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Obsessive-compulsive disorder – A question of conscience? An fMRI study of behavioural and neurofunctional correlates of shame and guilt

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ABSTRACT

Shame and guilt can be described as 'self-conscious emotions' and are an essential part of the psychopathology in obsessive-compulsive disorder (OCD). Our primary aim was to explore whether individuals with OCD are processing shame and guilt differently from healthy individuals (N = 20 in both groups; 50% female; age: 20–40 years) on the behavioural and neurobiological level.

For the experimental task, participants were scanned with functional magnetic resonance tomography (functional magnetic resonance imaging, 3 T) while imagining neutral, shame inducing and guilt inducing scenarios. In addition to clinical questionnaires, participants were asked to complete questionnaires measuring shame and guilt.

The functional data indicate an increased activity in OCD patients in the shame condition in the limbic, temporal and sub-lobar (hypothalamus) areas, in the guilt condition inter alia in frontal, limbic and temporal areas. In summary we found activity in OCD patients in neural networks which are responsible for stimulus filtering, emotion regulation, impulse control and memory. The results from our study may contribute to a better understanding of the origins and maintenance of OCD in association with the pathological processing of shame and guilt on different functional levels.

1. Introduction

In general, emotions like shame, embarrassment and guilt are termed "self-conscious" because individual comprehension and evaluation of the self are important to generate these emotions (Eisenberg, 2000). Shame and guilt are not only posited to be critical for self-development but also crucial for the development of malfunctioning self and maintenance of symptoms in psychological diseases like obsessive-compulsive disorder (OCD, Fergus et al., 2010). The neurobiological underpinnings of self-conscious emotions have been recently investigated in studies on healthy samples (e.g. Moll et al., 2002; Greene et al., 2001; Berthoz et al., 2006; Green et al., 2010; Takahashi et al., 2008). Convergent findings suggest the involvement of frontal, temporal and limbic brain regions in processing embarrassment and guilt which were provided by lesion studies in patients and imaging studies in healthy subjects (e.g. Basile et al., 2011; Beer et al., 2003; Berthoz et al., 2002; Devinsky et al., 1982; Krajbich et al., 2009; Takahashi et al., 2004). Functional brain imaging studies on processing guilt provide support for an associated distributed activation in frontal and temporal regions (Shin et al., 2000; Takahashi et al., 2004; Michl et al., 2014). Up to now, two studies have investigated the differences of shame and guilt in healthy subjects with functional brain imaging (Michl et al., 2014; Takahashi et al., 2004). Takahashi and colleagues found similarities for processing embarrassment and guilt in the

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medial prefrontal cortex, the left posterior superior temporal sulcus (STS) and the visual cortex (Takahashi et al., 2004). For embarrassment they found a distinctly greater activation in the right temporal cortex and hippocampus relative to guilt. In a German sample (Michl et al., 2014), activations were observed in distinct temporal networks for both emotions. Shame-specific neural responsiveness occurred in the medial and inferior frontal gyrus, whereas neural responsiveness in the amygdala and in the insula was associated with guilt.

Recent clinical research suggests that the aetiology of OCD is rooted in maladaptive strategies of thought control (Amir et al., 1997) and in a dysfunction of the neurotransmitter serotonin (e.g. Insel, 1985; Matsumoto et al., 2010; Pigott and Seay, 1999). Altered functional brain activity, particularly in prefrontal areas was found in OCD patients (e.g. Friedlander and Desrocher, 2006; Ogai et al., 2005). Hence, integrative models of the neurobiology of OCD include an executive dysfunction (Kis et al., 2007).

Main impairments in OCD seem to be a flawed impulse control and a disturbed modulation of socially appropriate behaviour. Pathophysiological models of OCD (e.g. Chamberlain et al., 2008; Graybiel and Rauch, 2000; Harrison et al., 2009; Modell et al., 1989; Saxena et al., 1998) suggest a dysfunction of a neural fronto-striatal circuit. Studies in paediatric OCD support this circuit model with an emphasis on the thalamus (Huyser et al., 2009; MacMaster et al., 2008). In adult studies crucial dysfunctions of the caudate nucleus and orbitofrontal structures are reported (Sakai et al., 2011).

Recent research in functional magnetic resonance imaging (fMRI) suggests that even more regions are affected in OCD, such as the anterior cingulate, insula, amygdala and hippocampus (Breiter et al., 1996; Cocchi et al., 2012; Menzies et al., 2008; Radua and Mataix-Cols, 2009; Schiepek et al., 2007). These regions are involved in emotional processing and its interaction with cognition and behaviour (Devinsky et al., 1995; Calder et al., 2000; Wiens, 2005; Bar-On et al., 2003; Schienle et al., 2005; Ciocchi et al., 2010; Richardson et al., 2004). Several studies have provided evidence for pathological emotional processing in OCD patients (Ursu and Carter, 2009; Mancini and Gangemi, 2004).

In summary, there is already evidence on dysfunctional interaction of emotional and cognitive/behavioural processes in OCD and OCD-specific changes in structural and functional brain networks related to emotions, memory and executive functions. The prior studies investigated basic emotion processing in OCD, however, moral or self-related emotions have not yet been considered to play a crucial role in OCD, though the role of self and an increased norm orientation have been reported (Fergus et al., 2010).

Hence, to our knowledge, this is the first study investigating OCD-specific differences in experiencing shame and guilt on different processing levels: neurobiological, measured as change of brain activation related to emotional imagination and subjective experiences, assessed by patients' self-reports. In OCD participants, we expect an increased activation in brain areas related to shame and guilt, such as prefrontal, temporo-parietal and limbic regions and augmented self-reported feelings of shame and guilt.

2. Method

2.1. Sample characterization

Overall, forty participants were included in the study. Twenty OCD patients were compared with twenty healthy controls matched for age, gender (50% women in each group) and education. The groups did not differ in their estimated verbal intelligence level assessed by using a German Vocabulary Test (see Table 1, Wortschatztest, Schmidt and Metzler, 1992).

Exclusion criteria for the study were as follows: aged below 18 or over 45, estimated IQ lower than 85, any physical or neurological diseases or mental retardation, magnetic metals in the body, current pregnancy.

All participants gave written consent. The study was accepted by the Ethics Committee of the Medical Faculty of the LMU Munich. Both experimental groups received an allowance up to 20 Euro for their expenses. OCD patients were recruited through the Clinic of Psychiatry and Psychotherapy of the Ludwig-Maximilians-University and the Clinic of Psychosomatic Medicine and Psychotherapy, Munich. Diagnoses were made according to the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV; Saβ et al., 1998) and the Structured Clinical Interview for the Diagnostic Schedule for Mental Disorders—Fourth Edition (SCID-I and -II; Wittchen et al., 1997). Severity of OCD symptoms was assessed with the Yale-Brown Obsessive Compulsive Scale (Y-BOCS, Goodman et al., 1991) (Table 1).

For assessing symptoms of comorbid depression and anxiety the Beck Depression Inventory (BDI, Hautzinger et al., 1995) and the German version of the State-Trait Anxiety Inventory were used (STAI, Laux et al., 1997). According to the SCID-I, none of the participants received any additional comorbid axis I diagnoses. However, OCD patients reported significantly more depressive symptoms on the BDI and more anxiety both on the state and the trait scale of the STAI (Table 1).

2.2. Questionnaires on general life-time experience of shame and guilt

For assessing shame, the Heidelberger questionnaire on shame (German: Heidelberger Fragebogen zu Schamgefühlen, HFS, Kämmerer et al., HFS, 2004) was used to capture the intensity of different feelings of shame, including two subscales, one reflecting shame with regard to one's own body and own sexuality, the second reflecting shame regarding achievement and social competence. To refer to the reliability Cronbach's Alpha constitutes 0.84, additional HFS has a good discriminative validity (Pearson's $r = 0.6$). To investigate the interpersonal feeling of guilt, a questionnaire by Albani and colleagues (German: Fragebogen zu interpersonellen Schuldgefühlen, FIS, 2002) was used, which consists of three subscales: survivor guilt (i.e., the belief that individual success leads to the suffering of others), separation guilt (i.e., when a person is convinced, that the own autonomy or separation, e.g. from one's parents, will hurt loved-ones), and guilt through responsibility or duty (i.e., an exaggerated sense of duty and responsibility and worry over the happiness and wellbeing of others). No information on validity and reliability is given by the authors up to now. In the original version of the Interpersonal Guilt Questionnaire (IGO) Kugler and Jones (1992) reported internal consistency for trait guilt of .89, and of moral standards .81. The reliabilities were .72 for trait guilt, .56 for state guilt, and .81 for moral standards (O'Connor et al., 1997).

2.3. Experimental stimuli and procedure

Thirty neutral, thirty guilt- and thirty shame-related already pre-evaluated sentences were presented to the participants (Michl et al., 2014). Presentation and timing were programmed using the Presentation® software (Neurobehavioral Systems, 2005). Inside the MR scanner, the participants viewed the stimuli over a head-coil compatible mirror system (300 cm screen to mirror, 15 cm mirror to participant's eyes). Stimuli were projected on a translucent screen in white capitals (font: arial, font size: 16) on black background by a commercially available video beamer (INTouch, resolution of 1024 × 768 pixel) protected by a faraday cage.