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Factors contributing to social cognition impairment in borderline personality disorder and schizophrenia

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ABSTRACT

Social cognition (SC) deficits have been described both in patients with schizophrenia and borderline personality disorder (BPD). However, while the former tend towards simplistic mental state attributions (undermentalizing), the latter are more likely to make overly complex mental state inferences (overmentalizing). Performance on complex SC tasks has been shown to correlate with neurocognitive ability, emotion perception, a history of trauma, and overconfidence in errors. However, it is unclear how these factors relate to different aspects of SC deficits. Aim of the present study was to examine the pathways of SC impairment by investigating performance profiles and their predictors comparatively in BPD and schizophrenia. Participants were 44 patients with BPD, 36 patients with schizophrenia, and 38 healthy controls. Undermentalizing and overmentalizing were assessed with an ecologically valid SC task. Patients with BPD exhibited increased overmentalizing, whereas patients with schizophrenia showed a more extensive deficit pattern, their main error type being undermentalizing. Overconfidence in errors was the most important predictor for overmentalizing, while undermentalizing depended mainly on verbal memory and emotion perception. Thus, BPD und schizophrenia exhibited different SC impairment patterns, and different types of SC errors were predicted by different factors. These findings have implications for the optimization of treatment approaches.

1. Introduction

Social cognition can be defined as the sum of mental processes associated with the perception and interpretation of stimuli pertinent for social interaction as well as with the response to these stimuli (Bell et al., 2010; Chung et al., 2010; Green et al., 2008).

In patients with schizophrenia, studies have provided solid evidence for impairments in various domains of social cognition: processing facial or prosodic information (Castagna et al., 2013; Chung and Barch, 2011; Silver et al., 2009); integrating emotional stimuli in their respective context in more complex, ecologically valid tasks (Bell et al., 2010; Chung and Barch, 2011; Koether et al., 2012); and using social cues to infer thoughts, feelings and intentions of others (Theory-of-Mind [ToM]: Brune, 2005; Harrington et al., 2005). These impairments are already present before the onset of the disorder (Addington et al., 2008; Davidson et al., 1999; Dworkin et al., 1993; Gibson et al., 2010; Green et al., 2012; Phillips and Seidman, 2008; Schiffman et al., 2004) and are rather stable over time (Addington et al., 2006; Green et al., 2012; Penn et al., 2008), independent of clinical fluctuations (Addington and Addington, 2008) and pharmacological treatment (Addington et al., 2008; Hempel et al., 2010; Kohler et al., 2010; Mueser et al., 1996; Salem et al., 1996; Wolwer et al., 1996). Importantly, social cognition has been shown to predict social and community functioning as well as vocational achievement (Bell et al., 2009; Couture et al., 2006; Green et al., 2008).

Research into social cognition is not limited to schizophrenia, however, but encompasses several other severe psychiatric disorders that affect psychosocial functioning. Of particular interest in this regard is borderline personality disorder (BPD), a chronic disorder characterized by pervasive instability of mood, interpersonal relationships and behavior (DSM-IV) with serious affects in social functioning (Zanarini et al., 2005). BPD is defined by three major symptom clusters: affective dysregulation, impulsivity, and disturbed relatedness (Sanislow et al., 2002; Skodol et al., 2005). Until recently, research on BPD has mainly focused on the two former dimensions. However, disturbed relatedness has been suggested to constitute the best discriminator for a diagnosis of BPD (Gunderson, 2007), and social situations have been shown to

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be potent triggers for another important dimension, affective instability (Ebner-Priemer et al., 2007), thus making research on social cognition particularly relevant for BPD.

The vast majority of social cognition studies in patients with BPD have concentrated of facial emotion recognition. Results have been rather equivocal, showing decreased (Bland et al., 2004; Levine et al., 1997), spared (Domes et al., 2008; Lynch et al., 2006), or even increased (Wagner and Linehan, 1999) ability to recognize facial emotions. More consistent impairment patterns have been reported with use of social cognition tasks of increased complexity that require integration of multiple cues (Dyck et al., 2009; Minzenberg et al., 2006; Preissler et al., 2010). With use of such more ecologically valid tasks, it has been shown that patients with BPD display a tendency towards 'overmentalizing' (i.e., overly complex or exaggerated attribution of mental states; Sharp et al., 2013', 2011). Interestingly, this may represent a different impairment pattern than that encountered in patients with schizophrenia. In the latter, a tendency for inappropriate mental state attributions has also been described (Okruszek et al., 2015), and reported to be associated with delusion severity (Fretland et al., 2015). However, these patients display more extensive impairment patterns, with a prominent tendency towards overly simplistic mental state inferences or even a complete lack of a mental concept ('undermentalizing'; Langdon et al., 2014; Montag et al., 2011; Ventura et al., 2015).

So far, possible underpinnings of impaired social cognition in BPD have received little attention. In the more widely researched field of schizophrenia, it has been shown that performance deficits on complex social cognition tasks do not only depend on impaired processing of simple facial/prosodic information, but also on non-social neurocognitive deficits such as verbal memory, executive functions or context processing (Bell et al., 2010; Chung et al., 2010; Koether et al., 2012; Langdon et al., 2014). In patients with BPD, studies have instead rather focused on trauma experiences and/or comorbidity with posttraumatic stress disorder (PTSD) (Dziobek et al., 2011; Preissler et al., 2010). So far, only one small study (Baez et al., in press) has assessed the contribution of cognitive deficits to impaired social cognition in BPD, despite the fact that patients with BPD have been suggested to exhibit similar neurocognitive deficits as patients with schizophrenia (LeGris and van Reekum, 2006; Mak and Lam, 2013; Ruocco, 2005).

A concept that might be relevant in this discussion is that of cognitive biases. Unlike elementary neurocognitive impairments (e.g., memory deficits), cognitive biases are best conceptualized as distorted thinking *styles*, i.e. deviations in stimulus integration and interpretation processes (Bell et al., 2006; Freeman, 2007). Examples of cognitive biases include a tendency to make strong judgments on the basis of poor evidence (jumping-to-conclusions (Huq et al., 1988)) or overconfidence in errors (Moritz et al., 2004). These abnormalities have been well replicated across patients with schizophrenia (Menon et al., 2008; Van Dael et al., 2006) and have been suggested to provide a link between social cognitive deficits and specific symptoms or behaviors in schizophrenia (Bell et al., 2006; Moritz and Woodward, 2006); for example, it has been suggested that theory-of-mind deficits and a jumping-to-conclusions bias might interact to give rise to persecutory delusions in schizophrenia (Garety and Freeman, 1999).

Previous studies have shown that cognitive biases, more particularly overconfidence in errors, extend into the social cognition domain in patients with schizophrenia. In two studies using different social cognition paradigms (Koether et al., 2012; Moritz et al., 2012), patients exhibited a clear pattern of increased confidence for error responses compared to healthy controls. It has been argued that overconfidence in errors is particularly relevant with respect to the behavioral consequences of false judgments: if a patient believes that a passenger in the bus *might* be looking angrily at them, it is unlikely that this will have a strong impact on their emotional status or behavior (at the most, they may switch seats); however, if the patient is highly *convinced* that their perception of the displayed emotion is accurate, behavioral consequences (e.g. withdrawal, open argument) and emotional distress (e.g. panic, anger, or intense insecurity) are more likely to follow (Moritz and Woodward, 2006). However, there is also another possibility, which has not been investigated so far – namely, that cognitive biases are not just relevant for behavioral consequences of social cognition deficits, but rather play a causal role in the misinterpretation of social stimuli (for example, by preventing the individual from assessing a given situation adequately). This question applies both to patients with schizophrenia and BPD, as the latter have been also reported to exhibit some of the cognitive biases typically encountered in schizophrenia (Moritz et al., 2011), especially overconfidence in errors (Schilling et al., 2013b).

In summary, evidence so far suggests that patients with BPD might exhibit social cognition deficits of a different nature than those observed in schizophrenia. Knowing which aspects of social cognition are impaired, and what the specific underpinnings of these impairments are, might be relevant for optimizing treatment approaches. In schizophrenia, treatment programs that specifically target social cognition deficits (Frommann et al., 2003; Horan et al., 2009; Penn et al., 2007) have been shown to ameliorate symptoms such as depression and anxiety, and produce moderate to large improvements in community function (Kurtz and Richardson, 2012). Thus, insights into the nature of social cognition deficits in BPD might help design more appropriate interventions for this disorder, the functional consequences of which persist over far longer time periods than actual symptoms (Gunderson et al., 2011), and in which pharmacological treatments have not shown robust effects (Leichsenring et al., 2011). Therefore, the aims of the present study were (a) to assess the patterns of social cognition deficits in patients with BPD compared to both healthy controls and patients with schizophrenia (as a well-characterized clinical control group), and (b) to investigate, using a wide set of variables, the predictors of two different aspects of social cognition deficits (undermentalizing and overmentalizing), which were expected to be differentially affected in the two patient groups.

2. Materials and methods

2.1. Participants

The present study included 44 patients with BPD, 36 patients with schizophrenia and 38 healthy control subjects. Patients were recruited from the in- and outpatient clinics of the Department for Psychiatry and Psychotherapy, University Hospital Hamburg-Eppendorf (Germany); healthy controls were recruited through advertisements and word of mouth. The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee; all participants were required to provide their informed consent prior to participation.

Patients of the two clinical groups were required to fulfill criteria of the respective disorder (BPD or schizophrenia) according to DSM-IV. Diagnoses (and absence thereof in healthy controls) were confirmed with the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) and the Structured Clinical Interview for DSM-IV Axis II (SKID-II; Wittchen et al., 1997). Exclusion criteria were a current major depressive episode of more than mild severity, neurological and developmental disorders, a history of alcohol or drug dependence, alcohol or drug abuse during the six months prior to testing, and the presence of uncorrected visual problems or hearing loss. Additional exclusion