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The effect of antipsychotic treatment on Theory of Mind

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ABSTRACT

Background. Deficits in a patient's 'theory of mind' (TOM) have been proposed to lead to psychosis. However, it remains unclear whether TOM deficits constitute a trait- or a state-related deficit and whether they respond to antipsychotic treatment, and also whether the change in TOM and change in psychosis are associated.

Method. In the cross-sectional component of this study, 71 patients with psychotic disorders were included and TOM ability was measured using a hinting task in which subjects had to infer real intentions behind indirect speech. In the longitudinal study, a different cohort of 17 drug-free patients were included wherein they received antipsychotic treatment for 6 weeks and the effect on psychotic symptoms and TOM was measured every 2 weeks. Associations between TOM and psychopathology were assessed and a mixed effects model was used to investigate the rate of change over time.

Results. Positive and Negative Syndrome Scale (PANSS) total scores were significantly associated with TOM scores. The hinting task was not associated with positive symptoms but was significantly associated with negative and general symptoms. The longitudinal arm of the study showed that both PANSS positive scores and TOM improved after medication was started, particularly during the first 2 weeks of antipsychotic treatment, but these changes were not associated. The TOM response at 2 weeks of antipsychotic treatment reached similar values to those obtained in the cross-sectional sample.

Conclusions. Although TOM and psychotic symptoms are related to each other, antipsychotic treatment impacts each independently, suggesting a dissimilar cognitive or neurobiological substrate for the two.

INTRODUCTION

'Theory of mind' (TOM) refers to the human ability to infer intentions of others and to understand that their actions are guided by their beliefs about the world. It taps into the ability to recognize and represent one's own and other persons' mental states. Persons with schizophrenia have been shown to have deficits in TOM abilities (Brune, 2005); however, the relationship between TOM deficits and the more routinely measured symptoms of schizophrenia is still unclear. In additional, whereas antipsychotics are a mainstay in the treatment of schizophrenia, little is known about their impact on these TOM deficits. Given that TOM is a fundamental skill for navigating our social world and that impairments in social functioning are among the hallmark characteristics of schizophrenia (Pinkham *et al.* 2003),

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understanding TOM deficits and their response to antipsychotic medications may prove to be key in optimizing treatment for persons with schizophrenia.

TOM deficits may be trait markers (Langdon & Coltheart, 1999; Herold *et al*. 2002; Janssen *et al*. 2003) of schizophrenia and/or related to the state of the illness (Corcoran *et al*. 1995; Frith & Corcoran, 1996; Sarfati & Hardy-Bayle, 1999; Sarfati *et al*. 1999; Pickup & Frith, 2001). For example, Janssen *et al*. (2003) showed that first-degree relatives perform between patients with schizophrenia and controls on a hinting task requiring TOM skills, suggesting a trait-like component to the impairment. However, the only prospective treatment study examining TOM changes showed that patients in an acute exacerbation of schizophrenia performed poorly on metaphor TOM tasks relative to a group of psychiatric controls before, but not after, remission (Drury *et al*. 1998). Thus, it is not yet clear whether TOM deficits are trait or state related and, more importantly, whether these deficits respond to antipsychotic treatment.

Crucial questions to address are therefore: (1) Does pharmacological treatment improve TOM? (2) Is improvement in TOM (if any) associated with improvement in the positive symptoms of psychosis? Several possible hypotheses can be formulated. First, impaired TOM could be a mediator of psychosis formation and maintenance, in which case an association between TOM measures and symptoms at baseline and in the change with treatment would be expected. In this case TOM would be a causal factor for psychotic symptoms, and as antipsychotics altered TOM, this would then alter the psychosis. Alternatively, TOM impairment could be a moderator of psychosis formation, in which case it would serve as a predictor of those patients who would show maximal response to antipsychotic medication. In other words, *baseline* TOM would be correlated with the change in psychosis. However, with the moderator hypothesis, unlike the mediator hypothesis, the *change* in TOM would not directly correlate with a change in PANSS. A third possibility is that the TOM and psychosis are neither mediator nor moderator variables, but instead are both downstream consequences of other illness variables and are not causally related to one another. Here, baselines impairments/presence of one do not predict change in the other; nor are the changes themselves associated with each other. The resolution of psychosis may be accompanied by improvements in TOM abilities but these improvements will not be associated.

These questions cannot be resolved by using the cross-sectional approach alone. They need to be addressed with studies using both cross-sectional and longitudinal designs that would allow (*a*) direct comparison between cross-sectional and longitudinal data and (*b*) a prospectively treated cohort study to understand the effects of antipsychotic medications. In the present study, TOM tasks were first administered to a large sample of patients with psychotic disorders. The data provided an opportunity to examine the relationship between TOM deficits and psychotic symptoms in a large cohort of subjects with psychosis. Changes in TOM and its association with psychotic symptoms were then explored by longitudinally following a sample of mostly first-episode patients over 6 weeks following the beginning of antipsychotic treatment. This provided the first prospective study to relate the drug-induced improvement in psychotic symptoms and TOM. The two data sets collectively allow us to choose between the three possibilities about the role of TOM in schizophrenia and how it is affected by antipsychotic medications.

METHOD

Subjects

Patients were recruited from the in-patient and out-patient services of the Centre for Addiction and Mental Health (CAMH), Toronto. All subjects gave their written consent after the study and its procedures had been explained to them. Patients were eligible to enter the cross-sectional part of the study if they were aged 15–65 years, had an IQ >65, met the DSM-IV criteria for a psychotic disorder (schizophrenia, schizophreniform and schizo-affective disorder), had no significant medical or neurological illness, and had no current history of substance abuse or dependence. In the longitudinal part of the study, patients were followed up to 6 weeks after the initiation of antipsychotic treatment. Patients were interviewed at baseline (drug free) and every 2 weeks thereafter.

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Measures

Diagnostic inclusion criteria were ascertained by a trained psychiatrist using the Mini-International Neuropsychiatric Interview (M.I.N.I.; Sheehan *et al.* 1998). Symptom severity was assessed with the Positive and Negative Syndrome Scale (PANSS; Kay *et al.* 1987). IQ estimates were obtained with the digit symbol and information test from the Wechsler Adult Intelligence Scale (WAIS; Sattler & Ryan, 1999).

TOM was assessed with the hinting task as described by Corcoran *et al.* (1995), in which an individual is required to infer real intentions behind indirect speech. The original task comprises 10 short passages presenting an interaction between two characters, ending with one of the characters dropping an obvious hint. The subject is then asked what the character really meant when he/she said this. An appropriate response given at this stage is given a score of 2 and the next story is read out. If the subject fails to give the correct response, an even more obvious hint is added to the story. The subject is then asked what the character wants the other one to do. If a correct response is given at this stage, the subject is given a score of 1. If the subject again fails to give a correct response, a score of 0 is given for that item. An example of an item would be: George arrives in Angela's office after a long and hot journey down the highway. Angela immediately begins to talk about some business ideas. George interrupts Angela saying: 'Oh, my! It was a long, hot journey down that highway!' *Question: What does George really mean when he says this?* Twenty is the maximal score. Mean scores for healthy controls have been reported in the range 16·7–19·9 (Corcoran *et al.* 1995). In the present study four different versions of the hinting task were used as described by Marjoram *et al.* (2005*a*, *b*) and were counterbalanced in the order of administration in the longitudinal part of the study.

Statistical analysis

Statistical analysis was carried out using general linear models. Pearson regression coefficients were used as a measure of association. Longitudinal data were analysed using mixed effects models. Subjects were included as a random effect in this model to take into account the relatedness of observations measured for the same individual. Bonferroni-adjusted pairwise comparisons were used as *post-hoc* tests.

RESULTS

Cross-sectional study

Seventy-one patients participated in the cross-sectional study (schizophrenia 82%, schizophreniform 3%, schizo-affective disorder 15%). The mean age was 33·02 (S.D. = 12) years and the majority were male (83%). The mean IQ estimate was 96·10 (S.D. = 12·83). At the time of the study most patients were receiving atypical antipsychotics (88·6%), with a minority receiving typical antipsychotics (11·4%). Their mean PANSS total score (PANSS-T) was 63·93 (S.D. = 12) and their mean PANSS positive (PANSS-P), negative (PANSS-N) and general (PANNS-G) scores were 16·23 (S.D. = 5), 15·65 (S.D. = 4·7) and 32 (S.D. = 6·2), respectively.

The mean hinting task score was 16·82 (S.D. = 2·8), in accordance with values reported for a similar population (mean 15·6, S.D. = 3·9) by Corcoran *et al.* (1995). Significant correlations were found between the score on the hinting task and the PANSS-T ($r = -0{\cdot}29$, $p < 0{\cdot}014$). In particular, the hinting task was not associated with positive symptoms (PANSS-P: $r = -0{\cdot}09$, $p = 0{\cdot}43$), but was significantly associated with negative symptoms (PANSS-N: $r = -0{\cdot}35$, $p < 0{\cdot}002$) and general symptoms (PANSS-G: $r = -0{\cdot}24$, $p < 0{\cdot}04$). These associations were not changed when IQ was added as a covariate.

Longitudinal study

Seventeen patients were included in the longitudinal study (schizophrenia 76%, schizo-affective disorder 6%, schizophreniform disorder 18%). The mean age of the subjects was 31 (S.D. = 12) years, and the majority were male (76%). Sixty per cent of this group were neuroleptic naive at the beginning of treatment, while the other 40% were drug free. Most subjects were started on atypical antipsychotic medications, except for two patients who were restarted on their previous clozapine dose (300 and 225 mg). The rest were started on risperidone 4 mg ($n = 4$), 3 mg ($n = 1$), 3·5 mg ($n = 1$), 1 mg ($n = 1$) or olanzapine 10 mg ($n = 4$), 20 mg ($n = 1$), 15 mg ($n = 1$), 2·5 mg ($n = 1$), and one

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Table 1. *Change in PANSS (total, positive and negative) and TOM over time*

	Baseline score (S.D.)	2 weeks' change	2–4 weeks' change	4–6 weeks' change	Total change
PANSS-T	86·68 (9·84)	−12·68*	−9·41	−7·1	−29·28*
PANSS-P	24·78 (3·04)	−4·21*	−3·1*	−2·73	−10·05*
PANSS-N	18·47 (5·25)	−2·42	−1·34	−1·23	−5*
TOM	14·57 (4·3)	2·92*	0·93	0·49	4·35*

PANSS, Positive and Negative Syndrome Scale: total (T), positive (P) and negative (N) scores; TOM, theory of mind.
* Bonferroni adjusted statistically significant $p<0·05$.

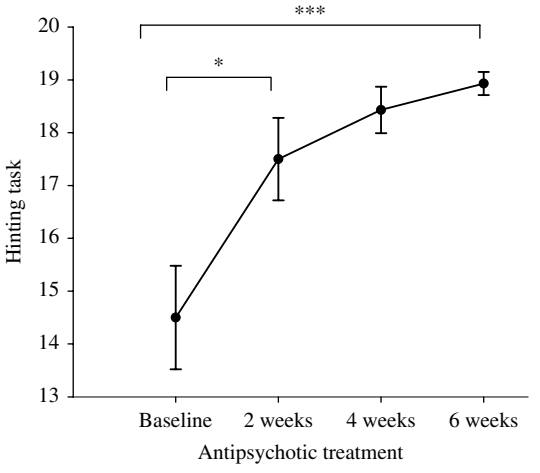

FIG. 1. Theory of mind (TOM) change after the beginning of antipsychotic treatment.

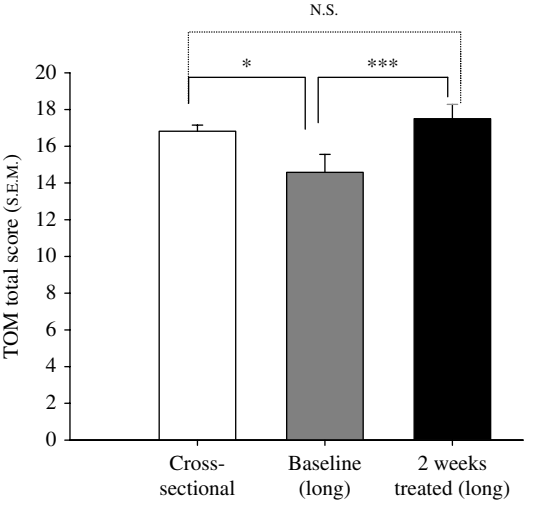

FIG. 2. Comparison of theory of mind (TOM) scores in the cross-sectional cohort and longitudinal cohort, before and after 2 weeks of antipsychotic treatment.

patient was restarted on her previous 35 mg of loxapine. Six patients were hospitalized, while the other 11 patients were treated on an outpatient basis.

The standard measures of PANSS showed the expected improvement over time (PANSS-P: $F=32·59$, df $=3·48$, $p<0·0001$; PANSS-N: $F=3·44$, df $=3·48$, $p=0·024$; PANSS-T: $F=25·12$, df $=3·48$, $p<0·0001$). The greatest change occurred during the first 2 weeks of antipsychotic treatment ($t=3·73$, df $=48$, $p=0·0005$), and kept improving thereafter (Table 1). The PANSS-N improvement was only significant between baseline and 6 weeks, suggesting that rapid improvement (i.e. within 2 weeks) was only seen for PANSS-P and PANSS-G.

Longitudinal analysis of the TOM data showed that performance on the hinting task changed significantly during the 6 weeks of antipsychotic treatment ($F=7·42$, df $=3$, $p=0·0004$). Scores on the hinting task were significantly different from baseline at 2 weeks ($t=–2·30$, df $=17$, $p=0·034$), and continued to improve thereafter, a similar pattern to that shown by PANSS-P (Table 1 and Fig. 1). The TOM response at 2 weeks of antipsychotic treatment reached similar values to that obtained in the cross-sectional sample [mean $=16·82$ (S.D. $=2·8$) for the cross-sectional and mean $=17·50$ (S.D. $=3·31$) in the 2-week treated longitudinal cohort; $t=–0·80$, df $=23·62$, $p=0·42$, equal variances not assumed]. However, patients in an acute psychotic episode (i.e. baseline in our longitudinal study) showed greater TOM deficits than that obtained in the cross-sectional sample [mean $=16·82$ (S.D. $=2·8$) for the cross-sectional and mean $=14·58$ (S.D. $=4·31$) at baseline in the longitudinal cohort; $t=2·14$, df $=22·28$, $p=0·043$, equal variances not assumed] (Fig. 2).

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Finally, we wanted to investigate how the change in psychopathology would be associated with the above-mentioned changes in TOM skills and how they would associate at baseline. At baseline, hinting task scores were not associated with PANSS-P ($r=0{\cdot}006$, $p=0{\cdot}98$) but were significantly associated with PANSS-G, PANSS-N and PANSS-T ($r=-0{\cdot}67$, $p=0{\cdot}002$; $r=-0{\cdot}52$, $p=0{\cdot}02$; and $r=-0{\cdot}61$, $p=0{\cdot}005$, respectively). This is consistent with the findings from the cross-sectional cohort (above). The changes observed in psychotic symptoms (percentage improvement in PANSS-P) and the changes observed in TOM skills (percentage improvement in TOM) were not significantly associated ($r=-0{\cdot}33$, $p=0{\cdot}22$ for PANSS-P and $r=-0{\cdot}10$, $p=0{\cdot}71$ for PANSS-T).

DISCUSSION

Although several studies have investigated TOM abilities in persons with schizophrenia (Brune, 2005), this longitudinal study is the first to follow a sample of drug-free subjects after the beginning of antipsychotic medications. The results from the cross-sectional arm of the study added to the growing body of evidence showing TOM to be impaired in those with schizophrenia. Furthermore, the association observed with negative symptoms in schizophrenia is in keeping with several studies that have shown a significant association between TOM difficulties and negative symptomatology in schizophrenia (Corcoran *et al.* 1995; Doody *et al.* 1998) or the 'psychomotor poverty syndrome' proposed by Liddle (Mazza *et al.* 2001). The present data also confirm the predictions of Frith & Corcoran (1996) that patients with prominent negative behavioural signs (i.e. poverty of speech, social withdrawal, flat affect) would perform poorly on TOM tasks. However, we did not find any association with positive symptoms; this may be related to the relatively low PANSS-P scores obtained in the cross-sectional sample or the lack of characterization by schizophrenia diagnosis subtypes (i.e. disorganized, paranoid), as has been reported previously (Greig *et al.* 2004).

The study adds unique data on the longitudinal effects of treatment on TOM indices, as indexed by the hinting task scores that approached the ceiling score after 6 weeks of treatment. This may explain why earlier cross-sectional studies have reported that schizophrenic patients in remission show quasi-normal TOM abilities (Corcoran *et al.* 1995; Frith & Corcoran, 1996; Sarfati *et al.* 1997, 1999; Drury *et al.* 1998; Sarfati & Hardy-Bayle, 1999). The pattern of change seems to supports TOM impairments as state (rather than trait) phenomena in first-episode patients; however, more difficult tasks (i.e. second-order tasks) that do not reach a ceiling effect or characterization by schizophrenia subtypes should be carried out to support this claim.

Of the three possibilities raised in the introduction (TOM as a mediator, moderator or co-occurring deficit), the data support the hypothesis that TOM and the psychosis are both downstream consequences of other cognitive processes or biochemical abnormalities co-occurring, but not causally related to one another. We found no relationship at baseline between psychosis and TOM, and while both improved with treatment, this improvement was not associated. It seems likely then that antipsychotic action in different brain areas that subserve social cognition and psychotic symptoms may explain this finding. Indeed, recent functional studies have identified the medial frontal cortex as the most consistent activated area in TOM tasks (Fletcher *et al.* 1995; Goel *et al.* 1995; Happe *et al.* 1996; Brunet *et al.* 2000; Calarge *et al.* 2003) and psychotic symptoms in schizophrenic individuals have been associated with the temporal cortex (Shamay-Tsoory *et al.* 2005; Suzuki *et al.* 2005) and the ventral portion of the striatum (Epstein *et al.* 1999). Reports of a functional overlap of TOM skills and the superior temporal sulcus and temporal poles qualify our conclusions (Vogeley *et al.* 2001; Frith & Frith, 2003; Vollm *et al.* 2006). Nevertheless, a similar mechanism of action of antipsychotic medications (i.e. dopamine receptor blockade) in different areas of the brain may explain this joint but uncorrelated improvement in TOM and psychotic symptoms. Alternatively, the lack of correlation observed in this study between TOM and psychosis improvement may be secondary to a distinct pattern of change of these two. For example, TOM may improve with treatment in a non-linear fashion, while psychopathology may follow a linear pattern of change.

The greatest change in TOM and psychosis occurred during the first 2 weeks of antipsychotic treatment. These findings are concordant with the recent considerations of an early onset of antipsychotic treatment (Abi-Dargham *et al.* 2000; Agid *et al.* 2003; Kapur *et al.* 2005; Leucht *et al.* 2005). This early improvement in TOM abilities, which shows the same pattern of improvement as in positive symptoms, suggests that antipsychotic treatment may be sufficient to improve TOM abilities in a group of subjects with first-episode psychosis and that the improvement is likely to be the primary effect of antipsychotic-induced dopamine blockade rather than secondary to some longer-term alterations.

There are several limitations that qualify our conclusions. First, no control group is available for either the cross-sectional or longitudinal part of the studies. Although we acknowledge this limitation, we would like to point out that the main focus of the study was to investigate TOM both cross-sectionally and longitudinally, and so far this is the only study to do this. Furthermore, the mean hinting task for the group with schizophrenia was comparable to previously published patient values and worse than published means for healthy subjects (Corcoran *et al.* 1995). Unfortunately, there are no normative data showing the test–retest profile for the hinting tasks. However, we have attempted to mediate the possibility of practice effects by using multiple versions of the task. However, if practice effects would have mediated the improvement in the hinting task, this would mean that practice in TOM tasks could improve social cognition, or at least social cognition test-taking skills. The longitudinal cohort does not have a placebo control group. A prospectively placebo-treated group of patients in their first episode of psychosis would be needed to avoid this limitation, which may not be feasible for ethical reasons. Finally, the degree of change presented in the longitudinal study may more accurately describe the response of the first-episode neuroleptic-naive patients experiencing their first trials with antipsychotic medications, rather than the response of chronically treated patients.

Our findings show that TOM impairments, as measured with the hinting task, are amenable to treatment with antipsychotic medication, at least in persons in their first episodes of psychosis. TOM is a crucial skill for living in a social world. Persons with schizophrenia are already socially marginalized in several ways. If TOM abilities are improved by antipsychotic medications, and as these do not seem to reflect only an improvement in psychotic symptoms, the data open up the road to investigate how antipsychotic treatment affects social cognition in patients with schizophrenia.

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DECLARATION OF INTEREST

None.

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