Insights into theory of mind in schizophrenia: The impact of cognitive impairment

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ABSTRACT

The ability to mentalize and attribute beliefs, intentions and desires to others has been found by the vast majority of studies to be impaired in patients with schizophrenia. However, it is not yet clear if this deficit in Theory of Mind (ToM) is independent of their also well established deficits in basic cognitive functioning. In the present study, we sought to clarify the above relationship by exploring patients' ToM impairment after controlling for their putative cognitive deficits. We examined 36 patients with schizophrenia and 30 healthy matched controls on first and second order tasks of ToM and on commonly used neuropsychological tests. Patients performed poorly on ToM tasks even after controlling for their cognitive deficits, particularly on second order ToM. The present findings contribute to the understanding of the mechanism of ToM, suggesting that ToM deficits are core characteristics in schizophrenia and relatively independent of patients' cognitive impairment.

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1. Introduction

Metacognition refers to one's knowledge concerning one's own cognitive processes. Theory of Mind (ToM) involves awareness that other people have beliefs, intentions and desires, which may differ from our own or from reality, and therefore, is considered a metacognitive process, since it requires the cognitive understanding of our own or other people's mental states. A crucial differentiation in ToM definition concerns whether a person has to reason for or decode social information (Shamay-Tsoory et al., 2007). Specifically, mental state reasoning occurs while explicitly inferring about other people's mental states, based on verbal or visual social cues; mental state decoding refers to the use of information such as facial expressions or emotional prosody in order to infer a mental state, while being based on more spontaneous/automatic inferential processing than Theory of Mind tasks involving effortful processing of social cues.

Over the last decade, numerous studies have explored ToM in schizophrenia and other psychotic disorders in an attempt to understand behavioral and psychotic symptoms in terms of patients' disturbed or impaired ability to reason or decode social information (Fletcher et al., 1995; Goel et al., 1995) and, predominantly, prefrontal thalamic region (Crespo-Facorro et al., 2000; Sanlipo et al., 2000; Shenton et al., 2001; Antonova et al., 2004). Interestingly, some of the same brain regions that have been associated with the cognitive deficits in schizophrenia have also been implicated in mentalizing functions in healthy adults (i.e., frontal (Fletcher et al., 1995; Goel et al., 1995) and, predominantly, prefrontal (Gallagher et al., 2000; Vogeley et al., 2001) cortices). Given the co-occurrence of cognitive impairment and deficient ToM in schizophrenia, as well as the potential overlap or contiguousness of the brain regions involved in both these functional domains, teasing them apart would help to further elucidate the nature of the latter. Are ToM deficits merely secondary to patients' overall cognitive dysfunction or are they related, yet independent?
To date, several studies have explored the putative relationship between ToM and other components of cognitive functioning in schizophrenia. These investigations have reported positive correlations between deficient ToM and particular cognitive functions: memory and attention (Druy et al., 1998; Greig et al., 2004); autobiographical memory (Corcoran & Frith, 2003); executive functions (Corcoran & Frith, 2003); context processing (Schenkel et al., 2005; Uhlhaas et al., 2006); and language (Sarfati et al., 1997). While these findings suggest a relationship between impaired ToM and cognitive dysfunction, any conclusions based on them are limited by their correlational nature.

Studies exploring the role of cognitive functioning in ToM deficits have yielded more informative results than the correlational studies. Several investigations examined ToM in patients with schizophrenia while controlling statistically for a particular cognitive function. For example, Abdel-Hamid et al. (2009) found that ToM deficits in patients with schizophrenia or schizoaffective disorder, as measured by a picture sequence task, persisted after controlling for IQ and executive functioning (separately). The minor effect of IQ on ToM deficits was also reported in other experimental studies (Pickup & Frith, 2001; Janssen et al., 2003), as well as in a meta-analytic study (Sprong et al., 2007), with one exception [in which ToM deficits in patients with disorganized symptoms disappeared once their verbal intelligence was taken into account (Bruné, 2003)]. Similarly, ToM deficits were found to persist even when memory and executive functions were taken into account (Langdon et al., 2001). While these studies would appear to support the independence of ToM from other cognitive functions, the variability in statistical methods (i.e., regression analyses vs. covariate analyses), as well as in the circumscribed cognitive domains used as statistical controls (i.e., IQ vs. executive functions, memory or attention), limit the comparability of their findings, and, consequently, impede the establishment of a robust theory concerning ToM and its relationship to basic cognitive functions.

In the present investigation, we explored the potential contribution of cognitive functioning to ToM in patients with schizophrenia. Given the multifaceted nature of cognitive functioning and of ToM, we included in our investigation several components of each, in order to discern which particular cognitive domains might account for aspects of impaired ToM. Based on the aforementioned studies on the putative relationship between ToM and higher cognitive functions, we expected that ToM would be independent of cognitive dysfunction and persist even after controlling for the latter.

2. Method

2.1. Participants

We recruited 36 patients with schizophrenia (29 men) and 30 healthy participants (24 men). The two groups did not differ in terms of age \([F(1, 64) = .116, p = .734]\), level of education \([F(1, 64) = .053, p = .819]\) and sex ratio \([\chi^2 (1) = .003, p = .955]\). The patients were assessed shortly before discharge. Healthy controls were recruited from the community and were screened with a semi-structured interview by one of the experimenters (MG) before entering the study under the close supervision of the psychiatrist (VPB). Healthy controls were asked about their medical and family history, possible psychiatric and neurological medication or past hospitalization. All participants gave their consent to participate in the study.

All patients met DSM-IV criteria (American Psychiatric Association, 1994). Diagnosis was confirmed with the Greek version (translation-adaptation to the Greek language by S. Beratis) of the Mini-International Neuropsychiatric Interview (4.4) (MINI) (Sheehan et al., 1998). All patients were receiving antipsychotic medication at the time of the study: twenty seven were receiving atypical antipsychotics, six a combination of two atypical antipsychotics, two were receiving typical antipsychotics and one a combination of atypical and typical antipsychotics. Anticholinergic drugs were administered to 10 patients and benzodiazepines to eleven patients. Given the cognitive disturbance that benzodiazepines might provoke, non parametric tests were conducted in order to investigate group differences between patients receiving and those not receiving benzodiazepines; these two subgroups were not different on any of the neuropsychological and theory of mind test.

Exclusion criteria were the following: non-native speakers of the Greek language, a history of neurological or developmental disorders, recent substance abuse (in the last six months), as well as a co-morbid psychiatric disorder, or a medical disorder which might compromise cognitive performance.

We assessed symptom severity (positive symptoms, negative symptoms and symptoms of general psychopathology) of the patients with schizophrenia with the Greek version (Lykouras et al., 1994) of the Positive and Negative Syndrome Scale (PANSS) (Kay et al., 1987). Rating of the PANSS was based on the Greek version (Lykouras et al., 1994) of the Structured Clinical Interview for PANSS (SCI-PANSS) and while blind to neuropsychological performance. Demographic characteristics of the two groups and patients’ clinical data are presented in Table 1.

2.2. Theory of Mind

We developed new ToM paradigms (cartoon and verbal stories) that were appropriate for the Greek population and language.

2.2.1. Cartoon stories

This task comprised 22 cards depicting short stories in cartoon form. After decoding the meaning of each story, participants had to choose which of the alternative sketches for each story provided a logical ending. The stories belonged to four different groupings (although they were presented in mixed order).

- **First order false belief**: four stories required 1st order ToM (i.e., how the protagonist will act, based on the fact that he/she had a false belief regarding the situation). Fig. 1 depicts an example of an item on this task.
- **Attribution of intention**: six stories required attribution of intention (i.e., what the protagonist is thinking of doing).
- **Attribution of desire**: six stories required attribution of desire (i.e., what the protagonist is going to do after perceiving another person’s desire). The subtle difference from the previous one (assignment of desire) is the presence of a second or third person.

The remaining six stories were included as controls, to test story comprehension without the need for mentalizing. The internal reliability of all 22 items yielded a moderately high coefficient (Cronbach’s alpha = 0.62).

2.2.2. Verbal stories

2.2.2.1. Comprehension of hinting. This task comprised six short stories involving two characters, in each of which the scenario ended with one character dropping a very obvious hint (Corcoran et al., 1995). Participants were asked what the character really meant by what he said.

Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Schizophrenia group (n = 36)</th>
<th>Healthy group (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD) range</td>
<td>M (SD) range</td>
</tr>
<tr>
<td>Age</td>
<td>36.72 (7.80) 23–52</td>
<td>37.40 (8.32) 23–55</td>
</tr>
<tr>
<td>Level of education (years)</td>
<td>12.19 (2.58) 9–18</td>
<td>12.33 (2.28) 6–18</td>
</tr>
<tr>
<td>Age at first diagnosis</td>
<td>25.78 (6.50) 16–42</td>
<td>25.78 (6.50) 16–42</td>
</tr>
<tr>
<td>Duration of illness (years)</td>
<td>10.92 (7.12) 2–29</td>
<td>10.92 (7.12) 2–29</td>
</tr>
<tr>
<td>PANSS*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive symptoms</td>
<td>16.44 (6.31) 7–33</td>
<td>16.42 (6.00) 7–30</td>
</tr>
<tr>
<td>Negative symptom</td>
<td>16.42 (6.00) 7–30</td>
<td>16.42 (6.00) 7–30</td>
</tr>
</tbody>
</table>

* PANSS: Positive and Negative Syndrome Scale.
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