# Electrophysiological Evidence of Attentional Biases in Social Anxiety Disorder

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## Abstract

**Background—**Previous studies investigating attentional biases in social anxiety disorder (SAD) have yielded mixed results. Recent event-related potential (ERP) studies employing the dot-probe paradigm in non-anxious participants have shown that the P1 component is sensitive to visuospatial attention toward emotional faces. We used a dot-probe task in conjunction with high-density ERPs and source localization to investigate attentional biases in SAD.

**Method—**Twelve SAD and 15 control participants performed a modified dot-probe task using angry-neutral and happy-neutral face-pairs. The P1 component elicited by face-pairs was analyzed to test the hypothesis that SAD participants would display early hypervigilance to threat-related cues. The P1 component to probes replacing angry, happy or neutral faces was used to evaluate whether SAD participants show sustained hypervigilance or rather decreased visual processing of threat-related cues at later processing stages.

**Results—**Compared to controls, SAD participants showed relatively (a) potentiated P1 amplitudes and fusiform gyrus activation to angry-neutral vs. happy-neutral face-pairs; (b) decreased P1 amplitudes to probes replacing emotional (angry and happy) vs. neutral faces; and (c) higher sensitivity ($d'$) to probes following angry-neutral vs. happy-neutral face-pairs. SAD participants also showed significantly shorter reaction times to probes replacing angry vs. happy faces, but no group differences emerged for reaction time.

**Conclusions—**The results provide electrophysiological support for early hypervigilance to angry faces in SAD with involvement of the fusiform gyrus, and reduced visual processing of emotionally salient locations at later stages of information processing, which might be a manifestation of attentional avoidance.

## Keywords

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## Introduction

Information processing studies suggest that attentional biases are related to anxiety (Williams *et al.* 1988; Eysenck, 1992), including social anxiety (Heinrichs & Hofmann, 2001). Some of these findings have been integrated into cognitive treatment models to explain the emergence and maintenance of social anxiety disorder (SAD) (Clark & Wells, 1995; Rapee & Heimberg, 1997). Specifically, it is assumed that socially anxious individuals overly attend to threatening, socially relevant cues, including angry faces. Such hypervigilance might lead to increased threat detection, likely exacerbating anxiety, and increased vulnerability to negative emotions (Eysenck, 1992; MacLeod *et al.* 2002; Mathews & MacLeod, 2002).

A widely used method to assess attentional biases is the dot-probe task (MacLeod *et al.* 1986), in which a neutral and a threat-related cue are presented simultaneously at different locations of a screen. After a brief delay, a probe replaces one of these cues, and the participant is instructed to press a button to indicate the detection of the probe. Decreased reaction times (RTs) to probes replacing threat-related relative to neutral cues suggest increased allocation of visual attention toward threat-related cues (MacLeod *et al.* 1986; cf. Fox *et al.* 2002).

Several dot-probe studies support the hypothesis of hypervigilance in SAD, especially if stimuli are briefly presented (≤500 ms) and if participants have no comorbid diagnosis of depression (Mogg & Bradley, 2002; Musa *et al.* 2003; Mogg *et al.* 2004; Vassilopoulos, 2005; Sposari & Rapee, 2007). Some studies using the dot-probe task and related paradigms, however, suggest that socially anxious individuals might avoid emotional stimuli (e.g., angry and happy faces) (Mansell *et al.* 1999; Horley *et al.* 2003; Vassilopoulos, 2005; Heuer *et al.* 2007).

Some of the conflicting results may be reconciled by the hypervigilance-avoidance hypothesis (Mogg *et al.* 1987; Williams *et al.* 1988; Amir *et al.* 1998), which assumes that SAD is characterized by early automatic hypervigilance followed by strategic avoidance of threat. Support for this hypothesis derives from studies using eye-tracking (Garner *et al.* 2006), homographs (Amir *et al.* 1998), and the dot-probe paradigm with varying stimulus-onset asynchronies (Vassilopoulos, 2005). However, other investigations have failed to find clear evidence for a vigilant-avoidant attentional pattern in SAD (Mogg *et al.* 2004). In addition to the mixed results regarding the direction of the bias (hypervigilance vs. avoidance), studies have found attentional biases to threat stimuli only (Mogg & Bradley, 2002; Mogg *et al.* 2004), to both angry and happy faces (Mansell *et al.* 1999; Heuer *et al.* 2007; Sposari & Rapee, 2007), or no biases toward external sources of threat (Pineles & Mineka, 2005).

A possible reason for these inconsistent findings is that behavioral measures provide an indirect measure of attentional processing (Horley *et al.* 2004), and can be confounded by post-perceptual processes (e.g. decision-making, motor responses) (Handy *et al.* 2001). Measurements of brain electrical activity through event-related potentials (ERPs) offer the possibility to investigate attentional processes more directly and thus circumvent some of the limitations of behavioral studies. Of note, recent studies using the dot-probe paradigm in healthy adults have shown that the P1 component to emotionally cued probes provides a sensitive measure to assess rapid spatial orienting towards threat-related stimuli. Specifically, Pourtois *et al.* (2004) and Santesso *et al.* (2008) reported increased P1 amplitudes to probes replacing fearful or angry faces as opposed to neutral faces. These findings are consistent with independent evidence indicating that P1 amplitudes are larger for stimuli presented at attended compared to unattended locations (Clark & Hillyard, 1996;

Hillyard & Anllo-Vento, 1998; Di Russo *et al.* 2003). In addition to P1 enhancements for probes cued by threat-related faces, negatively valenced emotional stimuli may also directly evoke increased P1 amplitudes compared to neutral stimuli (Streit *et al.* 2003; Klucharev & Sams, 2004; Pourtois *et al.* 2005). Importantly, these findings have also been linked to increased attention for threat during initial stages of processing (Vuilleumier & Pourtois, 2007).

The goal of the present study was to investigate attentional biases toward socially relevant cues and underlying brain mechanisms in a sample of SAD patients and matched healthy controls using the paradigm we recently developed in an undergraduate sample (Santesso *et al.* 2008). Based on behavioral findings reviewed above, we hypothesized that SAD participants would show initial hypervigilance toward threat-related cues, as manifested by potentiated P1 amplitudes to angry-neutral face-pairs compared to happy-neutral face-pairs. If such hypervigilance persists over time, we predicted that SAD participants would show significantly *increased* P1 amplitudes to probes replacing an angry face, as demonstrated in healthy participants (Santesso *et al.* 2008). Conversely, if SAD participants are characterized by attentional biases away from emotional faces (Mansell *et al.* 1999; Horley *et al.* 2003; Heuer *et al.* 2007) at later stages of the information processing flow, we predicted that they would show *decreased* P1 amplitudes to emotionally cued probes. We further examined whether these effects are restricted to probes replacing angry-neutral face-pairs (valence effect), or whether they generalize also to happy-neutral face-pairs (emotionality effect) (Martin *et al.* 1991).

To investigate the specificity of putative P1 findings, exploratory analyses focused on additional ERP components, including the C1, N170, and N1 components. The C1 originates from the primary visual cortex (DiRusso *et al.* 2003) and is typically unaffected by attention (Clark & Hillyard 1996), although threat-related stimuli have been found to modulate this component (Stolarova *et al.* 2006). The face-specific N170 (Bentin *et al.* 1996) was also analyzed, although it is still uncertain whether this component is affected by emotional facial expressions (Pizzagalli *et al.* 2002) or not (Eimer & Holmes, 2007). Finally, N1 amplitudes following probe presentation were evaluated in light of evidence that this component might be attenuated by exogenously cued probes (Fu *et al.* 2005).

As a behavioral measure we assessed RTs in response to the probe. If individuals with SAD are hypervigilant to threat (Bar-Haim *et al.* 2007), we predicted that they would react faster to probes replacing angry faces as opposed to probes replacing neutral faces. Conversely, if socially anxious individuals avoid angry and/or happy faces (Mansell *et al.* 1999), they should have longer RTs for probes preceded by emotional as opposed to neutral faces. Finally, using a signal-detection approach, we analyzed $d'$ to evaluate whether groups differed in their sensitivity toward probes cued by emotional faces.

## Methods and Materials

### Participants

Sixteen SAD and 18 control participants were recruited through an outpatient anxiety disorders clinic and advertisements, respectively. SAD was diagnosed using the Anxiety Disorders Interview Schedule for DSM-IV: Lifetime Version (ADIS-IV-L) (DiNardo *et al.* 1994). Control participants were screened with an abbreviated version of the ADIS-IV to confirm absence of psychopathology. All participants were right handed (Chapman & Chapman, 1987) and had no history of bipolar disorder, schizophrenia, psychosis, or delusional disorders. Additional exclusion criteria included a current diagnosis of posttraumatic stress disorder, major depression with severity greater than mild to moderate (as indicated by the presence of 4 or greater on the ADIS clinical severity rating), or current

active suicidal ideation. Participants reported no psychoactive substance abuse, no unstable medical illness, and no past or current neurological illness. The study was approved by the human subjects committees of Harvard University and Boston University. All participants gave informed written consent.

Seven participants (4 SAD and 3 controls) were excluded due to excessive artifacts in the ERP data, leading to a final sample of 15 control and 12 SAD participants. Comorbid diagnoses in the SAD group included generalized anxiety disorder (n=8), major depressive disorder (n=7), specific phobia (n=5), and obsessive compulsive disorder (n=3). Three SAD participants were receiving psychotropic medication at the time of the study (Paroxetine, Venlafaxine or Setraline). All assessments were done prior to the patient's receiving cognitive-behavioral therapy at the Center for Anxiety and Related Disorders at Boston University. Control participants reported birth control (n=2), asthma (n=2) and diabetes (n=1) medications. As shown in Table 1, the groups did not differ in sociodemographic characteristics. Relative to controls, participants in the SAD group reported higher levels of social anxiety, trait anxiety, and depression, as assessed by the Social Interaction Anxiety Scale (SIAS) (Mattick & Clarke, 1998), State-Trait Anxiety Inventory (STAI) (Spielberger & Gorsuch, 1983), and Beck Depression Inventory-II (BDI) (Beck *et al.* 1996), respectively.

## Dot-probe task

The task was a modified dot-probe task adapted from Pourtois and colleagues (Pourtois *et al.* 2004), and described in more detail in a recent independent study from our laboratory (Santesso *et al.* 2008). Participants had to maintain fixation on a centrally presented cross. A pair of face stimuli was presented for 100 ms (one face in the upper left and one face in the upper right visual field). Each pair consisted of one neutral and one emotional (either angry or happy) face taken from the Ekman series (Ekman & Friesen, 1976). Next, a black screen with the fixation cross was presented for a varying period of time (100–300 ms). Subsequently, a vertical or horizontal bar (the probe) was presented at either the location of the emotional face ("emotionally cued trial") or neutral face ("neutrally cued trial"), and one line of the fixation cross was thicker than the other. Participants were instructed to press a button whenever the thicker line of the fixation cross matched the orientation of the probe (go trial) and withhold a response otherwise (no-go trial). Trials were separated by intertrial-intervals of 1,250 ms, in which a black screen without a fixation cross was presented.

Participants first performed one practice block of 16 trials, followed by 9 blocks of 80 trials, which were separated by small breaks. Each block contained 24 go trials (30%), and 56 no-go trials (70%). The rationale for using the go/no-go paradigm with this particular trial ratio was to gather enough behavioral responses (derived from go trials) to allow reliable behavioral (RT and d′) analyses, while preserving a sufficient number of no-go trials for the ERP analyses (as elaborated below, only no-go trials were used to avoid movement-related artifacts). RT was recorded from probe onset. Trials with RTs that were <100 ms and >1,500 ms and incorrect responses were excluded from the analyses. Based on signal detection theory, sensitivity toward probes cued by emotional vs. neutral faces was calculated using the formula $d' = z(FA) - z(HR)$, where FA and HR are the false alarm and hit rates, respectively (Green & Swets, 1966).

## EEG recording and data reduction

The EEG was recorded using a 128-channel Electrical Geodesics system (EGI Inc, Eugene, OR) at 500 Hz with 0.1–200 Hz analog filtering referenced to the vertex. Impedance of all channels was kept below 50 kΩ. Data were segmented and re-referenced off-line to an average reference, yielding 129-channel EEG data. EEG epochs were extracted beginning 100 ms before and ending 350 ms after stimulus presentation. Data were processed using

Brain Vision Analyzer (Brain Products GmbH, Germany). Each trial was visually inspected for movement artifact and then automatically removed with a ±75 μV criterion. Eye-movement artifacts were corrected by Independent Component Analysis. To avoid movement-related artifacts, only no-go trials were used to compute ERPs. ERP amplitudes were derived from each individual's average waveform filtered at 0.1–30 Hz. For further details see Santesso *et al.* (2008).

Primary ERP analyses focused on the P1 elicited by the face-pairs (P1-Face) and the probe (P1-Probe), which were measured as the most positive peak in the time window of 80–150 ms following face or probe onset, respectively. In line with Pourtois et al. (2004), P1-Face and P1-Probe were measured at PO7 and PO8 (corresponding to channels 66 and 85 on the EGI net, Luu & Ferree, 2000). To test the specificity of P1 findings, exploratory analyses were performed on the peak amplitudes of the C1, N170, and N1 components. The C1-Face and C1-Probe were measured 50–80 ms after stimulus presentation at POZ (channel 68), N170 from 130–210 ms after face presentation at P7 and P8 (channels 59 and 92) and N1 from 150–210 ms after probe presentation at PO7 and PO8. For all analyses, a pre-stimulus baseline (−100 to 0 ms) was used.

## LORETA whole brain analyses

In case of significant scalp P1 findings, Low Resolution Electromagnetic Tomography (LORETA; Pascual-Marqui *et al.* 1994; Pascual-Marqui *et al.* 1999) was used to estimate intracerebral current density underlying such effects using information from all 129 channels. Validation for this source localization technique has been derived from studies combining LORETA with fMRI (Vitacco *et al.* 2002; Mulert *et al.* 2004), PET (Pizzagalli *et al.* 2004), and intracranial recordings (Zumsteg *et al.* 2005). [1] LORETA analyses reported in the current study closely mirror procedures previously described in detail (e.g., Pizzagalli *et al.* 2002, 2003, 2004).

At each voxel (N=2,394) current density (scaled to amperes per square meter, $A/m^2$)was computed as the linear, weighted sum of the scalp electric potentials during windows of ±20 ms around the global field power (GFP) peaks.[2] The GFP peaks for P1-Face (120 ms post-stimulus) and P1-Probe (96 ms post-stimulus) were similar to the latencies of the scalp P1 peaks (122 ms and 102 ms respectively). For each subject, LORETA values were normalized to a total power of 1 and then log transformed before statistical analyses.

## Statistical analyses

For RT and *d′*, $2 \times 2 \times 2 \times 2$ analyses of variance (ANOVAs) were performed with Group (SAD vs. control participants) as a between subjects factor and Visual Field of the emotional face (left vs. right), Emotion (angry vs. happy), and Probe-position relative to the cue (emotionally vs. neutrally cued) as within-subjects factors. For analyses of the ERP data, the factor Electrode-Position (left vs. right hemisphere) was added. Amplitudes of probe-locked ERPs were thus analyzed with a Group × Visual Field × Emotion × Probe-Position × Hemisphere ANOVA. Face-locked ERPs were analyzed with a Group × Visual Field × Emotion × Hemisphere ANOVA because the factor Probe-Position was not present. Significant ANOVA effects were further explored by post-hoc *t*-tests with Bonferroni adjusted alpha levels (α′=α/number of tests). In this report, only effects involving the factor Group are described (a full summary of the effects is available upon request).

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[1]For a critical discussion of this approach, including its limitation, the interested reader is referred to Hämäläinen (1995) and Pascual-Marqui (1995).
[2]The GFP-peak is assumed to index time points of maximal neuronal activity, and thus offers optimal signal-to-noise ratio (Lehmann & Skrandies, 1984).

For LORETA data, voxel-wise *t*-tests (two-tailed) were performed to compare current density between groups or conditions. To minimize Type I errors, only activation clusters of more than 5 voxels exceeding $p<0.01$ were considered significant.

## Results

### Reaction Time

The Emotion × Probe-Position interaction was significant [$F(1,25)=13.70$, $p<0.002$, partial $\eta^2=0.35$] (Fig. 1a). Follow up *t*-tests indicated that probes replacing angry faces were detected faster than probes replacing happy faces [$t(25)=3.83$, $p<0.001$] or neutral faces [$t(25)=2.42$, $p<0.025$], although only the first effect was significant after the Bonferroni correction ($\alpha'=0.0125$, based on four planned t-tests). A trend for longer RTs in response to probes replacing happy compared to neutral faces also emerged [$t(25)=2.12$, $p<0.045$]. There were no significant between-group effects.[3]

In light of prior findings indicating that anxious individuals attend preferentially to threat-related relative to neutral stimuli ("within subject bias") despite lack of differences between anxious and non-anxious subjects ("between-subject bias") (Bar-Haim *et al.* 2007), separate ANOVAs were conducted for each group. The Emotion × Probe-Position interaction was significant in the SAD group [$F(1,11)=32.0$, $p<0.0001$, partial $\eta^2=0.74$] but not control group [$F(1,14)=1.9$ $p=0.19$, partial $\eta^2=.12$]. As hypothesized, follow up *t*-tests in the SAD group revealed faster reactions to probes replacing angry faces versus happy faces [$t(11)=5.21$, $p<0.0002$].[4] Moreover a trend indicating hypervigilance to angry vs. neutral faces emerged [$t(11)=2.09$, $p<0.060$]. Finally, following happy-neutral face-pairs SAD participants reacted faster to probes replacing neutral vs. happy faces [$t(11)=3.30$, $p<0.008$]. For controls, none of these t-tests reached significance (all $ps>0.15$).

### Signal detection data (d′)

The Emotion × Group interaction was significant [$F(1,25)=5.35$, $p<0.030$; partial $\eta^2=0.18$] due to higher *d′* values for SAD participants after the presentation of an angry-neutral relative to happy-neutral face pair [$t(11)=0.41$, $p<0.042$] (Fig. 1b). No other effects emerged.

### Electrophysiological measures

**P1-Face**—P1 amplitudes were greater over the right hemisphere [$F(1,25)=9.91$, $p<0.005$, partial $\eta^2=.28$]. Importantly, the Emotion × Group interaction effect was significant [$F(1,25)=5.53$, $p<0.028$, partial $\eta^2=0.18$], indicating that groups differed in their relative P1 responses to angry vs. happy faces. Within-group analyses further revealed that participants with SAD had larger P1 amplitudes for angry-neutral as opposed to happy-neutral face-pairs, $t(11)=3.58$, $p<0.005$ ($\alpha'=0.0125$). No significant differences emerged for control participants or between the groups ($p$'s>0.1) (Fig. 2a,b).

**P1-Probe**—Similar to P1 evoked by the face-pairs, amplitudes were higher over the right hemisphere [$F(1,25)=6.31$, $p<0.020$, partial $\eta^2=0.20$]. Of particular interest, the Probe-Position × Group interaction effect was significant [$F(1,25)=11.39$, $p<0.003$, partial $\eta^2=0.31$], indicating that groups significantly differed in their relative responses to probes replacing emotional vs. neutral faces. Follow-up tests indicated that SAD participants

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[3]Based on studies reporting influences of concurrent depression on attentional biases in SAD (Musa *et al.* 2003), statistical tests for all behavioral and physiological variables were repeated as ANCOVAs with BDI-score as a covariate. With the exception of control analyses for the d′ measure, in which the Group × Emotion interaction became a trend (F(1,25)=3.24, p=0.084), all findings were confirmed (all *p*'s<0.05) and no effects involving BDI emerged.

[4]Note that these analyses are mathematically equivalent to testing whether bias scores (i.e. RT difference scores) are significantly different from zero within each group.

generated smaller P1 amplitudes for emotionally than neutrally cued trials [$t(11)$=3.42, $p$<0.007], whereas controls showed an opposite trend [$t(14)$=2.44, $p$<0.035] (Fig. 3). This effect was independent of the emotional expression [Probe-Position × Emotion × Group: $F(1,25)$<1.00, $p$>0.5, partial $\eta^2$=0.01]. No other significant main effects or interaction effects emerged.

**Control analyses**—To confirm that these results were not restricted to the electrode sites used, we repeated the ANOVAs with a cluster of surrounding electrodes (channels 59, 60, 66, 85, 86, 92 on the EGI net) and added Electrode as an additional factor. Both the Validity × Group interaction for the P1-Face [$F(1,25)$=8.36, $p$<0.037, partial $\eta^2$=0.16] and the Emotion × Group interaction for P1-Probe remained significant [$F(1,25)$=10.41, $p$<0.005, partial $\eta^2$= 0.28].

**Exploratory Analyses**—Relative to controls, SAD participants had smaller C1-Face [$F(1,25)$=7.73, $p$<0.010, partial $\eta^2$=0.24] and N170 [$F(1,25)$=6.11, $p$<0.021, partial $\eta^2$=0.20] amplitudes. For the C1 and N170 amplitudes, no further effects emerged. For the N1 component, significant Probe-Position × Group [$F(1, 25)$=11.23, $p$<0.004, partial $\eta^2$=0.31] and Hemisphere × Emotion × Group [F(1,25)=5.13, p=.032, partial $\eta^2$ = .17] interactions emerged. Follow-up tests revealed that the first effect was driven by decreased N1 amplitudes for emotionally versus neutrally cued trials in control participants [$t(14)$=2.90, $p$=0.012]. The Hemisphere × Emotion × Group interaction was due to larger N1 amplitudes to probes following happy-neutral face-pairs over the right hemisphere for control versus SAD participants.

## Source Localization

**P1-Face**—To localize the generator of the significant scalp amplitude differences between angry-neutral and happy-neutral face-pairs in SAD participants, current density following angry-neutral and happy-neutral face-pairs was compared within the SAD group. SAD participants showed higher activation after angry-neutral vs. happy-neutral face-pairs in a cluster around the right middle temporal gyrus, including the fusiform gyrus (FG) (BA 37) and the inferior temporal gyrus (BA 20/21/37) (Fig. 2c and Table 2a). No other regions were identified.

To explore whether this region was also more activated in SAD relative to control participants, current density to angry-neutral face-pairs was compared between groups. Independent voxel-wise t-tests revealed that following angry-neutral face-pairs SAD participants displayed significantly higher activation than control participants in the right FG (BA 20/21/37) (Fig. 2c and Table 2b). No other regions exceeded the statistical threshold.

**P1-Probe**—LORETA analyses evaluating potential neural generators underlying the scalp finding of smaller P1 amplitudes for emotionally than neutrally cued trials in SAD participants revealed no significant findings.

## Discussion

The goal of the present study was to investigate attentional biases in SAD during a dot-probe task using ERP and source localization techniques. Several findings relevant to the initial hypotheses emerged. First, SAD, but not control, participants showed increased P1 amplitudes and FG activation to angry-neutral vs. happy-neutral face-pairs, and a reliable Emotion × Group interaction indicated that SAD participants had a significantly larger P1 potentiation to angry faces relative to happy faces compared to control participants. Second, SAD participants had smaller P1 amplitudes to probes replacing emotional rather than

neutral faces, whereas control participants showed an opposite pattern. A significant Probe-Position × Group interaction indicated that SAD participants had significantly reduced P1 responses to probes replacing emotional vs. neutral faces compared to control participants. Third, SAD participants reacted faster to probes replacing angry vs. happy faces, although no group differences emerged from the RT data. Finally, SAD, but not control, participants showed higher sensitivity ($d'$ values) in response to probes following the presentation of angry versus happy faces.

Previous studies have shown that the P1 component is amplified in response to negatively valenced facial expressions (Streit *et al.* 2003; Klucharev & Sams, 2004; Pourtois *et al.* 2005) and that increased P1 to threat-related cues is larger for high compared to low trait anxious individuals (Li *et al.* 2008; see also Kolassa & Miltner, 2006). Similar to P1 enhancements due to heightened attention in studies with non-emotional stimuli (Hillyard & Anllo-Vento, 1998), P1 enhancements to threat-stimuli were found to originate from extrastriate generators (e.g. FG) (Pourtois *et al.* 2005) and have therefore been assumed to indicate increased attention to threat (Vuilleumier & Pourtois, 2007). In SAD participants, the finding of enhanced P1 amplitudes when an angry face was present might thus indicate initial hypervigilance to threat and mirrors (a) the reaction time data suggesting shorter reaction times to probes replacing angry than happy faces and (b) the increased visual sensitivity following angry versus happy faces. Analyses of $d'$ values indeed revealed that SAD participants were characterized by an increased visual sensitivity in both visual fields after the presentation of an angry face. Moreover, reaction times were shortened at locations cued by an angry face. Interestingly, SAD participants also reacted faster to probes preceded by neutral versus happy faces. When seen within the framework of prior findings indicating that SAD participants show increased activation relative to controls in anxiety-related brain regions in response to both neutral (Cooney *et al.* 2006) and angry (Straube *et al.* 2004) faces, the present ERP and behavioral findings converge in suggesting that in SAD, attention is initially oriented toward the relatively more threatening cue in the environment. These results are consistent with the cognitive model of SAD (Clark & Wells, 1995; Rapee & Heimberg, 1997; Hofmann, 2007).

In the present study, source localization analyses indicated that potentiated P1 responses to angry vs. happy face-pairs were associated with hyperactivation in the posterior FG. FG activation within the P1 time range has been reported in healthy controls in response to aversive stimuli (Pizzagalli *et al.* 2003; Streit *et al.* 2003). Moreover, P1 amplitudes have been associated with changes in posterior FG activation measured with positron emission tomography (Mangun *et al.* 1998). Importantly, the FG receives direct projections from the amygdala (Amaral *et al.* 1992), which has been found to (a) respond to facial stimuli as early as 120 ms after presentation (Halgren *et al.* 1994); (b) be sensitive to threat-related cues (Buchel & Dolan, 2000); and (c) be implicated in the pathophysiology of SAD (Etkin & Wager, 2007). Based on the convergence of these findings, we speculate that the P1 finding of hypervigilance to angry faces might be linked to increased amygdalar activation in SAD.

Extending prior fMRI findings highlighting FG hyperactivation in SAD (Etkin & Wager, 2007), the present results provide important insight into the temporal dynamics of brain mechanisms associated with early attentional biases in SAD. Specifically, we showed that functional abnormalities within the visual cortex unfold as early as 100 ms after stimulus presentation. ERP techniques cannot be used, however, to ascertain whether this potentiated activation reflects top-down influences from the fronto-parietal network or direct influences from the amygdala (Vuilleumier & Pourtois, 2007). Consequently, future studies that combine ERP and hemodynamic measurements in SAD should further investigate this important issue.

In contrast to the face-evoked P1 findings, this study also found evidence that individuals with SAD might show at later stages of the information processing flow reduced visual processing at emotionally cued locations. In SAD participants, probes replacing angry and happy faces in fact elicited smaller P1 amplitudes than probes replacing neutral faces. Control participants showed the opposite pattern. [5] Similarly, Santesso *et al.* (2008) showed that non-anxious adults exhibited larger P1s to emotionally vs. neutrally cued probes, but only following angry faces.

At least two interpretations for the P1-Probe effect in SAD can be advanced. First, it is possible that visual processing of the probes was disrupted by continuing processing of preceding emotional faces (Rossignol *et al.* 2007), resulting in smaller P1 amplitudes. Although plausible, this interpretation cannot explain the finding of *increased* amplitudes to emotionally cued probes in control participants. An alternative explanation is that SAD participants either attended the more ambiguous stimulus present in the visual field (i.e., the neutral face) (Cooney *et al.* 2006) or showed attentional avoidance away from emotional faces (Mansell *et al.* 1999) at later stages of the information processing flow. If the latter is true, it remains to be tested whether attentional avoidance in SAD participants might occur automatically or might be controlled by strategic influences (Amir *et al.* 1998). Regardless of the mechanisms, it is interesting to note that SAD participants showed significantly reduced overall face-locked C1 and N170 amplitudes relative to controls. This finding is consistent with the hypotheses that certain aspects of face processing might be avoided (Chen *et al.* 2002) or disrupted (e.g. Horley *et al.* 2004) in SAD (we note, however, that in contrast to P1, the C1 and N170 group differences were not modulated by emotions).

Similar to our previous study (Santesso *et al.* 2008), LORETA analyses of probe-evoked ERPs did not reveal differential activation in brain regions typically associated with attention-related P1-effects (i.e., extrastriate visual areas) (Mangun *et al.* 1998). A possible explanation is that differences between neutrally and emotionally cued probes emerging at the scalp level were not strong enough to reach statistical significance in the LORETA analyses, which used a higher statistical threshold and might be affected by additional sources of variance (e.g., assumption of a spherical head, issues with the inverse problem). However, it should be emphasized that the P1 effect to the probe was right-lateralized, replicating prior findings (Pourtois *et al.* 2004). Similarly, the P1 to the face was also more pronounced in the right hemisphere, in line with a substantial literature emphasizing right hemisphere dominance for face processing (Adolphs, 2002).

The limitations of the present study should be acknowledged. First, although ERP analyses provided evidence for abnormal attentional processes in SAD participants, no group differences emerged for RT data. This may be due to our relatively small sample size, which represents one of the main limitations of this study, and/or to the specific characteristics of our paradigm, particularly the chosen stimulus-onset asynchronies and its go/no-go component. Unlike classic dot-probe studies in which a behavioral response is required on each trial, our participants had to withhold responses on no-go trials, possibly introducing novel sources of variance (e.g. decision-making, inhibition of motor-responses). Moreover, by design, ERP waveforms were derived exclusively from no-go trials to avoid potential contamination of movement artefacts to early ERP components (e.g., C1), whereas RT data were assessed during go trials. Thus, although this particular version of the dot-probe paradigm has been found to induce reliable ERP correlates of attentional biases in two

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[5]In addition exploratory analyses revealed decreased N1 components for emotionally cued probes. Concomitant P1 increases and N1 decreases have been previously reported in response to attended stimuli (Fu et *al.* 2005a, 2005b) and may be due to a sustained P1-related positivity reducing the N1 component (Hopfinger & West, 2006).

independent control samples (e.g., Pourtois *et al.* 2004; Santesso *et al.* 2008), the integration of RT and ERP data is suboptimal.

An alternative explanation for the lack of group RT differences may be reduced power for the behavioral analyses, particularly since only 30% of the trials (i.e., go trials) could be used for behavioral analyses. Nevertheless, separate analyses for each group revealed that SAD - but not control - participants did react significantly faster to probes cued by angry as opposed to happy or neutral faces ("within-subjects bias"; Bar-Haim *et al.* 2007).

A final limitation of the present study arises from presenting probes with random interstimulus intervals (100–300 ms). Even though this technique reduces overlap from early and later ERP components, it also prevents a precise delineation of the time course of attentional effects. In order to better understand the temporal unfolding of attentional biases in SAD, ERP studies using both short (e.g., 100 ms) and long (e.g., 500 ms) stimulus-onset asynchronies in the same participants will be required.

Notwithstanding these limitations, the present study suggests that for participants with SAD, early (possibly amygdala-related) threat detection may trigger increased activation in visual areas (including the FG) leading to rapid hypervigilance, which was reflected in potentiated P1 responses, increased accuracy, and shortened RTs to angry faces. In addition to this initial hypervigilance, SAD participants were characterized by reduced visual processing of emotionally cued locations during the P1-Probe time-window. When seen within the framework of other studies (e.g. Amir *et al.* 1998; Vassilopoulos, 2005; Garner *et al.* 2006), the present ERP findings suggest the presence of a hypervigilant-avoidant pattern of attention in SAD. We recommend that future studies examine whether these ERP findings extend to emotional expressions of varying degrees and valences, and/or to other anxiety disorders in order to clarify how hypervigilance and avoidance play a role in the maintenance of these disorders and their potential cognitive treatments.

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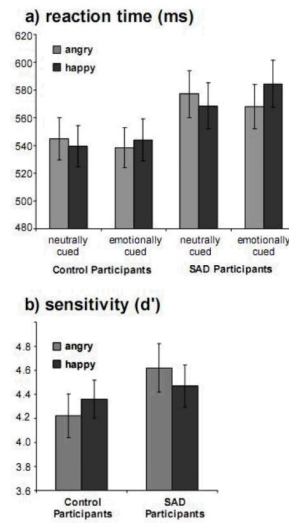
**Fig. 1.**
(a) Reaction times to the probe as a function of facial expression (angry vs. happy) and probe position (emotionally vs. neutrally cued) in both control participants and participants with social anxiety disorder (SAD). (b) Mean $d'$ values as a function of facial expression (angry vs. happy) in both control participants and participants with SAD. Bars denote standard errors.
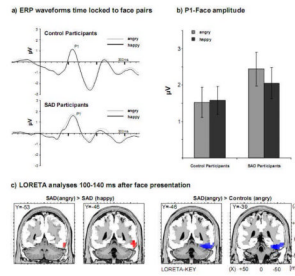
**Fig. 2.**
(a) ERP waveforms time-locked to the presentation of angry (grey lines) and happy (black lines) face-pairs at sensor 85 in the right hemisphere (equivalent to channel P08 in the 10/20 system) for control participants (controls) (top) and participants with social anxiety disorder (SAD) (bottom). (b) Mean P1 amplitude time-locked to angry (grey) and happy (black) face-pairs for participants with social anxiety disorder and control participants. Bars denote standard errors. (c) Left: Results of voxel-by-voxel paired *t*-tests contrasting current density 100–140 ms after presentation of angry-neutral face-pairs vs. happy-neutral face-pairs for participants with social anxiety disorder (SAD). Red: angry > happy. Right: Results of voxel-by-voxel unpaired *t*-tests contrasting current density 100–140 ms after presentation of angry-neutral face-pairs for participants with social anxiety disorder vs. control participants. Blue: participants with SAD > controls. Statistical maps are thresholded at *p*<0.01 and displayed on the MNI template.

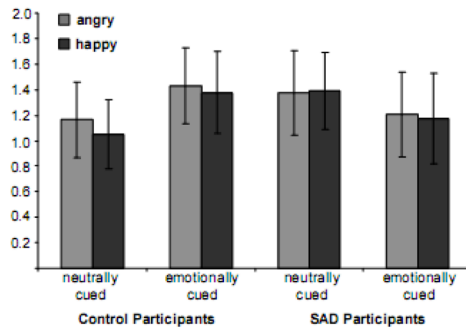**a) ERP waveforms to probes following angry-neutral face pairs**
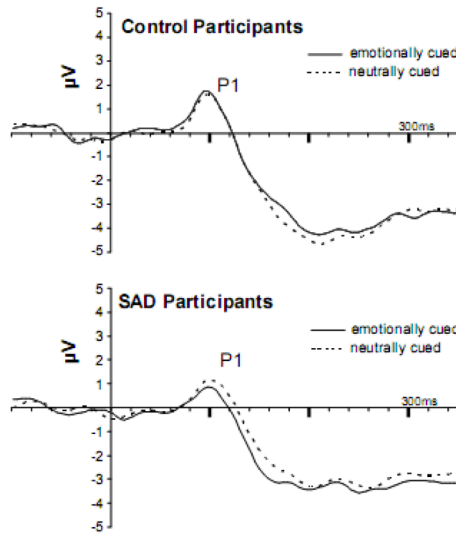
**Fig. 3.**
(a) ERP waveforms at sensor 85 (PO8) in the right hemisphere for control and social anxiety disorder (SAD) participants. ERPs are time-locked to the onset of probes presented in the left hemisphere replacing angry (solid lines) or neutral faces (dashed lines) of angry-neutral face-pairs. (b) Mean P1-Probe amplitudes as a function of facial expression (angry vs. happy) and probe position (emotionally vs. neutrally cued) in both control participants and participants with SAD. Bars denote standard errors.

## Table 1

Summary of sociodemographic and self-report measures of mood and symptom severity for participants with social anxiety disorder (SAD) and healthy controls.

| | SAD participants (*n*=12) | Control participants (*n*=15) | *t* or $\chi^2$-value | *p-values* |
|---|---|---|---|---|
| Age[*] | 30.3 (10.3) | 31.8 (9.8) | $t(24)=0.37$ | > 0.5 |
| Men/Women | 4/8 | 8/7 | $\chi^2(1)=0.33$ | > 0.5 |
| Education (years) | 16.3 (2.5) | 16.5 (1.3) | $t(24)=0.18$ | > 0.5 |
| Ethnicity (% Caucasian) | 75 | 93 | $\chi^2(1)=1.78$ | > 0.2 |
| Marital status (% married) | 20 | 25 | $\chi^2(1)=0.76$ | > 0.5 |
| Social Interaction Anxiety Scale[*] | 49.8 (11.1) | 8.2 (5.4) | $t(24)=-11.5$ | < 0.0001 |
| State-Trait Anxiety Inventory (trait)[*] | 53.8 (13.3) | 29.9 (6.7) | $t(24)=-5.5$ | < 0.0001 |
| Beck Depression Inventory-II | 12.8 (15.1) | 2 (2.8) | $t(25)=-2.4$ | < 0.05 |

*Note*: The Table shows means (standard deviations) and results of statistical tests.

[*] df=24 due to missing values for one participant.

**Table 2**

Summary of significant results emerging from whole-brain LORETA analyses 100–140 ms after presentation of face-pairs.

| Region | MNI Coordinates | | | Brodmann Area | Voxels | t-value | p-value |
|---|---|---|---|---|---|---|---|
| | **X** | **Y** | **Z** | | | | |
| *a. SAD (angry-neutral face pair) > SAD (happy-neutral face pair)* | | | | | | | |
| R. Middle Temporal G. | 60 | −46 | −6 | 20, 21, 37 | 6 | 4.41 | 0.001 |
| *b. SAD (angry-neutral face pair) > Controls (angry-neutral face pair)* | | | | | | | |
| R. Fusiform G. | 39 | −39 | −20 | 20, 36, 37 | 25 | 3.74 | 0.001 |

Note: (a) Results of paired *t*-tests contrasting LORETA activation to angry-neutral *vs.* happy-neutral face-pairs for participants with social anxiety disorder (SAD). Positive *t*-values are indicative of stronger current density for angry-neutral than happy-neutral face-pairs. (b) Results of unpaired *t*-tests contrasting LORETA activation to angry-neutral face-pairs for SAD vs. control participants. Positive *t*-values are indicative of stronger current density for SAD than healthy controls. The anatomical regions, MNI (Montreal Neurological Institute) coordinates, and Brodmann area of extreme *t*-values are listed. The numbers of voxels exceeding the statistical threshold (*p*<0.01) are also reported. Coordinates in mm (MNI space), origin at anterior commissure; (X) = left (−) to right (+); (Y) = posterior (−) to anterior (+); (Z) = inferior (−) to superior (+). L = left, R = right. G. = gyrus.