

Theory of mind disability in major depression with or without psychotic symptoms: A componential view

Yong-guang Wang^{a,b}, Yi-qiang Wang^b, Shu-lin Chen^b, Chun-yan Zhu^a, Kai Wang^{a,*}

^a Department of Neurology, The First Affiliated Hospital of Anhui Medical University, 218 Jixi Rd., Hefei, Anhui Province, PR China

^b Laboratory of Brain and Cognition Research, The Seventh Hospital of Hangzhou, Hangzhou, Zhejiang Province, PR China

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Abstract

Previous reports have conceptualized theory of mind (ToM) as comprising two components and questioned whether ToM deficits are associated with psychotic symptoms. We investigated 33 nonpsychotic depressed inpatients, 23 psychotic depressed inpatients, and 53 normal controls with the following measures: Eyes Task, Faux pas Task, Verbal Fluency Test (VFT), Digit Span Test (DST) and WAIS-IQ. The depressed patients were also evaluated with the Beck Depression Inventory-II (BDI-II) and the Brief Psychiatric Rating Scale (BPRS). The nonpsychotic depressed patients and the psychotic depressed individuals were significantly impaired on tasks involving ToM social-perceptual and social-cognitive components, as well as the VFT. The psychotic depressed patients performed significantly worse than nonpsychotic depressed patients on ToM tasks. An association was found between ToM performances and both BPRS total and hostile-suspiciousness scores in the depressed group. Both of the ToM components were impaired in depressed patients. Similar mechanisms and neurobiological substrate may contribute to schizophrenia and major depression.

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1. Introduction

Theory of mind (ToM) refers to the ability to judge one's own and other people's mental states (Premack and Woodruff, 1978). This ability is recognized as essential for social and behavioral functioning. Individuals with good ToM skills may outperform those who lack such skills with respect to social success (Brothers, 1990). However, there may be disadvantages to having a highly developed theory of mind (Brüne, 2001). ToM deficits have been reported in many neuropsychiatric disorders.

Moreover, previous reports have shown that ToM deficits contribute to poor social functioning in schizophrenia. For example, Brüne (2005a) reported that the schizophrenic patients' performance on ToM tasks was a predictor of severe abnormalities in social behavior. Bora et al. (2006) demonstrated that mental state decoding ability may be a best cognitive mediator of social functioning in schizophrenia.

The most familiar characteristic of depression is the impairment of social functioning (Levendosky et al., 1995). Depressed individuals exhibit a pattern of dysfunctional interpersonal interactions. For example, previous studies reported that a ToM deficit exists in depressed patients both in acute phases of illness (Kerr

* Corresponding author. Tel.: +86 551 2922418.

E-mail address: wangkai1964@yahoo.com (K. Wang).

et al., 2003) and in periods of symptomatic remission (Inoue et al., 2004). Depressed patients with ToM deficits may be at high risk for recurrence and lower social functioning (Inoue et al., 2006). Lee et al. (2005) reported that women with unipolar depression showed deficits in the “Reading the Mind in the Eyes Task”. In these studies, researchers defined ToM as a unitary skill and used different tasks to probe ToM abilities in depression.

Recent theories conceptualized ToM capacity as comprising two major components: the social–perceptual component and the social–cognitive component (Tager-Flusberg and Sullivan, 2000; Sabbagh, 2004; Sabbagh et al., 2004). The social–perceptual component involves the ability to decode the mental states of others based on immediately available observable information. The social–cognitive component involves the ability to reason about mental states in the service of explaining or predicting the actions of others. Evidence from children with Williams syndrome showed that these two components of ToM are detached; only the social–perceptual component of ToM is spared in Williams syndromes (Tager-Flusberg and Sullivan, 2000). The two components are linked to distinct neurobiological substrates. The neurobiological substrate for the social–perceptual component primarily involves the amygdala (Adolphs et al., 1994, 2002; Scott et al., 1997), the medial temporal structures (Puce et al., 1998; Haxby et al., 2001, 2002), and the frontal lobe (Hornak et al., 1996; Sabbagh et al., 2004). The brain region that was most consistently identified in studies aimed at delineating the neural correlates of the social-cognitive component was the medial frontal area (Frith and Frith, 2001; for review, see Siegal and Varley, 2002).

To date, only a few studies have explored whether both components of ToM are impaired in depression (e.g. Kerr et al., 2003; Inoue et al., 2004; Lee et al., 2005). Sabbagh (2004) concluded that the social–perceptual component may rely on right orbitofrontal/medial temporal regions, whereas the social-cognitive component may rely on left medial frontal regions. However, it is generally believed that depression is characterized by right-hemisphere dysfunction more than by left-hemisphere deficits (e.g. Heller and Nitschke, 1998). Thus, it is important to clarify whether both ToM components are affected in depression or whether the social–perceptual component is differentially impaired. The Eyes Task was first used as an advanced ToM task in adults by Baron-Cohen et al. (1997, 2001). In addition, Sabbagh (2004) (see also Tager-Flusberg and Sullivan, 2000) adopted the Eyes Task to test the social–perceptual component in an ERPs study. In the current study, we examined the social–perceptual component in our sample using a

Chinese version of the Eyes Task (Wang et al., 2006). The Faux Pas Task is another advanced ToM task that was also first used by Baron-Cohen and colleagues (Stone et al., 1998; Gregory et al., 2002). The task requires the subject to judge whether a Faux pas occurred in a social interaction story. As Stone et al. (1998) noted, the Faux Pas Task encompasses both cognitive and empathic affective components. Accordingly, we used a revised version of the Faux Pas Task to test the social–cognitive component in the subjects. To validate that the Faux Pas Task we used indeed tested the cognitive component of ToM, we omitted the last four Faux Pas Questions (empathic questions).

Clinically, psychotic symptoms can occur in patients with depression. Hallucinations and delusions are the most common psychotic symptoms in depression. Individuals with psychotic depression show neuropsychological deficits comparable to those seen in schizophrenia. For example, previous studies demonstrated that depressed patients with psychoses are more neuropsychologically impaired than depressed patients without psychoses (Jeste et al., 1996; Basso and Bornstein, 1999; Schatzberg et al., 2000; Hill et al., 2004). Moreover, depression may be the first episode of bipolar disorder. Ghaemi et al. (2000) demonstrated that 40% of unipolar depressed patients later received a diagnosis of bipolar disorder. With regard to genetics, multiple chromosomal regions, including 18p11, 13q32, 22q11, 10p14 and 8p22, have been identified that might be related to both bipolar disorder and schizophrenia (Berrettini, 2000, 2003). These findings suggest that there similar mechanisms and neurobiological substrates may contribute to schizophrenia and depression (especially, depression with psychotic symptoms). Moreover, Frith (1992) argued that the psychotic symptoms in schizophrenia are associated with the deficits in ToM. Psychotic symptoms can be interpreted as involving false attributions to one’s own and others’ mental states. To date, there is little research addressing whether psychotic symptoms in depression are associated with poorer performance on ToM tasks (e.g. Kerr et al., 2003; Inoue et al., 2004; Lee et al., 2005). Based on the literature reviewed, we predict that depressed patients with psychotic symptoms will perform worse than those without such symptoms in tasks that require ToM capacity.

2. Method

2.1. Participants

Fifty-six adult inpatients with major depression were referred from the Seventh Hospital of Hangzhou, the

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