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Theory of mind reasoning in schizophrenia patients and non-psychotic relatives

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ABSTRACT

Research consistently demonstrates that schizophrenia patients have theory of mind (ToM) impairments. Additionally, there is some evidence that family members of schizophrenia patients also demonstrate impairments in ToM, suggesting a genetic vulnerability for the disorder. This study assessed ToM abilities (i.e., sarcasm comprehension) in schizophrenia patients and their first-degree biological relatives during video-taped social interactions, to be representative of real-world interactions and to assess for disease-specific and/or genetic liability effects. Additionally, we assessed whether ToM abilities predicted social and global functioning in schizophrenia patients, and whether symptoms were associated with ToM deficits. Schizophrenia patients demonstrated impairments in sarcasm comprehension compared to controls and relatives, whereas relatives showed intact comprehension. Symptoms of schizophrenia significantly predicted worse ToM abilities. Furthermore, in schizophrenia patients, impaired ToM reasoning predicted worse social and global functioning. Given schizophrenia patients demonstrated impairments in ToM reasoning in a task that resembles real-life interactions, this might be a key area for remediation.

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1. Introduction

Theory of mind (ToM) refers to the ability of an individual to attribute mental states, such as beliefs and intentions, to another individual. ToM abilities are consistently found to be impaired in schizophrenia patients (reviewed in Bora et al., 2009a). Moreover, recent research suggests that ToM abilities are more strongly associated with social functioning than any other aspect of social cognition or neurocognition (Fett et al., 2011). Research is also beginning to assess whether ToM impairments are related to the genetic vulnerability for the disorder (e.g., Irani et al., 2006). Additionally, recent research has moved towards using more ecologically valid measures of ToM; however, these tools have been under-utilized to study schizophrenia, and have not frequently been used in genetic liability studies. The goal of the present study was to investigate ToM deficits using a more ecologically valid task in schizophrenia patients and non-psychotic first-degree biological relatives. Investigating both patients and family members allowed for a better examination of genetic (familial) liability, as well as disease-related processes.

Current research undoubtedly indicates the presence of ToM deficits in schizophrenia with large effect sizes of $d=1.21-1.26$ (Sprong et al., 2007; Bora et al., 2009a). Furthermore, ToM abilities have been associated with several aspects of social functioning in schizophrenia patients, namely better interpersonal communication skills and participation in more social activities (Bora et al., 2006). In fact, research shows that mental state attribution abilities in schizophrenia patients are one of the best predictors of difficulties with social interactions and behaviors (e.g., Bora et al., 2006; Brüne et al., 2007). One hypothesis is that a lack of integrity in fronto-limbic networks of the brain are related to both social cognitive (e.g., ToM) impairments, as well as deficits in emotion regulation abilities in schizophrenia patients (Rowland et al., 2012).

A more recent debate has focused on whether these symptoms are more state- or trait-related. To date there have been conflicting findings. Evidence for the state argument has come from findings that impairments in ToM abilities are strictly present during the acute stages of the illness (Drury et al., 1998), as well as findings that suggest state-dependent ToM deficits associated with specific behavioral symptoms of schizophrenia (e.g., Frith and Corcoran, 1996; Pickup and Frith, 2001). There is some evidence that suggests ToM deficits are present early in the course of the illness, and may be maintained throughout the course of the illness (e.g., Green et al., 2012); however, it has been postulated that these

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prolonged deficits may also be related to a more general neuro-cognitive impairment also present in schizophrenia (Green et al., 2012). ToM deficits have been found to be related to both positive and negative symptoms of schizophrenia (Brüne, 2005). For example, “over-attributing” the intentions of others may lead to the development of delusions, and individuals with paranoid symptoms may be unable to use contextual information to accurately understand others’ intentions (Brüne, 2005). In contrast, negative symptoms are hypothesized to be related to “under-attributing” or failing to utilize available information to interpret the intentions of others (Frith and Corcoran, 1996). Taken together, current research suggests that ToM deficits are likely present throughout the course of schizophrenia, are significantly related to both positive and negative symptomatology, and predict impairments in social situations.

In contrast, evidence for the trait-dependent view has identified similar ToM deficits in individuals with high levels of schizotypal traits as in those with schizophrenia (Langdon and Coltheart, 2004), while other studies have found impairments in schizophrenia patients with remitted symptoms compared to both healthy controls (Janssen et al., 2003) and psychiatric controls (Mitchley et al., 1998). In a recent meta-analysis, ToM impairments were described as being more pronounced in non-remitted patients, though remitted patients still performed significantly worse than healthy controls (Bora et al., 2009a).

One particularly useful method for resolving the state vs. trait debate is to assess whether ToM deficits are associated with the genetic liability for the disorder by studying the first-degree biological relatives of schizophrenia patients. Family studies are a particularly useful method for investigating genetic liability for the disorder, as non-psychotic relatives of patients share genes for the disorder, but not the illness process. Previous studies of family members of schizophrenia patients have demonstrated discrepant results. In some studies, first-degree relatives performed significantly worse than healthy controls at correctly interpreting hints in a series of vignettes (the Hinting Task; Janssen et al., 2003; Versmissen et al., 2008), and on a written second-order false belief task (Mazza et al., 2008), though it has been suggested that some of the relative samples used in this literature were experiencing sub-clinical psychotic symptoms themselves (Martin et al., 2014). Other studies have found no difference in ToM between relatives of schizophrenia patients and healthy controls on the Revised Mind in the Eyes Test (Kelemen et al., 2004), a cartoon picture task (Marjoram et al., 2006b), or on a written second-order false belief task, after controlling for the effects of IQ (Pentarakaki et al., 2008). A recent review has suggested the need for the literature to further expand upon the possibility of ToM deficits being an endophenotype (i.e., vulnerability marker) for schizophrenia (for review, see Martin et al., 2014).

A possible explanation for the inconsistent findings in schizophrenia family studies is the widely differing tasks used to measure ToM. In fact, an issue in the ToM literature is the heterogeneity amongst the tasks used (e.g., Bora et al., 2009a). The psychometric properties (i.e., reliability and validity) of ToM measures are widely under-studied, and there is a lack of task standardization across this literature. For example, though false-belief tasks are one of the most widely used tasks to tap into ToM abilities, there is a great deal of variability relating to the number of stories that are presented, how comprehension questions are phrased, and the method of story presentation (e.g., cartoon vs. written). In general, the majority of studies have used either written short stories or cartoon vignette tasks to assess ToM, which may not be representative of real-world encounters where tone and intonation cues, as well as facial expressions, give valuable information. The correct interpretation of the intentions of others goes beyond the simple understanding of language and

requires the ability to use contextual information in order to recognize non-literal or sarcastic utterances of speech (Langdon et al., 2002). Previous research has shown that the processes and strategies associated with detecting non-literal statements – including irony, sarcasm, and metaphor – differ depending on whether the statements are received in video/conversational form versus written form (Carvalho et al., 2009). As a result, ToM tasks utilizing written short stories and cartoons may not be adequately assessing the same abilities that are required in real-world social contexts. Taken together, this suggests further research is needed to determine whether ToM deficits are associated with schizophrenia using more ecologically valid tasks. Moreover, it can be argued that, in taking steps towards developing a standardized ToM assessment battery, video-taped stories should be included to improve ecological validity.

This study uses The Awareness of Social Inference Test (TASIT; McDonald et al., 2003), which consists of a series of videotaped vignettes that are designed to assess the detection of sarcasm, an important component of ToM (Kern et al., 2009). The TASIT has been used successfully to measure social perception abilities, and has the added benefit of being more representative of daily life encounters due to its video format (McDonald et al., 2003). Relatively few studies have utilized this task in schizophrenia research (e.g., Mancuso et al., 2011; Horan et al., 2012), with, to the best of our knowledge, no study utilizing the TASIT in a family study.

Given that ToM has been associated with critical aspects of social functioning (e.g., Roncone et al., 2002; Fett et al., 2011), it is important to understand the underpinnings of ToM deficits in schizophrenia and identify associated vulnerabilities in non-psychotic relatives. The present study investigated more ecologically valid ToM abilities in schizophrenia patients, first-degree biological adult relatives, and healthy controls in an effort to clarify whether ToM deficits are a state or trait marker for the disorder and whether the deficits are associated with the genetic liability for the disorder. Second, we investigated whether these more ecologically valid ToM impairments are associated with real-world social and global functioning in schizophrenia, as well as symptomatology. We hypothesized that schizophrenia patients would demonstrate ToM impairments compared to controls, and relatives would demonstrate an intermediate level of performance between schizophrenia patients and controls. Furthermore, we hypothesized that greater ToM impairment would predict worse social and global functioning and greater symptomatology in schizophrenia patients.

2. Methods

2.1. Participants

A total of 85 individuals participated: 30 schizophrenia or schizoaffective patients (hereafter referred to as schizophrenia patients), 28 adult non-psychotic first-degree biological relatives, and 27 healthy controls. Demographic characteristics are shown in Table 1. Schizophrenia patients were recruited through outpatient clinics at Foothills Hospital, and through community support programs in Calgary, Canada. Research staff identified first-degree biological relatives by completing a pedigree with the proband. Not all probands met recruitment criteria for the study; however, to enhance the sample, all first-degree biological relatives of schizophrenia or schizoaffective patients that met recruitment criteria were included. Thus, 22 of the 28 relatives were not related to the patients who participated in the study. A total of four relative-patient pairings were living together (1 patient-sibling pairing, and 3 patient-parent pairings). On average, the relative-patient pairings have lived together for 27 years (S.D. = 4.9 years). Healthy controls were recruited through flyers and advertisements around the community of Calgary. The University of Calgary ethics board approved the protocol and all participants provided informed written consent.

Inclusion criteria for all participants included: (1) age 18–65, (2) minimum IQ of 70, (3) no current diagnosis of drug or alcohol dependence or abuse, (4) no history of head injury or being unconscious for more than 20 min, (5) no history of electroconvulsive therapy, and (6) no history of stroke or other neurological

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