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Exploratory study of the association between insight and Theory of Mind (ToM) in stable schizophrenia patients

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to obtain the title of Doctor in Psychology

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“Toda vida verdadera es encuentro”

(Martin Buber, Yo y tú)

Aquesta tesi està dedicada al Toni,
el meu company, el meu amic, el meu estimat...

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1. GENERAL OVERVIEW

The research project included in this thesis stemmed from the author's clinical interest in understanding the phenomenology of schizophrenia, during the first years of working experience as a clinical psychologist with psychotic patients. First, theoretical and clinical knowledge in the area of insight in psychosis was gained through participation in the Spanish validation study of the Scale for Assessment of Unawareness of Mental Disorder (SUMD) (Ruiz et al., 2008). Listening to a large number of patients' answers on a detailed interview on their illness awareness was striking and raised a genuine interest in the understanding of such phenomena. In parallel, clinical experience in leading schizophrenia social skills training groups and working with individual clients towards the improvement of their global functioning also implied reflecting on factors that contributed to their functional status. Among these, particular interest arose regarding neurocognitive and social cognition deficits. This led to focus the first research project of the PhD course on a thorough review of the literature on ToM in schizophrenia and on the translation and cultural adaptation of an instrument to measure ToM in high functioning autistic patients (Pousa, 2002, unpublished). In this context, the idea that there might be some parallelisms between particular dimensions of illness unawareness and social cognition deficits arose and became the main study hypothesis for the PhD thesis.

At that time, while deficits in insight in schizophrenia had been widely and consistently reported in the literature, evidence on a ToM dysfunction in schizophrenia was scarce and its nature controversial. This was mainly due to methodological differences across studies as well as limitations regarding the instruments used for ToM assessment. Thus, the first aim of our research consisted of clarifying some of these issues -whether a specific ToM dysfunction existed in schizophrenia and whether this most suitably fitted in the state or trait deficit views- trying to overcome previous methodological drawbacks. We did so by the use of a well matched control group, by controlling for important confounds and by the inclusion of ToM

instruments of different nature (verbal and pictorial tasks). Results of this project were included in a first paper (Pousa et al., 2008) and also led to the publication of a letter discussing part of the conclusions of a recent meta-analysis on ToM in schizophrenia (Pousa, Ruiz & David, 2008).

Following this preliminary work and on the basis of a number of phenomenological parallelisms between insight and ToM dysfunctions that could be appreciated both clinically and in the literature, we decided to explore the relationship between insight and ToM. Since both phenomena are conceptually complex and a number of methodological difficulties exist around their measurement, it was a big challenge to go into such area of research. This, together with the scarcity of previous studies specifically focused on this issue, implied having to make several important decisions during its design and development. Thus, the nature of the study was mainly exploratory. Along the different stages of the project, an effort was punt into trying to be the most scientifically rigorous at the same time as not loosing phenomenological complexity. For all this reasons, the project implied a lot of preliminary analysis using distinct perspectives and methods, revealing a number of interesting results, the most relevant of which led to a second paper (Pousa et al., in press).

Besides the mentioned publications, two complementary published works are added in the present thesis for their relevance to the project (see Annex I). The first is the manuscript of the Spanish adaptation of the SUMD (Ruiz et al., 2008). The second is a chapter of a book on mental disorders from an evolutionary perspective, titled “Theory of Mind as an evolutionary brain module”. This chapter describes the concept of ToM, its measurement, as well as its neurobiological basis and philogenetic development, and was part of the literature review carried out while working on the design of the project (Obiols & Pousa, 2005).

2. INTRODUCTION

2.1. ToM in schizophrenia

2.1.1. ToM: Definition and terminology

Humans live in a social world and our everyday functioning as social beings entails seeing and understanding ourselves and others in terms of mental states. We need to see the desires, emotions, beliefs, intentions, and other inner experiences of ourselves and others, in order to understand and behave appropriately in social interaction. Because this understanding of the actions of others and one's self in terms of agency and intentional states is central to a child's socialisation and to the capacity of adults to empathically and correctly understand each other, it is not surprising that this topic has been a focus of scientific interest from different disciplines, including philosophy, anthropology, developmental and social psychology, psycholinguistics, psychopathology, neurobiology and cognitive neurosciences.

In line with the diversity of perspectives under which the issue has been approached, a variety of different terminologies have been adopted in the scientific literature to refer to the capacity to understand other people's minds or to refer to related abilities which include or entail this capacity. From a philosophical point of view, for example, the general ability to represent an object or thing in mind has been termed **metarepresentation**, and the idea includes both the ability to represent physical objects and mental states. Within the disciplines of anthropology and primatology, terms like **social intelligence** or **social reasoning** (Humphrey, 1976; Byrne & Whiten, 1988; Whiten & Byrne, 1997) are usually used, particularly in contrast to general intelligence or other instrumental cognitive abilities. Social psychology, on the other hand, has used the term **folk psychology** or **social cognition** to describe the domain of cognition that involves the perception, interpretation, and processing of social information (Ostrom,

1984), and to underline the fact that it represents a level of analysis of human functioning that differs from non-social cognition. In general psychology, a recently widely used term has been **emotional intelligence**. This term emphasises the idea that adaptive and successful behaviour in the present world does not rely on the classical idea of Intelligence Quotient (IQ), but in a more general way of interacting with the world. Emotional intelligence is a type of social intelligence that involves the ability to monitor one's own and others' emotions, to discriminate among them, and to use the information to guide one's thinking and actions (Salovey & Mayer, 1989). **Metacognition** is also a closely related term which has been used mainly by developmental psychologists when studying metacognitive development (Flavell, Miller & Miller, 1993). The concept includes knowledge about the nature of people as cognizers, about the nature of different cognitive tasks, and about possible strategies that can be applied to the solution of different tasks. It also includes executive skills for monitoring and regulating one's cognitive activities. The majority of studies in the area of metacognition have dealt with children's metamemory, and also children's metacognition regarding language and communication, perception and attention, comprehension, and problem solving.

Traditionally, however, the disciplines in which research on the particular topic of "understanding oneself and others in terms of mental states" has been central and most prolific over the last twenty-five years, have been developmental psychology and psychopathology. These have focused on how, when, and in what manner these abilities arise in child development, and on what takes place in developmentally delayed conditions such as autism. Among these disciplines, the term "**Theory of Mind**" (ToM) has been the most commonly used, together with others like "mindreading", "understanding of other minds" (Baron-Cohen, Tager-Flusberg & Cohen, 2000) or "mentalising" (Frith, Morton & Leslie, 1991). In the area of psychopathology, "ToM impairment", "abnormalities in understanding other minds", "ToM deficit" and "mindblindness" are used to refer to the lack of ToM.

The term Theory of Mind was first introduced by Premack and Woodruff in 1978 to define the “ability to attribute mental states to self and others in order to predict their behaviour” (Premack & Woodruff, 1978). ToM is considered to be a crucial part of social cognition and is thought to have evolved in primates with adaptive means to the increasing complex social environment (Brothers, 1990; Whiten, 2000). Moreover, a number of structural and neuroimaging studies exploring the neural systems underlying ToM have consistently reported activation in what seems to be a highly circumscribed mentalising network, comprising the medial prefrontal cortex, the superior temporal sulcus- especially around the temporo-parietal junction- and the temporal poles adjacent to the amygdala (Brüne & Brüne-Cohrs, 2006).

Although there is an agreement among researchers on the definition of ToM, in parallel with the accumulating research advances in developmental cognitive neurosciences in the last decades, the concept has progressively broadened, perhaps reflecting the fact that it’s not a unitary function. Brothers and Ring (1992) proposed that mentalising is a complex ability that involves two distinct capacities, one more “cognitive” and the other more “affective”, referred to as “cold” and “hot” aspects of theory of mind. Based on evidence from developmental psychology and psychopathology studies as well as from research on the neurobiological basis of ToM, Tager-Flushberg & Sullivan (2000) developed the “Componential model of Theory of Mind” which distinguishes between the capacity to make complex cognitive inferences about the content of mental states (social-cognitive component) from the on-line immediate judgement of mental states (social-perceptual component) (see Table 1). Further evidence of such distinction can be found in recent studies carried out by Shamay-Tsoory et al. reporting a differential impairment of cognitive and affective aspects of theory of mind in both individuals with Asperger's syndrome (Shamay-Tsoory et al., 2002) and in patients with pre frontal cortex damage (Shamay-Tsoory et al., 2003, 2004, 2005).

Paralleling the complexity of ToM, a wide variation in the operationalisation of the concept can be found in the research literature, with a variety of existing ToM tasks.

Table 1: Summary of Tager-Flusberg & Sullivan 's componential Model of ToM.

	Social-cognitive	Social-perceptual
Conceptual understanding	Original concept of ToM. Mind as a representational system.	Mentalising abilities Person perception and knowledge
Prototypical Tasks	False belief	Distinction between people and objects, on-line rapid judgements about people's mental states from facial and body expressions, intentional and other person-related knowledge.
Developmental links	Language and other cognitive capacities	Affective system
Emergence in child development	3 –4 years	Innate
Brain areas involved	Prefrontal cortex: orbito-frontal areas and medial-frontal areas	Amygdala and associated regions of medial temporal cortex
Selective affectation	Impaired in Autism	Impaired in high functioning autism or Asperger syndrome. Spared in Williams syndrome

2.1.2. Measuring ToM abilities in schizophrenia

Mindreading abilities in schizophrenia have been tested using a number of paradigms, usually adopted from tests used in developmental psychology and psychopathology. The ‘‘gold standard’’ test of comprehending other persons’ minds is grasping that others can hold false beliefs that are different from one’s own (correct) knowledge (Dennett, 1978). Thus the majority of ToM tasks involve false belief understanding. The classic Sally and Anne Test (Baron-Cohen, Leslie & Frith, 1985) involves experimental creation of a situation in which a test person has to distinguish his or her own knowledge that an object has been hidden by one character (Anne) in the absence of another person (Sally) from the knowledge of the other characters involved. The crucial question is where Sally would look for the object when she returned: the place it was before she left the scene, or the place where it had been moved by Anne.

Whereas false belief tasks are called **‘first-order’ tests** because they only involve inferring one person’s mental state, **‘second-order’ tests** have also been developed to examine a more elaborated ability consisting of understanding a false belief about the belief of another character. As is the case in first order false belief tasks, second order tasks have also been designed as stories that are read to the subject, usually using props to aid subjects’ concentration and comprehension, followed by a test question. Although there are multiple versions of second-order stories, ‘The Ice-Cream Van’ test (Baron-Cohen, 1989) and ‘The Burglar Story’ (Happé & Frith, 1994) are some of the most widely used of this type (see detailed description of these tests in Box I).

Other specific tasks for the assessment of more **advanced mindreading abilities** have consisted of tests that imply the use of **deception** (Sodian et al., 1992), the understanding of **jokes** (Baron-Cohen, 1997) and of **metaphors**, **sarcasm** and **irony** (Happé, 1994), as well as situations that involve the use of **pragmatics** in speech and communication (e.g. Surian, Baron-Cohen & Van der Lely, 1996) or the understanding of ‘**Faux Pas’**

(Baron-Cohen et al., 1999). To understand that a ‘faux pas’ has occurred, one has to represent two mental states: that the person saying it does not know that they should not say it and that the person hearing it would feel insulted or hurt.

When these tasks have been applied to schizophrenia they have usually been modified to better control for interference with attention, memory, ‘‘general’’ intelligence, and verbalization. Thus tasks have been complemented with comprehension aids, consisting of accompanying cartoon drawings depicting the story content and with control measures including both memory control questions (Frith & Corcoran, 1996, Drury, Robinson & Birchwood, 1998; Doody et al., 1998; Mazza et al., 2001) and matched non-mental representation control tasks (Pickup & Frith, 2001; Frith and Corcoran 1996; Langdon et al., 1997; Sarfati et al., 1997; Drury et al., 1998; Brunet et al., 2003). Some studies have also used new tasks specifically designed for schizophrenia. Examples of these are ‘The Hinting Task’, tests of understanding visual jokes, and picture sequencing tasks. In ‘The Hinting Task’, subjects are read ten short pieces of indirect speech presenting characters dropping a very obvious hint, and are then asked to comment on the character’s intention (Corcoran, Mercer & Frith, 1995). In tests of understanding visual jokes subjects are presented a series of ten pictures representing jokes that require mental state attribution and are asked to assess and explain them (Corcoran, Cahill & Frith, 1997; Happé, Brownell & Winner, 1999). Picture sequencing tasks, on the other hand, consist of a paradigm using comic strips which does not use verbalisation in either the material itself or in the answering procedure. Other tasks used with schizophrenic subjects are tasks of understanding the violation of conversational Gricean maxims and politeness convention (Corcoran & Frith, 1996; Tényi, Herold & Trixler, 2002). Examples of items included in some of these tests are shown in Box II. Finally, other type of tests that involve inferring mental states from looking at pictures of eyes such as the “Reading the Mind in the Eyes” test (Baron-Cohen et al., 2001) or the ability to judge mental states based on verbal and eye gaze cues have also been used in schizophrenia (e.g. Russell et al., 2000; Kelemen et al., 2004;

Shamay-Tsoory et al., 2007). Although these tasks have been referred to as ToM tasks, the construct being measured is different from that of other paradigms, perhaps assessing emotion recognition abilities or empathy rather than ToM.

Recent reviews of the literature on ToM in schizophrenia have consistently pointed out that discrepancies in the findings across studies and difficulties in their interpretation stem from methodological problems, mostly having to do with ToM assessment (Brüne, 2005; Harrington et al., 2005a; Sprong et al., 2007).

First, the problem of ‘‘real life’’ presentation of the tasks cannot fully satisfactorily be resolved in experimental laboratory ‘‘offline’’ test conditions. Therefore, persons with psychiatric disorders who on one hand are highly personally involved in their delusional systems, for instance, may in the test situation be relatively unaffected in abstract reasoning tasks (Simpson, Done & Vallé-Tourangeau, 1998), or may show intact ToM abilities in conversations with, for example, mental health professionals (Mc Cabe, Leudar & Antaki, 2004).

Second, comparability of ToM studies in schizophrenia may to some extent be limited because of differences in the demands of theory of mind tasks utilized in studies, perhaps involving different processes. For example, whereas performance on the false belief task requires cognitive understanding of the difference between the speaker's knowledge and that of the listener (knowledge about beliefs), irony and faux pas tasks require an additional empathic appreciation of the listener's emotional state (knowledge about emotions). As already mentioned, tasks such as the “Reading the Mind in the Eyes” test may be mainly measuring emotion recognition abilities.

Overall, the main problem is that tasks have generally been selected in studies on a theoretical basis, or because they have previously been used in developmental or autism research. Although this may confer them certain

construct validity, there is a serious lack of evidence on the psychometric properties of tasks in adult populations, such as test-retest reliability or criterion validity. Furthermore, a large proportion of the studies have only used one form of task, which precludes any evaluation of their convergent validity (ct. Harrington et al., 2005a).

Future studies need to further define the nature of different ToM tasks, to establish their construct and criterion validity, to develop mindreading tasks assessing more than intention attribution (e.g. needs, values of others), and to shed light to the relationships between various tasks. Further task validation might include investigating whether poor performance in the task is related to poor social skills and functioning using social functioning measures.

Box I: Description of examples of second-order ToM stories

'The Ice-Cream Van' test (Baron-Cohen, 1989):

John and Mary are together in the park. Along comes the ice-cream man. John would like to buy an ice-cream but has no money with him. The ice-cream man tells him to go home and get his money. In the meantime he will be staying in the park. When John comes home to get the money, the ice-cream man moves to the church. Later, John meets the ice-cream man in front of the church, but Mary does not know about that because she came back home before.

ToM Question: Where does Mary think that John has gone to buy an ice-cream?

Control Question: Does Mary know that the ice-cream man has talked to John?

'The Burglar Story' (Happé & Frith, 1994):

Burglar Bill has just robbed a bank and is running away from the police when he meets his brother Bob. Bill says to Bob, 'Don't let the police find me, don't let them find me!' then he runs off and hides in the church yard. The police have looked everywhere for Bill except the church yard and the park. When they come across Bob they were going to ask him, 'Where is Bill, in the park or in the church yard?' But the police recognise Bob and they realise that he will try to save his brother. They expect him to lie and so, wherever he tells them, they will go and look in the other place. But Bob who is very clever and does want to save his brother knows that the police don't trust him.

ToM Question: Where will Bob tell the police to look for Bill in the church yard or in the park? Why?

Control Question: Where is Bill really hiding?

Box II: Examples of items included in ToM tasks used in schizophrenic patients.

The Hinting Task Item (Corcoran, Mercer & Frith, 1995):

Paul has to go to an interview and he's running late. While he's cleaning his shoes he says to his wife, Jane: 'I want to wear that blue shirt, but it's very creased'.

Question: What does Paul really mean when he says this?

Extra information: Paul goes on to say: 'It's in the ironing basket'

Question: What does Paul want Jane to do?

Comic strip task item (Sarfati & Hardy-Baylé, 1999)

Image A: A man is walking with a fishing rod.

Image B: He arrives at a pond.

Image C: In front of the pond, he kneels down and starts to scrape away the earth

Answer card 1 (appropriate to the character's intention): He is standing with a worm in his hand.

Answer card 2 (a frequent situation in everyday life): He is standing with a bunch of flowers in his hand.

Answer card 3 (perceptive similarity with the last picture of the story): Kneeling in front of the pond, he is holding a teapot.

Answer card 4 (very unusual situation with no link with the comic strip): He is riding an elephant.

Conversational maxims item (Tényi, Herold & Trixler, 2002)

Violations of the Maxim of relevance:

Vignette 1: A boss at a workplace is asked to give his opinion about his co-workers. About X, he says:' I don't have any opinion about him'.

Vignette 2: A professor is asked to give his opinion about his junior lecturer. The answer is:'She is a female'.

Vignette 3: Students are asked about their teachers. A student says about teacher A: 'He's very young'.

Vignette 4: A teacher of art is asked about his students' talents. His answer about student B is: 'She has an attractive body'

2.1.3. Is there a ToM deficit in schizophrenia?

The Theory of Mind deficit in schizophrenia was first hypothesized by Frith (1992), who, drawing on diverse research findings from the study of higher primates, autism and neurophysiology, postulated that schizophrenia could be understood as a disorder of the representation of mental states. He suggested that the signs and symptoms of the disorder reflected underlying cognitive deficits within a system which enables the recognition and monitoring of one's own willed intentions as well as the attribution of intentions, thoughts, and beliefs to others (Frith, 1992, 1994). In particular, he argued that the specific psychotic signs or symptoms reflect the precise nature of the "metarepresentational" dysfunction. Thus, the inability to represent the intentions of others would contribute to paranoid delusions; a failure to monitor one's own intentions to act would lead to passivity phenomena (delusions of control, thought insertion, thought withdrawal) and auditory hallucinations; and impairments in patient's ability to consider others' perspectives during conversations (e.g. by omitting the appropriate speech referents) would underlie the patient's thought disorder manifested by incoherent speech. In addition, negative symptoms, such as social withdrawal and abulia, would arise from a complete failure to represent intentional behaviour, rather than impaired functioning, and would render the patient completely disinterested in social contact and impoverished in speech.

Ever since Frith's first proposal (Frith, 1992), the association between mentalising and the core symptoms of schizophrenia has been an important focus of research interest, with over thirty published studies in the last fifteen years comparing their schizophrenia sample with a control group. These have been critically reviewed twice (Brüne, 2005; Harrington et al., 2005a) and have been focus of recent a meta-analysis (Sprong et al., 2007), including 29 studies, and over 1500 participants (831 patients and 687 controls).

Data from these reviews and meta-analysis are fully consistent in indicating that schizophrenia patients show a significant and stable mentalising impairment. This is specific rather than secondary to the generalised cognitive impairment characteristic of the illness, and cannot be accounted for by the presence of general psychopathology. Moreover, the magnitude of the deficit is more than one standard deviation below that of healthy controls ($d=-1.255$, $p<0.0001$), which is conventionally considered a large effect size (Cohen, 1988).

Another consistent finding that comes out from this literature is that the severity of the ToM deficit in schizophrenia is not as strong as that found in autism, the majority of schizophrenic patients having an intact ability to solve first order false belief tasks, irrespective of their symptom profile. In contrast, strong support exists for a second-order ToM problem in schizophrenia. The different nature of the ToM impairment in schizophrenia and in autism may be explained by the age of onset of the disorders. While autism is present at a very early age, suggesting that there is an impairment in the development of the ability to represent mental states, the middle-age onset of schizophrenia suggests that people have experienced normal development of the metarepresentational ability. Impairments only occur at the illness onset, when the individual has had a long history of success using ToM, so keeping some residual skills.

A related question that has started to get attention by some researchers but that needs further understanding concerns the particular ToM process which might be impaired in schizophrenia. As previously mentioned, there is evidence to support a differentiation between cognitive and affective ToM abilities (Tager-Flusberg & Sullivan, 2000). The few existing studies exploring this distinction in schizophrenia have pointed out that high level of negative symptoms may demonstrate selective impairment of affective ToM (Shamay-Tsoory et al., 2007). A specific affective ToM impairment has also been revealed in patients with paranoid schizophrenia who are violent as compared to those who are not (Abu-Akel and Abushua'leh, 2004).

Other issues that deserve further investigation are the relationship between ToM impairment and particular symptom clusters, and the nature of the ToM impairment. Next, a brief account of the accumulated evidence on these will be presented. Firstly, an overview of the results regarding the association of ToM deficits to symptomatology will be described. Secondly, issues regarding the state versus trait deficit hypothesis will be outlined.

2.1.4. ToM and symptoms

Based on the above-mentioned Frith's metarepresentational model of schizophrenia, predictions were made about the performance of subgroups of schizophrenic patients on ToM tasks, and on the reasons underlying such performance (Corcoran, Mercer & Frith, 1995; Corcoran & Frith, 1996; Corcoran, Cahill & Frith, 1997; Frith & Corcoran, 1996) (See table 2). Thus, patients with positive or negative behavioural signs were expected to perform very poorly, those with paranoid symptoms relatively poor, and patients with passivity symptoms as well as remitted patients were expected to perform normally.

A series of experiments have been carried out to test this model, by Frith's group and others (For a review, see Brüne, 2005; Harrington et al., 2005a; Sprong et al., 2007). So far, strong support has been reported for the relationship between **negative symptoms** and defective mentalising (e.g. Doody et al, 1998; Mitchley et al, 1998; Langdon et al., 2001; Pickup & Frith, 2001). Although it has been shown that this relationship cannot be totally explained by general cognitive deficits, given that patients with negative symptoms present other prominent specific cognitive impairments in functions closely linked to ToM performance such as memory, the relationship between negative symptoms and ToM may not be specific (Corcoran & Frith, 2003). The relationship with **disorganized symptoms** has also been systematically revealed by Frith's group and in a series of studies by Sarfaty and colleagues on patients with prominent thought and language disorganization (Sarfaty & Hardy-Bayle, 1999; Sarfati, et al., 1997a, b; Sarfati et al., 1999). The nature of this relationship has proved to be temporal or state dependent, and again non-specific, as it is linked to impaired executive function and inability to extract relevant information from the context. Finally, support for the hypothesized specific and state dependent ToM deficit in patients with **paranoid symptoms** is more equivocal. To date, at least six studies have been able to show this association (Corcoran, Mercer & Frith, 1995; Frith & Corcoran, 1996; Corcoran, Cahill & Frith, 1997; Drury, Birchwood & Robinson, 1998; Craig et

al., 2004; Harrington et al., 2005b), whereas others have found negative results (Corcoran, Cahill & Frith, 1997; Langdon et al., 1997; Pickup & Frith, 2001; Randall et al., 2003). It must be noted, however, that these studies have only focused on persecutory delusions and not hallucinations. Moreover, except for Frith's studies which compared groups of patients with persecutory to groups with passivity delusions, the other studies only included patients with persecutory delusions, so that delusions of other types remain to be explored. Contradictory results in relation to paranoid symptoms may be explained by several conceptual and methodological factors. First, some studies may have found negative results as a result of not including actively enough deluded patients (e.g. Langdon et al., 2001). Second, it has been argued that because groups of patients with persecutory delusions tend to be less cognitively impaired as compared to groups of patients with negative or disorganized symptoms, they might use compensating general cognitive capacities when solving ToM tasks (Pickup & Frith, 2001). Alternatively, it has also been suggested that instead of ToM deficits, delusional patients tend to over-attribute mental states to others or "overmentalise", which would explain their intact performance on ToM tasks (Abu-Akel, 1999; Abu-Akel & Bailey, 2000; Walston et al., 2000).

Given methodological limitations regarding approaches to grouping symptoms and the different tasks used across studies, these findings remain tentative. Nevertheless, methodological limitations aside, it s clear that current evidence points to thought disorder and paranoid symptoms as being most consistently associated with the well-established ToM deficit in schizophrenia (ct. Harrington et al., 2005a). As for severity of ToM dysfunction, participants with symptoms of disorganization appear to be the most severely impaired (Sprong et al., 2007).

Table 2: Predictions about performance of subgroups of schizophrenic patients on ToM tasks (Frith, 1992):

Subgroup of patients	Predictions about performance	Reasons for ToM failure
Negative Behavioural Signs (abulia, poverty of speech, social withdrawal, flat affect)	Very poor and similar to autism	Failure in the development of ToM as a result of general neurocognitive deficits associated with negative schizophrenia.
Positive behavioural Signs (incoherent or inappropriate speech)	Very poor and similar to autism	Not a developmental ToM failure but rather a temporary malfunctioning associated with reasoning bias due to the psychotic state.
Paranoid Symptoms (persecutory delusions, delusions of reference, hearing other people's voices)	Relatively poor	Not associated with widespread or general cognitive deficit. Specific temporary breakdown of the mentalising ability.
Passivity Symptoms	Normal	
Remitted Patients (with no current signs or symptoms)	Normal	Evidence that the ToM deficit is state dependent in schizophrenia in patients with positive signs or positive paranoid symptoms.

2.1.5. State versus trait hypothesis

Another issue of crucial interest on the nature of ToM deficits in schizophrenia has been whether these are state or trait dependent. This is, whether they may be considered persisting characteristics of the disorder or alternatively, seen as intrinsically linked to psychotic symptoms, as hypothesised by Frith (1992). A first line of evidence on this are findings from studies comparing groups of patients with different symptom clusters and controls, and studies comparing remitted or stable patients with controls. If ToM deficits are linked to particular symptoms, then we should not expect to find them in asymptomatic patients in that particular symptom or in remitted patients. If ToM deficits are to be considered as traits, then they should to some extent be present in symptomatic, stable and remitted patients. From the literature linking ToM deficits to particular symptom clusters, and from the several studies that have also included a subgroup of stable patients, it is generally concluded that ToM deficits are only found in connection with the acute psychotic condition, failing to detect them during remission (Frith & Corcoran, 1996; Drury et al., 1998; Sarfati & Hardy-Bayle, 1999; Pickup & Frith, 2001), although some exceptions exist (Randall et al., 2003). 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. In addition, when these studies were recently meta-analysed, the remitted disease subgroup showed a significantly worse ToM performance than controls, with a medium mean effect size (d=− 0.692). Thus the authors concluded that mentalising impairment in schizophrenia may be trait dependent (Sprong et al., 2007).

However, this conclusion should remain tentative for several reasons. First, the number of existing studies comparing remitted patients and controls is limited. Second, the nature of the tasks in which remitted patients show impairment may imply higher interference with commonly impaired cognitive

areas in schizophrenia, such as executive functions and verbal memory. Thus, studies finding a ToM dysfunction in remitted patients assessed mentalising exclusively with verbal ToM tasks, and deficits were only observed on particularly complex tasks such as irony or the understanding of indirect speech, but not on less complex tasks such as 2nd order ToM tasks or the understanding of metaphors. Third, even though these studies controlled for general cognitive functioning, none of them used an equivalent control condition to their target ToM task, which may underscore weight to the validity of their results. Fourth, criteria for remission were unclear in these studies, leaving the possibility that patients were stable but still presented subtle or mild symptoms. This is clearly the case, for example, in the paranoid remitted patients of Randall et al. (2003), who presented paranoid-like attributional biases, and in spite of this they were included in the "remitted patients" subgroup of the meta-analysis. Reaching a consensus remission criteria for schizophrenia has been focus of recent work by experts in the field, proposing a definition based on low symptom severity of core symptoms (severity criterion), sustained over minimally 6 months (time criterion) (Andreasen et al., 2005; DeHert et al., 2007). The use of these criteria in future studies should help to overcome this limitation.

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 mentalising. 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).

In sum, the state versus trait hypothesis of the ToM deficit in schizophrenia is still a matter of debate. The limited existing evidence supports that the ToM dysfunction may be trait-like, but a number of methodological drawbacks indicate that the possibility of a state-like association should not be ruled out. The most methodologically sound means to explore this would be to carry out longitudinal studies comparing ToM abilities in different phases of the illness, defined by explicit criteria.

2.2. Insight in schizophrenia

2.2.1. Background

Since the fist descriptions of the illness in the 19th century, subjects suffering from psychotic disorders have been described as being unaware of their condition or showing lack of insight. It is a common clinical experience that schizophrenic patients do not admit that they are ill, do not perceive their specific symptoms as pathological and therefore they do not feel the need to search for medical help. Lacking or poor insight is frequently the factor that prevents treatment, destabilizes the patient and leads to a poorer prognosis. In line with this, assessment of the patient's degree of insight is a valuable clinical indicator, besides being of interest as a means of getting a better first –person understanding of the condition. Although symptoms or illness unawareness can be found in connection with different psychiatric and somatic illnesses, it is a central feature in the definition of psychosis. Comprehensive studies have shown that between 50% and 80% of individuals with a diagnosis of schizophrenia do not believe they have a disorder (Amador & Gorman, 1998; Husted, 1999), both in acute and stable phases (Mintz, Dobson & Romney, 2003).

Over the last 25 years there has been a surge of interest for the study of insight in schizophrenia, with an important body of research carried out into the conceptualization and assessment of insight, as well as on the its relationship with compliance, prognosis, psychopathological symptoms and neurocognitive impairment. This body of research has been subject of several recent literature reviews and meta-analysis, covering both general (Cooke et al., 2005; Dam, 2006; Lincoln, Lüllmann & Rief., 2007) and specific topics, such as the association between insight and symptoms (Mintz, Dobson & Romney, 2003), violence (Bjorkly, 2006), suicide (Pompili et al, 2004); quality of life (Karow & Pajonk, 2006) and neurocognitive function (Aleman et al., 2006; Shad et al., 2006). In addition, the 2nd edition

of the book “Insight and Psychosis” has comprehensively covered recent advances and controversial issues in the field (Amador & David, 2004).

In the present section, an overview of the concept, measurement, aetiological models and general findings regarding correlates of poor insight in schizophrenia will be presented.

2.2.2. Concept and measurement

The conceptualization of insight has evolved considerably, over the last decades, from simplistic and categorical views to more comprehensive and dimensional ones. Insight in psychosis was initially described as a binary, "all or nothing" phenomenon, which individuals either possessed or lacked. However, there is now a general agreement that insight is multi-dimensional and consists of several continua. In line with these changes, several detailed and valid measurement scales have been developed that encompass the dimensions that are held to be important by different conceptual frameworks.

Based on a review of the previous literature, David (1990) contended that there are three distinct but overlapping dimensions of insight: (i) recognition that one has a mental illness, (ii) compliance with treatment, and (iii) the ability to relabel unusual mental events as abnormal. Three scales have been developed to assess these dimensions; two structured interviews, the Schedule for the Assessment of Insight (SAI, David et al., 1992), and the SAI-Expanded (SAI-E, Kemp & David, 1996) and one self-report measure, the Insight Scale (Birchwood et al., 1994). Amador et al. (1991) define insight in terms of a five-dimensional construct, further divisible into current and retrospective components. Their definition is also derived from previous literature, and they have developed an interview, the Scale to assess Unawareness of Mental Disorder (SUMD, Amador et al., 1993) to assess these dimensions. The SUMD measures both current and retrospective awareness of having a mental disorder, the achieved effects of medication, the social consequences of mental disorder, the awareness of symptoms and the attribution of symptoms to a mental disorder. Self-report scales have been developed to assess still further aspects of insight, including "self-reflectiveness about unusual experiences", "capacity to correct erroneous judgments", "certainty about mistaken judgments" (Beck Cognitive Insight Scale, Beck et al., 2004) and awareness of more general changes occurring within individuals and their environments (Marková & Berrios Insight Scale, Markova et al., 2003).

Despite general agreement on the multi-dimensional nature of insight (if not its dimensions), isolated items from broad symptom rating scales are also frequently used as uni-dimensional measures of insight, including the G12 item from the Positive and Negative Syndrome Scale (PANSS, Kay, Fiszbein & Opler, 1987). A unidimensional measure of insight focussing on the need for treatment, the Insight and Treatment Attitudes Questionnaire (ITAQ, Mc Evoy et al., 1989) has also been used, particularly in earlier studies.

A review of the existing insight scales indicates that total scores in most of these scales show medium to high intercorrelations between them and between isolated insight items from broad symptom rating scales. This suggests that these measures target a common factor, and so allow some general conclusions to be drawn from the existing studies which have used different measures of insight (Lincoln, Lüllmann & Rief., 2007). Another point highlighted by this review is that self report scales do not generally differ in terms of internal consistency or reliability, but their association with observer-rated assessments is modest. Finally, it has also been outlined that the differentiation of dimensions has received consistent support from factor-analytical studies, so that the use of global or more detailed scales should be determined by the clinical or research purposes.

For the purposes of this thesis, we used the most widely accepted operational definition of insight in psychosis, namely that it consists of five components: (i) awareness of mental disorder, (ii) awareness of the social consequences of disorder, (iii) awareness of the need for treatment, (iv) awareness of symptoms of mental disorder and (v) attribution of symptoms to mental disorder. The assessment of insight regarding each particular symptom and the differentiation between the dimensions of symptom awareness and symptom attribution is especially relevant in the present research for its theoretical implications. First, the nature of insight deficits in different symptoms may differ. And second, the idea to further classify patients that are aware of their symptoms into those who correctly attribute symptoms to mental illness and those who incorrectly attribute them to other

causes is central to the study hypothesis, besides having received increasing support (Amador et al., 1994; Flashman & Roth., 2004; Mysore et al., 2007).

2.2.3. Models of poor insight in psychosis

Broadly, three different theoretical models have tried to explain the phenomenon of poor insight in psychosis. These have been comprehensively reviewed recently (Cooke et al., 2005).

The *Psychological Defence Model* posits that poor insight may reflect the tendency of individuals to use denial as a coping mechanism when facing a threatening situation such as the diagnosis of a chronic, lifelong and debilitating disorder (Moore et al., 1999). This model predicts that those who use denial as a coping strategy will have poorer insight, but will suffer less distress and maintain a greater degree of self-esteem. In contrast, those showing good insight will experience greater distress. Consistent with this model, there is considerable evidence from cross-sectional studies to suggest that possessing good insight in psychosis is associated with measures of distress, such as depression (see Mintz, Dobson & Romney, 2003; Lincoln, Lüllmann & Rief, 2007), hopelessness (Carroll et al., 2004, Eneman & Sabbe, 2006; Schwartz, Apter & Zalsman, 2006; Lysaker, Roe & Yanos, 2007), anxiety (Lysaker & Salyers, 2007) and suicidability (Schwartz et al., 2004, Crumlish et al., 2005; Pompili et al., 2007). Although some longitudinal studies suggest that increases in insight are associated with worsening measures of distress and depression (Carroll et al., 1999; Iqbal et al., 2000; Schartz, 2001; Drake et al., 2004; Lincoln, Lüllmann & Rief, 2007), the existing evidence is still weak so that the direction of causality needs to be explored in future well designed longitudinal studies. More direct testing of the hypothesis that poor insight is related to the use of denial as a coping strategy can be found in studies comparing insight measures with measures of coping on a variety of tests. Taken together, these give some evidence to suggest that there is an association between poor insight and coping styles aligned with denial. However, studies are difficult to compare due to their differences in the conceptualizations and measure of coping style (see Cooke et al., 2007).

The *Neuropsychological Deficit Model* views poor insight as arising from a cognitive impairment secondary to brain abnormalities (Lewis, 1934; David, 1999). This model developed out of an identified similarity between the symptoms of poor insight in schizophrenia and anosognosia, a neurological condition developing secondary to a specific lesion (such as focal traumatic brain injury) or diffuse brain damage (such as a stroke). Patients afflicted with anosognosia share striking similarities with psychiatric patients who have impaired insight (Amador and Paul-Odouard, 2000, Lele & Joglekar, 1998): both have a severe lack of awareness of their deficits, which persist despite all evidence to the contrary and both have a strong desire to prove their own assertions, and as such invent confabulations to explain away pathological symptoms.

While the deficit in cases of anosognosia is clearly related to either focal or diffuse brain damage (Mc Glynn & Schacter, 1989; Pia et al., 2004), that brain abnormalities are the sole causal explanation for poor insight in schizophrenia is not so well established. The neuropsychological deficit hypothesis has primarily been investigated by examining the correlations between measures of insight and performance on neuropsychological tests that index different domains of cognition or on global IQ tests. More recently, relationships with brain measures have been examined directly using neuroimaging.

Accumulated evidence on the relationship between generalized cognitive deficit (IQ) and insight is inconsistent, although a recent meta-analysis reveals a small albeit statistically significant positive relationship between IQ and insight (Aleman et al., 2006), which has been further supported by others (Cooke et al., 2007). Research on the relationship between insight and particular cognitive domains is less controversial, with recent reviews pointing as the most consistent finding a small positive association between poor insight and impaired executive function, as measured with the WCST (Drake & Lewis, 2003; Aleman et al., 2006; Shad et al., 2006). In particular, these studies indicate that poor insight relates to perseverative errors and reduced number of completed categories in the WCST, suggesting that it

might be mediated by deficiencies in conceptual organization and flexibility in abstract thinking. However, many questions regarding how deficits in executive functions lead to poor insight, as well as the implication of other cognitive functions need clarification. As for structural neuroimaging research, associations of insight with prefrontal cortex have been revealed in most studies (Laroi et al., 2000; Flashman et al., 2000; 2001, Shad et al., 2004, 2006; Sapara et al., 2007), with some exceptions (Rossell et al., 2003). Again, further research is needed to further specify the neurobiological basis of poor insight.

Another model of poor insight in psychosis that can be found in the current literature is the *Clinical Model*. This views poor insight as a general psychopathological manifestation, that is, a primary symptom of a disease process in its own right (Cuesta & Peralta, 1994) or related to how a particular symptom is formed (Marková & Berrios, 1995). Support for poor insight as a "primary symptom", in the terminology of Bleuler, comes from a number of studies showing an independence of insight from both positive and negative symptoms, and from results from a meta analytic review which show that even in those studies with moderate correlations between symptoms and insight, only 3-7% of variance in total insight scores seems to be accounted for by symptom measures (Mintz, Dobson & Romney, 2003). However, it has been argued that the lack of a strong relationship between psychotic symptoms and insight does not mean that insight is independent of the presence of other psychotic symptoms. One dimension of insight involves awareness of current symptoms, so one cannot possess this aspect of insight when symptoms are in remission (Marková & Berrios, 2001), and this aspect of insight must therefore be dependent on the presence of current symptoms rather than being independent of them. Such individuals can, however, be rated on insight into past symptoms. Furthermore, it remains possible that poor insight could be both weakly related to the presence of other psychotic symptoms and caused by factors other than the disease process itself. Indeed, both the neuropsychological model and the psychological denial model offer explanations for poor insight which are consistent with the lack of correlation between insight and symptoms. This is

incompatible with the view that poor insight is a primary Bleluerian symptom of psychosis, as these models view poor insight as secondary to a cognitive deficit, and coping style respectively (Ct Cooke et al., 2005).

After a thorough evaluation of evidence for and against each model of poor insight in psychosis, Cooke et al. (2005) conclude that integration of different aetiological models is necessary for a better understanding, and suggests that future research should assess multiple mechanisms in single investigations. The idea that different theoretical models are not necessarily mutually exclusive, and that different mechanisms may play part in explaining insight deficits in schizophrenia is shared by most experts in the field (see Amador & David, 2004) and has already received indirect support (Lysaker et al., 2003 a,b; Startup, 1996).

2.2.4. Correlates of poor insight in schizophrenia

A wide array of themes have been studied in relation to insight into mental illness, and there is now accumulated evidence on a number of clinical and functional correlates of poor insight in schizophrenia (Amador & David, 2004). First studies focused on the relationship between poor insight and symptoms, medication compliance and functional outcome. More recently, cognitive impairment and other topics such as assessment of needs, pre-morbid personality, interpersonal relationships and social context, suicidal thoughts and behaviour, recovery style, coping style, quality of life, duration of untreated psychosis, family attitudes, work performance, child care ability, competency and differential medication effects have also been approached, revealing some findings with important clinical implications (see David, 2004). Next, a brief overview of findings regarding variables which may be relevant to the present thesis will be presented.

Insight and psychopathology

Several studies have examined the relationship between insight and symptoms of schizophrenia, including global symptoms, positive and negative symptoms, as well as depressive symptoms (See reviews in Mintz, Dobson & Romney, 2003; David, 2004; Lincoln, Lüllmann & Rief., 2007). Mintz, Dobson and Romney (2003) conducted a systematic quantitative review of the existing body of published research investigating the relationship between insight and psychopathology and showed negative correlations of 0.25 between insight and positive symptoms and of 0.23 for negative symptoms, based on 22 and 20 studies respectively. In contrast, a positive correlation of 0.18 was found between depressive symptoms and insight, based on 15 studies. Apart from being small, these associations seemed to be moderated by other clinical variables such as illness status and age of onset. It has also been argued that the relationship between insight and both positive and negative symptoms are very ambiguous, and may be explained by different underlying mechanisms (Mohamed et al., 1999, Morgan & David, 2004).

As for depressive symptoms, both Mintz, Dobson and Romney‘s (2003) meta-analysis and other recent reports (Carroll et al., 2004; Sim et al., 2004) are consistent in showing positive associations with insight, with only some exceptions (Amador et al. 1994, Kim et al 1997). Moreover, insight has been consistently related with suicidal ideation or actions in cross sectional studies (see Lincoln, Lüllmann & Rief., 2007). However, as previously described, the strength and nature of these relationships are controversial, particularly regarding the causal path and mediation by a number of confounding factors. Since findings are correlational, it is both possible that depression leads to better insight (attributional explanation) and that better insight leads to depression (defence explanation). So far, longitudinal studies have offered controversial evidence in this respect, and unfortunately they have applied designs that do not allow inferring which of the postulated mechanisms is more appropriate. Although still preliminary, best designed studies seem to favour attributional views, which support the idea that depression leads to changes in attributional processes, resulting in a more accurate view of the self and thus a higher level of insight. In their review, Lincoln, Lüllmann and Rief (2007) conclude that the processes connecting insight to depression need further clarification before any conclusion can be drawn. They argue that it seems likely that insight and symptoms of depression are in a constant day-to–day interaction process that is difficult to assess by studies applying broad time frames and assessments. They also suggest that in order to estimate the risk associated with growing insight, it could be informative to distinguish between depression in the sense of an affective mental disorder and depression in the sense of a normal psychological reaction to a negative life event. In this respect, Cooke et al. (2007) investigated the relationships between insight, IQ, depression and self esteem in people with psychosis and showed that better self reported insight was associated with higher IQ and poorer self esteem, but not depression.

High consensus has been found in relation to symptom severity, with most studies finding that insight varies across the course of schizophrenic illness,

tending to be most impaired during the acute phase (Mintz, Dobson & Romney, 2003). However, it must be pointed out that no study has directly investigated the predictive value of insight in a suitable design, by analysing the relationship between insight at baseline and symptom severity at time two, while controlling for insight at time two. Also, studies on course of symptoms have largely failed to control for patient status (acute, in remission, remitted) as well as time of investigation (admission, discharge, follow-up), but this would be helpful as status and time of assessment seem to have an impact on the strength of the relationship (ct. Lincoln, Lüllmann & Rief., 2007).

Insight and adherence to medication

The majority of cross-sectional studies exploring the relationship between insight and adherence to treatment speak for a clear association of insight and treatment adherence. Longitudinal studies further reveal that the association between insight and adherence fades over time. This may be the result of successful clinical intervention, since patients with poor insight and adherence are more likely to be targeted for interventions such as long-acting injectable antipsychotic medication. It can also be explained by methodological reasons, such as lack of control of variance in the insight scores during hospital admission. However, it is also possible that insight is a necessary but not sufficient precondition for adherence, and factors other than insight probably gain more influence on the patient's adherence over time, such as medication side effects, weight gain, extrapyramidal symptoms or virility problems. In addition, attitude toward the disorder or the use of alternative dysfunctional coping strategies could be having a strong impact on adherence. In sum, the assumption that lack of insight leads to poorer adherence- despite being seemingly obvious- is not clearly supported (Lincoln, Lüllmann & Rief., 2007). Future studies should investigate shorter periods of time and control for relevant factors that might have an impact on adherence, such as attitude towards the disorder, use of alternative dysfunctional coping strategies, or treatment factors.

Insight and functional outcome

Existing cross sectional studies investigating the relationship between poor insight and global or more specific aspects of functioning -such as work or sociability- find controversial results, with some studies finding full or partial positive associations and some studies finding none. In contrast, all longitudinal studies find at least partially significant results, indicating that insight might not be related to present functioning, but that has an impact on improvements in functioning (Schartz et al., 1997). Research in this field also reveals that the long term relationship between insight and functional outcome is likely to be mediated by symptom severity (Lysaker et al., 1994).

2.3. Rationale for the study of the relationship between insight and ToM in schizophrenia

Traditionally, alterations in insight and ToM have been studied independently in the schizophrenia literature. However, conceptual, clinical and neurobiological perspectives suggest they may be linked. First, both concepts imply metacognition: thinking about one's morbid thoughts and experiences and acknowledging them as abnormal is clearly a metacognitive activity. ToM involves 'holding in mind' representations, in order to draw inferences about the nature of internal mental states, as well as to appreciate that other people's beliefs and intentions may differ from one's own (Frith & Frith, 1999; Gallagher & Frith, 2003). The idea of conceptual overlap between insight and ToM was implied in Frith's neurocognitive model of schizophrenia (Frith, 1992). He proposed that a failure in self-monitoring might underlie phenomena such as passivity experiences or formal thought disorder, while failure in monitoring others' thoughts may underlie paranoid delusions, suggesting that some dimensions of impaired ToM might be considered critical manifestations of impaired insight.

Second, from a clinical perspective alterations in both insight and ToM reasoning are prevalent in schizophrenia. Amador et al. (1994) found that nearly 60% of schizophrenic patients displayed moderate to severe unawareness of having a mental disorder in the DSM-IV field trial sample, which included over 400 subjects. Similarly, prevalence of ToM dysfunction has been estimated as reaching about 50% (Herold et al., 2002). In addition, these alterations of insight and ToM both have predictive value. In the insight literature unawareness has consistently predicted poorer treatment compliance, clinical outcome, social function, and response to vocational rehabilitation (Amador & David, 2004). More recently, it has been reported that alterations in ToM may directly account for the abnormalities in social behaviour seen in the illness (Brüne, 2005b; Ihnen et al., 1998; Penn et al.,

1999), which have in turn been shown to contribute negatively to prognosis (see Pinkham et al., 2003) and functional outcome (Brekke et al., 2005).

Third, there is some evidence suggesting a common neurobiological substrate underlying insight and ToM impairment in schizophrenia, based on specific frontal regions. Reviews on the neurocognitive correlates of poor insight in schizophrenia reveal as the most consistent finding an association with perseverative errors in the WCST (Drake & Lewis, 2003; Aleman et al., 2006; Shad et al., 2006). Similarly, dysfunctional ToM has been strongly related to poor performance on measures of executive functions (e.g. Langdon et al., 2001; Mitchley et al., 1998; Pickup & Frith, 2001). Furthermore, since the predictive power of neurocognitive tasks on poor insight has been shown to be limited, some authors have hypothesised that metacognitive abilities -such as reflecting on one's own cognitive abilities or on other's mental states- may contribute to unawareness of illness more than other cognitive skills (Lysaker et al., 2005; Aleman et al., 2006). In this line, Koren et al. (2004) found that first episode schizophrenic patients' self assessment of performance on the WCST was more closely related to insight than conventional WCST scores. Similarly, Bora et al. (2007) studied more directly the differential impact of ToM and executive functioning deficits on poor insight. They found that performance on second order false belief tasks was the best predictor of insight, and that WCST perseveration scores did not contribute to insight scores beyond that. In addition, the few structural neuroimaging studies that have explored neurobiological substrates of unawareness of illness in patients with psychosis have reported smaller brain size and frontal lobe abnormalities, including the cingulate gyrus and insula (see Flashman & Roth, 2004). Medial frontal areas have also been associated to ToM impairment in schizophrenia, with neuroimaging findings of under activation in left medial prefrontal cortex in these patients (Brunet et al., 2003; Russell et al., 2000; Shad et al., 2006).

Further evidence suggesting common neurobiological pathways comes from studies in different areas of cognitive neurosciences. Findings on cortical development have revealed close alignment in the emergence of self-

awareness, theory of mind, and related executive functions (Carlson & Moses, 2001; Gordon & Olson, 1998; Hughes, 2002; Perner, Lang & Kloo, 2002), although the order in which these functions develop is still unclear (Happé, 2003). It has also been suggested that in parallel with ToM impairment, autistic individuals show impaired ability to reflect on own's thoughts (Kazak, Collis & Lewis, 1997; Perner et al., 1989); and acquired frontal brain trauma may cause co-morbid deficiencies in self-awareness and social regulation (Bach & David, 2006; Mah, Arnold & Grafman, 2004). These and other findings from adult focal lesion research suggest that mental state attribution may be an extension of the capacity to conceive of oneself (Stuss & Anderson, 2004). This has been further supported by neuroimaging studies of areas of activation during attribution of mental states to others (see Frith & Frith, 2003; Gallagher & Frith, 2003; Siegal & Varley, 2002; Völlm et al., 2006) and self-reflection tasks (e.g. Gusnard & Raichle, 2001; Kelley et al., 2002; Lane et al., 1997; McGuire et al., 1996), which have consistently shown an overlapping activation area for both tasks involving the dorsal medial prefrontal cortex (Ochsner et al., 2004; Vogeley et al., 2001) (see also den Ouden et al., 2005; Happé, 2003; Prigatano & Johnson, 2003).

Although the findings reviewed above suggest some overlap between insight and ToM deficits in schizophrenia, to our knowledge, other existing preliminary reports have suggested lack of association. Thus, Langdon et al. (1997) explored whether different patterns of disturbed awareness of others in schizophrenia related to patterns of disturbed self awareness but results were inconclusive. Drake & Lewis (2003) looked at correlations between different insight measures (SUMD, SAI-E, ITAQ, BIS) and ToM in order to test whether poor insight could stem from an inability to decouple abstract representations from concrete meaning. They reported no significant linear or curvilinear relationships. Similarly, Boos (2003) in a study on the neurocognitive basis of poor insight found no relationship between several insight measures (SAI-E, IS, PSE insight item) and performance at a comprehensive battery of ToM tasks.

3. OBJECTIVE AND HYPOTHESIS

3.1. Objective

Objective I:
To explore whether schizophrenia patients on a stable phase show ToM impairment as compared to a control group with no mental illness.

Objective II:
To explore the relationship between distinct insight dimensions and ToM in a group of stable schizophrenia patients, taking clinical, neuropsychological and psychosocial functioning characteristics as possible modifiers of the relationship.

3.2. Hypothesis

Hypothesis I:
Performance on ToM tasks by stable schizophrenia patients will not differ from controls.

Hypothesis II: Symptom unawareness and misattribution of symptoms in stable schizophrenia patients will be associated to ToM impairment.

4. METHOD AND RESULTS (Published works)

This thesis is composed by two research articles published in indexed journals with impact factor, focused on each of the stated objectives and hypothesis, respectively. A letter to the editor discussing part of the conclusions reached by a recent meta-analysis on ToM in schizophrenia has also been added, since it has direct implications with our results. In addition, two publications with theoretical and methodological work on the issue have been included in Annex I. The first consists of the Spanish Validation of the SUMD, the second is a chapter of a book with part of the review work overtaken as the theoretical background of the thesis.

A detailed description of the sample characteristics, clinical, neuropsychological, psychosocial functioning, insight and ToM instruments, as well as statistical analysis can be found in the correspondent papers.

Work 1:
E Pousa, R Duñó, G Brébion, AS David, AI Ruiz, JE Obiols. (2008) Theory of mind deficits in chronic schizophrenia: evidence for state dependence. Psychiatry Research, 158: 1-10.

Work 2:
E Pousa, R Duñó, B Navarro, AI Ruiz, JE Obiols, AS David. (In press) Exploratory study of the association between insight and Theory of Mind (ToM) in stable schizophrenia patients. Cognitive Neuropsychiatry.

Work 3:
E Pousa, AI Ruiz, AS David. (2008). Mentalising impairment as a trait marker of schizophrenia?. Correspondence. British Journal of Psychiatry, 192, 312-315.

Additional related works (Annex I):

Work 4:

AI Ruiz, E Pousa, R Duñó, JM Crosas, S Cuppa, C Garcia-Ribera. (2008). Adaptación al español de la Escala de Valoración de la No Conciencia de Trastorno Mental SUMD. Actas Españolas de Psiquiatria, 36, 111-118.

Work 5:

JE Obiols, E Pousa. La Teoría de la Mente como módulo cerebral evolutivo. En J Sanjuan y CJ Cela Conde (Eds) 2005, cap 6, pp105-119. La Profecia de Darwin. Ars Médica. ISBN: 84-9751-090-9

Work 1

E Pousa, R Duñó, G Brébion, AS David, AI Ruiz, JE Obiols. (2008) Theory of mind deficits in chronic schizophrenia: evidence for state dependence. Psychiatry Research, 158: 1-10.

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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).

An important factor to take into account when interpreting this literature is the nature of tasks used for ToM assessment. A detailed review of these shows that, except for the studies of Langdon et al. (1997, 2001, 2002a), all studies supporting the trait-dependent view have used verbal tasks (Mitchley et al., 1998; Herold et al., 2002; Janssen et al., 2003). In addition, deficits were only observed on particularly complex tasks using irony or the understanding of indirect speech, but not on first and second order false belief recognition or metaphor mentalizing tasks. By contrast, studies approaching mentalizing abilities using non-verbal comic strips, picture sequencing or visual methodologies have not found differences between non-specific heterogeneous patient groups or subjects at risk and controls (Brüne, 2003; Kelemen et al., 2004), or did so only in relation to patients with particular symptom clusters (Sarfati et al., 1997a, 1999; Sarfati and Hardy-Baylé, 1999; Harrington et al., 2005b), or in relation to particular mental states (Sarfati et al., 1997b), thus supporting the state-dependent view.

In the light of these findings, the present study aimed to clarify the state- versus trait-dependent view of ToM deficits in schizophrenia using both verbal and non-verbal ToM tasks, introducing a more carefully controlled methodology. This was achieved by using strict criteria for matching controls, and by controlling for important confounds such as clinical status (Andreasen et al., 2005), IQ, illness duration (Sarfati et al., 1999; Harrington et al., 2005b) and type of antipsychotic medication (Kee et al., 1998). Based on previous findings discussed above, we hypothesised that remitted patients would not differ from controls on either modality of ToM tasks. In addition, the study also aimed to further examine the relation between mentalizing and individual or clusters of symptoms. In this respect, ToM deficits were expected to be associated with paranoid symptoms and disorganization symptoms, as well as with negative symptoms.

2. Methods

2.1. Participants

Sixty-one patients meeting DSM-IV criteria for schizophrenia (American Psychiatric Association, 1994) were recruited using a consecutive sampling technique between 2001 and 2004. All subjects were clinically stable at 5 months of discharge from the Day Hospital of the Psychiatry Department, Parc Taulí Hospital. This department manages a population of 400,000 inhabitants of the semi-urban area of Sabadell (Barcelona) and is part of a University General Hospital with 500 beds. All patients were taking antipsychotic agents, the majority atypicals, corresponding to mean equivalent doses of haloperidol of 8 mg/day (Schatzberg et al., 1997). Exclusion criteria included current or past CNS disease or history of head injury, physical disability (visual or auditory) that could limit the application of the tests or other chronic or acute condition that could interfere with cognitive performance. Additional exclusion criteria were the diagnosis of schizophreniform and schizoaffective disorder or other Axis I/II DSM-IV psychiatric conditions. Patients with an estimated pre-morbid IQ<70 as measured by the WAIS-III vocabulary subtest were also excluded.

In addition, 51 control subjects were included to compare their performance on ToM tasks with the schizophrenia group. Control subjects were recruited from the orthopaedics and surgery units of the same hospital at the same time as the schizophrenia group. They were matched by gender, age, educational level and current IQ, as obtained through the abbreviated form of the WAIS-III (Blyler et al., 2000). Exclusion criteria for this group were also current or past CNS disease, history of head injury or relevant physical disability, as well as the history of psychiatric disorders, the presence of psychopathology at the time of the evaluation according to the SCL-90 scale (Martinez-Azumendi et al., 2001) and medical prescription of psychoactive drugs. Demographic, background and clinical characteristics of schizophrenic patients and controls are shown in Table 1.

2.2. Clinical assessment

Clinical assessments were completed by the staff psychiatrist of the Day Hospital (RD). The experimental ToM tasks and the WAIS-III short form were administered by a trained clinical psychologist (EP).

The *Positive and Negative Syndrome Scale (PANSS)* (Kay et al., 1987) was used to assess symptoms. For the purposes of the present study, individual symptoms of interest were recoded as present or absent in order to be further analysed in relation to ToM performance. A score of ≥3 for a particular symptom was recoded as present and scores <3 as absent (range 1–7).

Global Assessment of Functioning (GAF) (DSM-IV) (American Psychiatric Association, 1994) was used to assess patient's global psychological, social and occupational functioning.

2.3. Theory of mind tasks

2.3.1. Verbal second order ToM tasks

Two second order ToM stories implying a false belief were used: "The Burglar" (Happé and Frith, 1994) and "The Ice-Cream Van" (Baron-Cohen, 1989). These

Table 1
Demographic, background and clinical characteristics of schizophrenia patients and controls

	Schizophrenia group		Control group		Significance test
N	61		51		
Gender, *N* (%)					
Male	48	(78.7)	38	(74.5)	$\chi^2=0.27$
Female	13	(21.3)	13	(25.5)	P=n.s.
Educational level (%)					
≤8 years	47	(77.0)	41	(80.4)	$\chi^2=2.86$
>8 years	14	(23.0)	10	(19.6)	P=n.s.
Age, mean (S.D.)	32.5	(7.8)	34.6	(8.3)	$T=1.39$ P=n.s.
IQ, mean (S.D.)	87.5	(20.6)	93.4	(23.5)	$T=1.41$ P=n.s.
Schizophrenia diagnosis, *N* (%)					
Paranoid	44	(72.13)			
Disorganized	7	(11.47)			
Residual	5	(8.20)			
Undifferentiated	5	(8.20)			
Family psychiatric history, *N* (%)					
Yes	39	(69.60)			
No	17	(30.40)			
Antipsychotic medication, *N* (%)					
Conventional	10	(16.40)			
Atypical	39	(63.93)			
Mixed	12	(19.67)			
Age at disease onset, mean (S.D.)	21.83	(5.49)			
Duration of illness (years), mean (S.D.)	10.69	(7.98)			
Global Assessment of Functioning (GAF), mean (S.D.)	62.69	(12.20)			
PANSS, mean (S.D.)					
PANSS-Positive (7–49)	11.80	(3.86)			
PANSS-Negative (7–49)	19.28	(11.01)			
PANSS-General (16–112)	32.57	(9.96)			
PANSS-Total (30–210)	63.87	(21.23)			

examined the ability to correctly deduce what one character thinks with regard to the thinking of another character of the story. Stories were read aloud by the examiner, and subjects were asked to listen and subsequently answer two questions. The first question had to be answered on the basis of the mental state of one of the characters (ToM question) and concerned that character's false belief within the situation. The second question (control question) reflected the subject's comprehension of the story. Scoring of these stories was 1 for a correct answer in both ToM and control questions and 0 for a wrong or incomplete answer to the ToM question while responding correctly to the control question. Please note that only exceptional participants were found to pass the ToM question while failing the control one, so exclusion of these subjects was decided to simplify the analysis. A *total ToM second order* score was obtained by adding up scores obtained at each ToM second order story (range 0–2).

2.3.2. Non-verbal ToM task

The picture-sequencing task (PST) is a non-verbal ToM task developed by Langdon and Coltheart (1999). The PST includes four different conditions or story types. Three of the conditions serve as control conditions and one is aimed to assess mentalizing. *Social script stories* (SC) represent people fulfilling routine social habits and evaluate the ability to reason logically based on the knowledge of social scripts. *Mechanical stories* (M) depict sequences of physical cause-and-effect events and evaluate the ability to infer causal relations. *False belief stories* (Fb) represent the story of a subject who does not know an event has occurred in the story and acts according to the false information about it. These stories imply ToM reasoning. Finally, *capture stories* (C) are designed specifically to include a salient "decoy" cue intended to mislead participants and require the ability to inhibit salient but inappropriate information in order to determine a logical sequence. There are four examples of each story type, each of them comprising four picture cards. The test consists of a total of 16 stories plus two additional practice sequences. Each of the four cards depicts a different black and white cartoon scene from the story and is placed face-down in front of participants. At a prompt, they are asked to turn the cards over and place them in a logical sequence. The scoring for each sequence ranges from 0 to 6 and a mean score is obtained for each story type. Time to complete the task is also recorded, obtaining a mean response time for each story type.

For the purposes of data analysis in the present study, a *PST ToM deficit* variable was calculated by averaging scores on control conditions and subtracting the score of the ToM condition from this mean, as follows: *PST ToM deficit*=(Sc+M+C/3)−Fb. The resulting score indicates the size of the difference between ToM and control conditions, and ranges from −6 to +6, with positive scores indicating worse performance on ToM as compared to control conditions and negative scores indicating better performance on ToM than on control conditions. The rationale for creating this variable was to obtain a single task measure indicative of "pure" ToM deficit, which also allowed for the possibility that scores on the ToM condition might be better than on the control conditions.

2.4. Statistical analysis

Data analysis was carried out by means of the SPSS for Windows, Version 12.0. Distribution of data was checked, and non-parametric tests were used for group comparisons and correlations in cases of non-normal distribution. An α level of 0.05 was used for all statistical tests.

3. Results

3.1. Group comparisons on ToM tasks

Table 2 shows descriptive statistics regarding patients' and controls' scores and response times when performing the four conditions of the picture-sequencing task (PST), scores obtained on the ToM deficit variable and total scores obtained on the verbal second order ToM tasks.

3.1.1. Verbal second order ToM tasks

No significant group differences were revealed by the non-parametric Mann–Whitney *U* test ($U=1363.00$, n.s.), indicating that patients were not significantly impaired relative to healthy controls in these tasks.

3.1.2. Picture-sequencing task

For the analysis of differences between patients and controls in the four conditions of the PST, we followed Langdon et al. (2001, 2002a) in order to facilitate comparison of results. This was, for both accuracy and response times, a (2×4) mixed design, with group (patients vs. controls) as a between-subject factor and story type (social–script, mechanical, false belief, capture) as a within-subject factor.

3.1.2.1. Performance scores. No main effect for group ($F(1,110)<1$, n.s.) was observed, indicating that patients were not significantly impaired relative to healthy controls in the PST. A highly significant main effect for story type was revealed ($F(3,330)=178.35$, $P<0.0001$), without any significant two-way group by story type interaction ($F(3,330)<1$, n.s.). This reveals that both groups had a similar pattern of performance. Post-hoc paired samples *t*-tests were conducted to investigate further the effect of story type in the whole group. It revealed significant differences in all contrasts. Higher performance was observed for the social script compared to mechanical stories ($t(111)=5.4$, $P<0.0001$), for mechanical stories compared to false beliefs ($t(111)=7.4$, $P<0.0001$) and for false beliefs compared to capture stories ($t(111)=8.4$, $P<0.0001$).

3.1.2.2. Mean response times. No main effect for group was observed ($F(1,110)=2.47$, n.s.), indicating that patients and controls did not differ in their response times at sequencing the stories. There was a significant main effect of story type ($F(3,330)=44.13$, $P<0.0001$), indicating that response times varied according to the different PST conditions. Post-hoc tests showed that longer times were observed on capture stories compared to false belief stories ($t(111)=-3.0$, $P<0.0001$), on false belief stories compared to mechanical ($t(111)=-5.8$, $P<0.0001$) and on mechanical compared to social-script ($t(111)=-6.8$, $P<0.0001$). No significant two-way group by story type

Table 2
Performance on ToM tasks by patients and controls

	Schizophrenia patients ($n=61$)				Healthy controls ($n=51$)			
	Mean	(S.D.)	Min	Max	Mean	(S.D.)	Min	Max
PST (scores)								
Social script	5.60	(0.79)	1.50	6.00	5.69	(0.49)	4.00	6.00
Mechanical	5.00	(1.23)	2.00	6.00	5.19	(0.92)	2.25	6.00
False belief	4.06	(1.55)	0.00	6.00	4.20	(1.14)	1.00	6.00
Capture	2.89	(1.24)	0.75	6.00	3.05	(1.08)	1.00	5.25
PST (time, sec.)								
Social script	19.65	(8.50)	7.25	45.25	18.05	(7.08)	6.50	42.25
Mechanical	24.83	(11.93)	8.25	83.25	23.26	(10.12)	12.50	68.50
False belief	30.22	(13.86)	11.00	87.00	27.31	(12.63)	12.25	83.25
Capture	37.20	(23.57)	9.75	159.00	29.84	(17.45)	15.00	128.50
PST								
ToM deficit variable	0.43	(1.27)	−2.08	4.17	0.50	(1.20)	−1.33	3.17
Total ToM 2nd order	1.23	(0.76)	0	2	1.41	(0.67)	0	2

PST: picture-sequencing task.

interaction was observed ($F(3,330)=2.09$, n.s.), which shows that both groups presented a similar pattern of response time.

3.1.2.3. PST ToM deficit variable. No main group effect ($F(1,110)<1$, n.s.) was observed, indicating that patients and controls did not significantly differ in their scores on the PST ToM deficit variable.

In order to unequivocally test the hypothesis that ToM deficits are state dependent, strict control for clinical status was required. Suspecting that lack of significant differences between patients and controls might stem from diversity in clinical severity in our sample – so that asymptomatic patients may mask the expected deficit for the whole patient sample – we proceeded to subgroup patients according to Andreasen et al.'s (2005) remission criteria (remission=severity of mild or less on three core symptom dimensions for at least 6 months). Based on those criteria, 33 (54%) patients met criteria for remission and 28 (46%) were classified as non-remitted. These groups did not differ significantly in age, sex, educational level, IQ or illness duration. Using the same methodology, group comparisons were carried out between controls, remitted and non-remitted patients.

Non-parametric Kruskal–Wallis tests revealed significant group differences ($\chi^2=7.84$, $P<0.05$) regarding performance on the verbal ToM second order tasks. Subsequent pair-wise comparisons showed that non-remitted patients were significantly impaired relative to controls (mean ranks 32.05 versus 44.36, $U=491.500$, $P<0.05$) and to remitted patients (mean ranks=25.38 versus 35.77, $U=304.500$, $P<0.05$). No significant difference emerged between remitted patients and controls (mean ranks=43.41 versus 41.91, $U=811.500$, n.s.).

As for the non-verbal task, using ANOVA, again, no significant group effects were found for performance scores on the PST including the four conditions ($F(2,109)=1.85$, n.s.) or on the PST ToM deficit variable ($F(2,108)<1$, n.s.). However, a significant group effect emerged in response time ($F(2,109)=3.29$, $P<0.05$) with non-remitted patients taking significantly longer to perform the PST than controls (31.08 versus 24.62, $F(1,77)=5.81$, $P<0.05$). The difference between non-remitted and remitted patients approached significance (31.08 versus 25.34, $F(1,59)=3.50$, $P<0.07$). No difference between remitted patients and controls was observed (25.26 versus 24.67, $F(1,82)<1$, n.s.).

3.2. Relationship with clinical symptoms

Associations between ToM performance and clinical symptoms were investigated. Considering that one of the ToM variables did not lend itself to correlational studies, as it was a three-level scale, our approach was to compare subgroups of patients with high or low levels of symptomatogy. Illness duration, IQ and type of antipsychotic medication were controlled for in the analysis.

3.2.1. Relationships with positive symptoms

The PANSS positive score was split along the median (11.5). The 30 patients with scores ≥11.5 were significantly impaired on the ToM second order task relative to the other 31 patients (1.02 versus 1.45, $F(1,56)=5.46$, $P<0.015$). When either hallucinations or thought disorganization was added as a covariate, the effect of subgroup was still significant ($F(1,55)=5.02$, $P<0.05$ and $F(1,55)=4.05$, $P<0.05$, respectively). However, when delusions were added as a covariate, the difference between subgroup was no longer significant ($F(1,55)=3.01$, n.s.). This suggests that the association with the positive symptom score was mainly accounted for by delusions. The same analyses were carried out with the PST ToM deficit variable and no significant differences were found between the two subgroups of patients with high versus low rating of positive symptoms (ToM score means=0.32 versus 0.54, $F(1,56)=0.28$, n.s.).

To further explore the association of ToM second order with positive symptoms, we studied the association with each particular symptom. Only symptoms that were present in at least 30% of the sample were explored. *T*-tests showed that all subgroups to be compared were not significantly different in age, educational level, IQ, illness duration and antipsychotic medication. As expected, the 46 patients with delusions were significantly impaired on the ToM second order task relative to the other 15 patients (1.13 versus 1.53, $F(1,56)=4.30$, $P<0.05$). However, no difference on the PST ToM deficit variable was revealed (0.47 versus 0.32, $F(1,56)<1$, n.s.). Analysis of the 23 patients with thought disorder and the 22 with inappropriate affect versus those without these symptoms did not reveal any significant subgroup difference on the ToM second order task or on the PST ToM deficit. The 13 patients with hallucinations were equivalent to the other 48 patients on both ToM measures (1.26 versus 1.13, $F(1,56)<1$, n.s.; and 0.66 versus 0.38, $F(1,56)<1$, n.s., respectively).

3.2.2. Relationships with negative symptoms

The PANSS negative score was split along the median (15). No significant differences in ToM second order tasks were found between the 30 patients with scores ≥15 and the 31 with scores <15 (1.13 versus 1.33, $F(1,56)=1.18$, n.s.). It should be noted that when IQ and illness duration were removed from the covariates, the group difference was significant ($F(1,58)=4.41$, $P<0.05$), which suggests

that these factors are confounders. The same analyses were carried out with the PST ToM deficit variable and no significant differences were found between the two.

4. Discussion

The present study focused on a sample of mainly paranoid chronic schizophrenia patients who were living in the community. Although they were all clinically stabilized, some 46% did not meet criteria for remission. The group of patients and a carefully matched control group were compared on performance on verbal and non-verbal ToM tasks. As expected, no statistically significant differences were revealed using either modality. These results are indicative that, *as a whole*, stable patients do not show ToM impairment on verbal and pictorial tasks. Subsequent subgrouping of patients by symptom severity into remitted and non-remitted showed that the non-remitted group performed significantly worse on the verbal ToM second order tasks than remitted patients and controls. These findings add weight to the state-dependent view of ToM deficits in schizophrenia, as they show that, unless symptomatic, patients do not differ from controls in ToM performance.

To our knowledge, excluding studies with a small subgroup of remitted patients and a study which compared subgroups of patients in different phases (Drury et al., 1998), only two previous studies have looked specifically at whether remitted patients and non-psychiatric controls differ in their ToM abilities (Herold et al., 2002; Janssen et al., 2003). These both found ToM to be defective in patients as compared to controls, so that our study is the first to find no such difference. However, of the previous studies, only Janssen et al. (2003) controlled for IQ. In addition, these studies explored ToM only by means of verbal tasks and reported patients to be impaired exclusively on complex tasks such as irony (Herold et al., 2002) or the understanding of indirect speech (Janssen et al., 2003), but not on the same second order ToM tasks as the ones used in the present study (Herold et al., 2002). Given that schizophrenia patients are known to present difficulties in processing and manipulation of verbal material (Kuperberg and Heckers, 2000), this could explain such differences with complex tasks rather than a specific ToM deficit. A further confound is that stability and remission tend to be ambiguously defined in previous studies (e.g. "total PANSS < 60", "no need for hospitalization" or "no prominent symptoms"), so that patients with active residual symptoms may have been included. In fact, our results clearly showed that, when stable patients were divided into remitted and non-remitted, the latter performed significantly worse than the former and than controls on the verbal ToM tasks. Explicit definitions of remission and relapse in schizophrenia will aid greatly in the interpretation of these studies (Andreasen et al., 2005). Apart from factors obscuring clear conclusions regarding the state- versus trait-dependent question, results from these studies and from ours seem to indicate that ToM deficits in schizophrenia may be genuinely linked to language. The relationship between defective pragmatic linguistic skills and ToM in schizophrenia has been widely discussed (e.g. Langdon et al., 2002a,b). Furthermore, it has recently been reported that verbal reasoning tasks typically failed by schizophrenic subjects, such as proverb comprehension, are more closely related to ToM than IQ (Brüne and Bodenstein, 2005).

Regarding the non-verbal ToM task, the present study showed that patients and controls did not differ in scores or in response time in any of the conditions of the PST, nor in the PST ToM deficit variable. Moreover, when remitted and non-remitted patients and controls were compared, no differences were found between groups except for slowed reaction time in the non-remitted group. Even though previous well-controlled studies have also failed to find differences between non-acute patients and controls with similar picture-sequencing procedures (Sarfati and Hardy-Baylé, 1999; Sarfati et al., 1999; Brüne, 2003), our results contradict the findings of previous studies using the same PST task (Langdon et al., 1997, 2001, 2002a). Examination of the samples and methods used in these studies revealed clear differences on three factors. First, educational level was clearly lower in both our patients and matched controls, while most of our patients had less than 8 years of education; this was an exclusion criterion in Langdon et al.'s studies. Second, previous studies used subjects with average IQs, whereas patients in the present study showed IQs which tended to be below average. Finally, although most patients in Langdon's studies were also chronic out-patients, clinical severity may have been lower in our patient sample. Since we showed no differences on PST performance between controls and the subgroup of non-remitted patients, we believe that differences from previous studies may be mostly accountable by educational level and IQs of the samples. Our data showed that lower educational level and lower IQs in both patients and matched controls contributed to a worse performance on all conditions of the PST, so that global sequencing difficulties may have obfuscated findings of more selective ToM difficulties in patients, suggesting that the PST may not be suitable for low IQ groups.

Given that clinical severity was shown to be related to mentalizing, at least as assessed by one of the ToM tasks, we proceeded to further explore the relationship between

ToM and symptoms. The subgroup of patients with higher scores on the PANSS positive symptom scale showed a worse performance on ToM second order tasks, which could be mainly accounted for by the presence of delusions. Subsequent single symptoms analysis further supported this, revealing the expected association between ToM and delusions. Unfortunately, we did not record type of delusions so the hypothesis that ToM would be related to persecutory delusions in particular could not be tested. Nevertheless, our results add weight to recent findings that ToM relates to delusions in general (Greig et al., 2004). Also, in line with previous studies was that the association between paranoid symptoms and defective ToM was only observed with verbal ToM tasks (Corcoran et al., 1995; Frith and Corcoran, 1996; Corcoran et al., 1997; Drury et al., 1998; Marjoram et al., 2004), but not with the PST (Harrington et al., 2005b). This is also in agreement with psychological models of delusions (Bentall et al., 2001; Craig et al., 2004), and with recent findings that delusions are linked to deficits in inductive reasoning (Corcoran and Frith, 2003; Simpson and Done, 2004).

In relation to disorganization symptoms, the lack of significant association with ToM was surprising (see Corcoran et al., 1995; Frith and Corcoran, 1996; Corcoran et al., 1997; Sarfati et al., 1997a,b, 1999; Sarfati and Hardy-Baylé, 1999). This may be explained by the scarcity of disorganised symptoms in our sample. As for negative symptoms, even though higher sensitivity was expected, no associations were revealed to any of the ToM tasks. However, when IQ and illness duration were removed from the covariates, the relationship reached significance for second order ToM tasks, suggesting that the association between ToM and negative symptoms may be accounted for by chronicity and general cognitive deficits (see also Drury et al., 1998; Mitchley et al., 1998; Langdon et al., 2001). Our results would further suggest that lack of control for illness duration may have led to spurious findings between ToM and negative symptoms in previous studies. In summary, results regarding the relationship between ToM and symptoms support the view that ToM deficits shown by patients with predominant negative symptoms are non-specific and that mentalizing deficits seen in delusional patients may more genuinely reflect a problem in forming representations of others' intentions.

In general, the different pattern of results obtained using verbal and non-verbal tasks in this and previous studies suggests that ToM deficits may be task specific. However, reasons other than task modality may underlie these findings, since mentalizing is intrinsically linguistic regardless of the nature of the stimuli used for its testing (e.g. Langdon et al., 2002a,b; Brüne and Bodenstein, 2005). Furthermore, there is evidence that when the same ToM task is administered in verbal and pictorial modalities, subjects do not differ in performance (Sarfati et al., 1999), which has been further supported by imaging studies revealing activation of the same key regions of the 'mentalising circuit' regardless of the modality of the concerned stimuli (Gallagher et al., 2000). Future studies comparing reliability and difficulty of different ToM tasks would help to clarify this issue. One limitation of this research is the scant evidence regarding validity and reliability of the ToM instruments used. Although tests with non-mental state control conditions were chosen, lack of psychometrically sound instruments continues to be a major problem in ToM research.

In conclusion, we propose that there are genuinely "representational" deficits in ToM reasoning that are state dependent in schizophrenia. These deficits are associated with delusions and possibly to other symptoms affecting verbal reasoning such as disorganization. When observed in patients with negative symptoms, ToM deficits may be less specific and accounted for by general cognitive impairment and illness chronicity, thus appearing more persistent.

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Work 2

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Exploratory study of the association between insight and Theory of Mind (ToM) in stable schizophrenia patients

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Background. Poor insight and impairment in Theory of Mind (ToM) reasoning are common in schizophrenia, predicting poorer clinical and functional outcomes. The present study aimed to explore the relationship between these phenomena.

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Methods. 61 individuals with a DSM-IV diagnosis of schizophrenia during a stable phase were included. ToM was assessed using a picture sequencing task developed by Langdon and Coltheart (1999), and insight with the Scale to Assess Unawareness of Mental Disorder (SUMD; Amador et al., 1993). Multivariate linear regression analysis was carried out to estimate the predictive value of insight on ToM, taking into account several possible confounders and interaction variables.

Results. No direct significant associations were found between any of the insight dimensions and ToM using bivariate analysis. However, a significant linear regression model which explained 48% of the variance in ToM was revealed in the multivariate analysis. This included the 5 insight dimensions and 3 interaction variables. Misattribution of symptoms—in aware patients with age at onset >20 years—and unawareness of need for medication—in patients with GAF >60—were significantly predictive of better ToM.

Conclusion. Insight and ToM are two complex and distinct phenomena in schizophrenia. Relationships between them are mediated by psychosocial, clinical, and neurocognitive variables. Intact ToM may be a prerequisite for aware patients to attribute their symptoms to causes other than mental illness, which could in turn be associated with denial of need for medication.

BACKGROUND

Since the first descriptions of the illness in the nineteenth century, persons suffering from psychotic disorders have been characterised by being unaware of their condition or showing deficits in "insight". Research over recent years has led to the conceptualisation of poor insight in psychosis as a continuous and multidimensional phenomenon, involving unawareness of illness, symptoms, or deficits; unawareness of need for treatment; and impaired understanding of the impact and consequences of the disorder (Amador, Strauss, Yale, Gorman & Endicott, 1993; David, 1990). In addition, it has been proposed that patients that are aware of their symptoms can be further classified into those who correctly attribute symptoms to mental illness and those who incorrectly attribute them to other causes (Amador et al., 1994; Flashman & Roth, 2004). Initial theories proposed that poor insight functions as an unconscious defence or coping mechanism that serves to preserve self-esteem and to minimise disability (e.g., Birchwood, Mason, MacMillan, & Healy, 1993; Lysaker & Bell, 1998; White, Bebbington, Pearson, Johnson, & Ellis, 2000). More recently, insight deficits have been thought to stem from a disturbed neurocognitive substrate (Aleman, Agrawal, Morgan, & David, 2006), paralleling observations on unawareness of deficits in some neurological disorders such as anosognosia seen in hemiplegic patients (see Amador & David, 2004). Both mechanisms may play a part in explaining insight deficits in schizophrenia (Lysaker, Byrson, Lancaster, Evans, & Bell, 2003; Lysaker, Lancaster, Davis, & Clements, 2003; Startup, 1996).

Additionally, there is growing evidence that schizophrenia patients show impaired Theory of Mind (ToM), which 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 & Woodruff, 1978). A number of studies over the last 20 years have shown that schizophrenics not only perform poorly on traditional ToM tests, but also on other verbal and pictorial experimental tasks requiring more complex mentalising abilities, such as the understanding of social hints and metaphors, irony, humour, and *faux pas*. Some studies have also shown that these deficits are specific rather than secondary to chronicity or general cognitive impairment (see review in Brüne, 2005b; Harrington, Siegert, & McClure, 2005). Evidence from high risk studies on first degree relatives (Janssen, Krabbendam, Jolles, & van Os, 2003; Wykes, Hamid, & Wagstaff, 2001) and subjects with schizotypal traits (Langdon & Coltheart, 1999, 2004; Pickup, 2006) has also shown that ToM impairment may be considered a trait in schizophrenia spectrum disorders. However, others have reported that deficits are exclusively linked to symptoms (e.g., Drury, Robinson, & Birchwood, 1998; Marjoram et al., 2005; Pickup & Frith, 2001; Pousa et al., in press), supporting a state-dependent view.

Traditionally, alterations in insight and ToM have been studied independently in the schizophrenia literature. However, conceptual, clinical, and neurobiological perspectives suggest they may be linked. First, both concepts imply metacognition: thinking about one's morbid thoughts and experiences and acknowledging them as abnormal is clearly a metacognitive activity. ToM involves "holding in mind" representations, in order to draw inferences about the nature of internal mental states, as well as to appreciate that other people's beliefs and intentions may differ from one's own (Frith & Frith, 1999; Gallagher & Frith, 2003). The idea of conceptual overlap between insight and ToM was implied in Frith's neurocognitive model of schizophrenia (Frith, 1992). He proposed that a failure in self-monitoring might underlie phenomena such as passivity experiences or formal thought disorder, while failure in monitoring others' thoughts may underlie paranoid delusions, suggesting that some dimensions of impaired ToM might be considered critical manifestations impaired insight.

Second, from a clinical perspective alterations in both insight and ToM reasoning are prevalent in schizophrenia. Amador et al. (1994) found that nearly 60% of schizophrenic patients displayed moderate to severe unawareness of having a mental disorder in the DSM-IV field trial sample, which included over 400 subjects. Similarly, prevalence of ToM dysfunction has been estimated as reaching about 50% (Herold, Tényi, Lenard, & Trixler, 2002). In addition, these alterations of insight and ToM both have predictive value. In the insight literature unawareness has consistently predicted poorer treatment compliance, clinical outcome, social function, and response to vocational

rehabilitation (Amador & David, 2004). More recently, it has been reported that alterations in ToM may directly account for the abnormalities in social behaviour seen in the illness (Brüne, 2005a; Ihnen, Penn, Corrigan, & Martin, 1998; Penn et al., 1999), which have in turn been shown to contribute negatively to prognosis (see Pinkham, Penn, Perkins, & Lieberman, 2003) and functional outcome (Brekke, Kay, Lee, & Green, 2005).

Third, there is some evidence suggesting a common neurobiological substrate underlying insight and ToM impairment in schizophrenia, based on specific frontal regions. Reviews on the neurocognitive correlates of poor insight in schizophrenia reveal as the most consistent finding an association with perseverative errors in the WCST (Drake & Lewis, 2003). Similarly, dysfunctional ToM has been strongly related to poor performance on measures of executive functions (e.g., Langdon, Coltheart, Ward, & Catts, 2001; Mitchley, Barber, Gray, Brooks, & Livingstone, 1998; Pickup & Frith, 2001). Also of interest is the finding that first-episode schizophrenic patients' self assessment of performance on the WCST is more closely related to insight than conventional scores from the WCST (Koren et al., 2004). In addition, the few structural neuroimaging studies that have explored neurobiological substrates of unawareness of illness in patients with psychosis have reported smaller brain size and frontal lobe abnormalities, including the cingulate gyrus and insula (see Flashman & Roth, 2004). Medial frontal areas have also been associated to ToM impairment in schizophrenia, with neuroimaging findings of under activation in left medial prefrontal cortex in these patients (Brunet, Sarfati, Hardy Baylé, & Decety, 2003; Russell et al., 2000; Shad, Tamminga, Cullum, Haas, & Keshavan, 2006).

Further evidence suggesting common neurobiological pathways comes from studies in different areas of cognitive neurosciences. Findings on cortical development have revealed close alignment in the emergence of self-awareness, theory of mind, and related executive functions (Carlson & Moses, 2001; Gordon & Olson, 1998; Hughes, 2002; Perner, Lang, & Kloo, 2002), although the order in which these functions develop is still unclear (Happé, 2003). It has also been suggested that in parallel with ToM impairment, autistic individuals show impaired ability to reflect on their own thoughts (Kazak, Collis, & Lewis, 1997; Perner, Frith, Leslie, & Leekam, 1989); and acquired frontal brain trauma may cause co-morbid deficiencies in self-awareness and social regulation (Bach & David, 2006; Mah, Arnold, & Grafman, 2004). These and other findings from adult focal lesion research suggest that mental state attribution may be an extension of the capacity to conceive of oneself (Stuss & Anderson, 2004). This has been further supported by neuroimaging studies of areas of activation during attribution of mental states to others (see Frith & Frith, 2003; Gallagher & Frith, 2003; Siegal & Varley, 2002; Völlm et al., 2006) and self-reflection tasks (e.g., Gusnard & Raichle, 2001; Kelley et al., 2002; Lane, Fink, Chau, & Dolan, 1997; McGuire, Paulesu, Frackowiak, & Frith,

1996), which have consistently shown an overlapping activation area for both tasks involving the dorsal medial prefrontal cortex (Ochsner et al., 2004; Vogeley et al., 2001) (see also den Ouden, Frith, Frith, & Blakemore, 2005; Happé, 2003; Prigatano & Johnson, 2003).

Although the findings reviewed here suggest some overlap between insight and ToM deficits in schizophrenia, to our knowledge, no previous study has specifically focused on this issue, and the few existing preliminary reports have suggested lack of association. Thus, Langdon et al. (1997) explored whether different patterns of disturbed awareness of others in schizophrenia related to patterns of disturbed self awareness but results were inconclusive. Drake and Lewis (2003) looked at correlations between different insight measures (SUMD, SAI-E, ITAQ, BIS) and ToM in order to test whether poor insight could stem from an inability to decouple abstract representations from concrete meaning. They reported no significant linear or curvilinear relationships. Similarly, Boos (MPhil thesis) in a study on the neurocognitive basis of poor insight found no relationship between several insight measures (SAI-E, IS, PSE insight item) and performance at a comprehensive battery of ToM tasks.

The aim of this study was to explore the relationship between insight and ToM in schizophrenia using uni- and multivariate regression analyses taking clinical, neuropsychological, and psychosocial functioning characteristics as possible modifiers of the relationship. Five distinct insight dimensions as well as distinct patterns of ToM performance were considered. Although our approach was mainly exploratory, on the basis of previous evidence that (a) aspects of insight such as general awareness of having a mental disorder, of the need for medication and of the social consequences are highly influenced by individual, psychosocial, and cultural factors (e.g., Lysaker, Bell, Byrson, & Kaplan, 1999; Saravanan, Jacob, Prince, Bhugra, & David, 2004; White et al., 2000), and that (b) in contrast, the dimensions of symptom awareness and symptom misattribution may most specifically reflect self-knowledge and metacognition (David, 1990; Marková & Berrios, 1995), a positive association was predicted between ToM and the latter dimensions. In particular, since ToM implies attributional abilities (Craig, Hatton, Craig, & Bentall, 2004; Frith, 1992; Randall, Corcoran, Day, & Bentall, 2003), associations were expected regarding the ability to relabel psychotic experiences as pathological.

METHOD

Design

This was a cross-sectional observational study of the relationship between insight and ToM in adult subjects with a diagnosis of schizophrenia.

Participants

Sixty-one patients meeting DSM-IV criteria for schizophrenia (American Psychiatric Association [APA], 1994) were recruited using a consecutive sampling technique. All subjects were clinically stable at 5 months of discharge from the Day Hospital of the Psychiatry Department, Parc Taulí Hospital. This department manages a population of 400,000 inhabitants of the semiurban area of Sabadell (Barcelona) and is part of a University General Hospital with 500 beds. All patients were taking antipsychotic agents, the majority atypicals, corresponding to mean equivalent doses of haloperidol of 8 mg/day (Schatzberg, Cole, & DeBattista, 1997). Exclusion criteria included current or past CNS disease or history of head injury, physical disability (visual or auditory) that could limit the application of the tests, or other chronic or acute medical conditions that could interfere with cognitive performance. Additional exclusion criteria were the diagnosis of schizophreniform and schizoaffective disorder or other Axis I/II DSM-IV psychiatric conditions. Patients with an estimated premorbid IQ <70 as measured by the WAIS-III vocabulary subtest were also excluded.

All participants gave their written consent before taking part in the study which was approved by the Ethics and Research Committee of Parc Tauli Hospital. Demographic, clinical, and neurocognitive functioning characteristics of the sample are shown in Tables 1 and 2.

Assessment

Clinical and insight assessments were completed by the staff psychiatrist of the Day Hospital (RD. Neuropsychological and ToM assessment was carried out by a trained clinical psychologist (EP.

Clinical assessment included: The Premorbid Adjustment Scale (PAS; Cannon-Spoor, Potkin, & Wyatt, 1982), the Positive and Negative Symptom Scale (PANSS; Kay, Fiszbein, & Opler, 1987), and the Calgary Depression Scale (Addington, Addington, & Schissel, 1990). The Global Assessment of Functioning (GAF; APA, 1994) was also used to assess patient's global psychological, social, and occupational functioning. Neurocognitive assessment included: Current intellectual functioning, as obtained through the abbreviated form of WAIS-III (Blyler, Gold, Iannone, & Buchanan, 2000); the California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 1987) for the assessment of verbal memory, and the Trail Making Test (TMT; Reitan & Wolfson, 1985), Stroop Test (Golden, 1994), and FAS Verbal fluency test (Benton & Hamsher, 1976) as measures of executive functioning.

TABLE 1
Demographic and clinical characteristics of the sample and their associations with PST ToM deficit ($n = 61$)

	N	*%*	*PST ToM deficit*
Gender			
Male	48	78.7	median = 0.0, IQR = 1.17
Female	13	21.3	median = 1.0, IQR = 2.58
Educational level			MWU ($p = .009$**)
Low	47	77	median = 0.16, IQR = 1.54
Medium/high	14	23	median = 0.16, IQR = 1.77
Schizophrenia diagnosis			MWU ($p = .8$)
Paranoid	44	72.1	median = 0.16, IQR = 1.54
Other (disorganised, residual, undifferentiated)	17	27.9	median = 0.5, IQR = 1.45
Family psychiatric history			MWU ($p = .6$)
Yes	39	69.6	median = 0.16, IQR = 1.50
No	17	30.4	median = 0.16, IQR = 2.50
Antipsychotic medication			MWU ($p = .9$)
Conventional	10	16.4	median = 0.29, IQR = 1.68
Atypical	39	63.9	median = 0.41, IQR = 1.66
Mixed	12	19.7	median = −0.16, IQR = 0.83
			K Wallis ($p = .5$)

	Mean	*SD*	*Median*	*Range*	*PST ToM deficit*
Age	32.5	7.9	32.1	19–50	rho = 0.28 $p = .031$*
Age at disease onset	21.8	5.5	20.2	13–39	rho = 0.10 $p = ns$
Duration of illness (years)	10.7	8.0	8.0	2–31	rho = 0.26 $p = .045$*
Premorbid Adjustment Scale (PAS) (0–1)	0.4	0.14	0.4	0.12–0.81	rho = −0.11 $p = ns$

*$p < .05$, **$p < .001$. MWU: Mann-Whitney U Test.

Theory of Mind

The Picture-Sequencing Task (PST). This is a nonverbal ToM task developed by Langdon and Coltheart (1999). The PST includes four different conditions or story types. Three of the conditions serve as controls and one is aimed to assess mentalising. *Social script stories* (Sc) represent people fulfilling routine social habits and evaluate the ability to reason logically based on the knowledge of social scripts. *Mechanical stories* (M) depict sequences of physical cause-and-effect events and evaluate the ability to infer causal relations. *False belief stories* (Fb) represent the story of a

subject who does not know an event has occurred in the story and acts according to the false information about it. These stories imply pure ToM reasoning. Finally, *Capture stories* (C) are designed specifically to include a salient "decoy" cue intended to mislead participants, and require the ability to inhibit salient but inappropriate information in order to determine a logical sequence. There are four examples of each story type, each of them comprising four picture cards. The test consists of a total of 16 stories plus two additional practice sequences. Each of the four cards depicts a different black and white cartoon scene from the story and are placed face-down in front of participants. At a prompt, they are asked to turn the cards over and place them in a logical sequence. The scoring for each sequence ranges from 0 to 6, with 2 points given for correct order in first and fourth positions and 1 point given for second and third positions. A mean score is obtained for each story type. Time to complete the task is also recorded, obtaining a mean response time for each story type.

A PST ToM deficit. This variable was calculated by averaging scores on control conditions and subtracting the score of the ToM condition from this mean, as follows: $PST\ ToM\ Deficit = (Sc + M + C/3) - Fb$. The resulting score indicates the size of the difference between ToM and control conditions, and ranges from –6 to +6, with positive scores indicating worse performance on ToM as compared to control and negative scores indicating better performance on ToM than on control conditions. The rationale for creating this novel variable was to obtain a single task measure indicative of a "pure" ToM deficit, which also allowed for the possibility that scores on the ToM condition might be better than on the control conditions.

Insight

Insight was measured with the full version of the *Scale to Assess Unawareness of Mental Disorder* (SUMD; Amador et al., 1993). The SUMD is a semistructured interview that assesses several dimensions of insight into illness. It is composed of three general items that evaluate (current and retrospective) (a) global awareness of mental disorder, (b) awareness of the effect of medication, and (c) awareness of the social consequences of having a mental illness, and two subscales that assess (current and retrospective) awareness and attribution of 17 specific signs and symptoms. It should be noted that according to the SUMD assessment guidelines, symptom ratings are only made for those symptoms present in a particular patient (i.e., symptoms rated as moderate or higher on the SAPS, SANS, and BPRS), and that attribution is not evaluated unless there is symptom awareness. All items scores range from 1 to 5, with higher scores indicating poorer awareness and attribution. Following previous studies, the

TABLE 2
Clinical and neuropsychological status of participants* ($n = 61$)

	Mean	*SD*	*Median*	*Range*
Global Assessment of Functioning (GAF)	62.7	12.2	60.0	40–90
PANSS				
PANSS-Positive (7–49)	11.8	3.9	11.0	7–23
PANSS-Negative (7–49)	19.5	11.0	15.0	7–46
PANSS-General (16–112)	32.6	9.9	33.0	16–56
PANSS-Total (30–210)	63.9	21.2	59.5	30–105
Calgary Depression Scale (0–27)	2.2	3.1	0.0	0–10
Short form WAIS-III (IQ)	87.5	20.6	85.3	51.05–140.25
Verbal Memory (CVLT)				
Learning	−1.58	1.15	−2.0	−4–1
Short term	−1.40	1.29	−1.0	−4–1
Long term	−1.97	1.43	−2.0	−5–1
Verbal Fluency (FAS) (total *n* of words)	29	10	26	13–53
Trail Making Test (B–A/A)	1.6	0.77	1.41	0.30–3.29
Stroop Test (interference)	−0.13	9.02	−0.42	−16.51–26.12

*None of these factors was associated significantly with PST ToM performance (all rho $< .16$).

first three subscales were treated as categorical, with scores 1 and 2 recoded as "aware" and scores 3, 4, and 5 as "unaware". Since scores on the Unawareness of symptoms and Misattribution subscales are mean scores of the ratings obtained at each symptom, these were treated as continuous variables. For the purposes of this study, only current awareness was assessed.

Statistical analysis

Data analysis was carried out using SPSS (Version 13.0). Distribution of data was checked and non parametric tests were used for group comparisons and correlations in cases of non-normal distribution. Data analysis included a descriptive study of all measures and bivariate analysis of the direct relationship between insight and ToM using Mann-Whitney U tests and Spearman correlations. Subsequently, multivariate linear regression analysis was carried out in order to estimate the explicative value of Insight for the outcome variable of ToM. This second analysis was done taking into account a total of 21 possible interaction/confounder variables, which included the following demographic, psychosocial, clinical, and neurocognitive characteristics: gender, academic premorbid adjustment (PAS), social premorbid adjustment (PAS), Global Assessment of Functioning (GAF), PANSS positive, PANSS negative, PANSS disorganisation item, PANSS General, PANSS Total, depression (CDS total score), duration of illness

(years), age at disease onset, Short form WAIS-III–Total IQ, WAIS-III Digit span subtest, WAIS-III Similarities subtest, Verbal Memory Learning (CVLT), Verbal Memory Short Term (CVLT), Verbal Memory Long Term (CVLT), Stroop test, Trail making Test (B-A), and Verbal Fluency (FAS).

Since the exploratory perspective of the study increases the number of potential interaction/confounders terms, simultaneous analysis of all of them in a sample of 61 patients would generate specification problems. Thus, following the proposal of Kleinbaum, Kupper, Muller, and Nizam (1998), we started the process of selecting the best model by analysing each interaction term separately. For each potential interaction variable, interaction components with the five insight measures were calculated, and a "chunk test" was applied to obtain the global statistical significance of the five interaction terms. If the obtained result reached statistical significance, each of the five interaction components was studied independently. If not, the five interaction components were eliminated from the model. From these initial analyses a group of three statistically significant interaction components were obtained, which included SUMD2 × GAF, SUMD5 × Age at onset, and SUMD4 × Illness duration. The variables not included in the interactions selected were evaluated as potential confounders, comparing the magnitude of the regression coefficient for the five insight measures between models with and without the confounder. None of these was superior to 10%, so following Maldonado and Greenland (1993), no confounders were added to the model. Finally, a regression model was estimated, taking as independent variables: (a) the five insight measures, (b) the three statistically significant interaction components, and (c) three more necessary variables to accomplish the hierarchy principle regarding the interaction components. The diagnostics of the final model were done by examining the distribution of residuals and the absence of influential observations.

RESULTS

Descriptives

Demographic, clinical, and neuropsychological functioning characteristics, as well as associations with ToM deficit, are shown are shown in Tables 1 and 2. In essence, females showed higher PST ToM deficits than males and higher age and illness duration were significantly associated to PST ToM deficits, using correlation analysis.

Patients' performance scores and response times when performing the four conditions of the Picture Sequencing Task, and scores obtained on the PST ToM deficit variable, are shown in Table 3. Patients' scores varied

AQ4

TABLE 3
Results obtained by patients on the Picture Sequencing Task (PST) ($n = 61$)

	Mean	*SD*	*Median*	*Range*
PST (Scores)				
Social Script (Sc)	5.6	0.8	6.00	1.50–6.0
Mechanical (M)	5.0	1.2	5.50	2.00–6.0
False Belief (Fb)	4.1	1.6	4.50	0.00–6.0
Capture (C)	2.9	1.2	2.75	0.75–6.0
PST (Time)				
Social Script	19.6	8.5	18.00	7.25–45.25
Mechanical	24.8	11.9	21.50	8.25–83.25
False Belief	30.2	13.9	27.25	11.00–87.00
Capture	37.2	23.6	32.00	9.75–159.00
PST ToM deficit* (−6–6)	0.43	1.27	0.17	−2.08–4.17

*PST ToM deficit = (Sc + M + C/3) − Fb.

considerably in the four PST conditions as well as in the PST ToM deficit variable, indicating absence of floor or ceiling effects.

Descriptive results obtained on the SUMD are shown in Table 4. None of the SUMD variables followed a normal distribution (Kolmogorov-Smirnov Test: all $< .01$). In general, the results show that patients had mild insight deficits in all SUMD subscales, and yet, moderate to high scores in current symptom misattribution were found among patients showing symptom awareness.

Bivariate analysis

None of the five insight subscales showed a significant association with ToM deficit (see Table 4). However, as these aspects of insight are interrelated it

TABLE 4
Patients' scores on the Scale of Unawareness of Mental Disease (SUMD) and Spearman correlations between insight dimensions and PST ToM deficit

	Mean	*SD*	*Median*	*Range*	*PST ToM deficit*
Unawareness mental disorder ($N = 61$)	2.21	1.42	1.00	1 to 5	rho = −0.00 $p = .99$
Unawareness medication ($N = 61$)	1.75	1.12	1.00	1 to 5	Rho = −0.15 $p = .24$
Unawareness social consequences ($N = 61$)	1.98	1.36	1.00	1 to 5	Rho = −0.27 $p = .31$
Unawareness of symptoms ($N = 59$)	1.77	1.07	1.21	0.88 to 4.50	rho = −0.15 $p = .25$
Misattribution of symptoms* ($N = 42$)	3.58	1.18	3.64	1 to 5	rho = −0.05 $p = .7$

*Only assessed in participants showing symptoms awareness.

was important to tease apart the impact of the different insight factors using multivariate linear regression, which could also take other potential confounding and interaction variables into account. Further, we carried out correlations between the various PST conditions and our neurocognitive measures in order to explore whether any relationship between the derived ToM deficit variable and insight could be explained by intervening neurocognitive scores.. Although some interesting results were found regarding PST conditions and executive functions, none of the SUMD dimensions showed associations with neuropsychological measures (see Table 5). Three of the four variables correlated with IQ with the strongest correlation found between the capture stories (requiring inhibition) and, as might be expected, the Stroop interference test.

Regression analysis

A statistically significant model which best explained the effect of insight on ToM was obtained, $R^2 = .460$, $F = 3.401$, $p = .002$ (see Table 6). This indicated that 46% of the variance of the PST ToM deficit could be predicted by the five insight dimensions, level of global functioning (GAF), age at onset, illness duration, and three interactions. In particular, misattribution of symptoms and unawareness of medication were found to have significant predictive value on the PST ToM deficit. However, the model also showed that this effect was mediated by interaction variables, so

TABLE 5
Nonparametric correlational analysis (Spearman's rho) between neuropsychological variables, Picture Sequencing Task (PST) conditions, and SUMD subscales ($n = 61$)

	Short form WAIS-III (IQ)	*CVLT Verbal Learning*	*CVLT Short Term*	*CVLT Long Term*	*Stroop Test interference*	*Trail Making Test (B–A)/A*	*FAS*
PST (Sc)	0.32*	0.10	0.05	−0.01	0.24	−0.32*	0.35**
PST (M)	0.29*	0.30*	0.06	0.19	0.25*	−0.22	0.24
PST (Fb)	0.26*	0.10	−0.07	0.06	0.03	−0.19	0.13
PST (C)	0.14	0.18	0.12	0.16	0.38**	−0.16	0.21
SUMD1	0.09	0.12	0.15	0.17	−0.06	−0.02	0.09
SUMD2	0.09	0.09	0.11	0.15	0.00	−0.15	0.10
SUMD3	0.07	0.05	−0.06	0.07	−0.02	−0.05	0.06
SUMD4[1]	−0.03	−0.09	−0.10	−0.01	−0.03	0.06	−0.08
SUMD5[2]	0.01	−0.15	−0.12	−0.03	−0.01	0.08	−0.10

*$p < .05$, **$p < .001$.
[1]$n = 59$, [2]$n = 42$.

TABLE 6
Regression model of insight as predictive of ToM

	B	*T*	*P*	*95% CI*
Constant	−2.465	−0.998	0.3	−7.441 to 2.512
SUMD1 (illness) (yes, no)	−0.284	−0.901	0.4	−0.918 to 0.351
SUMD2 (medication) (yes, no)	−3.732	−2.400	0.021*	−6.865 to −0.599
SUMD3 (social cons) (yes, no)	−0.182	−0.456	0.7	−0.986 to 0.623
SUMD4 (symptoms unawareness) (1–5)	0.172	0.758	0.5	−0.285 to 0.628
SUMD5 (symptoms misattribution) (1–5)	0.985	2.054	0.046*	−0.019 to 1.952
GAF	−0.046	−2.314	0.025*	−0.085 to −0.006
Age at onset	0.286	3.332	0.002*	0.113 to 0.458
Illness duration	0.149	3.481	0.001*	0.063 to 0.235
SUMD2 × GAF	0.057	2.337	0.024*	0.008 to 0.106
SUMD5 × Age at onset	−0.055	−2.637	0.012*	−0.097 to −0.013
SUMD4 × Illness duration	−0.069	−2.953	0.005*	−0.116 to −0.022

*$p < .05$.

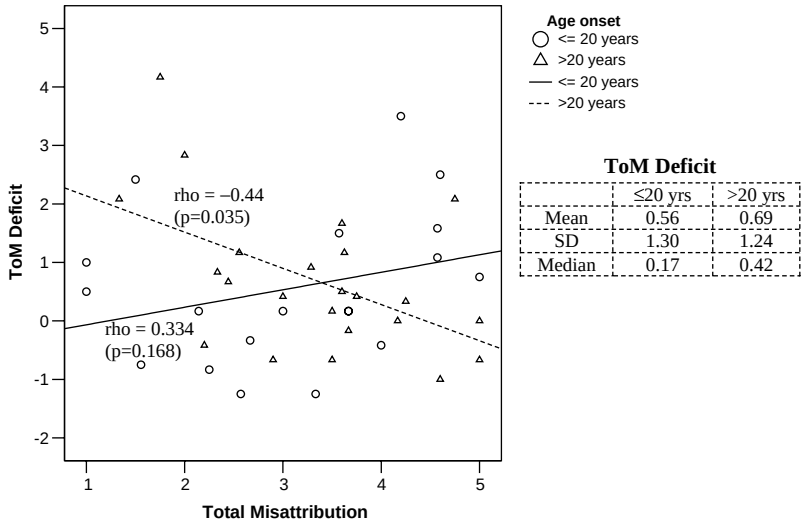
that misattribution of symptoms interacted with age at onset, and unawareness of medication with GAF. An interaction between unawareness of symptoms and illness duration was also observed.

Post hoc analyses

Given that two particular insight dimensions and three interaction terms showed significant predictive value in the model, post hoc analyses were carried out to further explore these.

Misattribution of symptoms and PST ToM deficit. Overall, for the 42 patients who showed awareness of symptoms, no significant correlations were found between PST ToM deficit and misattribution, rho = −0.05, $p = .7$. Subsequently, binary categorisation for age at disease onset was conducted using a median split. In the patients with age at onset >20 years, a negative significant correlation was revealed between misattribution and PST ToM deficit, $N = 23$, rho = −0.44, $p = .035$, indicating that higher misattribution of symptoms was related to lower PST ToM deficit (i.e., better ToM) in this group. This relationship was not found in those with an age at onset =20 years, $N = 19$, rho = 0.33, $p = .168$ (see Figure 1).

Awareness of medication and PST ToM deficit. No significant differences were found in ToM performance between medication-aware and unaware patients in the group as a whole, means: 0.51 vs. 0.25, MWU = 332.0, $p = .5$. Similarly, subsequent binary categorisation of GAF was conducted using a



ToM Deficit

	≤20 yrs	>20 yrs
Mean	0.56	0.69
SD	1.30	1.24
Median	0.17	0.42

Figure 1. Scatterplot of PST ToM deficit and misattribution.

median split. In spite of a different pattern of mean scores between medication-aware and unaware patients in the two GAF groups, no statistically significant differences were revealed: GAF $= 60$: $N = 32$, means 0.17 vs. 0.46, MWU $=$ 107.5, $p = .6$; GAF > 60: $N = 29$, means 0.78 vs. -0.25, MWU $= 33.0$, $p = .1$. These results were somewhat surprising, since the interaction was significant in the linear regression model. Thus, we explored the correlations between the original (noncategorised) awareness of medication variable and PST ToM deficit in the two GAF groups. For patients with GAF > 60, a significant negative correlation was revealed between unawareness of need for medication and PST ToM deficit, $N = 29$, rho $= -0.42$, $p < .025$. However, for patients with GAF $= 60$, this relationship was clearly nonsignificant, $N = 32$, rho $=$ 0.03, $p = .8$. Thus, unawareness of need for medication was related to lower ToM deficits (i.e., better ToM) in patients with average to normal functioning, but not in those showing low levels of functioning.

Unawareness of symptoms and PST ToM deficit. Overall, no significant correlations were found between PST ToM deficit and unawareness of symptoms, rho $= -0.15$, $p = .25$. Subsequently, binary categorisation for illness duration was conducted using a median split. In the patients with illness duration ≥ 10 years a significant negative correlation was revealed between unawareness and PST ToM deficit, $N = 28$, rho $= -0.49$, $p = .008$, while in those with illness duration < 10 years no significant association was found, $N = 31$, rho $= 0.07$, $p = .7$. Since these results were unexpected, further descriptive analysis was carried out. This showed that from the 28

patients included in the subgroup with illness duration $= 10$ years only 5 had unawareness of symptoms (score in SUMD4 > 2), so these results were considered irrelevant for clinical interpretation. Interestingly, however, it was noted that these five subjects all presented delusions or thought disorder (PANSS scores ≥ 3).

DISCUSSION

This exploratory study set out to test the relationship between insight and ToM in stabilised schizophrenia patients. Bivariate analysis of associations between the five insight dimensions and ToM showed no significant relationships, indicating that insight and ToM are two independent phenomena in schizophrenia. However, when the relationship was studied in more depth taking into account possible colinearity between distinct insight dimensions as well as the effect of a number of candidate interacting variables and confounds such as psychosocial, neurocognitive, and clinical characteristics, a significant regression equation was revealed. ToM performance was highly predicted by the five insight dimensions and some neurodevelopmentally related and clinical interaction variables. In particular, two dimensions—symptom misattribution and unawareness of need for medication—as well as age at onset, global functioning, and illness duration, reached significance in the prediction equation. In addition, significant interactions between these variables were revealed, so that the relationship between three dimensions of insight and ToM showed a different pattern for patients with young age at onset as compared to older age at onset, for patients with low GAF versus average to high GAF, and for patients with shorter as compared to longer illness duration, respectively. In sum, it was found that in symptom-aware patients without a later onset of the disorder, misattribution was associated with good mentalising skills. Similarly, good ToM skills were related to unawareness of need for medication in patients without severe psychosocial impairment.

Participants in the present study were stabilised schizophrenia patients living in the community who displayed mild to moderate symptom severity and predominantly negative symptoms. Global functioning and neurocognitive profile—including ToM performance—varied considerably. As a whole, mild unawareness was found regarding global illness, need for medication and social consequences. Similarly, most patients showed awareness of the symptoms they presented, and yet moderate to high misattribution of these to causes other than mental illness. This pattern of results is consistent with descriptions of insight in samples of similar characteristics using the SUMD (Amador et al., 1994; Laroi et al., 2000;

Mohamed, Fleming, Penn, & Spaulding, 1999; Schwartz, 1998; Smith, Hull, Israel & Willson, 2000).

Lack of direct significant associations between ToM and each of the insight dimensions was in line with previous preliminary findings in the literature using global (Boos, MPhil thesis; Langdon et al., 1997) or dimensional (Drake & Lewis, 2003) insight measures, indicating that clinical manifestation of these phenomena are distinct, although this should be interpreted with caution given substantial differences regarding sample characteristics and ToM measures between studies.

Taking into account that besides multidimensionality, insight is highly complex, with evidence showing close yet differential interaction with neurocognitive, psychological, and cultural factors (e.g., Lysaker et al., 1999; Saravanan et al., 2004; White et al., 2000), and that ToM performance may be influenced by neurocognitive and clinical status, it was considered that multivariate analysis would be most appropriate. In fact, preliminary evidence of some association between insight and ToM has been revealed in studies which included ToM and insight variables in their multivariate models. Langdon, Corner, McLaren, Ward, and Coltheart (2006) found a theory of mind difficulty among multiple pathways that contributed to poor insight in patients with persecutory delusions. Similarly, in a study with chronic stable patients, Lysaker et al. (2005) concluded that performance on metacognition (as measured by three subscales including Understanding of one's mind, Understanding of other's minds, and Mastery) could be predicted by neurocognition (particularly verbal memory), symptoms, insight, and quality of life.

When separate dimensions of unawareness were considered, the prediction that misattribution would be associated with ToM was supported, though in the opposite direction than expected. In particular, in patients who showed symptom awareness, misattribution of those symptoms to causes other than mental illness was related to better mentalising. This might indicate that in order to misattribute one needs to have mentalising abilities to some extent, and is in line with the suggestion that mentalising is a prerequisite for delusional thought (Abu-Akel & Bailey, 2000; Walston, Blennerhassett, & Charlton, 2000). These results might also be revealing in that patients with symptom awareness who at the same time are aware of the mental states of others (ToM), may feel resistant to labelling their own experiences as mental illness, since that would be harmful to their self image, and their assumed image in the eyes of others, thus leading to denial (e.g., Freeman & Garety, 2003). Altogether, it could be that both neurocognitive and psychological mechanisms play a part in explaining the relation between misattribution and good mentalising skills in symptom-aware patients. In previous studies poor insight has been independently associated with cognitive deficits and

avoidant/denial coping styles (Startup, 1996), as well as with ToM difficulties and externalising attributional bias (Langdon et al., 2006).

The finding that this association was only revealed in patients with age at onset >20 years—while in the group of early onset there was a tendency for the opposite (misattribution associated to worse ToM)—would suggest that early onset patients may have a more persistent deficit in metarepresentation, of both their own and others' mental states, resulting from the adverse developmental impact of the illness at an early age.

It is possible to reconcile the association between misattribution and good mentalising skills with that fact that in a high percentage of patients, preserved ToM goes hand in hand with symptom awareness. This might be so if we take into account that a condition for being assessed on symptom attribution is to show awareness of symptoms, and most patients in our sample were symptom aware. However, the correlation between the symptom-awareness measure of the SUMD and ToM was not significant perhaps because only a small number of subjects were symptom unaware ($n=17$), so that there was low statistical power to find such association. In addition, findings might have been obscured due to the fact that the score on symptom unawareness is the mean of scores obtained for different symptoms, which may individually relate differently to mentalising. For example delusions tend to be associated by definition with symptom unawareness, while they have also been related to intact mentalising skills (e.g., Abu-Akel & Bailey, 2000; Walston et al., 2000) and attributional biases (e.g., Langdon et al., 2006; McKay, Langdon, & Coltheart, 2005; Randall et al., 2003). In contrast, negative symptoms may be more commonly acknowledged by patients but have been consistently associated to poor ToM (e.g., Doody, Gotz, Johnstone, Frith, & Owens, 1998; Frith & Corcoran, 1996; Langdon et al., 1997; Mazza, de Risio, Surian, Roncone, & Casacchia, 2001). Interestingly, the five long-term unaware yet good mentalisers in our sample presented delusions or thought disorder. Future studies comparing ToM of aware and unaware patients on different individual symptoms would help to clarify this.

Significant associations regarding awareness of need for medication were unexpected. Findings showed that unawareness was predictive of better ToM, and that this was only the case for patients with relatively adequate global functioning (GAF >60). If as discussed earlier ToM is a prerequisite for misattributing symptoms, a possible explanation for this would be that patients with normal ToM functioning deny need for medication, in spite of being aware of symptoms, because they misattribute these to causes other than mental illness (if my symptoms are not mental, why take medication?). In contrast, patients with low global functioning—who may in turn be those most neurocognitively impaired—would tend to fail both normal reasoning and ToM tasks. It could be argued that patients with more severe global

impairment may be more prone to follow medical advice or would be more easily persuaded to agree with the need to do so. In fact, awareness of need for medication is likely to be the dimension most influenced by social and cultural factors and relatively independent from genuine awareness of illness. It is commonly seen that patients acknowledge need for medication at the same time as showing no illness or symptom awareness. Low psychosocial and cognitive functioning, including ToM impairment, might be among factors to explain this dissociation.

In summary, the present study showed that relationships between insight and ToM in schizophrenia are complex. In symptom-aware patients without early onset of the disorder, an association between misattribution of symptoms to causes other than mental illness and better mentalising was revealed. A small subgroup of long-term symptom-unaware patients with residual delusions and/or thought disorder also showed good ToM skills. While unawareness of need for medication was unexpectedly associated with preserved ToM skills in patients without severe impairment in global functioning, being aware of having a mental disorder and of its social consequences did not predict ToM performance.

It should be noted that this is an exploratory study of two highly complex phenomena, with a number of limitations that make interpretation of results tentative. First, the exploratory nature of the study implied the inclusion of many variables, increasing the risk for Type I error. Second, the use of a single task to measure ToM and the lack of specific information on psychometric characteristics of this instrument might have limited the findings. There did appear to be some shared variance between the non-false belief and false belief subtests of the PST, in terms of IQ but, given the lack of significant findings between insight and neuropsychological tests included in the present study, the method used to calculate the ToM variable should not have obscured relationships between ToM and insight based on shared neurocognitive abilities. Third, the fact that this research was based on stabilised chronic patients implied not only reduced variability in insight and ToM, but also that aspects that may play a crucial role in modulating the relationship between ToM and insight, such as clinical severity and presence of particular symptoms (e.g., depressive or delusional states) could not be explored.

In conclusion, while there are theoretical reasons to propose a connection between insight and ToM, their relationship in stable schizophrenia patients is complex and modulated by clinical factors. Rather, both insight and ToM should be seen as distinct cognitive abilities which may overlap when it comes to attributional reasoning in the presence of general illness awareness. Further studies are needed to help understand the nature of these clinical

associations, their developmental course and their neurobiological correlates.

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Work 3

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Contents

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- Month of birth in relation to suicide
- Reattribution for medically unexplained symptoms

Mentalising impairment as a trait marker of schizophrenia?

One of the most controversial issues in 'theory of mind' research in schizophrenia in recent years has been whether theory of mind impairment may be seen as a trait marker or rather linked to particular symptoms. Sprong *et al*[1] conclude that evidence to date seems to favour the notion that mentalising impairment represents a possible trait marker. We believe that their meta-analysis is an excellent piece of scientific work but that this conclusion should remain tentative.

First, the existing evidence on theory of mind abilities in remitted patients is limited and difficult to interpret because of methodological shortcomings, such as non-explicit criteria for remission and poor control of cognitive abilities in the experimental design. A recent study by our group revealed that as a whole, stable patients did not show theory of mind impairment compared with carefully matched non-psychiatric controls. When standard consensus criteria for remission were applied to the sample, half failed to meet criteria for remission and showed a significantly worse theory of mind performance than remitted patients and controls. Specific theory of mind deficits in this group were associated with delusions. Thus, specific theory of mind impairment could go hand-in-hand with the presence of symptoms.[2]

Second, findings of theory of mind impairment in schizophrenia high-risk groups seem to support the assumption that theory of mind deficits represent a trait marker of the disorder. However, since these studies are mostly correlational, it is possible that the continuity of theory of mind deficits among 'at risk' groups may in fact derive from an intrinsic relationship between a psychotic symptoms continuum and theory of mind impairment. A review of the literature of theory of mind and schizotypal personality traits reveals that studies finding a positive significant relationship do so mainly with respect to schizotypal positive traits such as the cognitive-perceptual and unusual experiences dimensions of the schizotypy instruments.[3] Regarding investigations of first-degree relatives, evidence is controversial,[1] with findings of impaired performance on the more common types of theory of mind tasks but not on the 'eyes' test. However, it should be noted from these studies that those controlling for subclinical symptoms or schizotypal traits conclude that the association may be linked exclusively to the presence of subclinical positive symptoms.[4,5]

In our opinion, the existing evidence in theory of mind research is still limited but the possibility of a state-like association should not be ruled out. The most methodologically sound means to explore this would be to carry out longitudinal studies comparing theory of mind abilities in different phases of the illness, defined by explicit criteria. Future studies also need to differentiate between the affective and cognitive aspects of theory of mind, since it is possible that these show a different pattern of relationship with symptom clusters or schizophrenia profiles. Furthermore, it is possible that future research reveals that state–trait interactions may be occurring.

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Authors' reply: Pousa *et al* comment that our conclusion that theory of mind impairment represents a possible trait marker for schizophrenia should remain tentative for two reasons. Regarding their first argument, data on remitted patients are indeed limited and have methodological shortcomings. Only five studies in remitted patients were available, and the number of remitted patients in each of these studies was small. We also remarked that the criteria for remission used may have varied across studies, and that other factors may have influenced the results. Thus, we agree that the conclusion that theory of mind impairment represents a trait marker for schizophrenia should be tentative. In fact, we did describe it as a 'possible' trait marker. It is important to note that meta-analyses are about effect sizes rather than significance levels. By synthesising data of multiple studies there is more statistical power to detect smaller group differences. Thus, although in three out of five studies the theory of mind impairment in remitted patients was not statistically significant, when the studies were combined, the overall effect was significant (mean $d=-0.692$, $P<0.01$). So when Pousa *et al* do no find theory of mind impairment in stable remitted patients, we are not only interested in the *P*-levels, but also in the effect size. We also agree with the second point that there is evidence of an association between psychotic symptoms and theory of mind impairment, but do not see why this would argue against our conclusion. Frith[1] already proposed associations between specific schizophrenia symptoms (e.g. paranoid delusions) and mentalising impairment, and in their upcoming paper Pousa *et al* apparently also find significant associations between theory of mind impairment and psychotic symptoms. Perhaps we should have stated that theory of mind impairment is a possible trait marker for psychosis rather than schizophrenia. We believe that theory of mind probably does not represent an 'all or nothing' skill, and that schizophrenia should perhaps be studied using a dimensional instead of a categorical approach.

1 Frith CD. *The Cognitive Neuropsychology of Schizophrenia*. Psychology Press, 1992.

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Month of birth in relation to suicide

Salib & Cortina-Borja[1] find that persons born during the spring–summer season of April, May and June were significantly more likely to die by suicide than those born during other months: they find a peak for May and a trough for October.

However, they misreport our earlier results in this field when they state in the introduction that 'Chotai *et al*[2] reported that people born in winter in Sweden were significantly more likely than those with other birth seasons to have used hanging as a suicide method'. They further misreport earlier findings of ours when they state in the discussion that: '. . . winter variations in serotonin reported by Chotai & Åsberg[3] are inconsistent with the findings of this study, essentially the opposite of the Swedish findings'.

Our earlier findings are in fact similar to and consistent with the results of Salib & Cortina-Borja. In Chotai *et al*[2] we clearly show that those who preferred hanging rather than poisoning or petrol gases were significantly more likely to be born during February–April. In Chotai & Åsberg[3] we demonstrate that those born during February–April had significantly lower levels of 5-hydroindoleacetic acid (5-HIAA).

We have also published cosine analyses of our data,[4] in which we found that the minimum of the month-of-birth curve for 5-HIAA was obtained for the birth month April (*t*-min 3.4, Table 1, where the interval 3–4 depicts April) and the maximum was obtained for October (*t*-max 9.4). We also reported that the maximum of the month-of-birth curve for preferring hanging was for March–April and the minimum was for September–October.

Low serotonin turnover has been implicated as a risk factor for suicidal behaviour, particularly with violent or lethal methods of suicide, as discussed by Salib & Cortina-Borja.[1] Thus, our findings are in line with those of Salib & Cortina-Borja regarding suicidality, since we obtained a peak for the birth month April comparable to their peak for May, and found a trough for 5-HIAA for the birth month April.

In another epidemiological study,[5] we report that season of birth association with suicide methods is found in those without a history of psychiatric contacts, but not in those with such a history. We have argued that season of birth associations for suicide methods are likely to be mediated to a large extent by a suicidality trait independently of specific major psychiatric disorders, with serotonin as the likely underlying neurotransmitter.

In our studies, the season of birth variation was found for hanging as the suicide method, but not for other methods often denoted as violent, for example firearms or drowning. Hanging is a more universal method of suicide, and gender differences in the proportion of hanging are much lower than for other methods. In this light, it would be of interest to analyse the data of Salib & Cortina-Borja, specifically with regard to whether there is a month of birth variation in suicide by hanging.

1 Salib E, Cortina-Borja M. Effect of month of birth on the risk of suicide. *Br J Psychiatry* 2006; **188**: 416–22.

2 Chotai J, Salander Renberg E, Jacobsson L. Season of birth associated with the age and method of suicide. *Arch Suicide Res* 1999; **5**: 245–54.

3 Chotai J, Åsberg M. Variations in CSF monoamine metabolites according to the season of birth. *Neuropsychobiology* 1999; **39**: 57–62.

4 Chotai J, Adolfsson R. Converging evidence suggests that monoamine neurotransmitter turnover in human adults is associated with their season of birth. *Eur Arch Psychiatry Clin Neurosci* 2002; **252**: 130–4.

5 Chotai J, Salander Renberg E. Season of birth variations in suicide methods in relation to any history of psychiatric contacts support an independent suicidality trait. *J Affect Disord* 2002; **69**: 69–81.

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Salib & Cortina-Borja[1] describe a disproportional excess of people who kill themselves when born in early winter and between late spring and midsummer, and a disproportional deficit when born in late autumn. This month of birth effect can be interpreted in the context of another unexplained characteristic, namely the increasing south–north gradient (i.e. the geographical latitude effect, as shown in different countries).

Optimal maturation of the oocyte in animals and humans has been proposed to occur during the prime time of the seasonally-bound ovulatory seasons and to lead to optimal development of the zygote leading to less morbidity during pregnancy, birth and adulthood. In contrast, non-optimal maturation would occur during the inherent transitional stages leading to errant early neural migration and/or developmental differentiation.[2] This seasonally-bound month of birth effect is recognised in the presented data, particularly in females (violent and non-violent methods) and males (non-violent methods), and in anencephalia, schizophrenia and related diseases such as eating disorders.[3] This concept also explains the shorter life expectancy for people born during the first part of the year *v.* the longer expectancy during the second part, and its mirror image on the southern hemispere.[4]

Seasonality of the ovulatory pattern as cause of month of birth effect on suicide can easily be connected with the geographical latitude effect. In fact, the consistent relation between timing of mating seasons in different animals and humans causes stronger transitional stages the further distanced from the equator and, thus, higher frequency of non-optimal maturation of the oocytes. This biological phenomenon explains the mentioned geographical latitude effect on suicidality, schizophrenia and congenital anomalies of the nervous system, diverging between both hemispheres. The highly biased tertiary gender ratio in both suicidality and schizophrenia, and other high-risk factors such as teenage motherhood, multiparity and intrauterine growth retardation,[5] are quite compatible with this concept. This month of birth factor, therefore, does not need to be interpreted in terms of the 'foetal origins' hypothesis, nor the 'maternal–foetal origins' hypothesis, as suggested by the authors, but rather of the 'oocyte origins' hypothesis.

1 Salib E, Cortina-Borja M. Effect of month of birth on the risk of suicide. *Br J Psychiatry* 2006; **188**: 416–22.

2 Jongbloet PH. The effects of preovulatory overripeness of human eggs on development. In *Aging Gametes. Their Biology and Pathology* (ed RJ Blandau): 300–29. Karger, 1975.

3 Jongbloet PH, Groenewoud HMM, Roeleveld N. Seasonally-bound ovopathy versus 'temperature at conception' as cause for anorexia nervosa. *Int J Eat Disord* 2005; **38**: 236–43.

4 Doblhammer G, Vaupel JW. Lifespan depends on month of birth. *Proc Natl Acad Sci USA* 2001; **98**: 2934–9.

5 Mittendorfer-Rutz E, Rasmussen F, Wasserman D. Restricted fetal growth and adverse maternal psycholosocial and socioeconomic conditions as risk factors for suicidal behaviour of offspring: a cohort study. *Lancet* 2004; **364**: 1335–40.

5. DISCUSSION

The present thesis focused on the study of the relationship between insight and ToM in a sample of 61 chronic schizophrenic patients during a stable phase. Previous to this, the project implied exploring ToM abilities of these patients as compared to a non-psychiatric control group, in order clarify some controversial issues on the nature of the ToM impairment in schizophrenia. Next, a general discussion of the main findings and contributions of this thesis will be presented, and suggestions for future research will be outlined.

As reviewed in the introduction, evidence of a ToM dysfunction in schizophrenia patients is widely documented. However, one of the most controversial issues in ToM research in recent years has been the nature of this dysfunction. Whereas the idea that ToM impairment is specific rather than secondary to general cognitive dysfunction and / or chronicity seems to be well established, the state versus trait views of the ToM dysfunction continue to be a matter of debate. Our study was designed so as to further explore this while trying to overcome some of the limitations of the previous studies, and we believe our findings made some interesting contributions.

Overall, data from our study revealed that when a more rigorous methodology and a larger sample were used, as a whole, stable patients did not show ToM impairment as compared with a carefully matched non psychiatric control group. Furthermore, when consensus criteria for remission were applied to the sample, not only half of it failed to meet criteria for remission but also showed a significantly worse ToM performance than remitted patients and controls. In addition, specific ToM deficits were associated with delusions. The fact that fully remitted patients did not show ToM impairment in one of the most sensitive ToM tasks in schizophrenia -2nd order- (Sprong et al., 2007) and that previous studies using a less strict remission criteria only found patients to be impaired in more complex verbal

ToM tasks - but not on 2nd order- may be indicative that under conditions of higher methodological rigour specific ToM impairment goes hand in hand with the presence of symptoms, and in particular positive symptoms.

These results add weight to the state dependent view of ToM deficits in schizophrenia, but at the same time they do not contradict recent accumulated findings of ToM impairment in schizophrenia high risk groups, which support the assumption that ToM deficits represent a trait marker of the disorder (see reviews in Brüne et al., 2005, Harrington et al., 2005., Sprong et al., 2007). This is so because it is possible that the continuity regarding ToM deficits along the “at risk” groups may in fact derive from an association between the positive psychotic symptoms continuum and ToM impairment. Thus, although commonly ignored, an important factor to take into account when interpreting the “at risk” literature is the control for subclinical symptoms.

First, a detailed review of the literature exploring the relationship between schizotypal personality traits and ToM reveals that studies finding a positive significant relationship do so mainly with respect to schizotypal positive traits such as magical thinking or ideation, and the cognitive-perceptual and unusual experiences dimensions of the schizotypy instruments. Thus, Langdon and Coltheart (1999) found that high schizotypals, as measured with the Schizotypal Personality Questionnaire (SPQ, Raine, 1991) did less well than low schizotypals on ToM tests, but they also reported a trend towards higher magical thinking and unusual perceptual experiences in the high schizotypal group. Data from another study by the same authors supported the view that positive schizotypal traits may be strongly associated with a ToM deficit, in particular with a difficulty in appreciating irony (Langdon & Coltheart, 2004). In the same line, Pickup (2006) revealed that individuals with high total schizotypy scores on the Oxford-Liverpool Inventory of Feelings and Experiences (O-LIFE, Mason, Claridge & Jackson, 1995) did not differ in ToM from those with low total scores, but schizotypal traits analogous to positive symptoms of schizophrenia (Unusual experiences scale of the O-LIFE) predicted poorer scores on the ToM task.

Similarly, Meyer and Shean (2006) found a significant relationship between two measures of social cognition -understanding of character intentions and the "Reading the Mind in the Eyes" test (Baron Cohen et al., 2001) - and a feature of schizotypal personality referred to as magical ideation. Other reports finding no differences in ToM performance between high and low schizotypal groups have used global schizotypy scores, obscuring possible findings regarding particular dimensions (e.g. Jahshan & Sergi, 2007).

Second, with regards to investigations into ToM abilities in relatives of subjects with schizophrenia, the existing evidence is still limited in number and with inconsistent findings, with reports of reduced performance of relatives on the more common types of ToM tasks (Wykes et al., 2001, Janssen et al., 2003; Marjoram et al., 2006a) but not on the "Reading the Mind in the Eyes" test (Kelemen et al., 2004; Irani et al., 2006). However, while most of these studies have only assessed mentalising abilities (Wykes et al., 2001, Kelemen et al., 2004; Janssen et al., 2003), those which have also included schizotypal or symptom measures have elucidated that the association may exclusively be linked to the presence of sub-clinical positive symptoms. Thus, Irani et al. (2006) compared ToM performance in stable chronic schizophrenia patients, their first-degree relatives and healthy controls. While initially they revealed no differences between these groups, after scores on schizotypal personality traits were added in the analysis, significant differences appeared, so that high schizotypal relatives showed ToM deficits similar to patients, while low schizotypals performed similar to controls. This was so particularly regarding the social interpersonal subscale of the SPQ. In addition, a recent study by Marjoram et al. (2006a) compared relatives of individuals with schizophrenia who had experienced psychotic symptoms, relatives who had not experienced symptoms and healthy controls on a battery of ToM tests. Significant group differences were seen only between controls and relatives who had experienced symptoms at or around the time of testing, concluding that ToM performance was related to the state effects rather than the enhanced risk for schizophrenia.

In sum, although studies exploring ToM abilities in schizotypy and first degree relatives of schizophrenia patients are mostly correlational and do not allow to elucidate issues on causality, findings of preserved ToM abilities in subjects with schizophrenia without active symptoms (remission) and in high risk populations showing no positive schizotypal traits, could be suggesting that it is psychopathology which impairs social cognition and not the other way around; although it could also be indicative that schizophrenia symptoms and mentalising alterations are subserved by a common neurobiogical substrate. In fact, findings from well designed prospective high risk studies of graded trait effects in several cognitive and psychopathological functions (Johnstone et al., 2005) as well as from high risk imaging studies (Marjoram et al., 2006b; Whalley et al., 2004) suggest that state-trait interactions may also be occurring (Brüne, 2003; Meyer & Shean, 2006).

Since there is evidence that positive and negative symptom clusters may be the result of different pathological abnormalities in schizophrenia (Smith et al. 2004; Brébion et al., 2002), and that cognitive and affective aspects of ToM can be differentiated, it might be hypothesised that different types of ToM impairment underlie different schizophrenia symptoms or patient subtypes (e.g. deficit syndrome). In his view of schizophrenia as a metarepresentational disorder, Frith (1992, 2004) proposed that whereas patients with negative features would tend to show impaired performance on ToM tasks because of lack of these abilities or ‘undermentalizing’, the results for patients with paranoid symptoms were predicted to be equivocal because of these patients' tendency to ‘overmentalize’ (Frith, 2004). Based on this model, it might be speculated that the tendency to ‘undermentalize’ specifically impairs the affective aspects of ToM while ‘overmentalizing’ impairs the non-emotional or cognitive ToM. In this line, Shamay-Tsoory et al. (2007) showed that schizophrenia patients presented a selective impairment in affective ToM as compared to normal controls, and that this was significantly correlated with negative symptoms. It is thus possible that ToM represents a core deficit in patients with a negative or “deficit syndrome” profile, whereas the ToM performance seen in delusional

patients may alter more specifically the cognitive ToM and may be state dependent.

As for the exploratory study of the relationship between ToM and Insight using uni- and multi-variate regression analyses, results partially supported our initial idea that, on the basis of several conceptual, clinical and neuroanatomical parallelisms, the two phenomena would show some overlap in schizophrenia patients. Results revealed that while no linear relationship existed between each of the five insight dimensions and ToM, these highly predicted ToM performance in a regression equation, together with some neurodevelopmentally-related and clinical interaction variables. In particular, two dimensions - symptom misattribution and unawareness of need for medication - as well as age at onset, global functioning and illness duration reached significance in the prediction equation.

Lack of a linear relationship between insight and ToM measures is in line with previous preliminary reports using correlational analysis (Langdon et al., 1997; Drake & Lewis., 2003), indicating that clinical manifestation of these phenomena are distinct. However, the significant predictive equation revealed in our study, and some other global associations suggested in previous studies using multivariate regression (Langdon et al., 2006; Lysaker et al., 2005), could be reflecting some conceptual overlap based on metacognition. In order to understand others' beliefs about another person (ToM) one may need to be aware of one's own thoughts and experiences (self-evaluation). Since both processes require the ability to think about mental states, the nature of the relationship between mental state reasoning and insight may be a manifestation of a common metacognitive underlying phenomenon. Support for this comes from studies exploring the neuropsychological correlates of poor insight, showing that metacognitive measures are more predictive of insight than traditional cognitive measures. Thus, both second order false belief tasks (Bora et al., 2007) and metacognitive scores of an adapted version of the WSCT (Koren et al., 2004) have shown a stronger association with insight than conventional WCST scores.

Besides this general relationship between insight and ToM measures, the distinction of the 5 different insight dimensions in our study allowed identifying more specific associations. In this respect, the most relevant finding concerned the prediction that the ability to make correct attributions of symptoms to the illness would be associated with mentalising. Although initially a positive association was predicted, we found that in patients who were symptom aware misattribution of these to causes other than a mental illness was associated with unimpaired mentalising, suggesting that in order to misattribute one needs to have mentalising abilities to some extent. This finding might also be revealing that ToM abilities mediate lack of insight as a psychological defence, since there is no reason for denial unless one is aware of one's own reality as well as others' thoughts or perspectives. Patients with symptom awareness who at the same time are aware of the mental states of others (ToM), may feel resistant to labelling their own experiences as mental illness, since that would be harmful to their self image, and their assumed image in the eyes of others, thus leading to denial (e.g. Freeman & Garety, 2003). This would also explain why denial of need for medication was associated with intact ToM abilities. Altogether, it could be that both neurocognitive and psychological mechanisms play a part in explaining these results. In previous studies poor insight has been independently associated with cognitive deficits and avoidant/denial coping styles (Startup, 1996), as well as with ToM difficulties and externalising attributional bias (Langdon et al, 2006). Further evidence in favour of an overlap between symptoms misattribution and ToM may in the future be found in neuroanatomical studies. Flashman et al. (2001) tested insight in schizophrenia patients as a function of neuroanatomic abnormalities in 15 sub-regions of the frontal lobe and correlated specific aspects of insight with each anatomic sub-region. They showed that overall unawareness of psychiatric illness was associated with smaller mid-frontal gyrus, right gyrus rectus, and left anterior cingulated gyrus, while misattribution of specific symptoms was associated with reduced superior frontal gyrus volume. So far specific ToM deficits have been associated with left medial frontal areas,

but future studies may identify more precise neuroanatomical basis for both functions.

Although no significant associations were found in our study between other insight dimensions -such as Illness and symptom unawareness- and ToM, this may have been due to the fact that our study was based on stabilised patients. The nature of ToM, insight, and of its relationships, may differ depending on the clinical status. We have previously discussed evidence supporting that the ToM dysfunction in schizophrenia may be state dependent, and particularly associated with positive symptoms. Although insight deficits are well documented in both stable and acute states, it is possible that Illness and symptoms unawareness during an acute state or in the first stages of the illness (first episode) may be of a different nature from unawareness shown following recovery. During an acute state or when psychotic experiences are encountered for the first time (before they can be confronted), poor awareness may simply be an extension of symptoms, and thus explained by direct psychopathological processes such as aberrant salience and alterations in self monitoring (see Kapur, 2005). In this line, it is very common to see patients that acknowledge they would label a particular experience as “abnormal” if told by another person, but “not abnormal” in their case because they have simply “experienced it”. In contrast, when the subject has progressively gone back to a “normal” way of perceiving or feeling and has achieved symptoms resolution, unawareness may be more related with psychological denial and/or with alterations in the metacognitive process necessary to acknowledge that a change of perspective has occurred, thus being able to label one’s own previous experience as pathological. Interestingly, there is some evidence that this process can take place while in a psychotic state, if the person is confronted by another’s point of view or is asked to shift perspective from first person to third person (Gambini, Barbieri & Scarone., 2004).

Finally, another issue that was not approached in our study but might nonetheless deserve consideration when exploring insight-ToM relationships is the distinction of awareness into particular symptoms or symptom

clusters. We have previously seen that the nature of ToM dysfunction may differ according to the type of symptoms. This may also be the case for insight and for insight-ToM relationships. For example delusions tend to be associated by definition with symptom unawareness and have been associated with increased misattribution (Cuesta et al., 1998, Kemp & Lambert, 1995), while they have also been related to intact mentalising skills (e.g. Abu-Akel and Bailey, 2000; Walston et al, 2000) and attributional biases (e.g. McKay, Langdon & Coltheart, 2005; Langdon et al, 2006; Randall et al, 2003). In contrast, negative symptoms may be more commonly acknowledged by patients but have been consistently associated to poor ToM (e.g. Doody, Gotz, Johnstone, Frith & Owens, 1998; Frith & Corcoran, 1996; Langdon et al, 1997; Mazza, De Risio, Surian, Roncone & Casacchia, 2001).

Besides the contribution of the particular results, our research helped to point out some relevant methodological and conceptual issues that should guide future research in the field. When studying mentalising abilities in schizophrenia issues such as the task specificity and the importance to accurately define symptom severity subgroups, include well-matched control groups, and control for variables such as illness duration, IQ and type of antipsychotic medication should be taken into account. As for future research on insight-ToM relationships, besides a multi-faceted approach to insight, studies should consider differentiating cognitive and affective aspects of ToM. In addition, the complexity of both phenomena makes it relevant to consider a wide range of possible confounders or interacting variables in their relationship. It would also be of interest to explore insight–ToM associations in different stages of the illness, and to explore particular symptoms or symptoms clusters separately.

Limitations:

The present thesis has approached the study of two highly complex phenomena in schizophrenia and holds a number of limitations that make interpretation of results tentative.

Lack of specific information on psychometric characteristics of ToM instruments might have limited the findings. Although tests with non-mental state control conditions were chosen, lack of psychometrically sound instruments continues to be a major problem in ToM research.

The exploratory nature of the study of the relationship between ToM and insight implied the inclusion of many variables, increasing the risk for type I error.

The fact that this research was based on stabilized chronic patients implied not only reduced variability in insight and ToM, but also that aspects that may play a crucial role in modulating the relationship between ToM and insight, such as clinical severity and presence of particular symptoms (e.g. depressive or delusional states) could not be explored.

6. CONCLUSION

Genuinely “representational” deficits in ToM reasoning may be state dependent in schizophrenia and associated with delusional thinking and possibly other symptoms affecting verbal reasoning such as disorganization.

Findings of a continuum of ToM deficits in the schizophrenia spectrum may reflect the continuum of psychotic experiences and in particular delusional thinking.

ToM deficits observed in patients with a negative symptom profile may be less specific and accounted for by general cognitive impairment and illness chronicity, thus appearing more persistent.

ToM deficits in schizophrenia may be task specific. While 2nd order ToM tasks may be sensitive to capture mentalising impairement, the PST may not be suitable to capture ToM deficits in low IQ populations.There is a clear need for validated ToM measures.

Future studies of ToM in schizophrenia should be very cautious with issues such as definition of subgroups by symptom severity, control for IQ and illness duration. Ignoring these may lead to spurious results.

Chronic stabilized schizophrenia patients have selective awareness of some attributes of their illness, but not others, supporting the multi-faceted model of insight, in which different insight aspects might correlate with different pathological, cognitive, or social deficits.

Insight and ToM are two complex and distinct phenomena in schizophrenia. However, relationships between particular aspects of insight and ToM exist, which are mediated by psychosocial, clinical and neurocognitive variables.

Intact ToM may be a pre-requisite for aware patients to attribute their symptoms to causes other than mental illness, which could be in turn associated with denial of need for medication.

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8. ANNEX I

Work 4

Al Ruiz, E Pousa, R Duñó, JM Crosas, S Cuppa, C Garcia-Ribera. (2008). Adaptación al español de la Escala de Valoración de la No Conciencia de Trastorno Mental SUMD. Actas Españolas de Psiquiatria, 36, 111-118.

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Adaptación al español de la escala de valoración de la no conciencia de trastorno mental (SUMD)

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Introducción. El objetivo del trabajo es examinar la fiabilidad y la validez externa de la versión en español de la Escala de valoración de la no conciencia de enfermedad mental (SUMD).

Metodología. Se utilizó un método de traducción-retrotraducción y la participación de un panel de profesionales para valorar equivalencia conceptual y naturalidad. La escala se compone de 3 ítems generales: conciencia de trastorno mental, conciencia de los efectos de la medicación y conciencia de las consecuencias sociales del trastorno, y de 17 ítems destinados a síntomas específicos que conforman dos subescalas: conciencia y atribución. Se valoraron 32 pacientes con trastorno esquizofrénico o esquizoafectivo, según criterios DSM-IV. Las evaluaciones fueron realizadas mediante el sistema de entrevista con observador. Se calculó la fiabilidad a través del coeficiente de correlación intraclase (CCI) y la validez externa mediante el coeficiente de correlación de Spearman entre las puntuaciones de la escala y una medida independiente de conciencia global de trastorno.

Resultados. El CCI fue siempre superior a 0,70. Los ítems generales conciencia de trastorno y conciencia de los efectos de la medicación y la subescala conciencia de los síntomas se correlacionaron significativamente con la medida global de conciencia. Contrariamente, el ítem general conciencia de las consecuencias sociales del trastorno y la subescala de atribución no se correlacionaron significativamente, lo que apoyaría la idea de que la conciencia de trastorno es un fenómeno multidimensional.

Conclusiones. La versión al español de la escala SUMD es conceptualmente equivalente y presenta una fiabilidad y validez similares a la original.

Palabras clave:
Conciencia. Trastorno mental. *Insight*. Esquizofrenia.

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Spanish adaptation of the Scale of Unawareness of Mental Disorder (SUMD)

Introduction. The aim of this paper is to examine the reliability and external validity of the Spanish adaptation of the Scale to Assess Unawareness of Mental Disorder (SUMD).

Method. A translation-backtranslation of the original scale was elaborated, and a panel of professionals participated to assess conceptual equivalence and naturality. The scale consists of 3 general items: awareness of mental disorder, awareness of the effects of medication and awareness of the social consequences of the disorder; and of 17 items related to specific symptoms, which make up two subscales: awareness and attribution. Thirty-two patients diagnosed of schizophrenic or schizoaffective disorder following DSM-IV criteria were evaluated. The evaluations were performed using interviews with an observer. Intraclass Correlation Coefficient (ICC) was calculated for the reliability analysis and the Spearman correlation coefficient between the SUMD scores and one independent score of global insight for external validity.

Results. The ICC were all over 0.70. Convergent validity with the independent global measurement of insight was found for the general items of awareness of mental disorder and awareness of the effects of medication, and for the subscale on awareness of symptoms. The awareness of the social consequences of the disorder and the subscale on attribution did not correlate significantly with the global measurement of awareness (insight). These results are consistent with the hypothesis that awareness (insight) is a multidimensional phenomenon.

Conclusion. The Spanish adaptation of the SUMD scale is conceptually equivalent and displays a similar reliability and external validity as the original version.

Key words:
Awareness. Mental disorder. Insight. Schizophrenia.

INTRODUCCIÓN

La falta de conciencia del propio trastorno (o falta de *insight*) es un fenómeno frecuente en los pacientes afectos de

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un trastorno psicótico. En el estudio piloto internacional sobre esquizofrenia de la OMS[1] se observó que el 97% de los pacientes presentaban déficit de conciencia. Posteriormente se hallaron resultados similares en pacientes esquizofrénicos crónicos hospitalizados[2]. En estudios más recientes con pacientes psicóticos no institucionalizados se ha hallado que el 57% de los pacientes con esquizofrenia presentan un déficit moderado-grave de conciencia de enfermedad, el 32% manifiestan una marcada alteración respecto a las consecuencias sociales de su enfermedad y el 22% niegan la necesidad o el beneficio de la medicación. Además, el déficit de conciencia es más prevalente y grave en pacientes con esquizofrenia que en pacientes con otro tipo de trastorno psicótico[3], aunque otros trabajos no han hallado diferencias entre pacientes esquizofrénicos y pacientes bipolares[4,5].

En la psicosis, la conciencia de enfermedad hace referencia a la conciencia de tener un trastorno mental, de necesitar un tratamiento y de las consecuencias sociales del trastorno, así como a la capacidad para reconceptualizar los síntomas como patológicos o realizar una atribución adecuada de los mismos[6,7]. Así, las conceptualizaciones actuales consideran la conciencia de enfermedad más como un fenómeno continuo y multidimensional que como dicotómico del tipo «presente o ausente». Asimismo se trata de un constructo complejo y no exento de ambigüedad que genera controversias teóricas importantes[8]. Desde un punto de vista empírico se han desarrollado diferentes escalas a fin de capturar este fenómeno. Las escalas más frecuentemente utilizadas han mostrado altos niveles de correlación y, por tanto, presentan validez concurrente, dando a entender que se trata de un fenómeno que, aunque parcialmente, puede ser medido y replicado[9,10].

Una escasa conciencia del trastorno se ha relacionado con peor cumplimiento terapéutico[11-13] y, de forma poco consistente, con inadecuada utilización de los recursos asistenciales[14,15]; también se ha asociado a hospitalización involuntaria[16,17], mayor distorsión en la percepción subjetiva de la calidad de vida[18,19], peor funcionamiento social[20] y, globalmente, a peor evolución[13,17,12,21,22]. No queda clara la relación de la falta de conciencia con la gravedad de los síntomas psicopatológicos de la psicosis, si bien de forma global los estudios son consistentes en encontrar correlaciones en el rango de débiles a moderadas entre puntuaciones globales de *insight* y puntuaciones de gravedad psicopatológica[23]. En cuanto a la asociación con subtipos de síntomas, parece existir una relación negativa pequeña entre *insight* y sintomatología tanto positiva como negativa y global[24]. Por otro lado, aunque la relación entre riesgo de suicidio y conciencia de trastorno es compleja, parece haber una asociación entre la falta de conciencia de trastorno y un mayor riesgo de suicidio en algunos pacientes[25-27].

Se han propuesto diferentes hipótesis para explicar la falta de conciencia en la psicosis. La hipótesis clínica contempla el déficit de conciencia como un síntoma más de la psicosis relacionado con la propia naturaleza del trastorno, ya sea como síntoma primario independiente[28] o como manifestación directa específica de los síntomas positivos, negativos o desorganizados[29]; en la hipótesis motivacional la falta de conciencia de trastorno o negación de la enfermedad se entiende como una defensa psicológica o estrategia de afrontamiento dirigida a la preservación de una percepción integrada del sujeto[21,30,31]; la hipótesis atribucional entiende la falta de conciencia desde la necesidad básica que tiene el ser humano de interpretar el mundo perceptivo y dotarlo de una explicación que le dé sentido[32], y, finalmente, la hipótesis neuropsicológica postula que la falta de conciencia sería el resultado directo de un déficit en los sistemas que registran la percepción consciente y que estaría relacionado con el deterioro cerebral que se produce en la esquizofrenia, en este sentido podría emparentarse con la anosognosia[7].

Todo ello hace que el estudio de la no conciencia de trastorno en la psicosis sea un objetivo de investigación legítimo; pero, además, el estudio de la conciencia nos lleva a plantear el abordaje de la subjetividad en la psicosis. Si bien la psicosis desorganiza de forma esencial el funcionamiento mental, reconocer los diferentes niveles de conciencia de trastorno nos acerca un poco más, durante el trabajo clínico, a lo que es esencialmente humano. En la Europa de entre guerras, bajo el dominio del pensamiento eugenésico, Aubrey Lewis se planteó el estudio de la conciencia de trastorno en la psicosis como una forma de rescatar la humanidad de los enfermos mentales[6,33]. Actualmente, en la época de los principios democráticos y el reconocimiento de los derechos de los pacientes, el estudio de la conciencia de trastorno en la psicosis es una oportunidad para avanzar en el conocimiento de los mecanismos de la conciencia y de la subjetividad humana.

Como ya ha sido mencionado, se han desarrollado diversas escalas a fin de capturar y tratar de cuantificar este fenómeno. Entre ellas, la escala de valoración de la no conciencia de trastorno mental *Scale of Unawareness of Mental Disorder* (SUMD)[12,34] se ha mostrado válida y fiable, siendo ampliamente aceptada y utilizada como medida multidimensional de la conciencia de trastorno. Así, desde su desarrollo, la SUMD, además de haber sido validada y estudiada en un amplio grupo de muestras clínicas, ha sido traducida al menos a 11 idiomas por investigadores de países no anglosajones. En España, si bien la escala ha aparecido traducida en diversas publicaciones de compilaciones de escalas y está siendo utilizada en contextos clínicos y de investigación, hasta la fecha no se disponía de ningún estudio de adaptación de la versión en español de la escala.

La SUMD es una escala estandarizada que se puntúa en base a una entrevista semiestructurada directa con el paciente. La escala se compone de tres ítems generales para evaluar la conciencia de tener un trastorno mental: la conciencia de los efectos de la medicación y la conciencia de las consecuencias sociales del trastorno mental, y de 17 ítems destinados a síntomas específicos. De cada uno de estos 17 ítems-síntoma se evalúa la conciencia y la atribución que el paciente realiza del mismo. Así, se conforman dos subescalas: la de conciencia de los síntomas, que es la media de las sumas de las puntuaciones según el número de ítems

puntuados, y la de atribución de los síntomas, que es la media de las puntuaciones de los síntomas que han podido ser evaluados por tener el paciente conciencia de los mismos. Además de considerar distintas dimensiones y distinguir entre conciencia y atribución de los síntomas, la SUMD en su concepción original también permite la valoración diferenciada entre conciencia presente y pasada de cada uno de ellos.

La escala permite dar un total de cinco puntuaciones, una por cada uno de los tres ítems generales, una cuarta por la subescala de conciencia y una quinta por la subescala de atribución. Todas las puntuaciones, tanto de los ítems generales como de las subescalas de síntomas, se sitúan en un rango de 1 a 5, indicando las puntuaciones más altas un nivel de conciencia de trastorno más bajo o de atribución más incorrecta (peor conciencia).

Mientras que los tres ítems generales han de ser siempre valorados, de los 17 ítems que hacen referencia a los síntomas sólo se valora la conciencia de los mismos si están claramente presentes. Seguidamente se valora la atribución sólo si el paciente ha mostrado conciencia total o parcial del síntoma, es decir, si ha recibido una puntuación de la conciencia del síntoma entre 1 y 3. Los síntomas de los que no se expresa conciencia no pueden ser valorados en su atribución, así la subescala de atribución es parcialmente dependiente de la subescala de conciencia.

Existe una SUMD abreviada de los mismos autores en la que sólo constan los tres ítems generales y seis ítems-síntoma de los que sólo se valora la conciencia y no la atribución[2]. La evaluación de los ítems se realiza en una escala del 0 al 3, correspondiendo las puntuaciones más altas a no conciencia del trastorno. Se ha asumido que la escala abreviada es válida y fiable como la original. Si bien la sencillez facilita la utilización de la escala, se pierde información, especialmente en relación a síntomas negativos, y se pierde la valoración de la atribución de los síntomas. Por otra parte también se utiliza un rango de puntuación diferente. Todo ello dificulta la comparación entre los estudios que están utilizando estas escalas.

El objetivo del presente estudio es evaluar las propiedades psicométricas de la adaptación al español de la SUMD. Como se ha mencionado más arriba, la SUMD fue diseñada para valorar la no conciencia de trastorno mental en relación a un episodio presente y también en relación a un episodio pasado del trastorno. En nuestro trabajo sólo se realiza una evaluación de las características de la escala para la sintomatología presente.

METODOLOGÍA

Se utilizó un método de traducción-retrotraducción, de forma que el manual de entrenamiento y la escala proporcionada por el autor (X. F. Amador) ya traducidos al español fueron volcados de nuevo al inglés por uno de los investigadores (S. Cuppa) bilingüe y vueltos a traducir al español junto con la participación de un panel de profesionales para valorar la equivalencia conceptual y la naturalidad. La versión española resultante de este proceso se encuentra disponible si se solicita a la autora. En el apéndice I se incluye un resumen de la escala.

En una primera fase se llevó a cabo un entrenamiento en el que se realizaron entrevistas a 15 pacientes y se recogieron las respuestas literales dadas por los pacientes a cada observador. A partir de éstas se consensuaron entre los investigadores la formulación de las preguntas y el valor dado a las respuestas de los pacientes.

Posteriormente se valoraron 32 pacientes diagnosticados de trastorno esquizofrénico o esquizoafectivo, según criterios DSM-IV, ingresados en régimen de hospitalización parcial y estabilizados a nivel psicopatológico. El grupo estaba formado por 28 hombres y 4 mujeres cuya edad media era de 36,3 años (desviación estándar [DE] = 7,1).

La sintomatología clínica fue evaluada por los psiquiatras de referencia mediante la *Positive and Negative Syndrome Scale* (PANSS), de forma independiente y previa a la administración de la SUMD, a lo largo de la misma semana. Las entrevistas para evaluar la no conciencia de trastorno mental fueron realizadas por los investigadores (dos psiquiatras y dos psicólogos), mediante el sistema de entrevista con observador, de forma que de cada entrevista se obtuvieron dos valoraciones independientes. Siguiendo la metodología propuesta por los autores originales, de todos los síntomas incluidos en la subescala de síntomas sólo se valoraron aquellos que estaban claramente presentes, teniendo como referencia las valoraciones de la PANSS, la observación directa y las referencias sólidas del personal asistencial y de la familia.

El análisis de datos se llevó a cabo mediante el paquete estadístico SPSS (versión 12.5). Para el estudio de la fiabilidad se calcularon los coeficientes de correlación intraclase (CCI), mientras que la validez externa se calculó mediante la correlación no paramétrica de Spearman.

RESULTADOS

Ítems generales

Las puntuaciones medias para los tres ítems generales fueron 2,4 (DE = 1,38), 2,09 (DE = 1,17) y 2,03 (DE = 1,40), respectivamente.

Se calculó el cociente de correlación intraclase (CCI) para las puntuaciones independientes obtenidas por entrevista. El CCI para el ítem 1 fue 0,85, para el ítem 2 0,87 y para el ítem 3 0,91.

Ítems de las subescalas

Los ítems 5, 14, 15, 16, 17 y 20 que corresponden, respectivamente, a los síntomas delirio, aplanamiento afectivo, desga-

Apéndice 1 | Instrucciones, lista de síntomas, ítems generales 1-3 y ítem 6 de la *Scale of Unawareness of Mental Disorder* (SUMD)

Instrucciones

Esta escala requiere que el sujeto tenga un trastorno mental con alguno de los síntomas que se detallan más abajo. Para cada síntoma ítem de la escala primero se debe comprobar que el sujeto ha presentado este síntoma particular durante el período bajo investigación. La gravedad del síntoma no es relevante, únicamente es necesario que esté claramente presente. La verificación de la lista de síntomas debe llevarse a cabo antes de rellenar la escala a fin de determinar qué síntomas ítems son relevantes. Los tres ítems «sumarios» (números 1, 2 y 3), que no corresponden a síntomas específicos, normalmente son relevantes y deben ser cumplimentados si ése es el caso.
En la columna actual «A», se califica el máximo nivel de conciencia apreciado durante la entrevista para la psicopatología actual.
En la columna pasado «P» se califica el nivel presente de conciencia por cada ítem acontecido durante un período de tiempo anterior a la investigación en curso. En otras palabras, cuando se pregunta acerca de un episodio particular del pasado el sujeto en el momento presente podría decir que entonces él estaba delirando, con trastornos del pensamiento, sin capacidad para relacionarse socialmente, mentalmente enfermo, etc.
Se pueden utilizar períodos de tiempo más cortos o más largos para la valoración actual y retrospectiva de la conciencia y la atribución, dependiendo de los objetivos de la investigación.
En los síntomas ítems (números 4-20) se debe valorar la comprensión del sujeto acerca de la causa de su síntoma (la atribución).

NOTA: Por cada síntoma los ítems de atribución serán evaluados sólo si el sujeto ha recibido una puntuación entre 1 y 3 en el ítem de la conciencia.

Lista de síntomas

Enmarque con un círculo la «A» para actual o la «P» para pasado, situadas junto al número de ítem, para señalar qué síntomas ítems y períodos de tiempo han de ser evaluados.

Ítem			Síntoma
4	A	P	Alucinaciones
5	A	P	Delirio(s)
6	A	P	Trastorno del pensamiento
7	A	P	Afecto inapropiado
8	A	P	Apariencia o vestimenta inusual
9	A	P	Comportamiento estereotipado o ritualista
10	A	P	Juicio social empobrecido
11	A	P	Control pobre de los impulsos agresivos
12	A	P	Control pobre de los impulsos sexuales
13	A	P	Alogia
14	A	P	Aplanamiento o embotamiento afectivo
15	A	P	Desgana o apatía
16	A	P	Anhedonía-asocialidad
17	A	P	Atención pobre
18	A	P	Confusión-desorientación
19	A	P	Contacto visual inusual
20	A	P	Relaciones sociales pobres

Items generales

1. **Conciencia de trastorno mental**
   ¿En términos generales, la persona cree que tiene un trastorno mental, un problema psiquiátrico, una dificultad emocional, etc.?

A	P	
0	0	No puede ser valorado
1	1	Conciencia: el sujeto claramente cree que tiene un trastorno mental
2	2	
3	3	Conciencia intermedia: está inseguro de tener un trastorno mental, pero puede considerar la idea de que pueda tenerlo
4	4	
5	5	No conciencia: cree que no tiene un trastorno mental

2. **Conciencia sobre los efectos obtenidos con la medicación**
   ¿Qué es lo que cree la persona sobre los efectos de la medicación? ¿La persona cree que la medicación le ha disminuido la intensidad o frecuencia de sus síntomas (si es aplicable)?

Apéndice 1 **Instrucciones, lista de síntomas, ítems generales 1-3 y ítem 6 de la *Scale of Unawareness of Mental Disorder* (SUMD) (continuación)**

A	P	
0	0	No puede ser valorado o ítem no relevante
1	1	*Conciencia:* el sujeto claramente cree que la medicación ha disminuido la intensidad o frecuencia de sus síntomas
2	2	
3	3	*Conciencia intermedia:* está inseguro de que la medicación haya disminuido la intensidad o la frecuencia de sus síntomas, pero puede considerar la idea
4	4	
5	5	*No conciencia:* cree que la medicación no ha disminuido la intensidad o la frecuencia de sus síntomas

3. **Conciencia de las consecuencias sociales del trastorno mental**
¿Cuál es la opinión de la persona acerca de las razones por las que ha sido ingresado en un hospital, involuntariamente hospitalizado, arrestado, desalojado, despedido, herido, etc.?

A	P	
0	0	No puede ser valorado o ítem no relevante
1	1	*Conciencia:* el sujeto claramente cree que las consecuencias sociales relevantes están relacionadas con tener un trastorno mental
2	2	
3	3	*Conciencia intermedia:* está inseguro acerca de que las consecuencias sociales relevantes estén relacionada con tener un trastorno mental, pero puede considerar la idea
4	4	
5	5	*No conciencia:* cree que las consecuencias sociales relevantes no tienen nada que ver con tener un trastorno mental

Ítem síntoma de las subescalas

6. **Conciencia de trastorno de pensamiento**
¿La persona se da cuenta de que su comunicación está desorganizada y es difícil de comprender para los demás?

A	P	
0	0	No puede ser valorado o ítem no relevante
1	1	*Conciencia:* el sujeto claramente cree que sus comunicaciones o sus pensamientos están desorganizados
2	2	
3	3	*Conciencia intermedia:* está inseguro de que sus comunicaciones o sus pensamientos estén desorganizados, pero puede considerar la idea
4	4	
5	5	*No conciencia:* cree que no tiene comunicaciones ni pensamientos desorganizados

6b. **Atribución: ¿cómo explica el sujeto esta experiencia?**

A	P	
0	0	No puede ser valorado o ítem no relevante
1	1	*Correcta:* el síntoma se debe a un trastorno mental
2	2	
3	3	*Parcial:* está inseguro, pero puede considerar la posibilidad de que se deba a un trastorno mental
4	4	
5	5	*Incorrecta:* el síntoma no está relacionado con un trastorno mental

na-apatía, anhedonía-asocialidad, atención y relaciones sociales pobres, estaban presentes en más del 50% de pacientes.

Entre el 20 y 50% de los paciente presentaban los síntomas alucinaciones, trastorno del pensamiento, afectividad inapropiada, estereotipias, juicio social pobre y alogia, que corresponde a los ítems 4, 6, 7, 9, 10 y 13.

Los ítems 8, 11, 12, 18 y 19, que corresponden a los síntomas apariencia o vestimenta inusual, control pobre de los impulsos agresivos, control pobre de los impulsos sexuales, confusión y contacto visual inusual, sólo pudieron ser evaluados en menos del 20% de pacientes.

Subescala conciencia de los síntomas

Medias y desviaciones estándar

La media de las puntuaciones totales de la subescala de conciencia fue 1,9 (DE = 1,04), con un rango de 1 a 5.

Por cada uno de los ítems el rango de las medias de las puntuaciones fue desde 1 (DE = 0,0) (ítem 4, alucinaciones, e ítem 18, confusión) a 5 (DE = 0,0) (ítem 8, apariencia o vestimenta inusual). El ítem 19, contacto visual inusual, tuvo la variabilidad mayor 3,40 (DE = 2,19), mientras que la menor variabilidad la presentaron los ítems 1, 8 y 18, que corresponde a los síntomas alucinaciones 1 (DE = 0,0), apariencia o vestimenta inusual 5 (DE = 0,0) y confusión 1 (DE = 0,0), respectivamente.

Coeficiente de correlación intraclase

El rango del CCI entre las dos valoraciones por ítem del mismo paciente fue de 0,72 a 1 (media: 0,86). El CCI para el valor del total de la subescala fue 0,97.

Subescala atribución de los síntomas

Medias y desviaciones estándar

La media de las puntuaciones totales de la subescala de atribución fue 3,33 (DE = 1,18), con un rango de 1,43 a 5.

Por ítems, el rango de las medias de las puntuaciones fue desde 2,60 (DE = 1,72) (ítem 4, alucinaciones) a 4 (DE = 1,00) (ítem 19, contacto visual inusual). El ítem 10, juicio social pobre, presentó la mayor variabilidad 3,2 (DE = 1,88), y el ítem 19, contacto visual inusual, presentó la menor variabilidad 4 (DE = 1,00).

Coeficiente de correlación intraclase

El rango del CCI entre las dos valoraciones por ítem del mismo paciente fue de 0,75 a 0,99 (media: 0,84). El CCI para el valor del total de la subescala fue 0,94.

La subescala de conciencia y la subescala de atribución no se correlacionaron de forma significativa (Rho = 0,22).

Existe una relación jerárquica entre la dos subescalas, ya que para valorar atribución de los síntomas debe existir al menos una conciencia intermedia del síntoma. Así, a más alta puntuación en la subescala de conciencia existe una menor posibilidad de valorar atribución y a más baja puntuación en la subescala de conciencia existe una mayor posibilidad de valorar atribución y de mostrar variación. La no correlación indicaría que las dos subescalas están valorando fenómenos al menos parcialmente independientes.

Validez externa

Se utilizó el ítem 12 de la subescala de psicopatología general de la PANSS, que había sido valorado de forma independiente en cada paciente, para realizar una validación externa de la escala.

La tabla 1 presenta los datos de correlación entre las puntuaciones globales de la SUMD y el ítem 12 de la PANSS. Los ítems generales 1 y 2, que valoran conciencia de trastorno y conciencia de los efectos de la medicación, y la subescala de conciencia de los síntomas mostraron correlaciones significativas positivas con el ítem que valora la conciencia de trastorno en la PANSS. Por el contrario, no se observaron correlaciones significativas con el ítem 3, que valora la conciencia sobre las consecuencias sociales del trastorno mental, ni con la subescala de atribución.

DISCUSIÓN

El presente estudio se centra en evaluar las propiedades psicométricas de la adaptación al español de la SUMD, examinando su fiabilidad y la validez externa en una muestra de 32 pacientes con esquizofrenia y trastorno esquizoafectivo, ingresados en régimen de hospitalización parcial y estabilizados a nivel psicopatológico. Los resultados muestran unos coeficientes de fiabilidad entre observadores altos en todas las subescalas del instrumento, así como correlaciones significativas entre el ítem de conciencia de trastorno de la PANSS y aquellas subescalas de la SUMD que evalúan más propiamente conciencia de trastorno, indicando todo ello que la versión en español de la escala de valoración de la no conciencia de trastorno mental SUMD presenta una fiabilidad y una validez externa comparables con la escala original.

Las puntuaciones de los ítems fueron muy variables, como es de esperar ante un fenómeno de carácter más dimensional que dicotómico. Además fueron comparables a los resultados obtenidos con muestras de similares características[35-38]. Por otra parte, las subescalas de conciencia y atribución, aunque parcialmente dependientes, no se correlacionaron entre ellas, corroborando la idea de que las dos subescalas están valorando fenómenos al menos parcialmente independientes.

Tabla 1 **Correlación entre las puntuaciones de la SUMD y la puntuación del ítem de conciencia de la PANSS en 32 pacientes psicóticos estabilizados**

Ítem 1. Conciencia de trastorno mental	0,427*
Ítem 2. Conciencia de los efectos de la medicación	0,374*
Ítem 3. Conciencia de las consecuencias sociales	0,288
Subescala de conciencia	0,505**
Subescala de atribución	0,239

Coeficiente de correlación de Spearman. *Correlación significativa al nivel 0,05 (bilateral). **Correlación significativa al nivel 0,01 (bilateral). SUMD: *Scale of Unawareness of Mental Disorder*. PANSS: *Possitive and Negative Syndrome Scale*.

En comparación con los datos descriptivos del estudio original, las puntuaciones medias obtenidas en las subescalas fueron discretamente inferiores, excepto en la subescala atribución. Esto podría explicarse por el hecho de que nuestro perfil de pacientes es ligeramente diferente a la muestra del trabajo original; se trata de pacientes un poco más mayores, con trastornos más evolucionados y en fase estabilizada de su trastorno que asisten a un dispositivo de hospitalización parcial en donde se supervisa el cumplimiento del tratamiento psicofarmacológico. Cabe esperar, por tanto, que estos pacientes puedan tener una discreta mejor conciencia de sus síntomas. Por otra parte, la mayor puntuación en la subescala de atribución podría entenderse desde una perspectiva de carácter contextual, ya que la atribución es una dimensión de la conciencia de trastorno más relacionada con los mecanismos interpretativos de la realidad externa y, por tanto, más relacionada con el contexto sociocultural que con la experiencia propiamente vivida.

La validez externa medida a través de la convergencia con una medida general de conciencia de trastorno, como es el ítem 12 de la subescala de psicopatología general de la PANSS, da resultados parciales, como ya era de esperar. La conciencia de las consecuencias sociales del trastorno mental y la atribución no se correlacionaron con el ítem general de la PANSS.

Todo ello apoya la idea de que el *insight* es un fenómeno multidimensional y que los mecanismos que subyacen a la capacidad de tener conciencia de una forma global del trastorno, de la necesidad de tratamiento y de las consecuencias sociales del trastorno puede que no sean los mismos que los que subyacen para la conciencia de los síntomas del trastorno o para la atribución de los mismos. Por ejemplo, existe evidencia de que los déficit neurocognitivos se asocian más a no conciencia de síntomas (o dimensión de conceptualización de los síntomas como patológicos) que con las dimensiones de conciencia de enfermedad y necesidad del tratamiento[39-41].

Incluso para cada uno de los síntomas puede que exista variabilidad en la conciencia y en los mecanismos subyacentes que podrían ser más o menos específicos. Un ejemplo de la falta de correspondencia entre la conciencia de síntomas de diferentes dominios es lo que ocurre entre la conciencia de movimientos anómalos en la discinesia tardía y la conciencia de trastorno mental en el mismo grupo de pacientes[42]. Disponemos también de evidencia preliminar respecto a la idea de que los mecanismos que subyacen a la conciencia de síntomas positivos y negativos son probablemente distintos.

Así, se ha observado que la conciencia evoluciona de forma diferenciada para ambos tipos de síntomas y que se correlacionan también de forma diferenciada con los déficit de funciones ejecutivas[36,43].

Se obtuvo una elevada fiabilidad entre observadores, con un CCI siempre por encima de 0,70. Los datos apoyan el buen diseño del instrumento, que se sostiene además en sugerencias para la exploración claras y concisas y en un manual de entrenamiento práctico y amplio.

Dadas las características de la escala, en donde los ítems síntoma solamente pueden ser valorados si están presentes y la atribución sólo se valora de los síntomas de los que se tiene conciencia, no se pudo realizar un estudio de consistencia interna de la misma, tanto en la versión original como en el presente trabajo, ya que cada uno de los pacientes puntúa en ítems diferentes.

La SUMD, así como otros instrumentos diseñados para valorar conciencia, probablemente captura de forma parcial este complejo fenómeno. La utilización de la escala en su versión completa de 20 ítems es lo más aconsejable porque permite distinguir, por una parte, la apreciación del paciente sobre sus síntomas de forma diferenciada y, por otra, las consecuencias sociales de su trastorno y la interpretación que realiza de sus síntomas. Ello facilita una visión más global del paciente y ayuda a focalizar mejor las intervenciones de rehabilitación o de mejora en la percepción de su trastorno.

Actualmente no tenemos una hipótesis comprensiva sobre este fenómeno, entendido a veces como un síntoma de la psicosis, otras como un déficit cognitivo o metacognitivo o también como una dimensión de la personalidad.

Aunque con instrumentos imperfectos, vale la pena adentrarse en el conocimiento de la conciencia de trastorno en la psicosis. Interesarse por qué piensa una persona sobre lo que le está pasando, como es la sensación de lo que le ocurre, donde encuentra una explicación a lo que le pasa, supone aceptar la subjetividad de cualquier experiencia humana y reconocer que nuestro trabajo va mas allá de identificar la existencia de síntomas y tratarlos según el protocolo.

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Work 5

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Capítulo 6

La Teoría de la Mente como módulo cerebral evolutivo

Jordi E. Obiols y Esther Pousa

INTRODUCCIÓN AL CONCEPTO

Un ladrón sale corriendo después de robar en una tienda. Mientras corre un policía que está de servicio observa que se le cae un guante. Él no sabe que ese hombre es un ladrón, solo quiere avisarle que ha perdido el guante. Pero cuando el policía grita:
"¡ Eh usted, pare!" El ladrón se gira, ve al policía y se entrega. Levanta hacia arriba sus manos y reconoce que acaba de robar en la tienda.

(Extraído de Happé, 1994)

El contexto social en el que se desenvuelve la vida humana implica que los sujetos vean y se comprendan a si mismos y a los demás no sólo en base a los hechos reales o conductas explícitas, sino en términos de las creencias, deseos e intenciones que los han motivado, es decir, en términos de estados mentales. De hecho, la mayor parte de la interacción humana tiene lugar en base a este tipo de lectura inferencial mentalística, y sólo a través de la misma pueden explicarse capacidades típicamente humanas como la manipulación de los pensamientos o creencias de los otros a través del engaño, la transmisión selectiva del conocimiento, la cooperación, la empatía, o el uso y interpretación de la prosodia.

Esta habilidad para distinguir la visión personal del mundo de la ajena y de interpretar la realidad en términos mentalísticos se conoce como Teoría de la Mente (ToM), término acuñado originariamente en 1978 desde el campo de la primatología por Premack y Woodruff, en su ya clásico artículo "Does the chimpanzee have a Theory of Mind?", para definir la "habilidad para atribuir estados mentales a uno mismo y a los demás con la finalidad de predecir su conducta" (Premack & Woodruff, 1978). En dicho trabajo los autores apoyaban la existencia de la ToM en primates superiores, a partir de una serie de

experimentos en los que unos chimpancés lograban predecir correctamente la conducta de otro individuo de su especie. Sus conclusiones fueron pronto puestas en tela de juicio, criticándose que la condición experimental utilizada permitía explicaciones alternativas. Esta polémica promovió que se definieran las condiciones mínimas necesarias para poder asumir que un sujeto mentaliza o dispone de la ToM, llegándose al consenso que tales requisitos los cumplía la situación de la "falsa creencia" (Dennett, 1978). En esta situación se le requiere al sujeto hacer una inferencia sobre una falsa creencia que otro sujeto tiene sobre una determinada realidad. Así, el sujeto experimental es expuesto a una determinada realidad, a la vez que es testimonio de cómo otra persona es sometida a información parcial o errónea sobre la misma realidad. Bajo estas condiciones, es posible separar, sin ambigüedades, el juicio basado en el estado mental del propio sujeto (su creencia real) del juicio basado en el estado mental del otro (su falsa creencia). Diferentes versiones experimentales de esta situación son las que, como se describe más adelante, se han venido utilizando en la investigación para evaluar la ToM.

Cabe dejar claro, pues, tal y como destaca Leslie (1987; 1994), que el funcionamiento de la ToM implica necesariamente que el sujeto lleve a cabo una representación de circunstancias imaginarias "desconectadas" ("decoupled") de la realidad. Así, cuando nos explicamos la conducta de otro en términos de un estado mental, por ejemplo, de una creencia, tenemos que reconocer que esa creencia puede no corresponder a la realidad e, incluso si la creencia entra en conflicto con la realidad, es la creencia y no la realidad la que determina la conducta. Pero tener ToM significa no sólo reconocer que los otros son agentes cuya conducta está determinada por sus estados mentales, sino que también implica el reconocimiento que los demás tienen perspectivas sobre el mundo distintas de las propias. Comprender la conducta ajena requiere ser capaz de inferir las creencias e intenciones de los demás, a la vez que darnos cuenta del estado real de las cosas según nuestra propia perspectiva. Implica considerar y separar dos perspectivas diferenciadas, la propia y la ajena.

En la literatura la habilidad para representar estados mentales ha estado referida, además de cómo ToM, mediante términos como el de conciencia reflexiva (Langdon et al; 1997), metarepresentación (Leslie, 1987; Perner, 1991), toma de instancia intencional (Dennett, 1978) y mentalización (Frith et al; 1991). En el presente texto utilizaremos indistintamente ToM y mentalización, por ser los dos términos más comúnmente utilizados desde las neurociencias cognitivas.

EVALUACIÓN DE LA TOM

Como hemos apuntado, la condición experimental que tradicionalmente se ha considerado por excelencia definitoria de la adquisición de la ToM ha sido la situación de la falsa creencia. En la primera tarea experimental de este tipo desarrollada (Wimmer & Perner, 1983) se le plantea al sujeto el siguiente escenario: Maxi deja su chocolatina en el armario de la cocina y sale a jugar. En su ausencia, su madre coge la chocolatina del armario y la guarda en un cajón. Maxi vuelve de jugar, deseando comerse su chocolatina. ¿Donde irá a buscarla, al cajón o al armario? Superar esta tarea con éxito requiere hacer el juicio de que Maxi buscará en el armario, aunque realmente la chocolatina esté en el cajón, dando evidencia de que el sujeto sabe que las acciones de Maxi dependen de su creencia sobre el estado de las cosas, y no simplemente del estado real de las mismas, ya que realidad y creencias difieren. Se considera que hacia los tres años los niños ya son capaces de superar este tipo de tareas con éxito. Se han desarrollado múltiples versiones de falsas creencias, entre las que destacan como más conocidas la “Sally-Anne Task” (Baron-Cohen, Leslie & Frith, 1985) o el “Smarties Test” (Hogrefe, Wimmer & Perner, 1986). Todas ellas tienen en común el hecho de requerir que el sujeto haga una inferencia sobre lo que otro sujeto piensa sobre hechos reales, por lo que han sido denominadas tareas que implican una inferencia de primer orden. Un tipo de tarea más compleja, que suele superarse a partir de los 5-6 años y para la que también existen distintas versiones, serian aquellas en las que se pide al sujeto que haga una inferencia sobre los pensamientos que un segundo sujeto tiene sobre lo que piensa un tercero, implicando estas una inferencia de

segundo orden. Entre las más utilizadas de esta categoría encontramos las tareas “The ice-cream Van” (Baron-Cohen, 1989) y “The burglar story” (Happé & Frith, 1994).

Para la exploración las habilidades mentalísticas más allá de los 5 o 6 años, se han diseñado, con formatos verbales y no verbales y metodologías más o menos naturalísticas, tareas experimentales ToM avanzadas. Estas plantean escenarios que implican el uso del engaño (Sodian, Taylor, Harris & Perner, 1992), la comprensión de chistes (Baron-Cohen, 1997), metáforas mentalísticas, sarcasmo y ironía (Happé, 1994), comprensión de la pragmática en el discurso y en la comunicación (Surian, Baron-Cohen & Van der Lely, 1996) o la comprensión de situaciones sociales de “metedura de pata” o “faux pas” (Baron-Cohen et al., 1999, Shamay-Tsoory, Tomer, Berger & Aharon-Peretz, 2003). Entre los instrumentos más utilizados de esta categoría destaca el ”Advanced Test of Theory of Mind” (Happé, 1994, Fletcher et al., 1995; Happé, Brownell & Winner, 1999), cuya traducción y adaptación al castellano ha sido recientemente llevada a cabo por nuestro grupo (Pousa, 2002).

El uso de procedimientos de evaluación de la ToM avanzada no solo ha sido común en psicología evolutiva, con el interés de explorar las fases del desarrollo de la ToM en la infancia y su funcionamiento en sujetos adultos y de edad avanzada (Ej. Happé, Winner & Brownell, 1998; Keysar, Lin & Barr, 2003), sino también en el contexto de estudio de condiciones psicopatológicas en las que aparecen déficit en ToM más sutiles, como en algunos trastornos del desarrollo (Ej. Happé & Frith, 1996) o en algunas condiciones psiquiátricas como la esquizofrenia (Ej. Corcoran, Mercer & Frith, 1995, Langdon & Coltheart, 1999, 2001) o trastornos de la personalidad (Ej. Fonangy & Target, 1998). También han sido utilizados en algunos de los recientes estudios de neuroimagen funcional sobre los circuitos neuronales implicados en la ToM (Happé, Brownell & Winner, 1999, Gallagher et al., 2000, Happé, Malhi & Checkley, 2001), aunque en este contexto también se han ido desarrollado otros paradigmas experimentales que intentan capturar “on line”, de forma implícita o explícita, los procesos de mentalización.

Con todo y salvo excepciones (Muris et al., 1999), no se han desarrollado tests validados desde un punto de vista psicométrico para la ToM sino que su evaluación ha consistido más bien en el diseño de tareas experimentales concretas y a medida de los distintos contextos, objetivos, intereses empíricos y tipo de población de estudio. Se trata pues de tareas con validez de criterio, pero no estandarizadas para un uso generalizado.

Por último, cabe notar que la evaluación de la ToM se ha enfrentado y se enfrenta a notables limitaciones ya que, no sólo resulta compleja la definición del fenómeno en sí - se ha discutido que la ToM significa más que ser capaz de resolver una situación de falsa creencia- (Bloom & German, 2000), sino que a la vez resulta difícil capturarlo de forma específica, sin el compromiso de otras funciones cognitivas frontales como la atención, memoria de trabajo, velocidad del procesamiento y planificación. Esto ha tendido a resolverse mediante el uso de tareas control, que plantean demandas cognitivas equivalentes a las tareas ToM, excepto en lo que se refiere concretamente a la mentalización. Un problema añadido es la dificultad de diseñar tareas sensibles a diferentes niveles de competencia de la función, es decir, instrumentos que permitan establecer gradación de niveles de dificultad y eviten el efecto techo. A ello se suman las limitaciones de la validez ecológica características de todo contexto experimental, agravadas en el caso que nos ocupa por el hecho de que el contexto social real es altamente complejo y muy difícilmente reproducible en situaciones experimentales concretas.

BASES NEUROBIOLÓGICAS DE LA TOM

Una hipótesis fundamental de la neurociencia cognitiva planteada por Fodor (1983) sugiere que el cerebro se estructura de forma modular. Los módulos son específicos para una determinada función, tienen un desarrollo ontogenético y un patrón de deterioro característico, así como una representación cerebral concreta. Esta visión modular del funcionamiento cerebral, opuesta a la hipótesis de la inteligencia general, propone que hay múltiples inteligencias que están específicamente diseñadas para resolver problemas de significación adaptativa (Brothers, 1990). En este contexto,

¿representa la mentalización un módulo innato, específico y organizado jerárquicamente, que ha evolucionado por presiones de la selección natural ante el ambiente social?

Disponemos de un creciente volumen de hallazgos que desde diferentes disciplinas van a favor de esta hipótesis sugiriendo que, en efecto, la ToM descansaría sobre un sistema cognitivo modular autónomo y específico, siendo el lóbulo prefrontal medial (Brothers, 1990, Siegal & Varley, 2002, Frith & Frith, 2003) una localización nuclear del mismo.

En primer lugar, desde la psicología evolutiva, una serie de estudios clásicos demostraron hace ya más de 40 años la preferencia innata de los neonatos por estímulos sociales (Franz 1961, 1963), existiendo también evidencia sugerente de que es a partir de estos mecanismos innatos, y en particular del reconocimiento de rostros humanos, que se gestan las funciones cognitivas precursoras del desarrollo de la ToM (Baron-Cohen & Cross, 1992). Además, desde esta disciplina también se demuestra que el desarrollo ontogenético de la ToM tiene lugar según una secuencia evolutiva preestablecida y universal, existiendo sólo mínimas diferencias individuales y culturales en la adquisición de las competencias mentalísticas (Avis & Harris, 1991, Wellman & Lagattuta 2000). Asimismo, existen también datos preliminares de que la ToM tiene un patrón de deterioro independiente del resto de las funciones cognitivas (Happé, Winner & Brownell, 1998), aunque esto ha sido puesto en duda recientemente (Maylor, Moulson, Muncer & Taylor, 2002).

Por otro lado, la cuestión de la especificidad de la ToM se defiende a partir de la evidencia desvelada desde diferentes condiciones psicopatológicas infantiles, en las que se manifiesta una clara disociación entre las habilidades de cognición social y otras funciones cognitivas básicas. Así, si revisamos la literatura experimental sobre los déficit de ToM en el autismo, encontramos un consenso muy consistente respeto a que los niños autistas pueden ser “ciegos a la mentalización” a la vez que inteligentes en otros aspectos. Por ejemplo, no logran comprender situaciones en las que otro sujeto tenga una falsa creencia sobre una determinada realidad, pero si reconocen falsas fotografías,

percatándose que no son una representación fidedigna de un determinado lugar. También en sus habilidades comunicativas sólo presentan déficit selectivos en aquellos aspectos que dependen de la mentalización, por ejemplo, utilizan gestos instrumentales y entienden expresiones literales, pero fracasan en el uso de gestos expresivos o en la comprensión de expresiones metafóricas. (Para una revisión, ver Baron-Cohen, Tager-Flusberg & Cohen, 2000). Esta disociación es sobretodo evidente en el autismo de alto rendimiento o Síndrome de Asperger, en el que se observa un elevado - y en algunos casos muy sofisticado- nivel intelectual en dominios cognitivos que no impliquen manipulación de estados mentales, junto con déficit muy acusados en lo referente a las capacidades mentalísticas. Se trata de sujetos que pueden llegar a tener carreras profesionales y académicas excepcionales - Baron-Cohen, por ejemplo, describe el caso de un profesor de matemáticas que llegó a ganar un equivalente al Premio Nobel (Baron-Cohen et al.,1999).- pero con una evidente torpeza en el contacto social. (Ver Happé, 2001, para la descripción autobiográfica de algunos casos recientes). Lo contrario ocurre en sujetos con Síndrome de Williams, quienes presentan una ToM intacta a pesar de un marcado retraso en el razonamiento lógico en otros dominios (Tager-Flusberg & Sullivan, 2000).

Más evidencia a favor de la especificidad de la ToM proviene de algunos hallazgos de estudios neuropsicológicos de pacientes con lesión cerebral traumática, que ponen de manifiesto, en pacientes con distintos tipos de lesión cerebral, la existencia de una disociación entre el nivel de ejecución en tareas de ToM y la competencia en tareas ejecutivas clásicas (Rowe, Bullock, Polkey & Morris, 2001; Varley, Siegal & Want, 2001; Pousa, Duñó, Cáceres & Ruiz, 2002; Stone, Baron-Cohen, Calder, Keane & Young, 2003; Shamay-Tsoory, Tomer, Berger & Aharon-Peretz, 2003) o en otras medidas de inteligencia cognitiva (Winner, Brownell, Happé, Blum, Pincus, 1998; Happé, Brownell & Winner 1999; Happé, Malhi & Checkley, 2001; Bar-On, Tranel, Denburg & Bechara, 2003).

Por último, la cuestión de si la ToM cuenta con una representación cerebral concreta se inscribe dentro de la línea de investigación más amplia

sobre la arquitectura del cerebro social (o de la cognición social), que constituye una de las líneas con mayor interés científico en las últimas décadas dentro de las neurociencias cognitivas, con aportaciones de estudios neurofisiológicos en primates no humanos (Brothers, Ring & Kling, 1990), estudios neuropsicológicos centrados en pacientes con lesión cerebral (Adolphs, 2001; Stuss, Gallup & Alexander, 2001; Damasio Tranel & Damasio, 1991; Damasio, 1994, Blair & Cipolotti, 2000), y de forma más reciente y creciente, con estudios de neuroimagen funcional (Gallagher & Frith, 2003). A modo de resumen, los hallazgos aportados hasta la fecha por esta serie de estudios ponen de manifiesto que la cognición social, usando una definición amplia del término que incluiría, además de la ToM, todas aquellas operaciones mentales que sustentan la interacción social, como el reconocimiento facial y de emociones, la autoreferencia, la memoria de trabajo, etc. (Brothers, 1990; Adolphs, 1999), está sustentada por un sistema neuronal ampliamente distribuido. Éste incluye un componente central circunscrito por estructuras amigdalares y áreas de conexión con estructuras prefrontales y temporales mediales, y otros componentes funcionales complementarios que comprenden regiones relacionadas con el lenguaje, funciones ejecutivas y áreas especializadas en la detección del movimiento animado (Adolphs, 2001; Siegal & Varley, 2002). (Para una revisión extensa, ver Frith & Frith, 2000, Grady & Keightley, 2002).

Por lo que se refiere más concretamente al substrato neuronal de la ToM, es decir a las áreas que sustentan la capacidad cognitiva concreta de representar estados mentales desconectados de la realidad, es a partir del volumen de estudios de neuroimagen funcional que han venido proliferando en los últimos cinco años que se ha llegado a delimitar de forma bastante precisa la localización de las áreas de activación asociadas a la ToM. Así, disponemos de más de 12 estudios de neuroimagen funcional que han utilizado concretamente tareas de mentalización, mientras que otros muchos, no haciéndolo directamente, también han aportado datos concluyentes al respeto de la circuiteria clave y específica para esta función. Una buena revisión reciente de esta serie de trabajos puede encontrarse en Gallagher & Frith

(2003) y en Frith & Frith (2003). A continuación se describen a modo de resumen los hallazgos más relevantes de esta línea de investigación.

En primer lugar, cabe destacar que se trata de estudios llevados a cabo con sujetos adultos sanos, que han utilizado paradigmas experimentales dispares así como tareas de mentalización de diferente naturaleza (verbales, no verbales). Así, algunos de estos estudios utilizan tareas ToM avanzadas, importadas del campo de la psicopatología del desarrollo como el test de Historias de Happé, comparándose la actividad cerebral de los sujetos cuando explican la conducta del protagonista de una historia que actúa determinado por una falsa creencia sobre una determinada situación, con la que se da cuando el sujeto explica la conducta de personajes de historias control que no implican mentalización, sino razonamiento lógico. Este tipo paradigma "off line" o retrospectivo, contrasta con el paradigma "on line", en el que se somete a los sujetos a tareas de mentalización en el tiempo real. En esta línea, bien se utiliza la metodología ToM /control, similar a la que acabamos de mencionar, bien se utiliza una misma tarea o estímulo, comparándose la actividad del sujeto ante la adopción de diferentes actitudes ante la misma, por haber cambiado la instrucción o consigna (por ejemplo, se le dice al sujeto que va a jugar contra un programa informático pre-establecido o contra una persona). Con todo, utilizando paradigmas multimodales y cognitivos dispares, hasta la fecha todos estos estudios han aportado resultados muy consistentes apuntando que las áreas de activación asociadas a la resolución de tareas mentalísticas son la corteza prefrontal medial, el surco temporal superior y los polos temporales bilaterales. Cabe notar, no obstante, que mientras que una región cerebral altamente circunscrita del córtex prefrontal medial, el córtex paracingulado anterior, -correspondiente al área de Broadmann (BA) 9/32-, se activa diferencialmente en todos los estudios sea cual sea la modalidad de tarea ToM utilizada, la activación del surco temporal superior y los polos temporales es menos exclusiva (y más modalidad-dependiente). Así, el surco temporal superior se activa ante tareas que incluyen a personas, estén o no relacionadas con mentalización (Gallagher et al., 2000), ante tareas que implican a personas que han de hacer juicios de causalidad o de intencionalidad (Brunet et al; 2000), y ante tareas que requieren la atribución de

intenciones al movimiento de figuras geométricas (Castelli et al., 2000), sugiriéndose que se trata de una zona específicamente dedicada a sustentar la percepción de intencionalidad, sea esta física o mental. Los polos temporales, por su lado, se activan durante tareas de recuerdo sobre escenas y caras familiares (Nakamura et al., 2000), reconocimiento de voces familiares (Nakamura et al., 2001), y recuperación de memorias emocionales (Dolan et al., 2000), siendo pues el sustento neuronal del almacenaje de recuerdos personales de naturaleza semántica y episódica.

Ante todo ello, Frith & Frith (2003) sugieren que la región específicamente implicada en la capacidad representar estados mentales "desconectados" de la realidad seria el córtex paracingulado anterior, mientras que las otras dos regiones, se asociarían al procesamiento de información conductual explícita, sobre la que cabría operar necesariamente para llegar a mentalizar. Así, tanto la percepción de la conducta intencional como los procesos memorísticos de recuperación de recuerdos sobre experiencias personales serían funciones consideradas requisitos esenciales para el desarrollo de la ToM, especulándose que ambas funciones precederían y serian aspectos clave a la hora de entender los orígenes del mecanismo de mentalización.

Es de particular interés comentar que la activación en los estudios de neuroimagen funcional del paracingulado anterior, además de asociarse específicamente a tareas que implican la representación de estados mentales de otros, también se ha descrito como área de activación diferencial ante tareas en que se pide a los sujetos que describan las emociones que están experimentando ellos mismos en un determinado momento (Lane et al., 1997, Gusnard et al., 2001), auto-reconocimiento visual (Kircher et al., 2000), memoria autobiográfica (Maguire, Mummery & Buchel, 2000), automonitorización verbal (McGuire, Silbersweig & Frith, 1996), pensamiento auto-generado (McGuire et al., 1996), y percepción del dolor (Rainville et al., 1997), por lo que seguramente se trata de una región crucial para la percepción de estados mentales, tanto propios como ajenos.

ORÍGENES DE LA TOM

¿Cuándo aparece la ToM en la filogénesis? ¿Existe una ToM en animales no humanos? y, más específicamente, ¿Existe una ToM en los primates superiores? ¿Existe una evolución de la ToM en el género Homo? ¿Cómo podemos saberlo?

Vistas ya la definición, funciones y base cerebral de la ToM, y partiendo de la hipótesis de su condición de "módulo cerebral", comparable a otros módulos como el lingüístico, veamos cuáles podrían ser sus orígenes evolutivos. Como ya hemos dicho, Premack y Woodruff (1978) fueron los pioneros del estudio y teorización de la ToM y lo fueron estudiando precisamente la conducta de chimpanzés. Parece, pues, apropiado iniciar este apartado con un resumen (Martí, 1997) de sus experimentos:

Un sujeto tiene una teoría de la mente cuando es capaz de atribuir estados mentales a los demás y a si mismo. Un sistema de inferencias de estas características merece el calificativo de teoría porque tales estados no son directamente observables y es posible utilizar el sistema para predecir el comportamiento de los demás. En lo que respecta a los estados mentales que un chimpancé puede inferir, considérense los que infieren los miembros de nuestra especie, tales como el propósito o la intención, el conocimiento, la creencia, la opinión, la duda, la suposición, la simulación, la simpatía y similares. Para determinar si los chimpancés pueden realizar este tipo de inferencias, mostramos a un chimpancé adulto una serie de escenas de video en las que un humano se enfrentaba a problemas de distinta índole. Algunos problemas eran sencillos: consistían en situaciones en las que determinado alimento resultaba inaccesible-plátanos situados vertical u horizontalmente fuera del alcance del sujeto, detrás de una caja y similares- como los problemas originales de Köhler. Otros eran más complejos: mostraban a un sujeto encerrado en una jaula de la que no podía salir, tiritando porque se le había apagado el termo o incapaz de poner en marcha un tocadiscos desenchufado. Con cada cinta se le daban al chimpancé varias fotografías, una de las cuales contenía la solución del problema planteado: un palo para los plátanos inaccesibles, una llave para el sujeto encerrado o una cerilla

encendida para el termo apagado. El hecho de que el chimpancé eligiera consistentemente las fotografías adecuadas se puede interpretar como que el animal era capaz de identificar los problemas representados en las grabaciones, entendía las intenciones de los protagonistas y elegía las alternativas compatibles con tales intenciones.

Desde la lógica evolucionista la concepción modular de la ToM implica, por una parte, la necesidad de hallarle un valor adaptativo y, por otra, que debemos constatar su presencia cada vez mayor en especies de animales que posean un sistema nervioso más y más próximo al humano actual. Si algo parece fundamental en el proceso de hominización es la creciente socialización, fenómeno observable ya en primates inferiores, más compleja en los simios y que llega a cotas máximas en el hombre actual. La pertenencia a un grupo tiene ventajas obvias como la posibilidad de acceso a mayores recursos de todo tipo y un menor riesgo de predación basado en una superior capacidad de defensa. Estudios recientes han demostrado que en los babuinos, un tipo de monos altamente social, las hembras más sociales (las que reciben más tiempo de aseo) tienen un número superior a la media de crias que sobreviven más de 12 meses. O sea, en las hembras babuino, la sociabilidad está correlacionada positivamente con la supervivencia de sus crias (Silk et al., 2003)

Pero la socialización y la organización jerárquica de los grupos sociales característica de los primates comporta también el conflicto de intereses individuales. Todo ello genera la necesidad de desarrollar una serie de recursos o habilidades sociales que permitan las interacciones interindividuales complejas que el grupo genera. Estas habilidades van a posibilitar el engaño, la simulación, la táctica, la manipulación y también la transmisión selectiva del conocimiento, la reciprocidad, la solidaridad y el altruismo con los miembros del grupo y congéneres. Es, por tanto, de la máxima importancia para un ser social el correcto desarrollo y adquisición de este repertorio de habilidades. Por todo ello, se ha sugerido que el sistema cognitivo humano ha estado modelado particularmente por la presión selectiva para la inteligencia social (Cosmides, 1989), y es obvio que la ToM subyace y es elemento básico de estos mecanismos mentales/conductuales claramente adaptativos en el desarrollo de la especie (Byrne & Whiten, 1988; Wellman et al, 2001). Desde el punto de

vista neurobiológico, estudios sobre la evolución cerebral,y en especial del neocórtex, también parecen sostener la hipótesis de un “cerebro social” claramente preformado ya en los primates (Dunbar, 1998).

Por otra parte, ya hemos visto como la expresión y comunicación de emociones es componente fundamental de la ToM. Estos procesos requieren de mecanismos básicos de descodificación y comprensión de las señales, a veces muy sutiles, que conllevan información emocional. Entender correctamente expresiones que denotan agresividad o seducción sexual en congéneres es esencial para la supervivencia y capacidad reproductiva del individuo. Es observación banal el notar que los simios superiores tienen un repertorio expresivo que recuerda a veces de modo sorprendente al de los seres humanos. El propio Darwin en su obra “The expression of the emotions in man and animals” (1889) fue pionero en el estudio sistemático y científico de este aspecto de la conducta animal. Ahora bien, si las expresiones son tan similares, tan antropomórficas, ¿podemos deducir que los estados mentales subyacentes son también similares o paralelos? Si bien la demostración puede ser compleja, al menos la sospecha parece fundada.

LA EVOLUCIÓN DE LA TOM EN PRIMATES NO HUMANOS

Los primates reconocen las relaciones de parentesco en conductas de compartir alimento y de aseado y son capaces de evaluar el estatus de un congénere en términos de rango social (Bergman et al., 2003).También forman alianzas para conseguir recursos, contra los predadores, para conseguir hembras (incluso un cómplice puede distraer la atención de un macho dominante) y en la venganza. Por lo tanto, pueden manipular la conducta de los demás y aprender a predecir su conducta en ciertas circunstancias (Tomasello y Call ,1994; de Waal, 1996). Los gorilas criados en un entorno humano son capaces de mirar a los ojos de otra persona para controlar lo que esta persona está atendiendo. De esta forma, a través de la coordinación de la mirada, el gorila se da cuenta de lo que otra persona desea y hasta puede modificar su conducta empleando la mirada (Gómez ,1991) .Gorilas y chimpancés son capaces de reconocer su propia figura ante un espejo en condiciones

experimentales, cosa que resulta imposible a los monos y a otros animales (Povinelli y Preuss, 1995).

En su conjunto, todas estas conductas apuntan a la existencia en los simios de unas capacidades mentales claramente similares a lo que definimos como ToM en los humanos. Existen otras conductas que sugieren capacidades afines. Señalar con el dedo es una conducta universal en el ser humano y emerge en la infancia. Los simios no lo hacen de modo natural pero aprenden a hacerlo si están en contacto intensivo con humanos. Otro repertorio conductual que sí exhiben los simios, como tirar excrementos, escupir o dirigir la orina hacia los cuidadores humanos reflejan unos componentes de malicia, jugueteo y travesura que apuntan a algún tipo de humor primitivo (schafenfreude). Finalmente, la habilidad de entrenar o enseñar que muestran madres chimpancés con sus crias, por ejemplo, para romper cáscaras de nueces,con una activa participación de la "maestra", se diferencia claramente del puro aprendizaje social imitativo u oportunista (Bradshaw,1997).

Humphrey (1976) acuñó la expresión "inteligencia maquiavélica" para designar conductas observables en primates superiores (chimpanzés, gorilas, bonobos) encaminadas a superar, controlar y manipular a iguales y a competidores por medio de la predicción de sus respuestas a nuestros propios cambios, relaciones y alianzas y también por medio de la interpretación de la conducta ajena, infiriendo estados tales como los deseos, intenciones y creencias.

Desde la teoría de la ToM en humanos, se considera que la simulación (pretence) y la adivinación del pensamiento (mindreading) son elementos básicos del constructo (Leslie, 1987). Existen observaciones naturalísticas de la conducta de chimpancés que sugieren fuertemente la existencia de una capacidad de adivinación de la mente de congéneres. Por ejemplo, Whiten y Byrne (1991) detallan, la siguiente cadena de actos, que califican de "contraengaño":

El chimpanzé A tiene acceso al cajón de comida

El chimpanzé B no tiene acceso y observa a A como simula no tener comida

El chimpanzé B simula que se va y se esconde detrás de un árbol desde donde sigue controlando a A

El chimpanzé A, confiado, saca la comida
El chimpanzé B sale de su escondite y arrebata la comida a A
Estos ejemplos de engaño natural entre simios indica una capacidad innata para conductas complejas que requieren de una ToM, aunque sea a un nivel rudimentario. Por otra parte, es notable el hecho de que no se han podido registrar conductas equivalentes en monos.

¿Existe algún tipo de evidencia que demuestre la existencia de simulación en simios? Veamos un ejemplo aportado por los estudios de Savage-Rumbaugh y McDonald (1988) sobre un joven chimpanzé llamado Kanzi: "...Kanzi pretende frecuentemente que esconde objetos entre las sábanas. Luego pretende que los saca y se los come, a veces incluso fingiendo que algo está malo. También involucra a otros en estos juegos, entregando el supuesto objeto y observando qué hacen con él...".

Es interesante notar que este tipo de conducta tampoco ha podido ser observada en monos, ni en estado salvaje ni en condiciones de entrenamiento humano. En estas especies de primates inferiores se observan conductas sociales complejas que recuerdan a la ToM. Por ejemplo, se ha descrito que los monos pueden distraer a otros congéneres haciendo ver que miran "como si hubiera" un predador en la distancia. Esta conducta, que ha sido denominada "engaño táctico" (Whiten y Byrne, 1986, 1988), implica la manipulación de la atención de otro individuo pero se considera que puede tener un componente rutinario, automático, que la aleja de una auténtica conducta de simulación. Por tanto, se puede dar interacción social compleja sin una ToM plenamente desarrollada. En cualquier caso, la capacidad de monitorizar la visión del mundo de otro individuo es, para un animal, un candidato a representar una etapa precursora en el desarrollo de la adivinación de pensamiento.

Se ha criticado a los ejemplos como el de Kanzi sobre la base de que son animales criados por humanos. Aparte de que se hace difícil aceptar que tales conductas pudieran emerger por el simple contacto con humanos, el hecho de que los monos no consigan incorporarlas, incluso con entrenamiento humano, demuestra su sustrato natural. Es interesante, pues, notar que se produce una co-evolución de las capacidades de simulación y de adivinación

del pensamiento desde una perspectiva filogenética, paralela a la que se puede observar desde una visión ontogenética en los seres humanos.

En resumen, estos experimentos y observaciones basados en la conducta de los primates parecen demostrar una serie de habilidades que, aunque no puedan ser tomadas como ejemplos inequívocos de destrezas mentalistas, sí constituyen precursores esenciales de una ToM. A pesar de la evidencia de engaño y de adivinación espontáneos, casi como la clara existencia de conductas sociales complejas, la mayoría de los autores son bastante cautos en atribuir competencias mentalistas a estos u otros primates. Existe consenso en reconocer que estas conductas están muy ligadas a individuos concretos y, por tanto, no son generalizadas. Además, incluso en estos casos, son relativamente infrecuentes. Todo parece conducir a la conclusión de que el engaño es básicamente comportamental pero no mentalista: el chimpancé sería capaz de actuar intencionadamente para lograr manipular la conducta de otro. Pero es más difícil aceptar que actúa intencionadamente para modificar su creencia (Martí, 1997).

LA EVOLUCIÓN DE LA TOM EN LOS HOMÍNIDOS

"No podemos afirmar que los chimpancés tienen capacidad de adivinar la mente pero podemos concluir que son unos muy buenos conductistas" (Smith, 1996) y, en cambio, observamos que los niños normales adquieren sin dificultad asombrosas
capacidades mentalístas hacia los 4 años de edad.

Se calcula que la separación evolutiva de los homínidos respecto a los chimpancés se produjo hace entre 6 y 5 millones de años. Teniendo en cuenta este dato y la conclusión del anterior apartado, se especula que la ToM humana debe haber evolucionado en este lapso de tiempo (Mithen, 2000). Si la investigación de la ToM con primates no humanos se enfrenta al problema básico de la ausencia de lenguaje verbal en estos animales, el objetivo de estudiar la ToM desde un punto de vista paleoantropológico se topa a un obstáculo no inferior, esto es, la desaparición de todas las especies del género Homo que precedieron al Homo Sapiens Sapiens actual. Desde el punto de

vista metodológico, este hecho lastra seriamente cualquier hipótesis sobre el tema, confiere un tono claramente exploratorio y especulativo a estas propuestas y seguramente explica la escasez de estudios en este campo (Wynn, 1993). Pero, al igual que en las hipótesis del origen del lenguaje, también podemos avanzar ciertas ideas sugerentes.

¿Cuáles son los argumentos que permiten especular con la presencia de ToM o de formas primarias de ToM en los orígenes del Homo Sapiens e incluso antes? Es probable que la ToM sea necesaria para la producción cognitiva no literal, fantasiosa o "creativa". Sabemos por ejemplo, que los niños autistas tienen grandes dificultades para pintar animales imaginarios. En la evolución humana tenemos evidencia de producciones artísticas complejas y de actividades ritualísticas en el paleolítico superior en Europa, hace entre 10 y 40.000 años. Estas actividades requieren de una cognición sofisticada, en la que el simbolismo, la abstracción y la "pretensión" (p.ej., que una pintura "representa" un venado con cuerpo de hombre, que a su vez simboliza un cierto tipo de fuerza o divinidad) sugieren la necesidad de una ToM plenamente desarrollada (Mithen, 1998).

Antes de este período, sobre los 250-600.000 años, sabemos de las capacidades y habilidades técnicas, en la manufactura de herramientas y demás, del hombre de Neanderthal. Esta tecnología implica necesariamente una transmisión cultural por medio de la tradición y del aprendizaje social. Los procesos de imitación, de enseñanza y de aprendizaje también sugieren la presencia de una ToM similar a la nuestra. Por otra parte, la posible existencia de habilidades lingüisticas en esta especie (Davidson, 1991), basada en la evidencia de un tracto vocal muy parecido al nuestro actual (y diferente del tracto en los grandes simios) y en el brote de crecimiento cerebral que se da en este periodo, plantea la siguiente cuestión: ¿es razonable pensar en la aparición del lenguaje complejo en ausencia de un ToM desarrollada? Y viceversa, ¿tiene sentido pensar en la aparición de una ToM compleja en una mente con niveles lingüisticos de chimpancé? Parece plausible sostener que ambas capacidades, módulos cerebrales paralelos, tuvieron una co-evolución puesto que se necesitan mutuamente. Si esto es así, podríamos deducir que ya en este período se está gestando una ToM afin a la actual (Mithen, 2000).

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