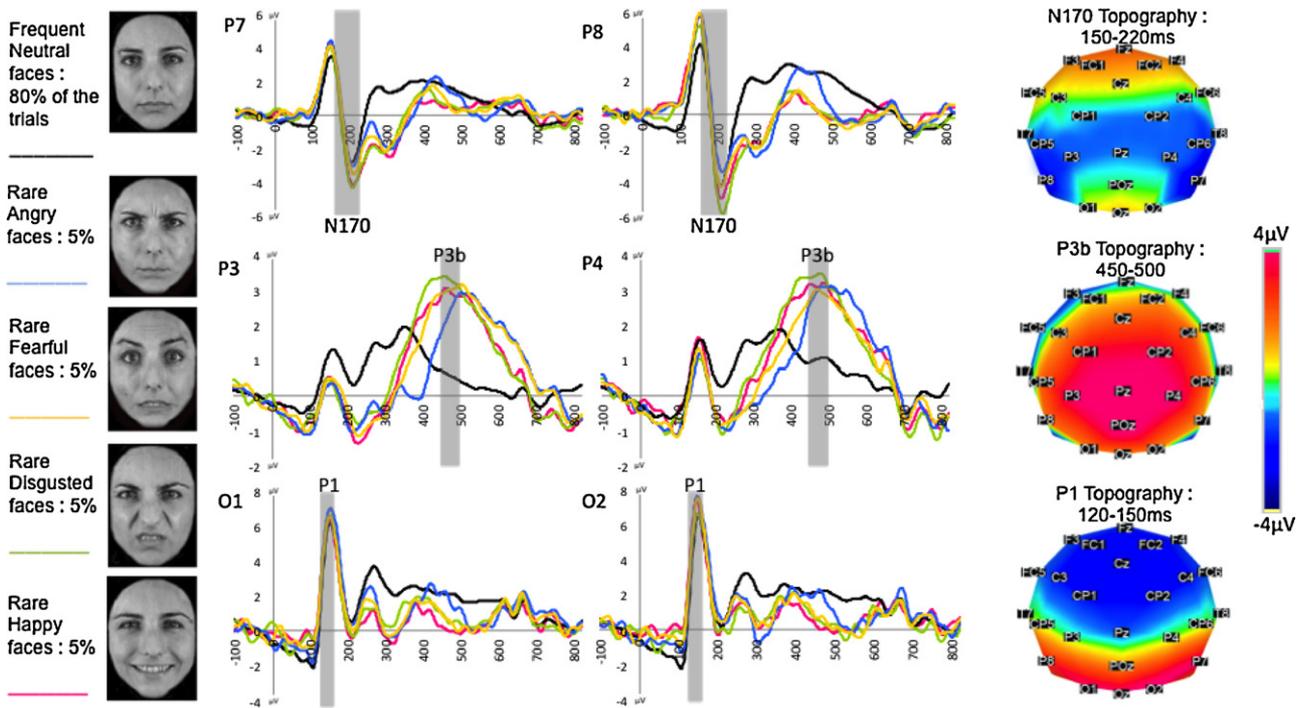
Amplitudes: A main effect of Hemisphere,  $F(1,22)=15.288$ ,  $p<.001$ , outlined higher P3b on the left electrode (P3: 4.04  $\mu$ V – P4: 3.97  $\mu$ V).

### 4. Discussion

The present study was designed to disentangle the early or late influence of social anxiety on the cognitive processing of different positive or negative facial expressions appearing amongst neutral faces. The major result is a positive relation between social anxiety level and P1 amplitude. Indeed, LSA participants showed an increased P1 specifically in response to EFE of anger, congruent with previous data [22]. In contrast, HSA displayed enhanced P1 responses to all facial changes as compared to neutral faces, but also a general P1 enhancement relative to non-anxious individuals.

The P1 enhancement in response to neutral faces in SAD allows exclusion of a selective improvement of facial change detection abilities. Rather, it suggests increased neural responses to faces regardless of their emotional expressions [15]. Indeed, neutral faces may have been perceived as more arousing in HSA, as sustained by the observation of amygdala activity for these stimuli [21]. An alternative explanation refers to a global hyper-vigilance in the visual cortex of anxious individuals, which enhances attention and the perceptual processing of incoming events [7]. Hence, further studies should be conducted to determine whether this enhancement is specific for faces or general to other visual stimuli.

<sup>1</sup> Since gender has been shown to influence emotional processing [6], we performed the same analyses by including the Gender as within-subjects variable. The main and interactional effects involving Gender were not significant, except for the P3b latency,  $F(1,20)=6.559$ ,  $p=.019$  which appeared shorter in women (425 vs. 459 ms).



**Fig. 1.** Illustration of the five categories of stimuli, grand mean baseline corrected ERP time courses at P7, P8, P3, P4, O1 and O2 for the different types of stimuli (colour legend appears on the left of stimuli and time windows are indicated by grey boxes), and mean ERP topographies during rare stimuli processing.

In contrast with this early perceptual effect, social anxiety did not influence the N170 or the P3b components. The N170 reflects the encoding of the structural characteristics of facial stimuli and appears as increased when an analytical treatment is induced by configural or spatial changes [6]. The lack of effect in connection with social anxiety level means that configural processing of human faces takes place normally in sub-clinical SAD [7,12]. However, one may postulate that EFE processing must be explicit to interact with social anxiety on facial structural encoding [9]. The comparison of implicit and explicit categorization tasks within a single study could address this question in further research.

Similarly, our results indicating a lack of social anxiety effect on P3b are congruent with previous reports [8,12,14], and the preservation of a response-related stage may be a property of emotional processing in sub-clinical SAD [20]. Nonetheless, a recent study by Sewell et al. and his collaborators [18] demonstrated a correlation between the score of social anxiety and the P3 amplitude in response to angry faces. However, these authors instructed the participants to selectively respond to angry or happy faces and focused their analyses on unattended stimuli. As the P3b is known to arise when an attended stimulus is detected [13], one may question whether the peaks measured in the present study are comparable to those measured in Sewell et al. [18].

Finally, depending on methodological criteria as well as on experimental paradigms, task instructions, exposure duration or intervals between stimuli, several behavioural and electrophysiological studies have been confronted with the lack of behavioural effect in SAD ([9,11], for a complete review, see [20]). In the present study, consistent with the absence of effect on P3b component, behavioural performances were not modulated by social anxiety. However, it is well known that ERPs are able to detect even minor neurocognitive restrictions that are undetectable at the behavioural level [13]. This is particularly important, as the absence of visible behavioural change is not incompatible with modifications of underlying cognitive processes. This study therefore supplied evidence that young socially anxious individuals

engage more early attentional resources than non-anxious participants when performing a visual task, as reflected by enhanced P1 component.

Interestingly, the influence of SAD on ERPs contrasts with those of high level of trait anxiety observed in previous studies. Trait anxiety is the general tendency to exhibit anxiety in different everyday-life situations [21]. Indeed, in two earlier studies using comparable oddball paradigms, high trait anxiety was not associated with disturbances in early perceptive ERPs but with a faster detection of deviant stimuli associated with faster P3b latencies, regardless of the emotional expression [16,17]. In the present study, HSA and LSA participants differed on social anxiety level but their self-reported trait-anxiety levels were comparable, which pleads for specific disturbances of emotional processing induced by social anxiety [20,21]. Conversely, both groups exhibited high grades of state anxiety, corresponding to their temporary level of worry [21]. As participants fulfilled the STAI after having completed the experiment, we can postulate that this experimental situation had enhanced their state anxiety despite normal levels of trait anxiety.

Concerning global emotional effects, we evidenced N170 sensitivity to the processing of the emotional load of faces, supporting previous observations [1,17]. Disgusted and happy faces gave rise to an enhanced N170, but that wave decreased for anger and fear. The same opposition was found in behavioural results. These results deviate from those reporting a faster detection of threat signals [22]. Since the participants were not asked to evaluate the stimuli's arousal value, an enhanced salience of happy and disgusted faces could have been responsible for their faster detection. However, such activation differences between the stimuli were controlled by the validation of that database [2]. Similarly, if the three negative emotions have led to similar delays as opposed to happy faces, we could have postulated the existence of a detection bias associated with the preponderance of negative events in our design, but that explanation does not seem to account here. However, the structure of the experience can provide a more reliable explanation to that result. Indeed, most studies reporting threat advantage contrasted

**Table 1**  
Mean amplitudes in  $\mu V$  (first line) and peak latencies in ms (second line) at the maximal point of temporal windows around the P1, N170 and P3 components, and Response Latencies in response to the different categories of facial displays (SD in parentheses).

Emotion	P100			N170			P300			Response latencies		
	LSA	HSA	Mean	LSA	HSA	Mean	LSA	HSA	Mean	LSA	HSA	Mean
Anger	4.8 (0.6) 147(3)	6.1 (0.6) 152(3)	5.3 (0.4) 150(2)	-0.8 (0.7) 209(3)	-1.3 (0.7) 207(3)	-1.0 (0.5) 208(2)	1.8 (0.2) 540(11)	2.1 (0.3) 489(13)	1.9 (0.2) 497(9)	535 (11)	550 (11)	542 (8)
Disgust	3.6 (0.6) 149(2)	6.1 (0.5) 147(2)	4.8 (0.4) 148(2)	-2.2 (0.8) 212(4)	-2.9 (0.8) 203(4)	-2.6 (0.6) 207(3)	2.0 (0.3) 437(13)	1.7 (0.3) 445(13)	1.8 (0.2) 441(9)	478 (12)	484 (12)	481 (9)
Fear	3.8 (0.6) 149(2)	6.2 (0.6) 149(2)	5.0 (0.4) 149(2)	-1.4 (0.9) 210(4)	-1.9 (0.9) 208(4)	-1.7 (0.6) 209(3)	1.9 (0.3) 482(11)	2.1 (0.3) 451(11)	2.0 (0.2) 466(8)	503 (11)	517 (11)	510 (6)
Happiness	3.7 (0.6) 147(3)	6.6 (0.6) 150(3)	5.1 (0.4) 149(2)	-2.2 (0.8) 207(4)	-2.5 (0.8) 204(4)	-2.4 (0.6) 205(3)	1.9 (0.3) 472(16)	1.9 (0.3) 447(16)	1.9 (0.2) 459(11)	484 (12)	494 (12)	489 (9)
Neutrality	3.2 (0.5) 150(3)	5.2 (0.5) 152(3)	4.2 (0.3) 151(2)	-1.4 (0.6) 203(3)	-1.7 (0.6) 203(3)	-1.5 (0.5) 203(2)						

positive and negative stimuli [17,22], but faster responses to disgust and happiness have already been reported in visual search tasks implying several types of EFE [4]. Authors have suggested that happy faces may be easier to recognize while EFE of anger and fear share several features and their recognition must be based on subtle elements that distinguish them [4]. Hence, in the present study, the more salient features of happiness and disgust may have facilitated their detection, while the similarities between fear and anger might elicit a longer categorization process.

To conclude, the present study is amongst the few using ERPs to explore SAD influence on cognitive processing of facial stimuli over time. We showed that social anxiety enhances early perceptual processing of neutral and emotional facial stimuli without modulation of late components or behavioural performance. First, these results highlight the necessity to explore early stages of cognitive processing to evidence SAD influence, as the utility of ERPs to provide sensitive information regarding these cognitive processes. Second, they have important implications for current definition of social anxiety as compared to trait anxiety. Finally, we show that neutral faces cannot be used as “neutral” control stimuli since they evoke enhanced neural response in SAD. That last result emphasizes the necessity for future studies to assess the specificity of enhanced processing of facial cues in social anxiety, which has not been questioned yet, by including non-facial stimuli.

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