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Theory of mind deficits in chronic schizophrenia: Evidence for state dependence

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Abstract

There is evidence that people with schizophrenia show specific deficits in theory of mind (ToM). However, it is a matter of debate whether these are trait or state dependent, and the nature of the relationship between ToM deficits and particular symptoms is controversial. This study aimed to shed further light on these issues by (1) examining ToM abilities in 61 individuals with chronic schizophrenia during a stable phase as compared with 51 healthy controls matched by gender, age, educational level and current IQ, and (2) exploring the relationship between ToM and symptoms. Second order verbal stories and a non-verbal picture-sequencing task were used as ToM measures. Results showed no differences in ToM performance between patients and controls on either measure. Subsequent subgrouping of patients into remitted and non-remitted showed a worse performance of non-remitted patients only on second order ToM tasks. Specific ToM deficits were found associated with delusions. Association with negative symptoms was found to be less specific and accounted for by illness chronicity and general cognitive impairment. The results from the present study are in line with models which hypothesise that specific ToM deficits in schizophrenia are state dependent and associated with delusions. Such associations may also be task specific.

Keywords: Mentalizing; Delusions; Negative symptoms

1. Introduction

Theory of mind (ToM) refers to the ability to understand mental states (e.g. beliefs, knowledge and intentions) of others in order to predict behaviour in social contexts (Premack and Woodruff, 1978). This ability is considered to be a crucial part of social cognition and is thought to have evolved adaptively in primates to complex social environments (Brothers, 1990; Whiten, 2000). ToM impairment has been proposed as a mechanism to explain clinical signs and symptoms of schizophrenia (see Frith, 1992; Gallagher, 2004), generating a prolific body of research over the last 20 years. Recent reviews of this literature have pointed out that, while consensus exists in support of a specific ToM disruption in at least some patients, controversy remains on issues such as the trait- or

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state-dependent nature of the deficits as well as their association with particular symptoms (Brüne, 2005a; Harrington et al., 2005a).

Interest in the study of ToM in schizophrenia arose from Christopher Frith's cognitive neuropsychological approach to schizophrenia as a "meta-representational" disorder (Frith, 1992). According to this model, three cognitive abnormalities could account for major signs and symptoms of the disease. In particular, disorders of willed action underlie negative and disorganized symptoms, disorders of self-monitoring account for passivity phenomena, and defective monitoring of other people's intentions or defective mentalizing account for the emergence of paranoid symptoms. In line with this, predictions were made about the performance of subgroups of schizophrenic patients on ToM tasks and on the reasons underlying such performance. Empirical evidence accumulated to date has tended to support Frith's idea that ToM deficits may be intrinsically distinct according to the different core schizophrenia symptoms. Negative symptoms have been consistently linked to defective mentalizing (e.g. Frith and Corcoran, 1996; Langdon et al., 1997; Doody et al., 1998; Mazza et al., 2001), but these symptoms are frequently confounded by general cognitive impairment, attention deficits and executive dysfunction. Disorganization symptoms have also been related to ToM impairment. The nature of this relationship has proved to be temporal or state dependent, and is modulated by executive function, inability to extract relevant information from the context (Sarfati et al., 1997a, b, 1999; Sarfati and Hardy-Baylé, 1999; Brüne, 2005b) and simply verbal intelligence (Brüne, 2003).

With regard to paranoid symptoms, evidence is equivocal. While some studies have found ToM skills to be similar between paranoid and non-paranoid groups (Corcoran et al., 1997; Langdon et al., 2001; Pickup and Frith, 2001; Randall et al., 2003), others have shown ToM impairment to be associated with paranoid delusions (Corcoran et al., 1995; Frith and Corcoran, 1996; Corcoran et al., 1997; Drury et al., 1998; Harrington et al., 2005b) or delusions in general (Greig et al., 2004). These contradictory findings may be explained by several methodological and conceptual factors. First, different ToM tasks and different methods of clustering symptoms have been used across studies. Second, it has been argued that because groups of patients with persecutory delusions tend to be less cognitively impaired than groups of negative or disorganized patients, they might use general cognitive strategies to compensate in solving ToM tasks (Pickup and Frith, 2001). Alternatively, it has been suggested that instead of ToM deficits per se, delusional patients may over-attribute mental states to others or "overmentalise", which would explain their normal performance on ToM tasks (Abu-Akel, 1999; Abu-Akel and Bailey, 2000; Walston et al., 2000). In spite of contradictory findings, from a theoretical basis, the most intuitive view continues to be that ToM deficits should be related to paranoid delusions since they are by definition alterations in the process of attributing mental states. Further, the nature of dysfunctional ToM in deluded patients ought therefore to be state dependent. Recent research by Corcoran and Frith (2003) and Simpson and Done (2004) has shown mentalizing to be related to reasoning processes and in particular inductive reasoning. In addition, research has shown that reasoning in delusional patients is associated with several attributional biases and that these in turn may relate to ToM (Bentall et al., 2001; Randall et al., 2003; Craig et al., 2004; McKay et al., 2005; Langdon et al., 2006).

A closely related issue is whether ToM deficits in schizophrenia are state or trait dependent; that is, whether they may be considered persisting characteristics of the disorder or linked to the presence of symptoms. Evidence on this can be traced from studies comparing groups of patients with different symptom clusters to controls and from studies comparing remitted patients to controls. From the first group of studies, it is generally concluded that deficits are only found in relation to some symptoms and in connection with the acute psychotic condition (e.g. Corcoran et al., 1995, 1997; Frith and Corcoran, 1996; Drury et al., 1998; Sarfati and Hardy-Baylé, 1999; Pickup and Frith, 2001). Paradoxically, the few studies exploring exclusively remitted patients have reported ToM to be impaired in patients as compared to healthy (Herold et al., 2002; Janssen et al., 2003) and psychiatric (Mitchley et al., 1998) controls.

An additional line of evidence regarding the state- versus trait-dependent view has come from studies of subjects at high risk for psychosis. Again, findings are equivocal. Langdon and Coltheart (1999, 2004) studied ToM in persons with high versus low schizotypy and found a negative association between schizotypy and mentalizing. Similarly, Wykes et al. (2001) reported that unaffected siblings of schizophrenia patients were likewise impaired in ToM as compared to controls. Furthermore, Janssen et al. (2003) found that schizophrenic patients were most impaired on ToM tasks relative to unaffected controls, and that first degree relatives performed somewhere in between patients and controls. However, no associations with impaired ToM were found in another study by our group on schizophrenia risk markers including schizotypy (Pousa et al., 2003) and in a recent study of unaffected first degree relatives (Kelemen et al., 2004).