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Theory of Mind in Schizophrenia Spectrum Disorders

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Abstract

Objective: To review studies that investigated theory of mind (ToM) deficits in schizophrenia spectrum disorders

Method: After a thorough literature search, 71 studies were included in this review. Data regarding the relationship between ToM, and other cognitive skills, symptoms, and the impact of the state of illness were reviewed.

Results: ToM instruments used in schizophrenia spectrum disorders have some major psychometric limitations; however, previous research was still able to provide some important findings regarding mentalizing impairments in schizophrenia. While ToM deficits are more pronounced in the acute phase of illness, it seems to persist during periods of remission. There is also evidence of ToM deficits in the healthy relatives of schizophrenics, patients with delusional disorder and bipolar disorder (BD), and individuals with high schizotypy scores. ToM dysfunction might be secondary to other cognitive deficits in patients with schizophrenia that have a good prognosis, asymptomatic schizophrenia, delusional disorder, and BD. Other cognitive deficits do not seem to explain ToM dysfunction in patients with psychosis and severe negative symptoms. These findings support the contribution of impairment in both domain-general and domain-specific mechanisms to ToM deficits in schizophrenia spectrum disorders. ToM deficits may be important for understanding poor social functioning and poor insight in psychotic disorders.

Conclusion: While ToM is influenced by state variables, it might be an endophenotype of schizophrenia; however, ToM is likely to be an indicator of other frontal lobe-related endophenotypes. Longitudinal studies conducted with high-risk individuals are particularly important.

Key Words: Theory of mind, mentalizing, schizophrenia

INTRODUCTION

One of the main characteristics of primate evolution was the development of cognitive skills necessary for adapting to an increasingly complex social environment. The term theory of mind (ToM) is used to define one of the essential cognitive abilities, which has a role in social interaction among humans. Originally this term was defined in 1978 by Premack and Woodruff with regards to one of their experiments with chimpanzees. ToM was described as the ability to explain the observed behaviors of others by referring to their mental states (Premack and Woodruff, 1978). Others referred to this concept as metallization (Langdon and Coltheart, 1999).

For the past 20 years researchers have shown interest in the role of ToM in human development and psychopathology. Following a study by Baron-Cohen et al. (1985), which reported ToM impairment in autistic disorder, a substantial number of studies supported the view that suggests there is severe dysfunction in mentalizing ability in autistic spectrum disorders (Baron-Cohen, 2001). Subsequent to studies of autistic spectrum disorders, other evidence suggesting a relationship be-

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tween ToM impairment and other mental disorders was published. One of these mental disorders is schizophrenia, and following the theory of Frith, who suggested that ToM impairment plays a role in the development of delusions, a large number of studies investigated the relationship between ToM deficits and schizophrenia (Brüne, 2005b; Harrington, 2005). Despite the fact that many studies have been conducted on this subject, there remain numerous highly debated points about the nature of ToM impairment in schizophrenia. Some of these unresolved issues follow:

1- Which symptoms are associated with ToM?

2- Are ToM deficits a temporary problem related to acute psychosis, or a trait characteristic of the disorder?

3- Is ToM impairment a primary impairment or is it secondary to cognitive deficits in other domains?

4- How does ToM impairment affect the functional capacity and clinical phenotype of the patient?

We aimed to review all studies that investigated ToM in schizophrenia and to discuss the nature of mentalizing impairment based on the questions above.

Relevant articles were chosen following a PUBMED search for articles published between January 1990 and December 2007 using the following keywords: Theory of mind, mentalizing, social cognition, schizophrenia, psychosis, and schizotypy*. Reference lists of the published articles and reviews were also searched.

In this manuscript we first summarize the different views regarding the concept of ToM. Later, tests that were developed to measure ToM and their weaknesses are discussed. Finally, ToM studies conducted with schizophrenia spectrum disorder patients are reviewed and their results are discussed in the context provided by the 4 questions listed above.

Theoretical Accounts Regarding ToM and Proposed Subtypes of this Ability

Examining the development of ToM from infancy to adulthood and the importance of mentalizing ability in primate evolution are beyond the scope of this review; however, we will briefly review the mental structure of ToM and the poposed subtypes of this ability, as this information is relevant to interpreting the findings of schizophrenia studies.

Based on autism studies, researchers suggest that there is a separate module in the brain that evolved specifically for mentalizing ability (Scholl and Leslie, 1999); however, other authors oppose the idea of a modular cognitive structure completely isolated from other cognitive processes. According to this account, mental structures that are important for other cognitive processes are also used for mentalizing. The most common explanations of ToM are theory theory (Perner, 1991; Apperley, 2008) and simulation theory (Gordon, 1986; Gallese et al., 2004). According to theory theory, there are some supraordinate rules and representations for describing mental states of self and others. Individuals create a theory to explain others' behaviors based on information about them. Simulation theory defines mentalizing in a less cognitive way; individuals use their own mental states to examine behaviors or information reflecting the mental state of others. Understanding the thoughts of another person is only possible by creating a similar experience inside the observer' own brain. Mirror neurons were suggested to mediate this process (Gallese et al., 2004). These 2 theories remain contentious and other alternative theories have been proposed (Apperly, 2008).

During the last 20 years many tests were developed to measure ToM ability, which have expanded the boundaries of the concept defined by ToM. It is difficult to understand ToM as a unitary concept. Some authors attempted to define subtypes of ToM. Tager-Flusberg and Sullivan (2000), and Sabbagh (2004a) defined 2 subtypes of ToM. One of these ToM subtypes is social-cognitive ToM, which can be defined as the ability to infer the mental state of others based on observation of their behavior. False-belief tasks are classical examples of this type of ToM. The second subtype of ToM defined by these authors is the ability to perceive the mental state of others based on directly observed information (social-perceptual ToM). The eyes task is most commonly used to measure this ability. Unlike social-cognitive ToM, social-perceptual ToM is suggested to be independent of other cognitive abilities, but dependent on emotion recognition skills. Normally, we require both abilities to understand the mental state of others. For example, to comprehend that what a friend said to us has an ironic meaning, we must pay attention to his mimicking, gestures, and tone of voice. In addition, we should examine the words he used and consider his past thoughts regarding the current subject. Nonetheless, it is also possible to suggest that social-perceptual ToM does not require building a theory and so it is another kind of social cognitive ability that cannot be referred to as ToM. In another study, affective and cognitive ToM subtypes were defined based on the content of inference (Shamay-Tsoory et al., 2007b).

TABLE 1. Studies that investigated ToM in schizophrenia spectrum disorders

Study	Sample	ToM test	Other	Outcome
Corcoran et al., 1995	55 Sch, 30 HC, 14 anxious/depressive controls	Hinting	Patients were grouped according to symptoms, 8 Sch patients in remission	ToM is impaired in Sch (even after IQ is corrected). No deficit in remission. IQ correlated to ToM
Frith and Corcoran, 1996	55 Sch, 22 HC, 13 anxious/depressive controls	1st and 2nd degree FB and deception	Patients were grouped according to symptoms, 9 Sch patients in remission matched for IQ	Sch: impaired only in 2nd degree FB
Corcoran et al., 1997	44 Sch, 40 HC, 7 anxious/depressive controls	10 ToM-jokes (7 FB-3 deceptions), 10 control-jokes	IQ associated with ToM Patients were grouped according to symptoms	Sch: impaired ToM No deficit in remission
Langdon et al., 1997	20 Sch, 20 HC	FB- picture-sequencing	SAPS, SANS 3 factors of Liddle	ToM impaired in sch. General sequencing deficit was related to all symptoms. Negative symptoms were associated with FB sequencing deficit
Sarfati et al., 1997a	12 Sch (6 FTD+), 12 depresssion, 12 HC	15 FB cartoon, 15 CIT	SAPS, SANS, TLC	Sch impaired ToM FDB+ group was more impaired in CIT
Sarfati et al., 1997b	24 Sch (12 FTD+), 24 HC 12 depression	CIT cartoon	PANSS	Sch impaired in 2nd degree ToM
Doody et al., 1998	28 Sch, 10 depressive, 19 mild learning deficit, 18 Sch + learning deficit, 20 HC	1st and 2nd degree ToM tasks		Sch: low performance
Drury et al., 1998	14 Sch, 10 psychotic, 12 depression	2. FB, metaphor, irony	Acute and remitted phases	Sch impaired in acute psychosis, no between-group difference in remission
Mitchley et al., 1998	18 Sch, 13 psychiatric controls	Irony	PANSS, IQ	Sch: low performance
Murphy, 1998	37 Sch, 23 personality disorder	1st and 2nd degree FB and deception	IQ, memory, executive functions	IQ, memory, and executive function deficits were related to ToM impairment
Langdon and Colthe-art, 1999	40 students	FB-picture sequencing	SPQ	Students with high schizotypy scores had ToM impairment
Pilowsky et al., 2000	12 early onset Sch, 12 autism, 12 HC	FB, deception		Sch impaired only in FB, autistic patients had the greatest impaired
Russell et al., 2000	5 sch, 7 HC	Eyes		Sch more impaired
Sarfati et al., 1999	13 disorganized Sch , 13 Sch, 13 depression, 13 HC	Comics		ToM impaired in disorganized patients
Sarfati and Hardy-Bayle, 1999	25 Sch (15 FTD+) 10 BD, 15 SK	CIT cartoon		

A series of imaging studies were conducted in order to identify the functional neuroanatomy of mentalizing ability. During ToM tasks the ventromedial frontal cortex, posterior superior temporal sulcus (STS), temporal pole, and temporoparietal junction were the most commonly activated regions (Fletcher et al., 1995; Goel et al., 1995; Gallagher et al., 2000; Vogeley et al., 2001; Brunet et al., 2003; Völlm et al., 2005). While the ventromedial frontal cortex specializes in mentalizing ability, other regions seem to be important for analyzing social cues (Frith and Frith 2003, Gallagher and Frith 2003). Only a few studies investigated the neuroanatomy of social-perceptual ToM. Sabbagh et al. (2004) demonstrated orbitofrontal and medial temporal cortex activation during the eyes task. The orbitofrontal cortex appears to play a more important role in social-perceptual aspect of ToM and empathy (Lee et al., 2004). Brain regions that mediate ToM abilities were less responsive to social cues in schizophrenia. Brunet (2003) showed that there was right prefrontal cortex activation in controls, but not in schizophrenics during nonverbal ToM tasks. Marjoram et al., (2006) reported decreased prefrontal cortex acti-

TABLE 1. *continued from previous page*

Study	Sample	ToM test	Other	Outcome
Sarfati et al., 2000	25 Sch, 25 HC	CIT	IQ, PANSS	ToM impaired in Sch
Langdon et al., 2001	32 Sch/SA, 24 HC	FB-picture sequencing	SAPS, SANS 3 factors of Liddle, executive functions	ToM impaired in sch. Negative symptoms were related to ToM deficits. Correlation was not significant after correction for executive functions
Mazza et al., 2001	35 Sch, 17 HC outpatients	1st and 2nd degree FB, deception	Patients were divided into 3 groups of Liddle. Executive functions, verbal fluency, memory	ToM impaired in Sch . Psychomotor retardation group was the most impaired No significant correlations with cognitive tests
Pickup and Frith, 2001	41 Sch, 35 HC, 18 anxious depressive control	1st and 2nd degree FB	IQ 8 remitted patients	Sch: impaired only in 2nd degree ToM
Herold et al., 2002	20 Sch, 20 HC	1st and 2nd degree FB, irony, metaphor	Remitted Matched for sex, education, and age	Sch: impaired in irony
Langdon et al., 2002	25 Sch/SA, 20 HC	FB-picture sequencing Irony metaphor	SAPS, SANS 3 factors of Liddle, executive functions	Patients impaired in all tasks Deficit in irony related to chronicity Executive impairment was associated with ToM deficits
Roncone et al., 2002	44 Sch	1st and 2nd degree FB	Social functioning	ToM was related to social functioning
Brüne, 2003	23 Sch, 12 HC	FB-picture sequencing	IQ, chronic disorganized Sch	ToM impaired in Sch Did not persist after correction for IQ
Brunet et al., 2003	25 Sch, 25 HC	Cartoon ToM	IQ	Sch impaired in verbal ToM
Corcoran, 2003	24 paranoid Sch, 15 remitted Sch, 44 HC	Hinting	IQ, pragmatic language task memory	Sch had ToM impairment Cognitive tasks were related to ToM in patients (especially in paranoid group)
Corcoran and Frith, 2003	59 Sch, 44 HC	1st and 2nd degree FB, deception, hinting	15 remitted IQ controlled memory	Sch impaired in all ToM tasks
Drake et al., 2003	33 Sch, acute phase	ToM-jokes	Other cognitive tasks, insight	Insight is not related to insight
Janssen et al., 2003	43 Sch, 41 Sch-healthy relative, 43 HC	Hinting, 1st degree FB	IQ, memory, attention, executive functions	Sch impaired in ToM, Relatives are impaired in Hinting
Randall et al., 2003	18 paranoid Sch/SA 14 remitted Sch/SA, 18 HC	1st and 2nd degree FB and deception	IQ, attention, attribution style	Sch: impaired in ToM Remitted and paranoid patients were not different
Abu-Akel and Abushua'leh. 2004	24 sch	1st and 2nd degree FB	Inpatients, aggression	Aggressive patients had better ToM ability and poorer empathy performance
Craig et al. 2004	16 paranoid patients (Sch and delusional disorder), 17 Asperger, 16 HC	Hinting, Eyes	Paranoia scale Sch inpatients	Both patient groups have poor performance in ToM

vation in positive symptom positive relatives of patients with schizophrenia. Russell et al., (2000) reported decreased inferior frontal and insular cortex activation in response to the eyes task in schizophrenic patients.

Methods Used to Assess ToM and Their Reliability

Many tasks have been developed for measuring ToM. Moreover, various tests have been developed by different researchers and in subsequent studies the same authors measured ToM with different assessment tools, which makes comparing the results difficult.

Most well known examples of ToM tests employ false belief tasks (Frith and Corcoran, 1996). The Sally and Anne test is the most widely known of these tasks. In this task, subjects are told (verbally or visually) a story in which they should understand that there was a change that is known by them, but not with another character

TABLE 1. *continued from previous page*

Study	Sample	ToM test	Other	Outcome
Greig et al., 2004	128 Sch or SA	Hinting	Attention, executive functions, PANSS, SAPS	Disorganized Sch had poorest ToM performance ToM deficit was related to FTD and PANSS delusion item
Kelemen et al., 2004	79 Sch-relatives (14 symptomatic) and 40 HC	Eyes		Poor eyes test scores only in symptomatic relatives
Langdon and Coltheart, 2004	18 high-schizotypy, 18 low-schizotypy	irony, metaphor	SPQ	Irony impairment schizotypy related
Brüne, 2005	23 Sch	FB sequencing and 1st and 2nd FB, deception	Social functioning, face recognition, other cognitive	ToM and duration of illness predicts social functioning
Brüne and Bodenstein, 2005	31 Sch	FB sequencing and 1st and 2nd Degree FB, deception	PANSS, executive function, proverbs	Proverbs variance 39% predicted by ToM
Harrington et al., 2005	25 Sch (13 persecution+ 12 persecution-) HC	FB sequencing and 1st and 2nd Degree FB, deception	IQ, SAPS, SANS	ToM impaired only in persecution+
Kelemen et al., 2005	52 Sch, 30 HC	Eyes	PANSS	ToM impaired both in acute phases and remission ToM related to negative symptoms
Marjoram et al., 2005	15 Sch, 15 affective psychosis, 15 HC	Hinting		ToM impaired in patients. Delusions were related to ToM
Marjoram et al., 2005b	20 Sch, 20 HC	Visual ToM comics	Inpatient, outpatient, positive symptoms	Sch impaired in ToM
Schenkel et al., 2005	42 Sch	Hinting	IQ, executive functions, visual/linguistic concept processing, childhood social functioning	ToM deficit was related to social functioning and concept processing,
Bell and Mishara, 2006	267 stabile Sch outpatients	Hinting	Executive functions, emotion recognition, memory, attention, negative symptoms	ToM related to negative symptoms (SANS r = 0.33)
Bonhstein et al., 2006	41 Sch, 22 HC, 24 mood disorders, 7 other psychotic	ToM cartoon	PANSS, theory of biology	Sch impaired in ToM
Bora et al., 2006	50 Sch	Eyes, Hinting	Social functioning	ToM related to social functioning, working memory and other executive functions
Bömmer and Brüne, 2006	21 delusional disorder, 22 HC	Proverbs, FB sequencing and 1st and 2nd degree FB	Executive functions	Impaired ToM in inpatients, did not persist after correction for executive function
Inoue et al., 2006	30 Sch, 30 HC	Picture sequencing	Near discharge or outpatients	Sch ToM is impaired
Irani et al., 2006	10 Sch, 10 Sch-relative, 10 HC	Eyes	Self face recognition	Patients and relatives are impaired in ToM

in the story. Subjects should predict the behavior of the other based on this information (first-degree ToM). Normal children acquire this skill by about age 3 or 4 years. In more complex versions of these tasks, the number of characters is increased and subjects should predict the behavior of the other based on the other's knowledge about another character (second-order ToM). In deception tasks subjects should recognize the deceptive behavior of the story character towards another character in the story. This task also has first-order, second-order, and third-order versions. Nonverbal versions of these tasks have also been developed (Langdon et al., 1997; Harrington et al., 2005). A different task developed by Sarfati et al. (1997a) asks subjects to infer the intention of characters in the story.

Indirect speech tasks measure the ability to understand hidden meanings (ironic, hinting, or metaphoric) behind what is said by one character in the story to another (Corcoran et al., 1995; Sprong et al., 2007).

TABLE 1. *continued from previous page*

Study	Sample	ToM test	Other	Outcome
Langdon et al., 2006a	22 Sch, 18 HC	FB Picture sequencing	outpatients	Sch ToM is impaired
Langdon et al., 2006b	34 Sch, 21 HC	FB Picture sequencing	outpatients	Sch ToM is impaired
Marjoram et al., 2006	13 Sch, 12 Sch relative psychotic +, 13 akraba psychotic-	Hinting, cartoon,		Sch and psychotic + relatives were impaired
Mazza et al., 2006	20 Sch, 20 HC, 18 frontal lobe injury	FB	Executive functions, social cognition	Both patient groups are impaired in ToM
Murphy, 2006	13 Sch, 13 Asperger, 13 personality disorder	Eyes, FB1, FB2	IQ	Sch and Asperger impaired in ToM
Pinkham and Penn, 2006	49 Sch, 44 HC	Hinting, FB deception	Outpatients with minimal symptoms	Social cognition contributed to functioning, independent of other tests Sch impaired in both ToM tasks
Pickup, 2006	62 university students	Strange stories	Schizotypy scale	Positive-schizotypy related to ToM
Russell et al., 2006	61 Sch, 22 HC	ToM- animation	Patients divided into subgroups, 13 remitted	Behavioral (negative and FTD+) most impaired, paranoid patients were also impaired
Zalla et al., 2006	40 Sch, 18 HC	ToM story	22 disorganized 18 non-disorganized	Sch impaired ToM, selective impairment in non-disorganized, ToM deficit was related to other cognitive deficits in disorganized patients
Bora et al., 2007	58 Sch	Eyes, 1st and 2nd degree FB	Insight, executive functions	ToM deficit was related to poor insight, working memory, and executive functions
Brüne et al., 2007	38 Sch, 29 HC	Picture sequencing ToM Questionnaire	Executive functions, PANSS and social skills	ToM was the best predictor of social skills in sch
Fiszdon et al., 2007	199 Sch, 73 SA	Hinting	Executive functions, memory, psychomotor speed, emotion recognition, stable outpatients	SA performed better than Sch in Hinting. Both groups performed similarly in other tests
Mizrahi et al., 2007	71 psychotic disorder (17 non-medicated)	Hinting	82% Sch, 15% SA, 6 weeks longitudinal in non-medicated	ToM was elated to negative symptoms Positive symptoms and ToM improved after antipsychotic treatment.
Shamay-Tsoory et al., 2007a	22 Sch, 55 HC	1st and 2nd degree cognitive and affective ToM	SANS, PANSS	Sch: only affective ToM is impaired. Affective ToM is related to negative symptoms
Jahnsan and Sergi, 2007	52 high- schizotypy, 40 low-schizotypy		Cognitive battery	ToM unrelated to schizotypy
Martino et al., 2007	21 stable Sch, 15 HC		Executive functions, negative symptoms, decision making	Negative symptoms related to ToM. SANS ($r = -0.68$) Sch impaired ToM
Savina et al., 2007	84 Sch, 24 HC (23 typicals, 18 clozapine, 20 olanzapine, 23 risperidone	Picture sequencing, 2. FB		ToM impairment was greater in patients using typical antipsychotics or risperidone

Tests that aim to measure social-perceptual aspects of ToM are quite rare. In schizophrenia studies the only test that has been used to measure this ability is the eyes task. In the eyes task a subject's ability to infer the mental state (not simple emotions) of another person by viewing his eyes is measured (Baron-Cohen et al., 2001).

The psychometric characteristics of all of these tests have not been investigated in detail. The number of items on these different tests are vary widely. While some tests include only one story, others have many more items. Even tests that assess the same type of ToM task vary in difficulty. Moreover, the same task is applied differently from one study to another (for example, some researchers present the ToM story visually, while others verbally, and some researchers repeat the task item). Furthermore, most of these tasks have been developed for autistic chil-

TABLE 1. *continued from previous page*

Study	Sample	ToM test	Other	Outcome
Shamay-Tsoory et al., 2007b	24 Sch, 43 brain injury, 28 HC	1st and 2nd degree cognitive and affective ToM		Sch and frontal lesion patients impaired affective ToM
Shamay-Tsoory et al., 2007c	26 Sch, 31 HC	1st and 2nd degree cognitive and affective ToM	Empathy, cognitive tests	Affective ToM related to social functioning, negative symptoms-empathy association
Stratta et al., 2007	20 Sch	Irony	PANSS	Irony related to positive signs
Bertrand et al., 2007	36 Sch, 27 HC	Hinting, 4 factors, social intelligence test	First episode Cognitive tests	Social cognition was impaired in schizophrenia, not related to symptoms
Bora et al., 2008a	91 Sch, 55 HC	Hinting, Eyes	Divided into 3 groups based on PANSS (asymptomatic, negative symptoms+, positive + negative symptoms +)	ToM impaired in all patient groups. ToM deficits did not persist after correcting for working memory in asymptomatic patients
Bora et al., 2008b	30 Sch, 30 HC	Hinting, Eyes	Empathy	Sch: empathy deficits in others' assessments. ToM impairment related to empathy deficit
Mo et al., 2008	29 Sch (remitted) 22 HC	Metaphor/irony 1st and 2nd FB	PANSS	Sch irony, hinting, 1st and 2nd degree FB impaired

Sch: Schizophrenia; HC: healthy controls; ToM: theory of mind; CIT: Character Intention Test; FB: false belief; FTD: formal thought disorder; SPQ: Schizotypal Personality Questionnaire.

dren. All of these factors should be considered when interpreting findings about ToM.

ToM Investigations in Schizophrenia

Frith suggested that many symptoms of schizophrenia could be explained by deficits in inferring the mental state of others and self. According to Frith, difficulties in inferring others' intentions and thoughts can cause reference and paranoid delusions, and very severe impairment in ToM can cause volitional difficulty (negative and disorganized symptoms). Frith suggested that the delusion of control is only related to self-monitoring deficits. This theory predicts that ToM ability should be relatively intact in patients that have the final group of symptoms or remitted patients.

ToM performance in patients with schizophrenia and healthy controls were compared in 46 studies (Table 1). Lower performance in patients with schizophrenia is the common finding in all these studies. In a recent study Sprong et al. (2007) reported findings of the first meta-analysis of ToM impairment in schizophrenia. According to this study, schizophrenia patients have very severe ToM impairment (Cohen D = 1.25). This effect size is comparable to the largest effect sizes reported for other cognitive tests; however, their study has some shortcomings. There was significant heterogeneity between the studies they analyzed. This is an expected finding when one considers that outcomes of very different tasks were combined. Additionally, the researchers used dichotomous data to calculate effect sizes, which for some is a controversial approach. Other studies used only a psychiatric control group (Drury et al., 1998; Mitchley et al., 1998).

ToM deficits have also been investigated in other schizophrenia spectrum disorders. A single study conducted with delusional disorder patients reported lower ToM performance in schizophrenia patients than in controls (Bommer et al., 2006). The relationship between schizotypal features and ToM has also been examined. Studies that investigated the association between schizotypy and ToM reported a moderate but positive relationship (Langdon and Coltheart, 1999; Langdon and Coltheart, 2004; Pickup, 2006); however, another study reported that was no relationship between schizotypy and ToM impairment (Jahnsan et al., 2007).

Relationship Between Symptoms and ToM

One of the main focuses of previous studies in schizophrenia is the relationship between mentalizing and symptoms. Theories regarding this association are rooted in the theory of Frith (1992). The 2 main methods used to investigate the ToM-symptom relationship are sub-grouping patients and correlational analysis. The first studies to examine this subject were conducted by Corcoran and

Frith in which they grouped patients according to Frith's theory (Corcoran and Frith, 1996; Frith and Corcoran, 1996; Corcoran et al., 1997). In these studies ToM deficits were only observed in patients with paranoid or behavioral symptoms. These studies appear to have used the same sample and have some contentious features, the most important of which is the hierarchical definition of symptom groups. In these studies, only in hierarchically above groups, subjects were allowed to have symptoms of other groups. As such, it is possible to suggest that the findings of these studies could be related to the severity of symptoms in some groups, rather than specific symptoms. Furthermore, studies that investigated the relationship between delusions and ToM reported contradictory findings (Table 1.). Many of these studies did not observe a positive relationship between the severity of delusions and ToM impairment (Langdon et al., 2001; Mizrahi et al., 2007); however, findings of other studies support the theory that there is a relationship between ToM deficit and delusions (Greig et al., 2004; Harrington et al., 2005).

Following Frith, Harde-Bayle suggested another well-known theory to explain ToM dysfunction in schizophrenia. This model posited that thought disorders are the source of ToM deficits in schizophrenia (Sarfati et al., 1997a, 1997b). According to this theory, ToM performance should be intact in non-thought-disordered patients. Consistent with this theory, studies by Frith, Harde-Bayle, and others (Greig et al., 2004; Russell et al., 2006) reported severe ToM impairment in patients with schizophrenia (Table 1.); however, there are contradictory findings regarding the second assumption of the theory, which is that patients without a thought disorder have intact ToM ability. Furthermore, as discussed below, studies that reported ToM deficits, even in remitted patients, contradict the theories of Frith and Harde-Bayle.

Other studies reported a relationship between ToM impairment and negative symptoms (Mazza et al., 2001; Bell and Mishara, 2003; Martino et al., 2007; Mizrahi et al., 2007), and the fact that were some similarities observed between patients with severe negative symptoms and autistic patients is noteworthy.

The above-mentioned studies suggest that all 3 symptom clusters of schizophrenia could contribute to ToM deficits in schizophrenia; however, these associations might have many possible causes and can change according to illness state. Furthermore, the relationship between symptoms and other cognitive deficits may play a role in the ToM-symptom relationship.

Could ToM Deficits Be a Trait Characteristic of Schizophrenia?

Most studies about ToM and schizophrenia were conducted with symptomatic patients. Some early studies included, in addition to symptomatic patients, a small number of remitted patients and did not find any ToM impairment in these patients (Corcoran and Frith, 1996; Frith and Corcoran, 1996). These findings support the notion that ToM impairment is a characteristic of acute psychosis; however, studies conducted in the years that followed did not support these earlier studies. Herold et al. (2002) reported ToM deficits in schizophrenia patients whose positive and negative symptoms were not significant. One of the most important of these studies was conducted by Jannsen et al. (2003). In these studies schizophrenia, both patients that were in remission and their healthy relatives were observed to have impaired ToM abilities.

Other studies conducted by Bell and Mishara (2006), Inoue et al. (2006), Martino et al. (2007), Mo et al. (2007), and Bora et al. (2008a) provided additional support regarding the prevalence of ToM deficits in remitted schizophrenia patients. The meta-analysis of Sprong (2007) also reported ToM impairment of medium effect ($d = 0.69$) in remitted patients. There are several possible reasons that these studies, but not earlier ones, observed ToM impairment in remitted patients:

(1) More difficult tasks like faux pas and hinting were more commonly used in later studies. Earlier studies tended to use easier and shorter tasks;

(2) Lack of power in earlier studies related to small samples;

(3) Earlier studies were conducted by researchers that suggested there was a relationship between symptoms and ToM impairment; however, a weak point of some later studies was a poor definition of remission and neglect of residual symptoms. For example, Inoue et al. (2006) examined patients before discharge, and Martino et al. (2007) recruited patients that had not been admitted to hospital and whose medications had not been changed in previous 4 months. We should expect residual symptoms in all these studies, except Herold et al.'s (2002).

In a study conducted by our group, ToM impairment was investigated in 91 stable schizophrenia patients (Bora et al., 2008a). The inclusion criteria of this study were similar to Martino et al.'s (2007); however, patients were subsequently further sub-grouped. According to the remission criteria used by Herold et al.

(2002), only 35% of the patients were free of positive and negative symptoms. In this study residual positive and negative symptoms were associated with more severe ToM impairment; however, patients with residual symptoms were also more impaired than controls. ToM abilities were also impaired in the relatives of schizophrenia patients (Jannsen et al., 2003; Irani et al., 2006); yet, ToM impairment in relatives was reported to be associated with psychosis-like features in some studies (Kelemen et al., 2004; Marjotam et al., 2006).

Another factor that can influence ToM abilities in remitted patients is antipsychotics, but very few studies have investigated this possibility. Salvina et al. (2007) reported better ToM performance in patients using clozapine or olanzapine than in patients using risperidone or typical antipsychotics. Nonetheless, it is not possible to conclude if these results are related to differences in treatment or other group characteristics. In another study (Mizrahi et al., 2007), improvement in ToM deficits was observed following 2 weeks of antipsychotic treatment.

Are ToM Deficits Independent of Other Cognitive Deficits?

Studies of healthy subjects showed relationships between ToM and other cognitive abilities, such as memory, executive functions, and working memory. Patients with schizophrenia are impaired in all these domains. This fact raises the possibility that ToM deficits are a secondary finding to other cognitive deficits. Corcoran et al. (2003) reported a relationship between cognitive deficits and ToM impairment. Brüne (2003) observed an association between IQ impairment and ToM deficits, and Langdon et al. (2001) reported that ToM deficits in schizophrenia might be due to deficits in executive functions. Other studies also reported associations between ToM impairment and executive functions/working memory (Murphy, 1998; Langdon et al., 2001; Bora et al., 2006; Bora et al., 2007), verbal memory (Murphy, 1998), and IQ deficits (Corcoran et al., 1995; Murphy, 1998; Brüne, 2003).

Explaining ToM impairment in schizophrenia as being exclusively due to other cognitive deficits does not seem possible, at least in symptomatic patients; however, in patients without residual negative and positive symptoms ToM deficits were reported to remit following correction of working memory deficits (Bora et al., 2008). In another study conducted in patients with delusional disorder, ToM impairment lost its significance, compared to controls, after correction for executive functions (Bömmer and Brüne, 2005). ToM deficits in schizophrenia were reported to be more severe than in schizoaffective disorder (Fiszdon et al., 2007) and mood disorders (Sarfati et al., 1997a); however, it is possible to suggest that lower performance in schizophrenia patients than in schizoaffective, delusional disorders, and affective psychoses patients is related to subgroups of patients with severe negative symptoms and poor prognosis. Furthermore, recent genetic findings, such as those reported by Owens et al. (2007), raise questions about the classification of schizophrenia and bipolar disorder. ToM deficits have been shown to persist in remitted BD patients (Bora et al., 2005; Olley et al., 2005). As in delusional disorder, ToM deficits in BD seem to be secondary to other cognitive deficits (Bora et al., 2005).

Impact of ToM Impairment on Clinical Presentation

One of the most significant findings in schizophrenia patients is poor social functioning. Social impairment is relatively independent of symptoms, which suggests that poor social functioning might be related to other factors, such as cognitive deficits. Most studies of schizophrenia have investigated the relationship between social impairment in such cognitive domains as attention, memory, and executive functions. In addition to these impairments, ToM deficits might be expected to contribute to social impairment in schizophrenia. This subject has not been sufficiently investigated; yet, several recent studies showed that ToM deficits are an important predictor of poor social functioning (Roncone et al., 2002; Brüne et al., 2005; Bora et al., 2006l Pinkham and Penn, 2006; Brüne et al., 2007). Empathy, an important ability for social functioning, might also be related to ToM (Bora et al., 2008b).

Another important characteristic of schizophrenia is poor insight. Both psychological defense mechanisms and cognitive deficits are suggested to play a role in poor insight. A substantial number of studies investigated the neuropsychology of insight deficits in schizophrenia and suggest that poor insight is related to only some executive functions. Among executive function tests, low Wisconsin Cart Sorting Test (WCST) scores have been most commonly associated with poor insight. Cognitive flexibility, which is measured with this test, is suggested to be important for self-awareness of illness; however, as a recent meta-analysis (Aleman et al., 2006) confirmed, this relationship is only moderately strong ($r = 0.25$). Other studies have shown that metacognition might be important for insight. For example, the ability to notice errors on the WCST was more strongly associated with poor insight than the task itself (Koren et al., 2004).

ToM might also be important for self-awareness of illness. ToM is essential to the ability to see one's self from the perspective of others, but an insufficient number of studies have tested this hypothesis. Drake et al. (2004) did not observe a relationship between mentalizing ability and the visual ToM task. Bora et al. (2007) reported a relatively strong relationship between ToM and poor insight in remitted schizophrenia patients. This is the first evidence to indicate a relationship between ToM and self-awareness of illness in schizophrenia patients. Nonetheless, this result should be confirmed by other researchers.

CONCLUSION

Severe ToM impairment is a ubiquitous feature of schizophrenia. While there is evidence that ToM deficits are exacerbated in acute psychosis, mentalizing impairment persists in remission. Furthermore, ToM dysfunction is observed in the healthy relatives of patients with schizophrenia. These results raise the possibility that ToM deficits might be an endophenotype of the illness.

These results should be interpreted with caution. The relationship between subsyndromal signs and ToM deficits in relatives has not been sufficiently studied. Moreover, ToM deficits in relatives and remitted patients seem to be secondary to other cognitive deficits, such as frontal lobe impairment, rather than being primary characteristics of the illness. A multidimensional explanation beyond the theories of Frith and Harde-Bayle is necessary to fully explain ToM impairment in schizophrenia. Neither a model based on temporary dysfunction in the ToM module during psychosis nor considering ToM impairment secondary to other cognitive deficits can entirely explain ToM impairment in schizophrenia. Moreover, ToM ability consists of multiple social cognitive skills. Examining the subcomponents of this ability is important.

Neuropsychological and imaging studies conducted with the healthy relatives of patients with BD and schizophrenia, and with other high-risk groups should be considered. These studies should be conducted with larger samples and other cognitive deficits should be controlled for. Additionally, developing new ToM measures with better psychometric features is important for future studies.

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