Metacognition in psychosis: Comparison of schizophrenia with bipolar disorder

Cumhur Tas, Elliot C. Brown, Omer Aydemir, Martin Brüne, Paul H. Lysaker

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

Keywords: Schizophrenia, Metacognition, Neurocognition, Bipolar disorder
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; Lahtera et al., 2008; Van Rheenen and Russell, 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 1 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, psychiatry 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
Table 1
Sociodemographic and clinical data between schizophrenia and bipolar patients.

<table>
<thead>
<tr>
<th>Group</th>
<th>Bipolar disorder</th>
<th>Schizophrenia</th>
<th>Stats.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Count (%)</td>
</tr>
<tr>
<td>Age</td>
<td>40.70</td>
<td>11.46</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>12(44.4%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>15(55.6%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education (years)</td>
<td>11.30</td>
<td>3.94</td>
<td></td>
</tr>
<tr>
<td>Duration of illness (years)</td>
<td>13.07</td>
<td>9.77</td>
<td></td>
</tr>
<tr>
<td>Number of hospitalization</td>
<td>1.85</td>
<td>1.83</td>
<td></td>
</tr>
<tr>
<td>PANSS – Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PANSS – Positive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PANSS – General</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamilton Depression</td>
<td>1.07</td>
<td>1.69</td>
<td></td>
</tr>
<tr>
<td>Young-Mania</td>
<td>0.56</td>
<td>1.01</td>
<td></td>
</tr>
<tr>
<td>MQ</td>
<td>8826</td>
<td>15.04</td>
<td></td>
</tr>
</tbody>
</table>

Notes for Table 1: PANSS; Positive and Negative Syndrome Scale, MQ; Memory Quotient.

2.2.2. Hamilton Depression Scale (HDRS)

The HDRS is a 17-item, clinician-rated scale used to evaluate the severity of depressive symptoms (Hamilton, 1967). The HDRS total scores range from 0 to 52, and higher scores indicate greater depression severity. HDRS was administered to bipolar disorder patients by a consultant psychiatrist (AED) who was working for the affective disorders unit.

2.2.3. Young Mania Rating Scale (YMRS)

The YMRS (Young et al., 1978) is an 11-item instrument in which a rater ranks the affective disorders unit.

2.2.4. Verbal memory

Age-corrected indices of the memory quotient (MQ) were calculated over total WMS-III (Wechsler, 1997). MQs were used as an outcome variable for verbal memory performance.

2.2.5. Executive functioning

Executive functioning was evaluated by the computerized version of Wisconsin Card Sorting Test (WCST). The WCST (Heaton, 1981) consists of four stimulus cards and 128 response cards that depict various symbols (crosses, circles, triangles, or stars), in different colors (red, blue, yellow, or green) and numbers of figures displayed (one, two, three, or four) whereby the aim is to sort out the rule for matching the series. We used perseverative errors and the number of completed groups as outcome variables for executive functioning.

2.3. Metacognition assessment

2.3.1. The Indiana Psychiatric Illness Interview (IPII)

The IPII (Lysaker et al., 2002) is a semi-structured interview developed to assess how individuals understand their experience with mental illness. A trained psychiatrist conducted the interview, which typically lasted between 30 and 60 min. All original procedures from the IPII were strictly followed, after the interview was translated into Turkish (IPII; Lysaker et al., 2002). The interview is conceptually divided into five sections. First, rapport is established and participants are asked to tell the story of their lives, beginning with their earliest memory. Second, participants are asked if they think they have a mental illness and, if so, whether or not this condition has affected different facets of their life. Third, participants are asked if and how their condition controls their life and, alternately, how they control their condition. Fourth, they are asked how their condition affects, and is affected by others. Finally, participants are asked about their expectations for the future.

2.3.2. The Metacognition Assessment Scale-Abbreviated

The Metacognition Assessment Scale-Abbreviated (MAS-A) is a rating scale designed to systematically identify the extent to which persons can engage in increasingly complex and integrated metacognitive abilities. The original MAS was developed to detect growth within psychotherapy transcripts (Semerari et al., 2003) and, in consultation with the authors, has been abbreviated and adapted for the study of IPII transcripts (Lysaker et al., 2005). The MAS-A differs from other more structured assessments of metacognition in that it focuses on metacognitive functions that arise spontaneously rather than cued, as in a task or referenced in a questionnaire. The MAS-A contains four scales: “Self-reflectivity”, “the comprehension of one’s own mental states;” “understanding of others’ minds”, “the comprehension of other individuals’ mental states;” “decentration” or the ability to see the world as existing with others having independent motives; and “mastery”, defined as the ability to work through one’s representations and mental states, with a view to implement effective action strategies in order to accomplish cognitive tasks or cope with problematic mental states. The MAS-A asks the rater to indicate whether the participant has successfully used or failed to use a function for each task. For example, the rater must determine if the participant can identify different emotions they feel and recognize that their understanding of life events is subjective. The full presence of a function is awarded a score of “1”, whereas a score of “0.5” is awarded for the partial presence of a function. The highest score obtainable for “self-reflectivity” is “9”, for “understanding of others minds”, “7”, for “decentration” a “3” and for “mastery” a “9”. For all scales, higher scores indicate a greater capacity to create or use increasingly more complex and integrated representations of oneself or others. Findings were evaluated by a psychiatrist (CT) who had received formal training of MAS before the experiment.

2.4. Statistical analyses

In the preliminary step, variables were checked for the assumptions of parametric statistical testing by the visual analyses of distribution plots and Kolmogorov–Smirnov tests. Differences in the socio-demographical and clinical variables were tested by t-tests and chi-square. Statistically significant differences were treated as a covariate in the next step. In order to compare executive functioning and metacognition subdomains between bipolar disorder and schizophrenia patients we conducted a MANCOVA, which controls the type I errors caused by multiple comparisons. In this step, a planned logistic regression analysis, including the significantly different variables and diagnosis groups, was performed to determine whether any specific metacognitive subdomain or executive functioning could discriminate schizophrenia from bipolar patients. Lastly, series of Pearson’s correlation analyses were conducted to explore the associations among outcome variables. The criteria for statistical significance was set to 0.05 and all analyses were performed with a commercially available statistical analyses software (SPSS 20).

3. Results

3.1. Group differences in MAS and WCST

MANCOVA analysis was performed to compare the metacognition and executive functioning levels of the patient groups. Due to
with number of correctly identified groups in the WCST in the both groups. Neither the Hamilton depression and Young-mania score were significantly correlated with MAS-A scores. However, metacognition subdomains such as self-reflexivity, understanding others mind and decentration were highly correlated with the positive symptoms of schizophrenia patients. Lastly, the duration of illness was only associated with the decentration subdomain of metacognition in the bipolar patients but not schizophrenia patients. Correlation coefficients are presented in Table 3.

### 4. Discussion

One aim of this study was to explore whether deficits in metacognition are specific to schizophrenia. A patient group with bipolar disorder was chosen as a clinical comparison group. As predicted, we found that patients with schizophrenia had substantially poorer self-reflexivity as compared to bipolar disorder, but did not differ on other subdomains of metacognition. Specifically, participants with schizophrenia tended to produce less complex accounts of themselves relative to bipolar patients. This is consistent with another study that demonstrated that impairments in the cognitive self-reflexiveness of deluded schizophrenia patients were graver than those with bipolar disorder (Engh et al., 2007). In addition, our findings add to the previous literature indicating greater metacognitive deficits in both early and later stages of schizophrenia compared to other psychiatric populations (Vohs et al., 2014). Taken together, our data suggests that

---

**Table 2**

<table>
<thead>
<tr>
<th>Group</th>
<th>Bipolar Disorder Mean</th>
<th>S.D.</th>
<th>Schizophrenia Mean</th>
<th>S.D.</th>
<th>Stats. F</th>
<th>Sig.</th>
<th>$\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>WCST-Pers. errors</td>
<td>29.41</td>
<td>17.16</td>
<td>30.10</td>
<td>13.62</td>
<td>0.002</td>
<td>0.958</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>WCST-Completed</td>
<td>2.20</td>
<td>1.97</td>
<td>2.62</td>
<td>2.21</td>
<td>0.469</td>
<td>0.496</td>
<td>0.009</td>
</tr>
<tr>
<td>Self-reflexivity</td>
<td>6.07</td>
<td>1.33</td>
<td>4.98</td>
<td>1.55</td>
<td>4.140</td>
<td>0.047</td>
<td>0.071</td>
</tr>
<tr>
<td>Understanding others</td>
<td>4.28</td>
<td>1.20</td>
<td>4.43</td>
<td>1.34</td>
<td>0.423</td>
<td>0.518</td>
<td>0.008</td>
</tr>
<tr>
<td>Decentration</td>
<td>1.72</td>
<td>0.92</td>
<td>1.53</td>
<td>0.83</td>
<td>0.016</td>
<td>0.901</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mastery</td>
<td>5.48</td>
<td>1.25</td>
<td>4.75</td>
<td>1.81</td>
<td>2.360</td>
<td>0.131</td>
<td>0.042</td>
</tr>
</tbody>
</table>

Notes for Table 2: WCST: Wisconsin Card Sorting Task; MAS: Metacognition Assessment Scale.

**Table 3**

The correlations between metacognition and outcome variables between groups.

<table>
<thead>
<tr>
<th></th>
<th>MAS-SR</th>
<th>MAS-UAM</th>
<th>MAS-D</th>
<th>MAS-M Mean</th>
<th>WCST Pers. Err. Mean</th>
<th>WCST Comp.</th>
<th>MQ</th>
<th>Age</th>
<th>Edu</th>
<th>Dur</th>
<th>HAM</th>
<th>YOUNG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bipolar Disorder</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAS-Self refl.</td>
<td>0.57**</td>
<td>0.43*</td>
<td>0.68**</td>
<td>0.07</td>
<td>0.27</td>
<td>0.3</td>
<td>0.45*</td>
<td>0.40*</td>
<td>0.29</td>
<td>0.07</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>MAS-Understanding</td>
<td>–</td>
<td>0.47*</td>
<td>0.58**</td>
<td>0.16</td>
<td>0.19</td>
<td>0.29</td>
<td>0.11</td>
<td>0.14</td>
<td>0.26</td>
<td>0.23</td>
<td>–0.15</td>
<td></td>
</tr>
<tr>
<td>MAS-Decentration</td>
<td>–</td>
<td>0.48*</td>
<td>0.18</td>
<td>0.31</td>
<td>0.46*</td>
<td>0.34</td>
<td>0.25</td>
<td>0.49**</td>
<td>–0.01</td>
<td>–0.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAS-Mastery</td>
<td>–</td>
<td>0.44*</td>
<td>0.50**</td>
<td>–0.01</td>
<td>0.47*</td>
<td>0.15</td>
<td>0.24</td>
<td>0.04</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Schizophrenia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAS-Self refl.</td>
<td>0.83**</td>
<td>0.69**</td>
<td>0.83**</td>
<td>–0.46**</td>
<td>0.35</td>
<td>0.60**</td>
<td>0.26</td>
<td>0.24</td>
<td>–0.02</td>
<td>–0.21</td>
<td>–0.74**</td>
<td>–0.34</td>
</tr>
<tr>
<td>MAS-Understanding</td>
<td>–</td>
<td>0.59**</td>
<td>0.78**</td>
<td>–0.54**</td>
<td>0.31</td>
<td>0.42*</td>
<td>0.08</td>
<td>0.29</td>
<td>–0.16</td>
<td>–0.25</td>
<td>–0.73**</td>
<td>–0.41*</td>
</tr>
<tr>
<td>MAS-Decentration</td>
<td>–</td>
<td>0.45*</td>
<td>0.3</td>
<td>0.02</td>
<td>0.28</td>
<td>0.23</td>
<td>–0.03</td>
<td>0.06</td>
<td>0.09</td>
<td>–0.36</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>MAS-Mastery</td>
<td>–</td>
<td>–0.45*</td>
<td>0.39*</td>
<td>0.11</td>
<td>0.34</td>
<td>0.04</td>
<td>–0.29</td>
<td>–0.74**</td>
<td>–0.36</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes for Table 3: WCST: Wisconsin Card Sorting Task; MAS: Metacognition Assessment Scale; SR: Self Reflexivity; UAM: Understanding others mind; D: Decentration; M: Mastery; Pers. Err: Perseverative Error; Comp: Completed; Edu: Education; HAM, Hamilton depression scale total score; Young, Young mania scale total score.

* $p<0.05$.

** $p<0.01$ (2-sided). $P$ values indicated the significant correlations between metacognition and outcome variables between groups.

the observed group differences, age was treated as a covariate in this step and the univariate statistical results were presented following the bonferroni correction. Significant multivariate effects were found for age (Wilks Lambda; $F(6,49)=2.93$; $p=0.02$; $\eta^2=0.26$) and the diagnosis (Wilks Lambda; $F(6,49)=2.47$; $p=0.04$; $\eta^2=0.23$). Univariate testing found the effect to be significant for the self-reflexivity subscale of MAS ($F(1,54)=4.14$; $p=0.047$; $\eta^2=0.07$) but not for understanding others mind, decentration, and mastery subscales of MAS. In addition, executive functioning as measured with the perseverative errors and the number of completed groups in WCST did not differ between diagnosis groups (see Table 2 for details). Lastly, the binary logistic regression analyses revealed that self-reflexivity subscale of metacognition ($Beta: –0.517$, $SE:0.201$) significantly discriminated schizophrenia cases from bipolar patients ($Wald\chi^2(df=1) 6.61; p=0.01; Exp(B):0.596$). This regression model correctly classified 85.2% of patients with schizophrenia and 50.7% of patients with bipolar disorder, with an overall classification success rate of 66.7%.

### 3.2. Correlational analyses

Moderate to high correlations were found among metacognition subdomains in both diagnostic groups, though the correlation coefficients were higher in schizophrenia patients. The number of perseverative errors in the WCST was negatively correlated with understanding others mind and self-reflexivity subdomains of schizophrenia patients. Metacognitive mastery was associated

---

The page contains tables of statistics and text discussing the results of a study comparing executive functioning and metacognition between bipolar disorder and schizophrenia patients. The tables include means, standard deviations, and significance levels for various measures such as perseverative errors, self-reflexivity, understanding others mind, and mastery. The discussion section highlights the findings, noting that metacognitive deficits are specific to schizophrenia, with particular emphasis on self-reflexivity and understanding others mind. The data indicates that schizophrenia patients have poorer performance in these areas compared to bipolar disorder patients, suggesting that metacognition is a significant factor in schizophrenia. The study also notes correlations and variances among these metacognitive subdomains, providing a nuanced view of how these components interact within the diagnostic groups. The findings are consistent with previous research and add to the understanding of the cognitive profiles in schizophrenia.
disturbances in self-experience are a hallmark of schizophrenia (Lysaker and Lysaker, 2010; Lysaker and Dimaggio, 2014).

In addition, we also explored whether these two groups had similar kinds of relationships between metacognition, other neurocognitive, clinical and demographic variables. Here most interestingly, we found that metacognition had different kinds of correlates in the schizophrenia as compared to bipolar disorder group. Deficits in verbal memory and executive function were linked with lower levels of self-reflectivity and understanding others mind in the schizophrenia, but not in the bipolar disorder group. Deficits in verbal memory were linked to lesser levels of decentration only in bipolar disorder group. By contrast metacognitive mastery was associated with executive functioning and verbal memory in both groups. It is worth noting that we did not find a correlation between negative symptoms and metacognition, possibly due to the selection of a clinically stable schizophrenia group. Considering the strong link between negative symptoms and executive functioning in previous studies (i.e. Lysaker et al., 2005), one can argue that the effects of neurocognition on metacognitive faculties in our study were relatively independent of negative symptoms. Turning to demographics, greater age and years of education was significantly related to greater levels of self-reflectivity in the bipolar disorder group, but not the schizophrenia group. Greater duration of illness also predicted better decentration in the bipolar disorder group only. Considering psychopathology metacognition appeared strongly associated with positive and general psychotic symptoms in schizophrenia, but was not associated with affective symptomatology in the bipolar disorder group.

Our findings are also consistent with the possibility that patients with schizophrenia may develop metacognitive deficits through unique pathways. In particular, one interpretation of the findings is that deficits in verbal memory and executive functioning play a role in the development of impairments in self-reflectivity in schizophrenia. For example, perhaps with fewer available cognitive resources, persons with schizophrenia are less able to form mental representations of themselves and others. Of note, the lack of correlation between neurocognition and some metacognitive subdomains such as self-reflectivity and understanding others’ minds in bipolar disorder does not prove these variables are unrelated and the causality cannot be determined for certain in the schizophrenia group given the correlational nature of these analyses. Our results are nevertheless consistent with Philips et al. (2008) who proposed that self-regulating capacities, another facet of self-reflectivity, are linked with different kinds of disturbance in brain activity in schizophrenia vs. BD patients. They argued that the impairments in self-regulation capacity of BD patients originates from the misinterpretation of emotions at the subcortical level, which leads to cognitive biases. On the other hand, such impairments seen in schizophrenia were suggested to be related to a dysfunction in prefrontal cortex (PFC) leading to deficits in basic cognitive processes such as the coding of the discrepancy between expected and actual outcomes (i.e. prediction error), which is thought to impair associative learning, and can lead to abnormal reappraisals of environmental stimuli (Brown and Brüne (2012)). Such prediction error-related activity in the PFC has been shown to be reduced in schizophrenia (Corlett et al., 2007).

One issue unaddressed in the literature concerns when and how metacognitive deficits develop. Consistent with recent findings (Vohs et al., 2014), we did not find evidence of progression of deficits over time. While age and duration of illness were linked with metacognitive levels of BD they were not in schizophrenia patients. This finding is also in line with some recent studies. For instance, Barbato et al. (2013) recruited a clinical high risk for psychosis (CHR) group and tested metacognitive beliefs at baseline, and then again six months later. When comparing the CHR group to a young help seeking population (HSP), metacognitive beliefs did not differ. Most interestingly, those in the CHR group who later converted to psychosis had poorer metacognitive beliefs than the HSP group at baseline. Moreover, metacognitive deficits in schizophrenia have been shown to be independent from duration of illness in the previous cross sectional studies (Brüne et al., 2007; Lysaker et al., 2008). Results as a whole thus are consistent with models which to consider metacognitive deficits in schizophrenia to be one core feature, or trait of the illness.

Our study also yielded some unexpected findings. Although previous studies have demonstrated poorer neurocognition in schizophrenia than BD, we did not find any group differences in executive functioning and verbal memory. This may have been due to the selection of a clinically stable schizophrenia group with less symptom severity and relatively better functioning. Additionally one domain of metacognition, decentration was linked with neurocognition in the BP group. This may suggest that in this group, losses in neurocognition play a unique role in disrupting their abilities to see events from multiple perspectives. Additionally the capacity for metacognitive mastery was equivalent in both groups and moreover linked with neurocognition in both groups. This may suggest that while impairments in self-reflectivity are a central feature of schizophrenia, the loss of the ability to use metacognitive knowledge to cope is with distress is a common feature in psychiatric disorders with psychotic like episodes in general and linked with deficits in neurocognition. This is consistent with Vohs et al. (2014), which failed to find group differences in mastery when comparing schizophrenia and substance abuse patients. Lastly, regarding the link between greater duration of illness and poor decentration in the BD group, it is possible that, in the presence of mood swings that have serious negative socio-economical consequences, these patients may have developed a self-serving attributional bias, that is, assigning negative life experiences to other factors rather than the self. Alternatively, this may also relate to a treatment effect, by learning the impact of their psychological condition on others. As with all unexpected findings replication is needed and all interpretations should be taken as speculative at best.

This study has several strengths and limitations. The comparative design of the study allows us to draw firm conclusions on the development of these deficits. However, in order to make causal inferences, longitudinal studies would be required. In addition, metacognitive deficits in this study were tested by the MAS, which was scored by evaluating the transcripts of personal narratives. We observed that interviews tended to be longer in our non-psychotic population, and also the impairments of BD patients appeared to be more qualitative rather than quantitative. This is a critical issue, which requires additional attention for the evaluator. However, we only had one rater (CT) for both groups to try to control for this issue, but this may have been problematic in large samples where multiple diagnosis groups were compared on the MAS, and in which multiple raters were used.

To the best of our knowledge this is the first comparative study investigating the specificity of metacognitive deficits in schizophrenia and bipolar disorder. Our results support the ongoing efforts to link metacognitive deficits with various clinical and functional outcome variables in schizophrenia. In addition, these results may also support the importance of psychotherapy and psychosocial interventions that aim to remediate metacognitive impairments in schizophrenia (e.g. Lysaker et al., 2011). Looking at our results, efforts to improve self-reflectivity together with cognitive rehabilitation therapies that remediates neurocognitive deficits may have a substantial potential on the road to recovery of our patients. Regarding patients with bipolar disorder, more work is needed to understand the potential causes of metacognitive
disturbances including disruptions in attachment patterns following profound elevations and collapses of mood.

References


