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Dissociation of cognitive from affective components of theory of mind in schizophrenia

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Abstract

Patients suffering from schizophrenia show impaired emotional and social behavior, such as misinterpretation of social situations and lack of theory of mind. However, there is conflicting evidence regarding their ability to perform on theory of mind tasks. Based on previous findings with patients suffering from prefrontal damage, the present study suggests that the behavioral deficit of schizophrenic patients may be due to impaired 'affective theory of mind' abilities, rather than to a general impairment in theory of mind. To test this hypothesis we assessed the ability of 22 schizophrenic patients and 55 age-matched healthy controls, to judge first and second order affective vs. cognitive mental state attribution, based on eye gaze. The relationships between negative and positive symptoms of schizophrenia, and affective and cognitive theory of mind were also assessed. Results indicated that while healthy controls made fewer errors on affective as compared to cognitive theory of mind conditions, schizophrenic patients showed a less prominent trend. Although the pattern of reaction time did not differ significantly between groups, the patients made significantly more errors in the affective conditions, as compared to controls. Furthermore, correlation analysis indicated that impaired affective theory of mind in these patients correlated with their level of negative symptoms. These results indicate that individuals with high level of negative symptoms of schizophrenia may demonstrate selective impairment in their ability to attribute affective mental states. These findings offer new insight into the affective facets of social behavior that may underlie the profound behavioral disturbances observed in schizophrenia.

Keywords: Schizophrenia; Theory of mind; Affective processing; Social cognition

1. Introduction

1.1. Social cognition and its neural correlates in schizophrenia

Impaired social cognition has been consistently observed in patients with schizophrenia and was found to link to their social functioning and behavior (Penn et al., 1999). Recently it has been suggested that specifically measures of social cognition, rather than a general cognitive deficit, may account for these patients social incompetence (Brüne, 2005a; Roncone et al., 2002). Furthermore, Brüne (2005b) have recently shown that almost one third of the variance of social behavioural abnormalities in schizophrenic patients may be explained by impaired theory of mind, but not by impaired emotion recognition abilities. This idea is supported by finding that schizophrenic patients show consistent

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impairments in social cue and emotion perception (Morrison et al., 1988; Corrigan and Addis, 1995; Bell et al., 1997; Mandal et al., 1998). Yet, whereas impaired social behavior in schizophrenia has been repeatedly described, there is little agreement in the scientific literature as to the neurocognitive and emotional correlates of these deficits.

Moreover, important corroboration for the idea of impaired social cognition in schizophrenia arises from the comparable neuroanatomical bases of both social cognition dysfunctions and symptoms of schizophrenia. Symptoms of social dysfunction have been strongly associated with prefrontal (PFC) damage (Stuss and Benson, 1986). Evidence from clinical groups (Anderson et al., 1999; Blair and Cipolotti, 2000) show that individuals with PFC damage show impaired social behavior and functioning, despite the retention of intact cognitive skills. Patients with PFC damage may show disinhibition and misinterpretation of social situations, especially when the damage is located in the orbitofrontal/ventromedial (VM) PFC region (Rolls, 1996). This selective deficit can suggest that a unique neural circuit that involves the PFC subserves social cognition (Pinkham et al., 2003; Shamay-Tsoory et al., 2004).

PFC dysfunctions have been also widely reported in schizophrenic patients (Gold et al., 1992; Bunney and Bunney, 2000). Hypofrontality, especially in the dorsolateral PFC, is one of the main findings of the disorder (Weinberger, 1988; Taylor, 1996). This hypofrontality is thought to be related to a decrease in dopaminergic activity and to negative symptoms such as social withdrawal (Carlson, 1998). Furthermore, it is the dysfunction of the PFC that is suggested to be the neural correlate for the different clinical schizophrenic subsyndromes (Vogeley et al., 1999).

Additionally to the dorsolateral PFC, schizophrenic patients also show deficits in the ventral and orbital PFC regions. Neuroimaging studies have consistently found anatomic alterations in the PFC, including the medial and orbitofrontal cortex in individuals with schizophrenia (Crespo-Facorro et al., 2000; Convit et al., 2001). Decreased activation was also found in the medial PFC during the Wisconsin card sorting task and in rest condition (Kawasaki et al., 1993). Finally, individuals with schizophrenia show evidence of hypoactivity of the right medial PFC during cognitive tasks such as time estimation and discrimination (Volz et al., 2001).

1.2. 'Theory of mind' and schizophrenia

Several proposals have been offered to account for the social impairment observed in schizophrenia. Among these, the suggestion that failure to understand other people's mental states — the ability termed "theory of mind" — has been studied most widely.

Theory of mind is defined as the capacity to make inferences regarding other people's mental states: their knowledge, needs and intentions (Premack and Woodruff, 1978).

It has been recently suggested that the development of theory of mind is part of a constellation of functions considered to be impaired in schizophrenia (Lee et al., 2004), for which the orbitofrontal and medial PFC regions are likely to be important (Happaney et al., 2004).

Given the importance of theory of mind for healthy social functioning, it is not surprising that individuals with schizophrenia perform poorly on theory of mind tasks relative to clinical and non-clinical control groups (Frith and Corcoran, 1996; Sarfati et al., 1997; Drury et al., 1998). Furthermore, it has been shown that the performance of schizophrenic patients on theory of mind tasks is comparable to that of individuals with autism and Asperger's Syndrome (Craig et al., 2004), though less pronounced (Pilowsky et al., 2000).

Theory of mind deficits in schizophrenia are believed to be specific rather than an indication of a general cognitive impairment (Pickup and Frith, 2001). Although there is no dispute regarding the existence of theory of mind impairments in schizophrenia, their nature is still unclear.

Theory of mind abilities in schizophrenia have been tested mostly using first order 'false belief' (the ability to understand that people may hold false beliefs) and second order 'false belief' (belief about belief) paradigms. Overall, the literature has focused so far mainly on cognitive theory of mind tasks and there is conflicting evidence regarding patients' ability to pass basic as well as more complex theory of mind tasks. In some cases theory of mind deficits are reported to appear specifically on second order false belief tasks (Doody et al., 1998; Pickup and Frith, 2001), whereas other studies offer evidence of impaired performance even on simple first order tasks (Frith and Corcoran, 1996; Drury et al., 1998). Additionally, Langdon et al. (2002) report that understanding of irony is impaired in patients with high ratings of positive formal thought disorder and is weaken in high-schizotypal adults (Langdon and Coltheart, 2004). On the other hand by analyzing encounters between mental health professionals and people with chronic schizophrenia McCabe et al. (2004) showed that individuals with schizophrenia demonstrate intact theory of mind abilities when tested in real life during conversational interactions. Abu-Akel and Abushua'leh (2004) have recently reported that patients with paranoid schizophrenia who are violent performed better than non-violent schizophrenics on high-level mentalizing tasks (second