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Metacognition in psychosis: Comparison of schizophrenia with bipolar disorder



Cumhur Tas^{a,b,*}, Elliot C. Brown^{b,c}, Omer Aydemir^d, Martin Brüne^b, Paul H. Lysaker^{e,f}

^a Department of Psychology, Uskudar University, Istanbul, Turkey

^b LWL University Hospital, Division of Cognitive Neuropsychiatry and Psychiatric Preventative Medicine, Ruhr University Bochum, NRW, Germany

^c Maryland Psychiatric Research Centre, University of Maryland School of Medicine, Baltimore, MD, USA

^d Department of Psychiatry, Celal Bayar University, Manisa, Turkey

^e Roudebush VA Medical Center, Indianapolis, IN, USA

^f Indiana University School of Medicine, USA

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ABSTRACT

While deficits in metacognition have been observed in schizophrenia (SZ), it is less clear whether these are specific to the disorder. Accordingly, this study compared metacognitive abilities of patients with schizophrenia and bipolar disorder (BD) and examined the degree to which neurocognition contributed to metacognitive deficits in both groups. Participants were 30 patients with SZ and 30 with BD. Metacognitive capacity was measured using the Metacognition Assessment Scale Abbreviated (MAS-A). This scale comprises four domains: self-reflectivity, understanding others' minds, decentration and mastery. Verbal memory, executive functioning and symptoms were concurrently assessed. Group comparisons revealed that SZ patients had greater deficits in metacognitive self-reflectivity, which correctly classified 85.2% of patients with SZ in a logistic regression. Self-reflectivity and understanding others' minds were related to verbal memory and executive functioning in the SZ group, but not in the BD group. Furthermore, greater positive and general psychotic symptoms were associated with poorer metacognition in SZ. Results suggest SZ involves unique deficits in the ability to self-reflect and that these deficits may be uniquely linked with neurocognition.

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1. Introduction

Schizophrenia is an often prolonged mental disorder that has debilitating effects on the cognitive faculties of the affected individuals. In the past, descriptions of cognitive deficits in schizophrenia were studied mostly in terms of impairments in memory (Gold et al., 1992), attention (Braff, 1993) and executive functioning (Green et al., 2000). However, a growing body of evidence has revised the architecture of these cognitive deficits by including synthetic cognitive capacities that allow persons to construct the kinds of integrated representations of self and other necessary to adapt to the changing demands of the social environment (reviewed in Lysaker et al., 2013).

“Metacognition” refers to a set of skills necessary for identifying mental states and ascribing them to oneself and others, which includes the deciphering of expressions of emotion, the reasoning about mental states and the use of mentalistic information in

order to decide, solve problems and interpersonal conflicts, and to master subjective suffering (Carcione et al., 2011; Brüne et al., 2011). Historically, metacognition was described as the memory processes that are related to the insights people have about their own cognitive processes. Developmental psychologists proposed that an increase in the ability of children in predicting their memory spans pave the ground for developing successful communication skills and problem solving capacities (Flavell, 1979; Brown, 1987). This view was expanded when researchers turned from discrete mental acts to more synthetic ones in which an array of pieces of information were integrated into the complex representations of one's own mental states and those of others (Semerari et al., 2003).

While deficits in metacognition have been linked with a range of poor outcomes in schizophrenia, it is unclear whether impairments in metacognitive thinking are unique to schizophrenia. For instance, patients with borderline and narcissistic personality disorders have been reported to have difficulties in reflecting on social exchanges from multiple perspectives (Dimaggio et al., 2007). Along the same lines, Carcione et al. (2011) have reported persons with a number of different personality disorders have significant difficulties using metacognitive knowledge to respond

* Corresponding author at: Department of Psychology, Uskudar University, Altunizade Mah. Haluk Türksoy Sk. No:14 PK:34662 Üsküdar, Istanbul, Turkey. Tel.: +90 216 400 22 22; fax: +90 216 474 12 56.

E-mail address: cumhur.tas@uskudar.edu.tr (C. Tas).

to psychological and social challenges. Several researchers have also found association between difficulties naming emotions in the presence of cluster C traits (Nicolò et al., 2011). Patients with Major Depression have been reported to have difficulties in making social decisions on the basis of metacognitive knowledge (Papageorgiou et al., 2003) and to be generally less aware of their own emotional states (Honkalampi et al., 2001). More recently, Ladegaard et al. 2014 replicated these findings and found that depressed patients had significantly more difficulties in forming complex integrated representations of themselves and other than healthy controls. Moreover, patients with somatic and substance abuse disorders have been reported to lack awareness of emotions and the events which evoke negative emotions (Taylor et al., 1997; Honkalampi et al., 2001; De Rick, Vanheule 2007; Lane, 2008). Lastly, a more complex relationship among metacognitive function has been suggested in substance disorders with one report finding that the ability to use metacognitive knowledge moderated the impact of unawareness of emotions on the severity of cluster C traits (Lysaker et al., 2014).

While metacognitive dysfunction has been found in a range of disorders, one possibility is that more severe metacognitive deficits are unique to schizophrenia. In support of this assumption, one study found schizophrenia patients in both early and later phases of illness had more difficulties forming complex representations of self and other than substance abuse patients, though no significant differences were found between groups for the ability to use metacognitive knowledge to respond to psychological and social challenges (Vohs et al., 2014). One potential unique cause of more severe deficits in metacognition in schizophrenia could relate to patients' greater impairments in neurocognitive functioning, including impairment in verbal memory and executive function that distinguish this disorder from other forms of psychosis (Lysaker et al., 2008). For instance, it is possible that schizophrenia patients with poor executive functioning are less able to formulate alternative ways to understand life events and shift fluidly between different perspectives. Similarly, patients with deficits in verbal memory may have difficulties remembering and integrating different life experiences in a nuanced manner and thus may lose a sense of previous experience, which provide a context for relatively richer understanding of oneself and others. Consistent with this notion, several studies have found that greater impairments in verbal memory, executive functioning, intelligence and processing speed were linked with the ability to form complex representations of the self (Lysaker et al., 2005; Nicolò et al., 2012). In another study, different forms of executive function were related to different forms of metacognition in schizophrenia with self-reflectivity more closely linked to mental flexibility and the ability to use metacognitive mastery more closely linked to the ability to inhibition (Lysaker et al., 2008).

Of note, while there is some evidence that deficits in neurocognition are linked to metacognitive deficits in schizophrenia, the question of the relationship of neurocognition to metacognition in other disorders is a matter of open debate. For one, it is possible that potentially lesser levels of neurocognitive compromise found in other forms of chronic mental disorders with psychotic like episodes such as bipolar disorder (BD) are also linked with the albeit lesser levels of metacognitive compromise. In a recent comparative study, both schizophrenia and bipolar patients demonstrated profound deficits in some domains of social cognition compared to controls, yet neurocognitive impairment was less in bipolar patients than in schizophrenia patients (Caletti et al., 2013). However, deficits in theory of mind (ToM), a construct related to metacognition, which reflects the ability to make attributions about the mental states of others, did not differ between bipolar patients and healthy controls in the same study. As an alternative explanation, mood swings of bipolar patients

may affect metacognitive capacity. In line with this, Bora et al. (2009), as well as Wolf et al. (2010), demonstrated that ToM deficits were a state rather than a trait marker in bipolar patients. One explanation for this is that bipolar patients in a depressive episode may detach their attention from the social cues in the environment or their internal world, and in the manic stage, the over-activated behavioral approach systems may compromise the appraisal and understanding of mental states (Alloy and Abramson, 2010). Given all, it is possible that metacognitive compromises in non-psychotic patients have other causes and are not that tightly linked with neurocognition (Olley et al., 2005).

To explore this possibility, the current study sought to determine whether persons with schizophrenia experience more severe deficits in metacognition than persons with another form of severe mental disorder, namely bipolar I disorder patients in the euthymic stage, i.e. a stage where the mood of the affected patients is relatively stable. We predicted that the schizophrenia group would demonstrate graver impairment in four domains of metacognition: "Self-reflectivity," the comprehension of one's own mental states, "Understanding the Mind of the Other," the comprehension of other individuals' mental states, "Decentration," the ability to see the world as viewable from multiple perspectives, and "Mastery", the ability to use metacognitive knowledge to address social and psychological dilemmas. Evidence supporting these possibilities includes research suggesting that in contrast to schizophrenia patients, patients with Bipolar I disorder had milder deficits in ToM (Kerr et al., 2003; Lahera et al., 2008; Van Rheenen and Rossell, 2013). These findings were further supported in bipolar I patients with known psychotic features by a recent study (Thaler et al., 2013).

We were also interested in whether levels of neurocognitive deficit have similar relationships with metacognition in patients with schizophrenia and bipolar I disorder. Here we considered two rival hypotheses. First, it was possible that in both groups poorer neurocognition would be related to poorer metacognition. An alternative hypothesis was that it is also possible to predict that there should be no relationship between executive function and metacognitive function exclusively in the bipolar disorder group.

2. Method

2.1. Participants

Thirty patients with schizophrenia and 30 patients with bipolar I disorder in euthymic stage were recruited from the Celal Bayar University, psychosis and affective disorders units. The patients met DSM-IV criteria for schizophrenia and bipolar I disorder as determined by medical records and diagnosis was confirmed with the Structured Clinical Interview for DSM-IV – Patient Edition (SCID; First et al., 2002). According to the medical reports, 3 patients in the bipolar group who were receiving high doses of second generation antipsychotics had experienced psychotic like episodes without any affective component and, therefore, were excluded from the study prior data analyses. Inclusion criteria included clinical stability as defined by no change in medication dosage in the last three months, and no hospitalization in the last 6 months before recruitment for the study. Exclusion criteria included neurological comorbidities such as epilepsy and comorbid drug and alcohol abuse. All schizophrenia patients received second-generation antipsychotic medication. Mean chlorpromazine equivalent dosages (CPZ) were 402.19 ± 225.53 mg per day (see Rijcken et al., 2003 for calculation). All bipolar patients were medicated and received mood stabilizers (35% received lithium; 40% valproate; 20% lamotrigine; 5% second generation antipsychotics with mood stabilizing features i.e. quetiapine). All patients provided written informed consent. The study was approved by the local Institutional Review Board. The socio-demographical and clinical data of the study group with statistics are summarized in Table 1.

2.2. Clinical and neuropsychological measures

2.2.1. Positive and Negative Syndrome Scale (PANSS)

The PANSS (Kay et al., 1987) is a 30-item rating scale that was administered by clinically trained research staff using a chart review and a semi-structured

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