

# Theory of mind deficit in bipolar disorder: Is it related to a previous history of psychotic symptoms?

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## Abstract

It has been hypothesized that a Theory of Mind (ToM) deficit could be a vulnerability marker for psychosis. Recent studies, however, have shown ToM deficits in affective relapses of bipolar disorder as well as in the euthymic phase. This study analyzes the relationship between ToM and a previous history of psychotic symptoms in bipolar disorder. ToM, sustained attention and executive functions were analyzed in 75 bipolar euthymic patients with three or more previous relapses (42 of them had a history of psychotic symptoms and 33 did not) and 48 healthy subjects. ToM was assessed with the Advanced Test by Happé. ToM performance was similar in bipolar patients with or without a history of psychotic symptoms, and in both cases it was significantly reduced as compared with the healthy control group. Similarly, both bipolar groups showed impaired sustained attention and executive functions. This general cognitive deficit partially explains the differences obtained in ToM. The ToM instrument used shows low sensitivity for assessing ToM in bipolar patients and it could partially reflect general cognitive functioning rather than a specific deficit in psychosis. ToM deficit is not a trait marker for psychosis, given that it is present in bipolar disorder regardless of a previous history of psychotic symptoms.

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## 1. Introduction

Psychotic symptoms frequently occur in bipolar disorder, particularly in the manic phase, when they are present in more than half of the cases (Pope & Lipinski, 1978). Delusions (paranoid, megalomaniac, ruin, hypochondriacal, referential and bizarre) and

auditory hallucinations (reviewed in Goodwin and Jamison, 1990) are the predominant psychotic symptoms. This appearance of psychotic symptoms is associated with a higher rate of family history of other psychotic disorders (Potash et al., 2001), higher comorbidity (Strakowski et al., 1995), worse clinical outcome (Coryell et al., 1990) and worse psychosocial adjustment. Some other studies have suggested that the development of psychotic symptoms could be associated with an early age at onset (Strober, 1996) and alterations in brain structure (Reite et al., 1999). Despite the above findings, there is currently a lack of specific

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vulnerability markers to predict the development of psychotic symptoms in the course of bipolar disorder.

The relationship between Theory of Mind (ToM) and the development of psychosis has been increasingly debated in the last decade. ToM is defined as the *cognitive ability to infer mental states to oneself and to others*, in terms of thought, emotion and intention (Premack and Woodruff, 1978; Baron-Cohen et al., 1985). Frith (1992) proposed that a ToM abnormality could underlie the development of psychotic symptoms in schizophrenia, stimulating a considerable body of research (for review, see Brüne, 2005; Harrington et al., 2005). The association between a deficit in ToM and both negative (Frith and Corcoran, 1996; Langdon et al., 1997; Mazza et al., 2001) and disorganization symptoms (Sarfati et al., 1997; Sarfati et al., 1999) is well established, whereas the association with paranoid symptoms seems to be less certain (Corcoran et al., 1995; Pickup and Frith, 2001; Randall et al., 2003; Harrington et al., 2005). A supplementary line for debate has been whether a ToM deficit is a *state* marker (Pousa et al., 2006) or a *trait* marker (Janssen et al., 2003) in relation to psychosis.

There is a paucity of studies about ToM in bipolar disorder. Some affective patients were included as a clinical control group in schizophrenia studies (Frith and Corcoran, 1996; Drury et al., 1998; Sarfati et al., 1999; Fletcher et al., 1995; Mazza et al., 2001), but these samples were small and heterogeneous. Kerr et al. (2003) found ToM deficit in 20 manic patients and 15 depressed patients *versus* 13 bipolar patients in remission and a control group. Inoue et al. (2004) studied 50 patients with remitted mood disorder (mostly with major depression) and 50 healthy control subjects, and detected a ToM deficit in the affective group. The authors suggested that remission after affective episodes is not complete and that a disturbed understanding of social interactions, paramount for later social adjustment, remains. These same authors have recently shown (Inoue et al., 2006) that patients who maintain a ToM deficit after a depressive episode constitute a group at risk for poor outcome, with a higher relapse rate and worse social functioning at 1 year.

More recent studies (Olley et al., 2005; Bora et al., 2005) have confirmed a ToM deficit in euthymic bipolar patients associated to other cognitive deficits, mainly in executive function, but a relation to psychotic symptoms has not been assessed. After studying performance in a hinting task in 15 schizophrenic, 15 affective and 15 control patients, Marjoram et al. (2005) found that a ToM deficit was related to the presence of delusions and hallucinations, regardless of the disorder. Nevertheless,

the hypothetical association between a ToM deficit in remitted bipolar patients and a previous history of psychotic symptoms has only been specifically explored in one *post hoc* analysis (Bora et al., 2005). This association would indicate that a ToM abnormality constitutes a (social) cognitive marker of vulnerability to psychosis, which would be consistent with the meta-representational, deficit-based neurocognitive model of psychosis (Frith, 1992).

The primary objective of this study is to evaluate ToM performance in euthymic bipolar patients and assess whether that relates to a previous history of psychotic symptoms. A secondary objective is to analyze the possible influence of other cognitive functions on ToM performance.

## 2. Methods

### 2.1. Participants

Seventy-five euthymic bipolar I patients (42 with a history of psychotic symptoms [BP+] and 33 without a history of psychotic symptoms [BP-]) were recruited at two Mental Health Centers (Alcalá de Henares and Torrejón de Ardoz, Madrid) and at the Lithium Clinic of the Ramón y Cajal University Hospital (Madrid). Patients included in the study met the DSM-IV-TR criteria (American Psychiatric Association, 2000) for the diagnosis of bipolar I disorder. Evaluation was completed with the Lifetime version of the structured interview, the Schedule for Affective Disorders and Schizophrenia (SADS-L; Endicott and Spitzer, 1978). Bipolar disorder type II and schizoaffective disorder were excluded. In the SADS, the description of psychotic symptoms includes delusions, hallucinations, thought broadcasting, insertion or withdrawal and marked formal thought disorder. Patients were required to have a history of at least three major affective episodes, according the lifetime history section of the SADS interview. In this way we tried to avoid including false negative cases in the BP- group (subjects that might develop psychotic symptoms in the future). Age limits were established at 18 and 70 years of age, the upper limit being necessary to avoid the confusion factor due to the physiological decline in cognitive performance. Patients had to be stable and under outpatient follow-up for at least the 3 previous months. The current euthymic state was controlled by a score  $\leq 7$  on the Hamilton Depression Rating Scale (HAM-D; Hamilton, 1960) and a score  $\leq 7$  on the Young Mania Rating Scale (YMRS; Young et al., 1978). The *exclusion criteria* were: personal history of schizophrenia,

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