# Neural Correlates of Anticipation and Processing of Performance Feedback in Social Anxiety

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**Abstract:** Fear of negative evaluation, such as negative social performance feedback, is the core symptom of social anxiety. The present study investigated the neural correlates of anticipation and perception of social performance feedback in social anxiety. High (HSA) and low (LSA) socially anxious individuals were asked to give a speech on a personally relevant topic and received standardized but appropriate expert performance feedback in a succeeding experimental session in which neural activity was measured during anticipation and presentation of negative and positive performance feedback concerning the speech performance, or a neutral feedback-unrelated control condition. HSA compared to LSA subjects reported greater anxiety during anticipation of negative feedback. Functional magnetic resonance imaging results showed deactivation of medial prefrontal brain areas during anticipation of negative feedback relative to the control and the positive condition, and medial prefrontal and insular hyperactivation during presentation of negative as well as positive feedback in HSA compared to LSA subjects. The results indicate distinct processes underlying feedback processing during anticipation and presentation of feedback in HSA as compared to LSA individuals. In line with the role of the medial prefrontal cortex in self-referential information processing and the insula in interoception, social anxiety seems to be associated with lower self-monitoring during feedback anticipation, and an increased self-focus and interoception during feedback presentation, regardless of feedback valence. *Hum Brain Mapp 35:6023–6031, 2014.* © 2014 Wiley Periodicals, Inc.

**Key words:** anticipation; feedback; medial prefrontal cortex; social anxiety

## INTRODUCTION

Social anxiety refers to fear of social and performance situations, and particularly fear of negative evaluation by others; high levels of social anxiety can not only be found in subclinical populations, but also in clinical samples such as patients with social anxiety disorder (SAD) [DSM-IV-TR; American Psychiatric Association, 2000; Stangier and Fydrich, 2002]. Thus, social anxiety seems to be represented on a continuum ranging from subclinical behavior (e.g., shyness) to clinical manifestation (SAD) which may rely on the same underlying dysfunctional mechanisms [Stein et al., 2000]: Individuals suffering from social anxiety tend to interpret negative evaluation in a destructive

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manner and expect negative consequences and rejection by others [Clark and Wells, 1995; Turner et al., 1992]. Accordingly, negative criticism is particularly painful for socially anxious subjects [Gregorich et al., 1986]. Cognitive models of social anxiety [Clark and Wells, 1995] predict that socially anxious individuals are significantly biased toward dysfunctional self-focused processing in situations of social threat-like negative social evaluation.

Several neuroimaging studies have investigated the neural underpinnings of social anxiety by analyzing the brain circuits involved in the processing of (potential) social threat. SAD has been shown to be associated with hypersensitivity of the amygdala in response to threatening social stimuli and symptom provocation (for an overview see Miskovic and Schmidt, 2012; Schulz et al., 2013]. Altered activation patterns were also observed in further emotion-related limbic areas such as the insula [Straube et al., 2004, 2005], and in cortical areas such as the prefrontal cortex (PFC) [Gentili et al., 2008; Hahn et al., 2011; Quadflieg et al., 2008].

Remarkably, despite the obvious relevance of feedback processing for socially anxious subjects, as yet only Blair et al. [2008, 2011] have investigated the matter with functional imaging, although outside the context of performance feedback. They reported heightened sensitivity within the bilateral amygdala and the medial prefrontal cortex (mPFC) to self-referential general criticism (e.g., "You are ugly") in SAD. Interestingly, these results were not replicated in subclinical social anxiety, possibly indicating that this pattern may be specific only to full-blown SAD [Abraham et al., 2013]. The emotional and neural basis of the processing of performance-related feedback in social anxiety is unknown, as is whether altered emotional and neural responses already emerge during the anticipation of performance feedback. Fear of negative evaluation as main concern in socially anxious subjects may promote avoidance behavior and thus lead to maintenance of the disorder [Salters-Pedneault et al., 2004], rendering anticipatory anxiety an essential feature of social anxiety [Clark and Wells, 1995; Mellings and Alden, 2000].

In healthy subjects, the neural correlates of anticipatory processing have been investigated repeatedly: some studies [Bermpohl et al., 2006; Herwig et al., 2007; Nitschke et al., 2006; Simmons et al., 2004; Ueda et al., 2003] report overall similar activation patterns during anticipation of and confrontation with emotional stimuli, but also identify distinct brain areas uniquely activated during anticipation of aversive stimuli, for example, within the PFC and anterior cingulate cortex (ACC) [Herwig et al., 2007; Nitschke et al., 2006]. Others, for example, Critchley et al. [2001], report activations of mPFC, ACC, and insula during anticipation of monetary gain and loss. But to our knowledge, to date there are no studies on anticipatory processing of verbal performance feedback in SAD. Crucially, anxiety affects anticipatory processes. In subclinical samples of anxious individuals, anticipatory anxiety modulates activation within ACC [Straube et al., 2009], mPFC and insula [Holtz et al., 2012; Simmons et al., 2006; Simpson et al., 2001]. In clinical anxiety, for example, spider phobia, anticipation of pictures of spiders was associated with greater activity of ACC, insula, thalamus, bed nucleus of the stria terminalis, and extrastriate visual cortical areas [Straube et al., 2007], reflecting processes of appraisal, increased vigilance and attention, negative affect, and heightened arousal [Straube et al., 2007]. In SAD, anticipation of public speech evokes exaggerated activations within insula and amygdala, and decreased activation within the ventral striatum [Boehme et al., 2013]. Brühl et al. [2011] report increased activity in the amygdala and thalamus, and decreased activity in the left orbitofrontal cortex during anticipation of afore known negative or unknown ambiguous emotional stimuli in SAD. These findings suggest altered neural networks subserving anticipation of threat in anxious and anxiety-prone subjects. However, even though fear of negative evaluation is a core symptom of social anxiety, studies concerned with the neural basis of anticipation and perception of performance feedback in social anxiety are lacking. The present study investigated brain activation while subjects with high (HSA) and low (LSA) social anxiety anticipated and processed positive and negative performance feedback or neutral feedback-unrelated messages. HSA subjects were expected to experience anticipation of negative feedback as more negative, more arousing and more anxiety-inducing than LSA subjects. Accordingly, HSA as compared to LSA subjects were hypothesized to show differential activation patterns within brain regions involved in anticipatory and feedback processing, for example, prefrontal, temporal and limbic regions, and particularly the mPFC, ACC, amygdale, and insula.

## MATERIALS AND METHODS

### Subjects

Participants were selected from a volunteer database of the Institute for Biological and Clinical Psychology at the University of Jena, Germany. Six hundred and thirty-nine persons had previously participated in a large-scale screening for social anxiety by completing an online version of the Liebowitz social anxiety scale (LSAS; German version; Stangier and Heidenreich, 2004]. We defined strict inclusion criteria to ensure investigation of two groups of individuals with distinct social anxiety scores. Two extreme groups were built based on the screening data.16 subjects with a high score (cutoff $> 60$) in the LSAS were assigned to the HSA group, and 18 subjects with a score $< 20$ were assigned to the LSA. Note that HSA subjects did not complete full diagnostic evaluations and thus cannot be labeled SAD patients. Nevertheless, an LSAS score $> 60$ indicates symptom levels commonly observed in clinical samples [Mennin et al., 2002; Rytwinski et al., 2009]. All participants were right-handed, had normal or

**TABLE I. Mean age and mean scores (± standard deviation) on social anxiety related questionnaires (LSAS, SPAI, SANB, and FPE) and depression inventory (BDI) for high socially anxious subjects (HSA) and low socially anxious subjects (LSA)**

| | HAS (M ± SD) | LSA (M ± SD) | *t*-value | *P*-value (two-tailed) |
|---|---|---|---|---|
| Age | 22.85 ± 2.58 | 23.67 ± 2.64 | −0.79 | 0.44 |
| LSAS | 70.38 ± 8.43 | 10.5 ± 4.68 | 21.69 | ≤0.001 |
| SPAI | 3.41 ± .57 | 1.04 ± .44 | 11.62 | ≤0.001 |
| SANB | 58.46 ± 7.54 | 32.92 ± 4.37 | 10.25 | ≤0.001 |
| FPE | 28.15 ± 5.43 | 20.75 ± 3.42 | 4.04 | ≤0.001 |
| BDI | 11.15 ± 7.4 | 2.67 ± 2.43 | 3.78 | ≤0.001 |

M = Mean; SD = standard deviation; LSAS, Liebowitz Social Anxiety Scale; SPAI, social phobia anxiety inventory; SANB, fear of negative evaluation; FPE, fear of positive evaluation; BDI, beck depression inventory.

corrected-to-normal vision, no history of neurological or psychiatric diseases, and met the general MRI-requirements, for example, no ferromagnetic implants. At time of testing, all subjects completed the German versions of the beck depression inventory [BDI, Hautzinger et al., 1995], the social phobia and anxiety inventory [SPAI; Fydrich, 2002], the fear of negative evaluation [SANB; Vormbrock and Neuser, 1983] and the fear of positive evaluation [FPE; Weeks et al., 2008] questionnaire. To further ensure two distinct groups, HSA subjects with a score $< 3$ in the SPAI (three HSA subjects) and LSA subjects with a SPAI-score $> 2$ or a discrepancy of $\geq 2$ standard deviations on the BDI, SANB, or FPE (four LSA subjects) were excluded. Furthermore, two LSA subjects were excluded from the statistical analyses due to head movements >3 mm during fMRI-scanning. Data of 13 HSA subjects and 12 LSA subjects, matched for age and gender (all participants were female), were analyzed. Mean age and mean scores on the clinical questionnaires are provided in Table I. As expected, HSA compared to LSA subjects showed significantly higher scores on all questionnaires sensitive to social anxiety (Table I). BDI scores were also significantly higher in HSA compared to LSA, but reached only mild clinical significance.

The study conforms to the Declaration of Helsinki and was approved by the ethics committee of the University of Jena, Germany. Written informed consent was obtained from each participant prior to the experiment.

## Experimental Task

The task consisted of two separate experimental sessions completed within 2–3 days from one another. During the first session, all subjects gave a speech about a personally relevant topic (favorite book/movie). The speech lasted 5 min and was recorded. In the second session, participants were told that two persons had evaluated their speech and that they would receive feedback on their performance in the MRI-scanner. Participants were also informed that aside from the two evaluators' feedbacks, a third person's statements would provide information about the weather. This latter condition was introduced as a neutral control condition as similar to the feedback statements as possible, because feedback as such is never completely neutral. To this end, brief statements, for example, weather reports matching the feedback statements in terms of length, were selected. Feedback and control statements were preceded by a letter (A, B, or C), announcing the feedback category or neutral statement. Cue-feedback type associations were counterbalanced across the two groups, and subjects were informed about these associations (e.g., A = positive feedback, B = negative feedback, C = control condition) prior to scanning. Participants were instructed to concentrate on the cue and to anticipate subsequent feedback.

Unknown to the participants, feedback was standardized. Each participant received 14 positive feedback statements (e.g., "Your speech was fluent!"), 14 negative feedback statements (e.g., "Your voice was unclear!"), and 14 neutral control messages (e.g., "It's 32°C in Istanbul!"). To ensure personal relevance of feedback, and to prevent inadequacy, appropriate positive and negative statements for each subject were chosen from a set of 28 standardized feedback sentences by two independent raters (members of staff at the Institute for Biological and Clinical Psychology in Jena, Germany) who had previously watched the movies of the speech performances.

Stimulus presentation was controlled with Presentation software (version 9.90, Neurobehavioral Systems Incorporation, Albany, CA). Stimuli were presented in white color in the center of a black screen. Trials started with presentation of a letter cue for 2 s. The anticipation phase, marked by a central fixation cross, lasted on average 10 s (range: 4–12 s). Subsequently, during the feedback phase, a positive or negative feedback statement or a neutral message was presented for 4 s, followed by 14 s intertrial-interval. Feedback types were pseudorandomized, with a maximum of one immediate repetition of the same feedback type. The whole paradigm lasted approximately 12 min.

After scanning, participants rated the anticipation phases using nine-point Likert-scales to separately assess valence (1 = "very negative" to 9 = "very positive"), arousal (1 = "not arousing" to 9 = "very arousing"), and the anxiety they had experienced during the anticipation period (1 = "not anxious" to 9 = "very anxious"). Behavioral data were analyzed by means of repeated measures analysis of variance (ANOVA) with feedback type as within-subjects factor (positive or negative feedback or control condition) and group as between-subjects factor (HSA or LSA) using the software SPSS (version 21, SPSS). Post hoc *t*-tests were applied to resolve interactions. For the ANOVAs, a probability level of $P \leq 0.05$ was considered statistically significant, and for subsequent *t*-tests, a Bonferroni corrected probability level of $P \leq 0.012$ was

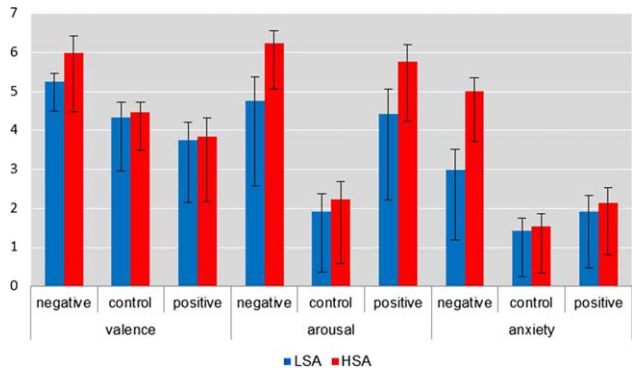


**Figure 1.**

Differential ratings (M ± SE) for the anticipation of positive and negative feedback as well as the control condition in high socially anxious subjects (HSA) and low socially anxious subjects (LSA).

selected to account for multiple comparisons. As this study concentrates on differences between HSA and LSA subjects, only main effects and interactions including the factor group will be presented.

### Functional Magnetic Resonance Imaging

fMRI data were recorded with a 1.5-T scanner ("Magnetom Vision plus," Siemens Medical Systems), using a T2-weighted echo-planar sequence (TE = 50 ms, flip angle = 90°, matrix = 64 × 64, FOV = 192 $mm^2$, TR = 2,800 ms). Four hundred and thirty-four volumes of 30 axial slices (thickness = 3 mm, no gap, in plane resolution = 3 × 3 $mm^2$) were acquired. To minimize susceptibility artifacts in inferior parts of anterior brain areas, the volumes were tilted 30° from the AC/PC line. The first three volumes of each run were discarded to secure steady-state tissue magnetization. Additionally, a high-resolution T1-weighted anatomical volume was acquired.

Preprocessing and analysis of functional data were performed using Brain Voyager QX software (version 2.3.1, Brain Innovation, Maastricht, Netherlands). The volumes were realigned to the first volume to minimize effects of head movements, and slice time correction was applied. Further preprocessing comprised spatial (8 mm full-width half-maximum isotropic Gaussian kernel)and temporal (low pass filter: 2.8 s; high pass filter: 0.005 Hz, linear trend removal) smoothing. The anatomical and functional images were coregistered and normalized to Talairach space [Talairach and Tournoux, 1988].

Statistical analyses were performed analogously for the anticipation phase and the presentation phase. A multiple linear regression of the signal time course at each voxel was calculated. The expected blood oxygen level-dependent signal change for each predictor was modeled by a hemodynamic response function. The three predictors of interest were negative feedback, positive feedback and control condition. Statistical comparisons were conducted using a mixed-effect analysis. First, voxel-wise statistical maps were generated and predictor estimates (beta weights) were computed for each individual. Contrasts of predictor estimates were then analyzed across subjects with 3 × 2 repeated measures ANOVAs with feedback type (positive or negative feedback or control condition) as within-subjects factor and group (HSA or LSA) as between subjects factor. As the study was aimed to investigate differences between HSA and LSA subjects, only interaction effects were calculated.

Analyses were conducted for specific regions of interest (ROIs) as defined a priori using the Talairach daemon software [Lancaster et al., 2007]. These ROIs were the amygdala, the insula, the ACC and the mPFC. Statistical parametric maps resulting from the voxel-wise analysis were considered significant for clusters surviving cluster-based correction for multiple comparisons. Voxel-level threshold was initially set to $P \leq 0.005$ (uncorrected). Thresholded maps were then submitted to a ROI-specific correction criterion which was based on the estimate of the map's spatial smoothness and on an iterative procedure (Monte Carlo simulation) used to estimate cluster level false-positive rates [Forman et al., 1995]. After 1,000 iterations, the minimum cluster size threshold that yielded a cluster level false-positive rate of 5% was applied to the statistical maps. Furthermore, post hoc *t*-tests were calculated to resolve the significant interaction effects of the ANOVA.

## RESULTS

### Behavioral Data: Valence, Arousal, and Anxiety Ratings

Mean ratings of feedback valence, arousal and anxiety are provided in Figure 1. The ANOVA yielded a feedback type × group interaction for anxiety [$F(2,46) = 5.107$, $P = 0.007$]. Post hoc group comparisons revealed significantly higher anxiety ratings for HSA compared to LSA subjects during anticipation of negative feedback [$t(23) = 3.202$, $P = 0.004$]. For positive feedback and the control condition, anxiety ratings did not differ between groups (both $P \geq 0.674$). The main effect of group failed to reach statistical significance ($P = 0.064$). For valence and arousal, there were no significant main effects or interactions involving the group factor (all *P*-values $\geq 0.056$).

### fMRI Data: Feedback Anticipation

For the anticipation phase, the ANOVA showed a significant interaction effect of feedback type × group (Table II) in a large cluster in the left mPFC which extended to the left and right ACC, and in a separate cluster in the right ACC. *T*-contrasts yielded significantly decreased activation in HSA compared to LSA within these clusters during

**TABLE II. Significant interaction effect of feedback type × group during anticipation and presentation of performance feedback (*P*-values ≤ 0.05 corrected)**

| Region | Lateralization | Talairach co-ordinates | | | Cluster size ($mm^3$) | *F*-value | Effect |
|---|---|---|---|---|---|---|---|
| | | *x* | *y* | *z* | | | |
| Anticipation phase | | | | | | | |
| mPFC[a] | L | −6 | 54 | 14 | 3201 | 8.09 | LSA > HSA |
| ACC[a] | L | −3 | 48 | 11 | 383 | 6.86 | LSA > HSA |
| ACC[a] | R | 3 | 49 | 16 | 139 | 6.83 | LSA > HSA |
| ACC | R | 3 | 45 | 16 | 388 | 6.52 | LSA > HSA |
| Presentation phase | | | | | | | |
| mPFC[b] | L | −3 | 51 | 14 | 2058 | 7.54 | HSA > LSA |
| ACC[b] | L | −3 | 51 | 7 | 240 | 6.92 | HSA > LSA |
| ACC | L | −6 | 38 | 19 | 342 | 6.72 | HSA > LSA |
| Insula | L | −42 | 12 | −10 | 232 | 7.44 | HSA > LSA |

[a,b]These activation clusters are respectively interconnected.

anticipation of negative feedback versus control messages (left mPFC: $t = -4.715$, connected left ACC: $t = -4.119$, connected right ACC: $t = -4.189$, separated right ACC: $t = -3.735$, all *P*-values ≤ 0.05 corrected, see Fig. 2), and also for negative versus positive feedback in the mPFC cluster ($t = 3.436$, $P \leq 0.05$ corrected, see Fig. 2). ROI-analyses of the amygdala and insula did not reach significance.

### fMRI Data: Feedback Presentation

For feedback presentation, the ANOVA showed a significant feedback type × group interaction (Table II) in a main cluster within the left mPFC, expanding into the left ACC, and in a separate cluster within the left ACC and in the left insula. *T*-contrasts reflected hyperactivation in HSA compared to LSA subjects during presentation of negative feedback versus control messages (left mPFC: $t = 3.739$, connected left ACC: $t = 3.63$, separated left ACC: $t = 3.3$, left insula: $t = 3.594$, all *P*-values ≤ 0.05 corrected, see Fig. 3), and positive feedback versus control messages (left mPFC: $t = 3.716$, connected left ACC: $t = 3.364$, separated left ACC: $t = 3.347$, left insula: $t = 3.718$, all *P*-values ≤ 0.05 corrected, see Figs. 3 and 4). No significant amygdala activation was found for the presentation phase.

## DISCUSSION

The present study investigated the neural correlates of anticipation and processing of performance feedback in high and low socially anxious individuals. Participants gave a speech on a personally relevant topic and received standardized but appropriate performance feedback. Neural activity was measured during anticipation and presentation of negative and positive performance feedback and a neutral control condition. As expected, anxiety ratings were higher in HSA compared to LSA subjects during anticipation of negative feedback, supporting the fear-provoking quality of negative feedback for socially anxious individuals [Gregorich et al., 1986]. Anxiety is therefore increased already prior to confrontation with disorder-related material in socially anxious individuals. This effect is also reflected on the neural level: During anticipation of negative feedback versus the control condition, HSA compared to LSA subjects showed significant deactivation within the mPFC/ACC. This is in accordance with previous studies reporting anxiety-driven modulation of anticipatory processing, for example, in spider phobia or social anxiety [Boehme et al., 2013; Holtz et al., 2012; Simmons et al., 2006; Simpson et al., 2001; Straube et al., 2007, 2009].

Activation patterns during feedback presentation differed considerably: HSA compared to LSA subjects showed hyperactivation of mPFC/ACC and insula in response to positive and negative feedback relative to the control condition, suggesting differential mechanisms during feedback anticipation and presentation, which is consistent with differential processing of emotional stimuli during anticipation and presentation [Herwig et al., 2007; Nitschke et al., 2006; Simmons et al., 2006]. Processes of preparation, adaption and increased vigilance for external stimuli may dominate during anticipation of emotionally relevant external stimuli, while during presentation, processes of stimulus appraisal and emotion expression prevail [Gross, 1998; Straube et al., 2009]. In accordance with these assumptions, and with fear of negative evaluation as the core symptom of social anxiety, anticipatory processing differed between HSA and LSA subjects only for negative feedback, while during feedback presentation, group differences emerged for negative and positive feedback. Specifically, mPFC/ACC and insula activation was increased during presentation of negative and positive feedback, while during anticipation mPFC/ACC activation was decreased only for negative feedback. Findings for the presentation phase may reflect increased relevance of both negative and positive feedback in social anxiety, since anxiety ratings showed exaggerated fear of positive evaluation in

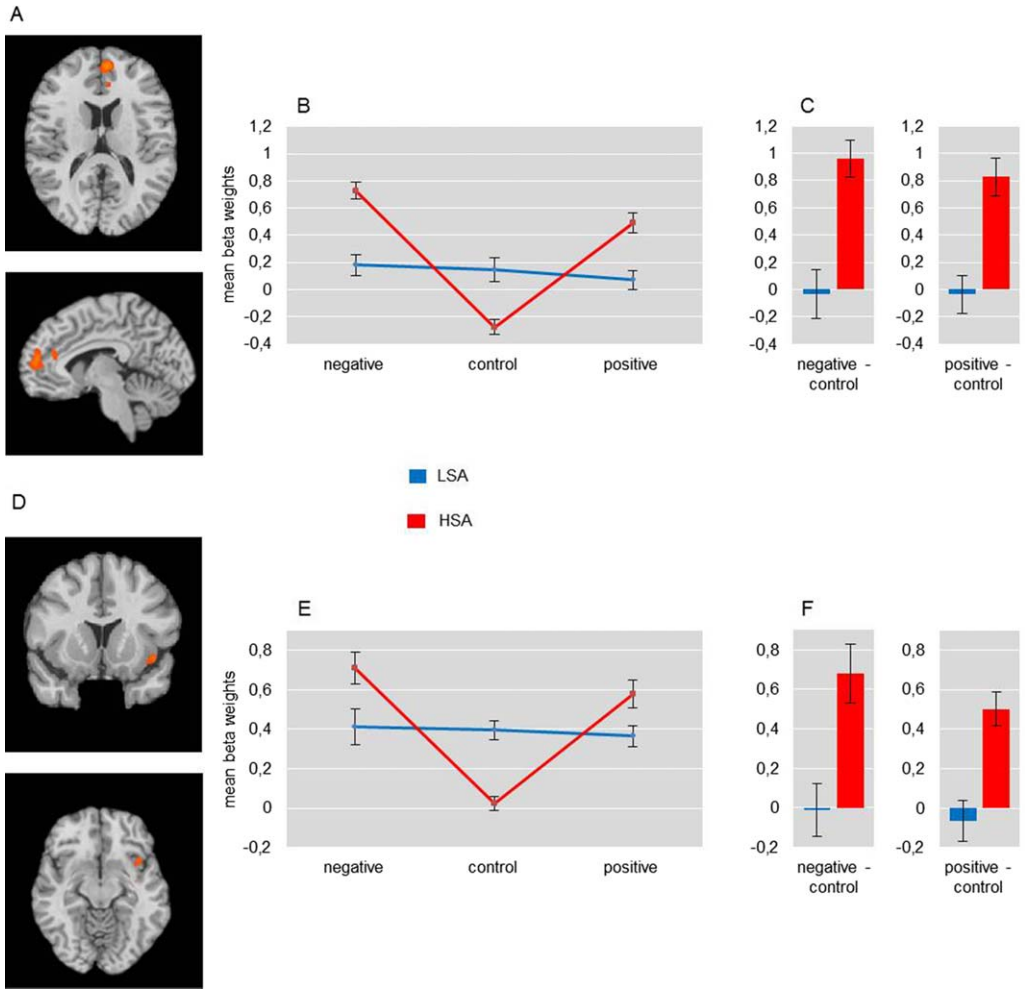


**Figure 2.**

Significant interaction of group × condition during the anticipation phase within the left medial prefrontal area. Brain activation overlayed on a t1 scan [transversal ($z = 14$) and sagittal ($x = -7$), **A**]. Diagramms show average parameter estimates for the the significant ANOVA cluster (**B**) and the subsequent *t*-contrasts (**C**).

HSA relative to LSA subjects. This is in line with overestimation of the costs of positive social outcomes [Gilboa-Schechtman et al., 2000] and heightened anxiety after positive feedback [Alden and Wallace, 1995; Wallace and Alden, 1997]. Wallace and Alden [1995, 1997] proposed that upon receiving positive feedback, socially anxious individuals believe others to expect more in upcoming interactions. Dysfunctional processing during presentation of positive performance feedback may relate to more negative interpretation of positive events [Alden et al., 2008] and the absence of a positive inferential bias [Hirsch and Mathews, 2000]. This notion is also supported by mPFC/vACC hyperactivation in response to positive feedback in individuals with low self-esteem [Somerville et al., 2010]. MPFC deactivation in anxious individuals during anticipation of aversive stimuli has been interpreted as a neural correlate of reduced self-monitoring [Simmons et al., 2006]. Indeed, mPFC deactivation may be accompanied by more externally focused attention [Raichle et al., 2001]. Medial prefrontal regions are assumed to be part of the so-called "default mode network" which is deactivated during externally oriented tasks [Gusnard et al., 2001]. This complements evidence for an important role of the mPFC and ACC for self-referential processing and self-focused attentional allocation [Gusnard et al., 2001; Mitchell et al., 2005; Northoff et al., 2006; Ochsner et al., 2004; Raichle et al., 2001]. MPFC deactivation in anxious individuals may lead to reduced capacity to properly regulate emotional responsiveness [Simmons et al., 2006]. In view of mPFC deactivation during anticipation in HSA subjects, hyperactivation during presentation of feedback may reflect increased self-monitoring [Raichle et al., 2001]. Indeed, Blair et al. [2011, 2008] reported increased

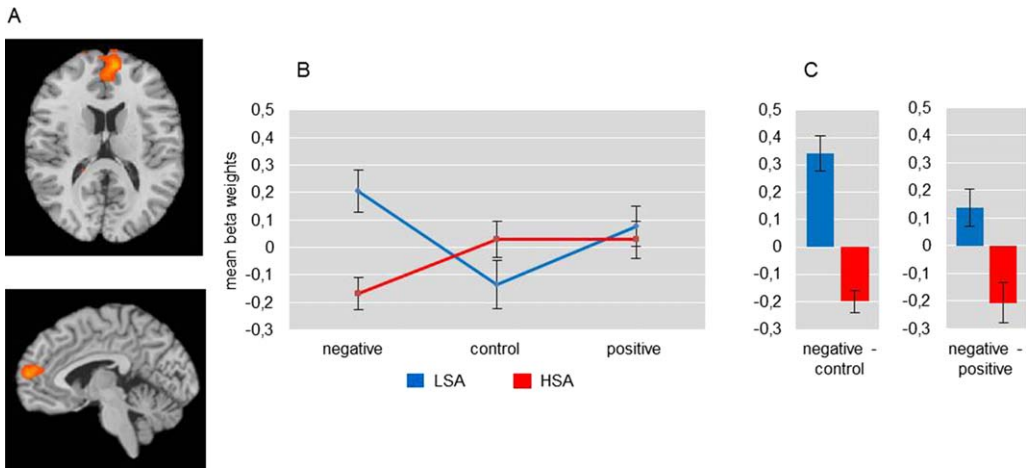


**Figure 3.**

Significant interaction of group × condition during the presentation phase within the left medial prefrontal area and insula. Brain activation overlayed on a t1 scan [mPFC transversal ($z = 13$) and sagittal ($x = -5$), **A**; insula coronal ($y = 12$) and transversal ($z = -12$), **D**]. Diagramms show average parameter estimates for the the significant ANOVA cluster (mPFC: **B** and insula: **E**) and the subsequent *t*-contrasts (mPFC: **C** and insula: **F**).

mPFC activation as a correlate of self-referential processing in social anxiety. This agrees with the cognitive model of social anxiety [Clark and Wells, 1995], suggesting increased self-focus in social situations to cause enhanced anxiety and dysfunctional processing. Insular activation in the present study is likely related to interoceptive processing, possibly reflecting increased self-focus in response to performance feedback [Critchley et al., 2004; Paulus and Stein, 2006], and evaluation of the emotional salience of interoceptive stimuli [Reiman, 1997].

Activation patterns during anticipation and presentation of feedback appear to indicate differential allocation of self-focused attention in HSA subjects. Both increased and decreased self-focus have previously been associated with dysfunctional processing of external information [Ingram, 1990]. Although reduced self-monitoring may lead to lead to reduced self-control [Carver and Scheier, 1981], increased self-monitoring may increase anxiety [Clark and Wells, 1995; Spurr and Stopa, 2002]. Possibly, there is an inverted u-shaped function between self-focus and functional processing of threat, in which extreme expressions of self-focus are associated with inadequate and dysfunctional threat processing. Consequently, lower self-monitoring in HSA subjects may have decreased self-regulation and increased vigilance during feedback anticipation, while during feedback presentation feedback, anxiety was increased due to an increased self-focus.

The present results appear to contradict a recent study failing to replicate previous reports of anxiety-modulated feedback processing in subclinically anxious individuals [Abraham et al., 2013]. However, the present study used performance feedback rather than self- or other-related criticism. It is conceivable that, in line with the cognitive model of SAD, performance feedback is particularly relevant in both SAD and subclinical HSA. Nevertheless, the generalizability of the present results is limited due to a rather small final sample, although populations of 11–14 participants are not uncommon in SAD research [e.g., Campbell, 2007; Evans et al., 2008]. More research is needed to better characterize feedback processing in clinical and subclinical social anxiety, also with regard to the subjective assessment of feedback presentation, which was not obtained due to the present study's focus specifically on anticipation of feedback. Moreover, no group effects were observed in brain regions other than mPFC and insula, including the amygdala, possibly due to processing of cued, that is, perfectly predictable, performance feedback. Future research will need to investigate the role of feedback expectancy and the neural correlates of unpredictable feedback in social anxiety. Lastly, hormonal levels influence reactivity to socially relevant stimuli [Gingnell et al., 2014], and may modulate SAD symptom levels [van Veen et al., 2009]. Menstrual cycle phase was not assessed in the present study but should be considered as an influencing factor in future studies.

## CONCLUSIONS

Feedback processing in socially anxious individuals seems to rely on distinct mechanisms during anticipation and presentation of performance feedback. Although HSA and LSA subjects differed with regard to anticipation of only negative feedback, differential processing of negative and positive feedback was observed during feedback presentation. In view of the role of the mPFC in self-

referential processing, activation patterns potentially reveal dysfunctional allocation of self-focused attentional resources in social anxiety, with decreased self-focus during anticipation and increased self-focus during presentation of performance feedback. Further research is needed to clarify the differential role of self-focusing during anticipation and presentation of threat in social anxiety.

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