The role of negative affectivity and social inhibition in perceiving social threat: An fMRI study

Mariska Esther Kret a,b,1, Johan Denollet b, Julie Grèzes c, Beatrice de Gelder a,d,∗

a Cognitive and Affective Neurosciences Laboratory, Tilburg University, Tilburg, The Netherlands
b CoRPS - Center of Research on Psychology in Somatic diseases, Tilburg University, Tilburg, The Netherlands
c Laboratoire de Neurosciences Cognitives, U960 INSERM & Département d’Etudes Cognitives, École Normale Supérieure, Paris, France
d Martinos Center for Biomedical Imaging, Massachusetts General Hospital, Harvard Medical School, Charlestown, MA, USA

1. Individual differences in emotion perception

Social communication includes intuitively grasping signals of hostility and reacting to signals of distress. Humans are especially sensitive to the gestural signals and facial expressions of other people, and also use these signals to guide for their own behavior. Previous research has largely focused on the perception of emotions from static faces (Adolphs, 2002a; Haxby, Hoffman, & Gobbini, 2000). But our communicative ability also relies heavily on decoding messages provided by body movements. Dynamic presentations of facial stimuli facilitate processing (Sato, Fujimura, & Suzuki, 2008; Sato, Kochiyama, Yoshikawa, Naito, & Matsumura, 2004). Moreover, dynamic information is useful for a better understanding of the respective contribution of action components in body expressions (Grèzes, Pichon, & de Gelder, 2007; Pichon, de Gelder, & Grèzes, 2008).

People vary in how they perceive emotions and their brain activity patterns differ. For example, healthy individuals with high trait anxiety show increased amygdala activity when they look at threatening faces (Etkin et al., 2004). Yet observers not only differ in how they perceive emotions, but also in how they act in threatening situations. Whereas some of us may fight back when confronted with aggression, others flee or freeze (Schmidt, Richey, Zvolensky, & Maner, 2008). These differences may be mediated by the orbitofrontal cortex (Rolls, 2004). Eisler and Levine (2002) provided evidence that the orbitofrontal cortex is the pivotal area for choice between a fight or flight or other responses in a threatening situation. Since the orbitofrontal cortex plays a role in linking sensory events and positive or negative affective valuation, behavioral selection may be biased by an individual’s personality and by the presence of a stressor (Damasio, 1994; Rolls, 2004).

Socially anxious people are afraid of possible scrutiny and negative evaluation by others and strive towards social acceptance. Research supports a positive link between anxiety levels and orbitofrontal cortex activity during threat perception (Stein, Simmons, Feinstein, & Paulus, 2007). Observing another person in a distressed or aggressive state evokes stress in the observer (Hatfield, Cacioppo, & Rapson, 1994). The stress response includes facilitation of neural pathways that subserve acute, time limited adaptive functions, such as arousal, vigilance and focused attention, and inhibition of neural pathways that subserve acutely non-
adaptive functions (Chrousos, 2009). However, this response can become maladaptive when the anxiety response is disproportionate to the situation because of hyper- or hypo-responsiveness at any of a variety of points in the complex network of neural pathways that serve the stress response. Through its mediators, stress can lead to acute or chronic pathological, physical and mental conditions (Chrousos, 2009).

Individuals with a Type D (distressed) personality (21% of the general population) are more likely to experience feelings of depression and anxiety (Denollet, 2005). They tend to experience negative emotions across time and situations (negative affectivity component of Type D) but also inhibit the expression of these emotions due to fear of rejection or disapproval (social inhibition component of Type D). Type D personality is associated with hyper-reactivity of the hypothalamic–pituitary–adrenal axis, increased inflammatory activity, decreased endogenous neural progenitor cells and eventually, poor prognosis in cardiovascular patients (Denollet, de Jonge et al., 2009; Denollet, Martens, Nyklicek, Conraads, & de Gelder, 2008; Denollet, Pedersen, Vrints, & Conraads, 2006; Denollet, Schiffer, & Spek, 2010; Habra, Linden, Anderson, & Weinberg, 2003; Molloy, Perkins-Porras, Strike, & Steptoe, 2008; Sher, 2005; Van Craenendonc et al., 2009). Because Type D personality affects the course and treatment of cardiovascular conditions (Denollet et al., 2010), this personality construct qualifies for the DSM-IV classification Psychological Factors Affecting Medical Condition (DSM, 2000). Whereas depression is an episodic risk marker, Type D is a chronic risk marker for clinical manifestations of coronary disease (Denollet, de Jonge et al., 2009). Type D and depression are partly overlapping (for a recent meta-analysis, see Denollet et al., 2010).

Type D refers to the combination of negative affectivity with social inhibition, but these two subcomponents may be reflected differently in the brain. Learning more about how the two sub-scales independently and jointly influence emotion processing in the brain will provide new insights into the Type D construct. Still very little is known about the neurofunctional basis of negative affectivity and social inhibition. A study by de Gelder, van de Riet, Grèzes, and Denollet (2008) reports a negative correlation between negative affectivity and amygdala activation following static threatening vs. neutral facial and bodily expressions. The authors focused only on the amygdala as region of interest but other effects may be detected in a whole brain analysis and also in relation to the social inhibition personality trait.

The amygdala is viewed as a key area in the social brain network and responds to salient signals such as faces (Adolphs, 2009). We recently compared the neurofunctional network of dynamic facial expressions with that of dynamic bodily expressions and showed that the amygdala was more active for facial than bodily expressions. But bodily expressions triggered higher activation than facial stimuli in a number of regions including the cuneus, fusiform gyrus, extrastriate body area, tempo-parietal junction, superior parietal lobule, primary somatosensory cortex as well as the thalamus. We found no major differences between fearful and angry expressions. Emotion related activations were primarily observed in the superior temporal sulcus and gyrus as well as in the extrastriate body area and the middle temporal gyrus. The absence of the amygdala here may be surprising. However, most studies using dynamic naturalistic expressions (not morphs between a neutral and emotional static face), reported similar results (Grosofski & Pauss, 2006; Kilts, Egan, Gideon, Ely, & Hoffman, 2003; Simon, Craig, Miltnner, & Rainville, 2006; van der Gaag, Mindera, & Keyser, 2007), possibly because of the relevance of a dynamic neutral face (these results are in detail discussed in Kret, Pichon, Grèzes, and de Gelder (2011)). But this explanation may not be complete.

The current study investigates the relation between negative affectivity and social inhibition and the neural responses to threatening signals provided by videos of facial and bodily expressions in a healthy population. Our main questions were threefold. First, we wanted to know whether the earlier reported decrease in amygdala activation associated with threat perception in high negative affectivity scorers (de Gelder et al., 2008) would persist when using dynamic, more naturalistic stimuli and examine whether this decreased activity would extend to other brain areas known to be important for emotion perception. Second, we wanted to examine whether socially inhibited individuals would over activate the cortical social brain network including tempo-parietal junction (which is involved in mentalizing) and the orbitofrontal cortex (which is involved in social decision making). Third, since Type D personality is associated with a broad range of health issues and somatic responses, we were specifically interested in the combined influence of social inhibition and negative affectivity because these subscales together have much predictive value in health outcomes.

2. Methods

2.1. Participants

Twenty-eight students (14 females, mean age 19.73 years old, range 18–27 years old; 14 males; mean age: 21.69 years old, range 18–32 years old) were recruited via an advertisement at Maastricht University. The advertisement stated that we were looking for healthy, right-handed students without a neurological or psychiatric disease or psychological problems. As part of the standard protocol at Maastricht university, before inviting them to participate in the experiment, they were sent additional information about the general and the aim of the study to fill out a standard medical questionnaire developed at Maastricht university, in order to check if their psychological or medical condition was normal and if they were medication free. In addition, the experimenter asked all participants whether they had been diagnosed with a psychiatric disorder or whether they suffered from psychological problems. All were eligible and took part in the experiment in September 2007.

None of the participants reported having a neurological or psychiatric history, all were right-handed, as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971), healthy, and had normal or corrected-to-normal vision. The students were randomly assigned to one version of the experiment (anger-neutral or fear-neutral). The groups did not differ in age ($M_{\text{anger}}=21.81$, $SD=5.63$ vs. $M_{\text{fear}}=21.83$, $SD=1.80$, t(26)=1.168, $p=.251$) and male and female participants were equally distributed. All participants gave informed consent. The study was performed in compliance with national legislation and in accordance with the declaration of Helsinki and was approved by the local ethics committee. Two participants were discarded from analysis, due to (1) task miscomprehension and attention deficit disorder (2) neurological abnormalities, so 26 participants were included.

2.2. Stimuli and validation

Video recordings were made of 26 actors expressing six facial and bodily emotions. For the body video sessions all actors were dressed in black and filmed against a green background. For the facial videos, actors wore a green shirt, similar as the background color. Recordings used a digital video camera under controlled and standardized lighting conditions. To coach the actors to achieve a natural expression, pictures of emotional scenes were, with the help of a projector, shown on the wall in front of them and a short emotion induction story was read out by the experimenter. The actors were free to act the emotions in a naturalistic way as response on the situation described by the experimenter and were not put under time restrictions. Fearful body movements included stretching the arms as if to protect the upper body while leaning backwards. Angry body movements included movements in which the body was slightly bended forward, some actors showed their fists, whereas others stamped their feet and made resolute hand gestures. Additionally, the stimulus set included neutral facial and body movements (such as pulling up the nose, coughing, fixing one’s hair or clothes). Distance to the beamer screen was 600 mm. All video clips were computer-edited using Ulead and After Effects, to a uniform length of 2 s (50 frames).

We filmed several versions per actor and condition. A total of 380 face and body videos of all six basic emotions and in addition neutral stimuli were included in a large validation study. The face and body videos were validated separately. All videos were presented twice to 20 independent raters who had to categorize the emotion (and choose among seven categories) and rate its intensity (how intense is the emotion being displayed?) on a 5-point scale. The in this experiment included angry facial expressions were recognized for 84% (SD 19) with intensity 2.90 (SD .23), fearful facial expressions for 86% (SD 7) with intensity 3.31 (SD .22), neutral facial expressions 79% (SD 21), angry bodily expressions for 85% (SD 15) with intensity 3.56 (SD .62), fearful bodily expressions for 83% (SD 16) with intensity 3.64 (SD .47) and neutral bodily expressions for 80% (SD 20) with intensity 2.09 (SD .21). Angry and fearful expressions were rated as more intense than neutral ones ($M_{\text{ang}}=6.417$, $p<.001$) and $M_{\text{fear}}=6.028$, $p<.001$ respectively.
Intensity scores did not differ between fearful and angry expressions ([t(19) = 1.532, p = .165]. Actors’ age closely matched that of the participants.

The faces of the body videos were covered with Gauussian masks so that only information about the face was perceived. To check for quantitative differences in movement between the movies, we estimated the amount of movement per video clip by quantifying the variation of light intensity (luminance) between pairs of frames for each pixel (Grèzes et al., 2007). For each frame, these absolute differences were averaged across pixels that scored (on a scale reaching a maximum of 255) higher than ten, a value which corresponds to the noise level of the camera. These were then averaged for each movie. Angry and fearful expressions contained equal movement (M = 30.64, SD 11.99 vs. M = 25.41, SD 8.71) ([t(19)] = 776, p < .005) but more movement than neutral expressions (M = 10.17, SD 6.00) ([t(19)] = 3.78, p < .005) and ([t(19)] = 4.093, p < .005). By using Matlab software, we generated scrambled movies by applying a Fourier-based algorithm onto each movie, a technique that has been used in many other studies (Hoffman, Gothard, Schmid, & Logothetis, 2007). This technique scrambles the pixel spectra of each frame and generates video clips that served as low level visual controls and prevented habituation to the stimuli.

2.3. Experimental design

The experiment consisted of 176 trials, presented in two runs, with 80 non-scrambled (10 actors × 2 expressions (threatening (fear or anger), neutral) × 2 runs × 2 repetitions), 80 scrambled videos and 16 oddballs. There were 80 null events (blank, green screen) with a duration of 2000 ms. These 176 stimuli and 80 null events were randomized within each run. A trial started with a fixation cross (500 ms), followed by a video (2000 ms) and a blank green screen (2450 ms). An oddball task was used to control for attention and required participants to press a button on a keypad, positioned on the right side of the participant’s abdomen each time an inverted video-clip appeared so that trials of interest were uncontaminated by motor responses. Half of the participants viewed neutral and angry expressions and the other half neutral and fearful expressions. They were pseudo-randomly assigned to one of the two versions of our experiment but we made sure that the male–female distribution was exactly equal. In this way, the participants saw an equal number of emotional and neutral movies and this design allowed us to pool the results without gender effects at the first level.

2.4. Description of the Type D questionnaire

After the scanning session participants completed the DS14 scale as a standard measure of Type D personality (Denollet, 2005). All forty items are answered on a five-point range from zero (false) to four (true). Seven items refer to ‘negative affectivity’ or the tendency to experience negative emotions (‘I am often down in the dumps’, ‘I often find myself worrying about something’). The other seven items refer to the participants’ level of ‘social inhibition’ or the tendency to inhibit the expression of emotion/behavior in social relationships (‘I am a closed kind of person’, ‘I often feel inhibited in social interactions’). People who score ten points or more on both dimensions are classified as Type D and have the tendency to experience increased negative emotions across time and situations and tend not share these emotions with others, because of fear of rejection or disapproval. These personality scales were earlier found reliable (Cronbach’s = .80/8.86) and stable over time (Denollet, 2005; Martens, Kupper, Pedersen, Aquarius, & Denollet, 2007).

2.5. fmRI data acquisition

2.5.1. Parameters of the functional scan

Functional images were acquired using a 3.0-T Magnetom scanner (Siemens, Erlangen, Germany). Blood Oxygenation Level Dependent (BOLD) sensitive functional images were acquired using a gradient echo-planar imaging (EPI) sequence (TR = 2000 ms, TE = 30 ms, 32 transversal slices, descending interleaved acquisition, 3.5 mm slice thickness, with no interslice gap, FA = 90°, FOV = 224 mm, matrix size = 64 × 64 × 64 mm). An automatic shimming procedure was performed before each scanning session. A total of 644 functional volumes were collected for each participant (total scan time = 10 min per run (2 runs with the anatomical scan in between)).

2.5.2. Parameters of the structural scan

A high-resolution T1-weighted anatomical scan was acquired for each participant (TR = 2250 ms, TE = 2.6 ms, FA = 9°, 192 sagittal slices, voxel size 1 x 1 x 1 mm, inversion Time (TI) = 900 ms, FOV = 256 mm × 256 mm, 192 slices, slice thickness = 1 mm, no gap, total scan time = 8 min).

2.6. Statistical parametric mapping

Functional imaging data were preprocessed and analysed using SPM2 Functional images were processed using SPM2 software (Wellcome Department of Imaging Neuroscience; see www.fil.ion.ucl.ac.uk/spm). The first five volumes of each functional run were discarded to allow for T1 equilibration effects. The remaining 639 functional images were reoriented to the anterior/posterior commissures (AC–PC) plane, slice time corrected to the middle slice and spatially realigned to the first volume, resampled to an isotropic voxel size of 2 mm, normalized to the standard MNI space using the EPI reference brain and spatially smoothed with a 6 mm full width at half maximum (FWMH) isotropic Gaussian kernel. Statistical analysis was carried out using the general linear model framework implemented in SPM2 (Friston et al., 1995).

As the first level analysis, nine effects of interest were modelled: four represented the trials where subjects perceived emotional expressions or neutral face and body videos; four represented the scrambled counterparts and one the oddball condition. Null events were modelled implicitly. The BOLD response to the stimulus onset for each event type was convolved with the canonical haemodynamic response function (HRF) and high-pass filtered using a cut-off frequency of 1/128 Hz. We computed the images of parameter estimates of the eight contrasts of interest with a 6-mm FWHM isotropic Gaussian kernel and estimated the following main effects at the first level:

(1) Main effect of body vs. face [emotion + neutral (body vs. face)];
(2) Main effect of face vs. body [emotion + neutral (face vs. body)];
(3) Main effect of emotion vs. neutral [emotion vs. neutral (face + body)].

At the second level of analysis, we performed within-subjects correlation analyses examining the contrast between threatening and neutral videos and social inhibition and negative affectivity scores. Social inhibition and negative affectivity were included in the same regression model. We performed a correlation analysis with social inhibition and one with negative affectivity and four conjunction analyses to investigate areas that are (1) positively correlated with both scales, (2) negatively correlated with both scales, (3) positively correlated with social inhibition and negatively with negative affectivity and, (4) negatively correlated with social inhibition and positively with negative affectivity. Our goal was to study common modulations by threat in areas involved in processing faces and bodies, rather than studying specific modulations by fear or anger. As such, we decided to include only six contrasts in the final analysis, i.e., six covariates were included to capture residual movement-related artefacts (three rigid-body translations and three rotations determined from initial spatial registration), and a single covariate representing the mean over scans. To remove low frequency drifts from the data, we applied a high-pass filter using a cut-off frequency of 1/128 Hz. We estimated the images of parameter estimates of the eight contrasts of interest with a 6-mm FWHM isotropic Gaussian kernel and estimated the following main effects at the first level:

For all statistical maps, we report activations that survived the threshold of p < .001, uncorrected, with a minimum cluster extent of 15 contiguous voxels. Statistical maps were overlaid onto the SPM’s single subject brain template with MNI space, i.e., Colin27 (Holmes et al., 1998) in the anatomy toolbox (www.fz-juelich.de/ime/spm/anatomy toolbox (Eickhoff et al., 2005)). The atlas of Duverynoy (1999) was used for macroscopical labeling.

3. Results

Scores on the negative affectivity trait ranged from 0 to 19 (M = 6.54, SD = 4.28), five individuals scored ≥10. Scores on the social inhibition trait ranged from 1 to 16 (M = 7.65, SD = 4.41), eight individuals scored ≥10. The two trait subscales were correlated (r = .402, p < .05). The scores on the questionnaire of the participants who participated in the anger-neutral version of the experiment were similar to the scores from the students that participated in the fear-neutral version (negative affectivity: M = 6.21, SD = 4.08 vs. M = 6.92, SD = 4.66. t(24) = .410, p = .69; social inhibition: M = 7.50, SD = 5.33 vs. M = 7.83, SD = 3.25 t(24) = .188 p = .85). Three individuals met criteria for Type D personality. These scores are similar to norms for this age group. In a study that included 167 students, scores were as follows: negative affectivity: M = 7.49, SD = 5.34; social inhibition: M = 9.06, SD = 5.24 (Kupper & Denollet, 2007).

3.1. Negative affectivity

We observed a negative correlation between the negative affectivity score and activity in amygdala, right hippocampus, orbitofrontal cortex, cingulate cortex, temporal pole, right insula,
Fig. 1. Neural correlates with negative affectivity and social inhibition. A negative correlation between negative affectivity and activity following threatening versus neutral videoclips was observed in amygdala ($r^2(2, 23) = .338$) and insula ($r^2(2, 23) = .378$). The correlations appear to be driven by an outlier (an individual with an negative affectivity score beyond that of the group mean). However, these results are to be considered very robust, since after this subject was removed and the analysis was performed again, the correlations remained significant. Increased activation following threatening versus neutral videoclips stimuli was observed in the left temporo-parietal junction with more social inhibition traits ($r^2(2, 23) = .372$). See Tables 1–3 for the full list of activations.

3.2. Social Inhibition

A broad network was positively related to social inhibition; orbitofrontal cortex, left superior frontal gyrus, inferior frontal gyrus pars Triangularis (Brodmann area 45), right medial temporal pole, right primary somatosensory cortex (Brodmann area 3a), superior temporal sulcus, left temporo-parietal junction, inferior temporal gyrus, right fusiform gyrus, left middle occipital gyrus and the visual cortex. See Fig. 1 and Table 2 for the full list of activations.

3.3. Negative affectivity and social inhibition

We did not find areas similarly correlating with social inhibition and negative affectivity. However, a conjunction between a negative correlation with negative affectivity and a positive correlation with social inhibition showed common patterns in the orbitofrontal cortex, inferior frontal gyrus (Brodmann area 45), primary somatosensory cortex (Brodmann area 3b), right medial temporal pole, left middle temporal gyrus, superior temporal sulcus and left temporo-parietal junction (see Table 3). There were no regions that were positively correlated with negative affectivity and negatively with social inhibition.
Positive correlation with social inhibition and threatening versus neutral videoclips.

<table>
<thead>
<tr>
<th>Brain regions</th>
<th>MNI coordinates</th>
<th>Cluster</th>
<th>z-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left/right superior medial gyrus</td>
<td>±14.62 ±14.62</td>
<td>2585 *</td>
<td>3.48</td>
</tr>
<tr>
<td>Left/right rectal gyrus</td>
<td>8.50 ±8.50</td>
<td>44</td>
<td>2.98</td>
</tr>
<tr>
<td>Left middle frontal gyrus</td>
<td>−30 52 ± 16</td>
<td>2585 ± 3.58</td>
<td></td>
</tr>
<tr>
<td>Right/left orbitofrontal cortex</td>
<td>±24 ±30 ±10</td>
<td>2585 ± 3.60</td>
<td></td>
</tr>
<tr>
<td>Right middle frontal gyrus</td>
<td>26 42 ±6</td>
<td>2585 ± 3.51</td>
<td></td>
</tr>
<tr>
<td>Left middle frontal gyrus</td>
<td>−36 ±34.50</td>
<td>35</td>
<td>3.11</td>
</tr>
<tr>
<td>Right inferior frontal gyrus pars Triangularis (BA 45)</td>
<td>48 ±30 ±14</td>
<td>2585 ± 4.00</td>
<td></td>
</tr>
<tr>
<td>Right/left temporal pole</td>
<td>44 16 ±22</td>
<td>599 ± 3.16</td>
<td></td>
</tr>
<tr>
<td>Right insula</td>
<td>34 ±22</td>
<td>138 ± 3.75</td>
<td></td>
</tr>
<tr>
<td>Right insula</td>
<td>44 ±10 ±10</td>
<td>56 ± 3.03</td>
<td></td>
</tr>
<tr>
<td>Right rolandic operculum (BA 3a)</td>
<td>54 ±12.26</td>
<td>299 ± 4.69</td>
<td></td>
</tr>
</tbody>
</table>

MNI coordinate: * = right hemisphere.
MNI coordinate: = left hemisphere.

Table 2
Positive correlation with social inhibition and threatening versus neutral videoclips.

<table>
<thead>
<tr>
<th>Brain regions</th>
<th>MNI coordinates</th>
<th>Cluster</th>
<th>z-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left orbitofrontal cortex</td>
<td>−16.62 ±16.62</td>
<td>47</td>
<td>2.97</td>
</tr>
<tr>
<td>Right/left inferior frontal gyrus pars Triangularis (BA 45)</td>
<td>±52 ±12.6</td>
<td>403 ± 3.29</td>
<td></td>
</tr>
<tr>
<td>Right inferior frontal gyrus pars Orbitalis (BA 45)</td>
<td>52 30 ±2</td>
<td>403 ± 3.36</td>
<td></td>
</tr>
<tr>
<td>Right medial temporal pole operculum/rolandic operculum (BA3a) (SI)</td>
<td>42 8 ±30</td>
<td>46 ± 3.03</td>
<td></td>
</tr>
<tr>
<td>Right/fusiform gyrus</td>
<td>54 ±12 ±26</td>
<td>131 ± 3.93</td>
<td></td>
</tr>
<tr>
<td>Left tempo-parietal junction</td>
<td>50 ±30 ±20</td>
<td>303 ± 3.38</td>
<td></td>
</tr>
</tbody>
</table>

MNI coordinate: * = right hemisphere.
MNI coordinate: = left hemisphere.

4. Discussion

A growing literature demonstrates that different personality traits are associated with specific activity patterns in the brain when people are faced with threat (Campbell-Sills et al., 2010; Canli et al., 2001; Cremers et al., 2009; Ebanks et al., 2009; Jimura, Konishi, Asari, & Miyashita, 2010; Kugel et al., 2008; Perez-Edgar et al., 2007; Reker et al., 2010). Our main results are threefold. First, the observed amygdala decrease in high negative affectivity scorers for threatening facial and bodily expressions is similar to what we found earlier by the use of static stimuli. Second, the orbitofrontal cortex, left temporo-parietal junction and right extrastriate body area showed increased activity for threatening stimuli in high scorers on the social inhibition scale. Third, the orbitofrontal cortex, superior temporal sulcus, inferior frontal gyrus (Brodmann area 45) and temporal pole correlated negatively with negative affectivity and positively with social inhibition. The first two findings are in line with our expectations, but the third one is different from what we first predicted. Below we elaborate on these findings in more detail.

In line with our expectations, we observed decreased activity for threatening videos in the amygdala in a whole brain analysis along with right hippocampus, orbitofrontal cortex, cingulate cortex, temporal pole, right insula, inferior frontal gyrus (including Brodmann area 44/45), fusiform gyrus, superior temporal sulcus, and temporo-parietal junction. These areas are widely reported in the emotion literature and also in studies on structural abnormalities in depression (including a reduced volume of the orbitofrontal and cingulate cortex, insula and amygdala/parahippocampal region) (Lee et al., 2007). Whereas some studies report a decrease in activity (Thomas et al., 2001) or no difference (Lee et al., 2008), others report increased amygdala response to threatening versus neutral expressions related to depressive symptoms (Canli et al., 2005; Peluso et al., 2009). Beesdo et al. (2009) observed amygdala hypoactivation in major depressive disorder patients while passively viewing fearful faces, and amygdala hypoactivation when actively rating how afraid they were when seeing a fearful face. Similarly, healthy behaviorally inhibited adolescents, relative to non-inhibited peers, showed exaggerated amygdala response during subjective fear ratings and deactivation during passive viewing of emotional faces (Perez-Edgar et al., 2007). These inconsistencies may thus originate from differences in the specific task and attention load between studies. Importantly, our results are in line with those using implicit tasks and show that deactivation of the amygdala was paired with deactivation of other emotion areas in the brain.
Whereas we found cortical but also subcortical structures correlating negatively with negative affectivity, we found a broad but exclusively cortical network which activity pattern was positively correlated with social inhibition. These regions (including temporo-parietal junction, superior temporal sulcus, inferior frontal gyrus (Brodmann area 45) and orbitofrontal cortex) are jointly involved in perceiving the action goal of the observed (Van Overwalle & Baetens, 2009). Observing and imitating facial expressions both activate the inferior frontal gyrus (Brodmann area 45) similarly (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003). The temporo-parietal junction plays an important role in mentalizing and computes the orientation or direction of the observed behavior in order to predict its goal (Decety & Lamm, 2007). As predicted, we observed a positive correlation between threat-related activity in these areas and scores on the social inhibition scale. Pelphey, Morris, and McCarthy, (2005) report that observing whole-body motion and gaze engage the posterior superior temporal sulcus and most likely reflect an orientation response in line with the action or attention of the observed. So, the observed increased activity in the superior temporal sulcus may indicate increased vigilance in individuals who have a tendency to inhibit socially. This explanation is plausible since there was also a positive correlation with V1 which may point to increased attention (Somers, Dale, Seiffert, & Tootell, 1999). Hyperactivity in these cortical structures does not necessarily mean, and probably does not mean, better function.

Since Type D personality is associated with a broad range of health issues and somatic responses, we were specifically interested in the combined influence of social inhibition and negative affectivity on threat-related brain activity. We did not find areas with activity patterns correlating positively or negatively with both social inhibition and negative affectivity. Instead, whereas the orbitofrontal cortex and somatosensory cortex correlated positively with social inhibition, they correlated negatively with negative affectivity. The orbitofrontal cortex is connected with areas that underlie emotional function and empathy (Hynes, Baird, & Graf ton, 2006) and interprets somatic sensations (Bechara, Tranel, & Damasio, 2000) mediated by internally generated somatosensory representations that simulate how the other person would feel when displaying a certain emotion (Adolphs, 2002b). Without the ability to re-activate emotion-related somatic markers in the orbitofrontal-limbic circuit, behavior lacks planning. Whereas the orbitofrontal cortex and somatosensory cortex correlated positively with social inhibition, they correlated negatively with negative affectivity. The function of the orbitofrontal cortex is complex and dependent on the exact location, and functional asymmetry in emotional processing has been reported earlier (Kringelbach & Rolls, 2004). With fMRI, we can never be sure of the exact time frames of the two networks underlying social inhibition and negative affectivity. It may well be that the decrease in activity as observed in high negative affectivity scorers in a number of (subcortical) regions preceded the increase in cortical regions in participants with higher social inhibition scores.

A number of limitations should be considered when interpreting the results of our study. One limitation of our study is the small sample size which resulted in statistical comparisons losing significance when correcting for multiple comparisons. A bigger sample size would have allowed us to investigate the full Type D personality construct in more detail and also differentiate between fear and anger. Further research is needed to link threat related brain activity in different personality traits including the Type D personality trait which is characterized by high negative affectivity and high social inhibition. In a follow-up experiment it would be interesting to include participants based on their scores on the DS14 and compare participants that score high on social inhibition and high on negative affectivity with participants that score low on both scales, but also with participants that score extreme on one scale and not on the other. Moreover, further studies need to demonstrate the validity of present findings in clinically relevant samples. One of our participants, whom we excluded from the analyses, reported suffering from ADD. It turned out later, that he misunderstood the task instructions (he pressed a button following each scramble instead of during the presentation of inverted videos). To prevent this from happening again in future studies, we strongly recommend a clinical interview such as the SCID (Structured Clinical Interview for DSM-IV Axis I Disorders) (Gibbon, Spitzer, Williams, Benjamin, & First, 1997) beforehand, and the use of additional questionnaires such as the Beck Depression Inventory (Beck, 2006). To summarize, the current study investigates the normal variance in negative affectivity and social inhibition scores in healthy participants and relate the between-subject differences to between-subject differences in brain reactivity. This makes clear that subclinical individual differences in negative affectivity, characterized by the tendency to worry and feel unhappy, etc., are also related to differences in reactivity of the emotional brain. Moreover, the present findings reveal that social inhibition may be marked by a sensitivity to over-mentalize and empathize when perceiving threat. Altogether, this study demonstrates that negative affectivity and social inhibition are differentially related to emotion-specific brain activation that may be relevant to both physical and mental health. Our results support that the network of brain regions involved in emotion regulation may be relevant to the relationship between medical and psychological disorders. Therefore, their assessment should be considered in neuroimaging studies on emotion regulation and stress reactivity.

Acknowledgements

This study was partly supported by Human Frontiers Science Program RGP54/2004, NWO Nederlandse Organisatie voor Wetenschappelijk Onderzoek 400.04081 to BdG, VICI grant 453.04004 (to JD) and EU FP6-NEST-COBL043403 and FP7 TANGO to BdG. We thank three reviewers for their helpful and insightful comments and suggestions.

References


