

## Theory of mind and language comprehension in schizophrenia

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### Abstract

**Background:** Theory of mind (ToM) is the natural ability to attribute/infer mental states about ourselves and others. The study of the limits of this capacity in autism-spectrum disorders has been projected more recently to the case of schizophrenia. **Method:** We review the studies on ToM deficiency in schizophrenia, based on the link observed by Chris Frith between psychotic symptoms and mentalizing anomalies, with particular attention to the implications of ToM in linguistic communication in the field of figurative language comprehension. **Results:** The data support a connection between ToM deficits and psychotic symptoms. In schizophrenia, the deficit in ToM appears to be specific and not dependent on more general cognitive abilities, and according to the evidence examined, it resembles a trait more than a state condition. The analysis of results shows that anomalies in ToM have projections on pragmatic aspects of language comprehension. **Conclusions:** ToM deficits showed by schizophrenic patients are especially linked to difficulties in understanding figurative language, beyond the influence of intelligence and executive functions.

**Keywords:** schizophrenia, theory of mind, language comprehension, executive functions.

### Resumen

**Teoría de la mente y comprensión del lenguaje en la esquizofrenia.**

**Antecedentes:** la teoría de la mente (TM) es la capacidad natural que nos permite atribuir/inferir estados mentales respecto de nosotros mismos y de los demás. El estudio de los límites de dicha capacidad en los trastornos de espectro autista se ha proyectado más recientemente al caso de la esquizofrenia. **Método:** se revisan los estudios sobre el déficit de TM en la esquizofrenia partiendo del vínculo establecido por Chris Frith entre sintomatología psicótica y anomalías mentalistas, prestando especial atención a las implicaciones de la TM en la comunicación lingüística en el ámbito de la comprensión del lenguaje figurado. **Resultados:** los datos apoyan una conexión entre déficits mentalistas y síntomas psicóticos. En la esquizofrenia el déficit en TM se ha mostrado específico y no dependiente de capacidades cognitivas más generales y, por la evidencia examinada, parece que se asemeja más a una condición de rasgo que de estado. Los resultados analizados muestran que las anomalías mentalistas se proyectan en los aspectos pragmáticos de la comprensión del lenguaje. **Conclusiones:** los déficits en TM que muestran los pacientes con esquizofrenia están especialmente ligados a dificultades en la comprensión del lenguaje figurado, más allá de la influencia de la inteligencia y las funciones ejecutivas.

**Palabras clave:** esquizofrenia, teoría de la mente, comprensión del lenguaje, funciones ejecutivas.

The study of psychological problems in schizophrenia has traditionally focused on basic cognitive processes, such as attention, memory and other executive functions. It was not until the 1990s that researchers began to take an interest in studying social dysfunction and its impact on the daily life of people who suffer from this illness. Several studies (Corrigan & Penn, 2001; Couture, Roberts, & Penn, 2006) already indicate the difficulties patients with schizophrenia face in terms of social interaction, their ability to live independently, hygiene and personal care, and their effectiveness at resolving common social problems. These contributions signify a new direction in the study of psychological impairment in schizophrenia, and they demonstrate the progression from a viewpoint that was excessively focused on basic

neurocognitive impairments to a wider perspective in which the social limitations of patients take on a significant role. It should be mentioned that when the consequences of neurocognitive deficits in the social functioning of schizophrenia patients were assessed (Green, Kern, Braff, & Mintz, 2000), the data revealed that they only explain between 20 and 60% of the variance found, leaving the accompanying range (40-80%) without explanation. These results suggest that, in addition to the domain of basic psychological processes, there is a mediating psychological domain related to social-cognitive aspects that would account for at least part of the unexplained variance. This explanatory gap establishes the social functioning of people with schizophrenia as a key to understanding their overall cognitive functioning.

Within the realm of social cognition, a discipline that studies the psychological processes involved in handling social information, research into schizophrenia has placed an emphasis on *emotion perception, social cognition, attributional style and theory of mind* (ToM). This article intends to focus on studies that explore anomalies in the ToM capacity of schizophrenia patients, selecting fundamental aspects of Frith's meta-representational deficit theory

as a point of departure for the study of mentalizing anomalies in schizophrenia. Relevant works that research the specific nature of ToM deficit in this type of patient will be reviewed in this context, as will the question of whether or not this deficit corresponds to a state or trait of the illness. After that, we will focus on the studies that connect mentalizing deficits with anomalies in the pragmatic aspects of language comprehension (LC), and last section provides a short summary of the article as a conclusion.

#### Theory of mind and meta-representational deficit in schizophrenia

ToM has been defined as the natural capacity to attribute/infer mental states in order to explain and predict behaviour in situations that involve social interaction (Baron-Cohen, Tager-Flusberg, & Cohen, 1993). The term was originally coined by primatologists Premack and Woodruff (1978) to suggest the possibility that chimpanzees are capable of inferring the mental states of humans and members of their own species. Subsequently, a considerable number of developmental psychologists adopted the term to describe the ontogenetic development of the capacity to pretend or simulate (fictional games) in babies and children at a developmental age (Leslie, 1987; Perner, 1991). In the area of schizophrenia, Chris Frith and colleagues (Corcoran, Mercer, & Frith, 1995; Frith & Corcoran, 1996) began the empirical study of ToM deficit in the mid 1990s. The same author theorized in an earlier 1991 publication in collaboration with his wife (Frith & Frith, 1991) that autism and schizophrenia could share a common deficit, one in which ToM or, in their terms, the mechanism used to generate meta-representations, is severely compromised. This idea stems from the observation that the negative signs that appear in schizophrenia (social isolation, poor communication, absence of creative/spontaneous behaviour and stereotyped behaviour) are similar to Wing's triad (Wing & Gould, 1979) characterizing autism (*socialization, communication and imagination disorders*). Chris and Uta Frith suggest that, unlike autism, in which ToM anomalies are congenital, in schizophrenia, the impaired capacity for mentalizing usually appears in adulthood and is generally associated with the first psychotic episode, when all cognitive and linguistic capacities have already been fully developed. In their opinion, this difference could explain why these two pathologies show clearly differentiated symptomatology. In fact, characteristic signs of schizophrenia, such as hallucinations and delusions, do not usually appear in autism, whereas in the latter illness, it is common to find islands of ability that are rarely seen in schizophrenia.

In what is probably his most referenced work, Frith (1992) develops the theory that the fundamental disorder suffered by patients with schizophrenia is a deficiency in *being aware of oneself* and *being aware of others*. In short, Frith suggests that, in schizophrenia, there is an underlying reflective awareness disorder, in other words, a disorder in the capacity to think about one's own thoughts and about those of other people. In his opinion, patients with schizophrenia present a dysfunction in the psychological mechanism that makes it possible to generate meta-representations about mental states, that is, representations of intentional attitudes.

The link Frith makes between awareness and the meta-representational mechanism is worthy of critical analysis, as is his insistence on attributing the cognitive anomalies of schizophrenia to the mentalizing deficit alone. In the first instance, it does not seem justified to state that awareness is essential for the mechanism

to fail, while in the second, he is perhaps guilty of reductionism when he causally reduces the heterogeneity of deficits observed in schizophrenia to one sole mechanism, however fundamental it may be. In any case, his proposals can be used to contextualize early research into ToM in schizophrenia, and it is on the basis of his first works (Frith, 1987, 1992, 1994) that Frith establishes a tentative connection between *schizophrenia, reflective awareness* and *meta-representation*. Using this connection as a point of departure, he develops the theory that the signs and symptoms seen in schizophrenia patients are the expression of the absence of, or a dysfunction in, the mechanism used to process meta-representations. According to this theory, psychotic symptomatology can be explained in terms of variable degrees of severity or impairment in the capacity to think about (or be aware of) one's own thoughts and those of other people (see Table 1).

Frith (1992) distinguishes between the *signs* and *symptoms* of schizophrenia. The former refer to the observable behaviour of patients and the latter to the internal experience (non-observable) inherent to suffering from this illness. His model makes predictions about how patients with schizophrenia should behave in ToM assessment tasks. Specifically, both patients with *negative behavioural signs* (avolition, poverty of speech, social isolation, flat affect) and *positive behavioural signs* (incoherent thoughts, disorganized behaviour) should achieve very low scores in ToM tasks, showing a deficit similar to that of the autism spectrum disorder, due to their prevailing difficulty in representing aims and intentions (their own and those of others). Frith also predicts that patients with *symptoms of paranoia* (delusions of self-reference, delusions of persecution, and conspiracy) will find it difficult to resolve mentalizing tasks, because even though they can interpret the intentions of others, they commit attributional/inferential errors that lead to a mistaken interpretation. In his opinion, patients with *passivity symptoms* (thought insertion/theft, delusions of control) should not be differentiated from *patients in remission* or control subjects, because although they find it difficult to represent their own mental states, they have no problem in representing the mental states of others.

The first experimental studies carried out by Frith and colleagues (Corcoran et al., 1995; Frith & Corcoran, 1996; Corcoran & Frith, 1996) widely confirmed the predictions of the model, although it has not been possible to reproduce some of the results since. Frith found very severe difficulties in patients with *behavioural signs* (negative and positive) and less severe problems in patients with *paranoid symptoms* and *passivity symptoms*. The connection between *paranoid symptoms* and ToM deficits discovered by Frith's team could not be corroborated by later studies (Langdon et al., 2001; Mazza et al., 2001).

Table 1  
Anomalies in awareness of intention associated with psychotic signs and symptoms. From C. D. Frith (1994)

| Loss of awareness of... | Positive features                                     | Negative features                  |
|-------------------------|-------------------------------------------------------|------------------------------------|
| Own goals               | Grandiose ability                                     | Depersonalization, lack of will    |
| Own intentions          | Delusions of control, thought insertion               | Poverty of thought, loss of affect |
| Others' intentions      | Delusions of persecution, third-person hallucinations | Derealisation, social withdrawal   |

While attributing psychotic symptomatology to ToM deficits, Frith also suggested a connection between *mentalizing capacity*, *symptomatology* and difficulties with the *pragmatic use of language*. Frith proposed that mentalizing deficits or meta-representational problems are connected to the type of symptoms that develop in a patient with schizophrenia. In turn, he connected theory of mind deficits in schizophrenia to problems with language use, by which he meant the pragmatic aspects of language and not linguistic capacity itself. Together, Frith's hypotheses gave a new focus to the cognitive problems that had traditionally been observed in schizophrenia patients and led to a prolific line of research into this area.

#### Specific deficit or derived from general abilities?

It could be said in general terms that, beyond nosologic differences, a considerable number of studies have revealed deficits associated with the mentalizing capacity of patients with schizophrenia (Corcoran et al., 1995; Frith & Corcoran, 1996; Corcoran, Cahill, & Frith, 1997; Langdon et al., 1997, 2001, 2002; Mazza et al., 2001; Brüne, 2003; Couture et al., 2008; Bora et al., 2008). This discovery is consistent throughout these studies, despite the diversity of the researchers' hypotheses and aims, and heterogeneity when grouping patients by symptomatology.

One of the most sought-after goals when studying ToM in schizophrenia has been to confirm whether the mentalizing deficit seen in patients is specific, or whether it is secondary and dependent on other more general cognitive abilities, such as intelligence and/or executive functions. This idea was borrowed from the modular concept of ToM proposed and supported by autism spectrum disorders. One of the pioneering works in the study of the relationship between mentalizing deficit and general cognitive capacity was carried out by Fine and colleagues in 2001. They studied a patient with very early or congenital damage to the left side of the amygdala who, in adulthood, was first diagnosed with schizophrenia and then with Asperger's syndrome. In the first stage of cognitive assessment, the study determined that the patient (B.M.) had severely impaired ToM capacity. In the second stage, the researchers assessed B.M.'s executive capacity. The patient underwent various neurocognitive tests and it was found that he had normal intelligence (IQ= 103) and did not suffer from executive dysfunction. The main conclusion of the study is interesting because it revealed that ToM is not simply an aspect of more general executive functioning, and that executive functions can develop and function independently from ToM. The difficulty in confirming the patient's diagnosis was likely due to the dissociation of capacities observed.

Langdon and colleagues were interested in the specific nature of ToM deficit in patients with schizophrenia. A 2001 article (Langdon et al., 2001) reviewed the degree to which mentalizing deficits can be explained by executive planning deficits or deficits in inhibiting salient information. Patients in the study presented ToM, executive planning and inhibition deficits. However, the authors confirmed that mentalizing deficits continued predicting the probability of subjects to belong to the patient group, when planning and inhibition were controlled statistically. The study concluded that ToM in schizophrenia is impaired in specific terms, although it co-occurs with planning deficits.

In general, research that has aimed to clarify the specific v. general deficit debate suggests that, regardless of the tasks used,

mentalizing anomalies are domain specific and not the result of general cognitive deficits, though this specific nature does not have the same magnitude as that found in autism (Corcoran, 2000). Although these results are considerably consistent, it cannot be concluded that intelligence or executive functioning are not needed to perform mentalizing tasks, rather that once their influence is removed from the ToM deficit-schizophrenia relationship, the correlation between these variables remained statistically significant. Therefore, taking into consideration the specific nature of mentalizing deficit in schizophrenia, it can be confirmed that general cognitive ability is necessary, but not sufficient, for patients to be able to infer/attribute mental states suitably.

#### The state or trait controversy

Another aspect that has been of great interest to researchers is the degree to which mentalizing deficits in schizophrenia can be considered to be a *trait* associated with the illness or a *state* dependent on symptomatic exacerbation. Although some data support the notion that ToM deficits are heightened in acute phases of the illness, several lines of research have yielded evidence suggesting that the deficit is a trait. On one hand, research shows that schizophrenic patients in remission perform significantly worse in ToM tasks than their close family members and control subjects (Herold et al., 2002; Janssen, Krabbendam, & Jolles, 2003). On the other hand, there are studies, based on continuity models between schizotypy and schizophrenia (Eysenck, 1960; Crow, 1990), that have found a significant relationship between mentalizing anomalies and subclinical schizotypy traits (Langdon & Coltheart, 2004; Meyer & Shean, 2006), which seems to indicate that ToM impairment is not restricted to the acute phases of the illness. Finally, further studies have found a clear relationship between ToM deficiencies and the chronic nature, or years of duration, of the illness (Harrington et al., 2005). Two recent meta-analyses (Sprong et al., 2007; Bora, Yucel, & Pantelis, 2009) also showed that patients *in remission* had significant mentalizing anomalies in comparison with the control groups assessed. Despite the fact that Frith's initial work as well as some more recent studies (Pousa et al., 2008) lead to the conclusion that mentalizing deficits are best identified with a *state* dependent on the intensity and quality of psychotic symptoms, the review of studies on patients in remission, patients at risk of suffering from the illness and close family members seems to consistently relate mentalizing impairment with the characteristics of a variable *trait*.

#### ToM and LC in schizophrenia

The past decade has seen growing interest in understanding how different impaired mentalizing capacities are interrelated in patients with schizophrenia. Linguistic ability, and particularly its *semantic-pragmatic* aspects, has been on researchers' agendas from the time when schizophrenic disorder was first defined. Studies on linguistic ability have benefited from the recent boom in linguistics and psycholinguistics, and although these disciplines have focused on problems from different theoretical bases and heterogeneous methodologies, they have provided a theoretical solidity that has made it possible to reach decisive conclusions in the study of language in schizophrenia. One of the conclusions that will be addressed in this review states that, in their most basic aspects, linguistic structures do not seem to be significantly impaired in

schizophrenia (Frith, 1994); therefore any observed linguistic anomalies must be located not in patients' *linguistic competence* but in their *communicative competence*. In this theoretical context, special importance is attached to the level of central processing in which the linguistic system has to interact with other cognitive systems such as ToM, *memory*, *attention* and other *executive functions* (EF) to achieve its communicative aim.

Within the interface of the concurrent processes that constitute central processing, dysfunctional figurative language comprehension (FLC) in schizophrenia has been thought to be dependent on dysfunction in the attribution/inference of mental states. This article will now review studies that were interested in, to a greater or lesser degree, the relationship between ToM and LC in schizophrenia, taking into account variables related to intelligence and EF.

Langdon and colleagues (2002a, b) were interested in the relationship between ToM ability and pragmatic language capacity in schizophrenia. They initially (2002a) assessed a patient group and a control group on mentalizing tasks and figurative language comprehension (FLC). They assessed ToM by using the *Picture Sequencing Task* (Langdon & Coltheart, 1999; Langdon et al., 2002a, b) while pragmatic capacity was assessed through different tasks that required the interpretation of figurative language in different stories. The group of patients found it difficult to complete the *false belief* tasks and also showed different degrees of difficulty in understanding *metaphor* and *irony* in the story comprehension task. These difficulties were found to be independent of verbal IQ and executive dysfunction in inhibiting salient information.

In a parallel study published at a later date (2002b), Langdon et al. proposed establishing the degree to which difficulties in attributing/infering mental states and intentions caused pragmatic deficits in comprehension and language production in patients with schizophrenia. To achieve this, they again assessed a patient group and a control group on their performance in different mentalizing tasks. ToM and pragmatic capacity were assessed with the same tools used in the previous study (Langdon et al., 2002a). The results of this research consistently showed that patients' ToM and *irony* and *metaphor* comprehension were selectively impaired. They also found that deficits in mentalizing capacity were associated with difficulties in interpreting *irony*, but not with understanding *metaphor*. In this second work, the authors concluded that mentalizing anomalies may contribute to the positive aspects of formal thought disorder (FTD) and impaired comprehension of *irony*, while the negative characteristics of FTD and comprehension of *metaphor* are better explained by semantic anomalies.

The two aforementioned studies led to the conclusion that ToM deficits have specific effects on irony comprehension but not on metaphor comprehension, although both types of comprehension are impaired. For this reason, the authors suggest a possible dissociation between the capacities underlying the comprehension of metaphor and irony. Patients also show anomalies in general intelligence, inhibition and executive planning, but controlling them statistically does not eliminate the capacity of FLC deficits (of both metaphor and irony) to predict whether a subject belongs to the patient group or not.

Langdon and colleagues' research into the relationship between mentalizing processes, language pragmatics and general cognitive processes have acted as incentive to several research teams. Two recent studies, removed from the Anglo-Saxon context,

are reviewed below that were also interested in variables that coincide with the difficulties schizophrenics have in understanding figurative language.

Champagne-Lavau and Stip (2010) explored the extent to which the characteristic pragmatic deficits (problems with FLC) of schizophrenia coexist alongside mentalizing deficits and/or executive deficits. They assessed a group of patients and a control group on three different abilities: *pragmatic capacity*, ToM, and finally EF. The variables were put into operation as follows: to assess *pragmatic capacity*, they used a *metaphor comprehension* task (new and crystallized) and an *indirect request comprehension* task from the *Protocole Montréal d'évaluation de la Communication* (Joanette, Ska, & Côté, 2004). ToM was assessed using a verbal comprehension task that included false belief stories, and EF using the *Stroop Test* (inhibition) (Golden, 1978), the *Trail Making Test* (planning) (Reitan, 1958), the WCST (cognitive flexibility) and *verbal fluency* using one of the tasks of the *Neurosensory Center Comprehensive Examination for Aphasia* (Spreen & Benton, 1977). The main results obtained by these authors clearly demonstrate that patients have difficulties in *pragmatic comprehension* tasks that co-occur with *executive dysfunction* (lack of flexibility) and ToM deficits. Covariance analysis suggests that ToM plays a role in the pragmatic comprehension of language, while the latter does not appear to be related to cognitive flexibility.

Our research group (Gavilán & García-Albea, 2011) recently carried out a study to assess the incidence of mentalizing deficits in the FLC of schizophrenic patients, while also evaluating the influence of *general intelligence* on the relationship we expected to find. To do this, a group of patients and a control group were assessed. They both were paired in terms of *age*, *gender*, *years of schooling*, and *linguistic capability*. Verbal (false belief stories, Happé, 1994) and non-verbal tasks (*Picture Sequencing Task*; *Graphic Jokes*, Fletcher et al., 1995) were used to assess ToM, while language comprehension was evaluated in its most basic aspects (lexical, syntactic and semantic-pragmatic level without figurative language) and its figurative aspects (metaphor, irony and proverbs) to assess the patients' pragmatic ability. A correlational analysis indicated a connection between ToM deficits and language comprehension problems. A discriminant analysis determined that the variables that best differentiated between patients and control participants were those that corresponded to the critical false belief items in the three ToM tests, and then those that corresponded to the FLC tasks. The results led us to conclude that ToM impairment is primarily associated with the *semantic-pragmatic* level of language processing and that this relationship presents as genuine and independent of IQ. In other words, in patients with schizophrenia, mentalizing anomalies contribute negatively to understanding the figurative aspects of language, beyond impairment to general intelligence.

## Conclusions

Widespread interest in the social dysfunction of patients with schizophrenia has brought a new perspective to the study of the cognitive functioning of this type of patient. The work of Chris Frith can be viewed as pioneering to the extent that he was the first to associate mentalizing deficits with the signs and symptoms experienced by people who suffer from this illness; prior to his proposals, research had principally focused on prototypically neuropsychological processes (attention, memory, executive



functions). Empirical research stemming from Frith's proposals has highlighted that, among other things, ToM deficit in schizophrenic patients is specific and cannot be better explained by limited intelligence and/or executive ability. This result is interesting because it means ToM can be thought of as a modularized mental capacity that is not limited to being an aspect of general cognitive ability, although it is modulated by it. Similarly, the results point towards a dissociation between capacities used in social and non-social processing. It should be mentioned that the study of ToM in schizophrenia has provided a complementary viewpoint to the study of cognition in schizophrenia, and has not replaced research projects in which capacities such as attention, memory, inhibition or executive functions are considered to be of primary importance in explaining the social and communicative anomalies observed in patients. Viewed from the perspective of nearly two decades of study, and taking into account the large number of deficient cognitive processes seen in schizophrenia, perhaps it is more correct to view schizophrenia as an illness caused by "multiple deficits" rather than a "sole deficit", as Frith proposed.

Another factor that stands out in this review is the consideration of mentalizing deficits as a *trait* (rather than a state associated with exacerbation phases) of the illness that is already present, though in an attenuated form, in schizotypal patients, close family relations, and in asymptomatic patients or patients in remission. This would agree with theories that propose that schizophrenia is a neurodevelopmental disorder (very early neuronal damage that

remains "dormant" until different developmental factors "connect" it) and would open the door to early detection of the illness if the relevant observations and tests were applied to children at critical ages of development.

Language comprehension in schizophrenia has been thoroughly researched; however, the functional relationship between mentalizing capacity and language comprehension (controlling the contribution of intelligence and EF) has received less attention. This relationship appears to be decisive in understanding cognition in schizophrenia and, by extension, the human mind. Work by Langdon's team, Champagne-Lavau and Stip's team and that of our own group may be the start of a path that proves to be extremely fruitful in understanding how ToM, LC and general cognitive ability coincide. The reviewed studies have shown that ToM impairment is genuinely connected to difficulties in processing the pragmatic aspects of language, and that connection is not primarily due to the impairment of general cognitive abilities such as intelligence and/or executive functions. Therefore, it can be concluded that, to a certain degree, ToM is necessary, although not quite sufficient, to understand the figurative aspects of language.

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#### References

- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. (1993). *Understanding other minds: Perspectives from autism*. Oxford, England: Oxford University Press.
- Bora, E., Gokcen, S., Kayahan, B., & Veznedaroglu, B. (2008). Deficits of social-cognitive and social-perceptual aspects of theory of mind in remitted patients with schizophrenia. Effect of residual symptoms. *Journal of Nervous and Mental Disease* 196, 95-99.
- Bora, E., Yucel, M., & Pantelis, C. (2009). Theory of mind impairment in schizophrenia: Meta-analysis. *Schizophrenia Research*, 109, 1-9.
- Brüne, M. (2003). Theory of mind and the role of IQ in chronic disorganized schizophrenia. *Schizophrenia Research*, 60, 57-64.
- Champagne-Lavau, M., & Stip, E. (2010). Pragmatic and executive dysfunction in schizophrenia. *Journal of Neurolinguistics*, 23, 286-296.
- Corcoran, R. (2000). Theory of mind in other clinical samples: Is a selective theory of mind deficit exclusive to autism? En S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.
- Corcoran, R., & Frith, C.D. (1996). Conversational conduct and the symptoms of schizophrenia. *Cognitive Neuropsychiatry*, 1(4), 305-318.
- Corcoran, R., Cahill, C., & Frith, C.D. (1997). The appreciation of visual jokes in people with schizophrenia: A study of "mentalizing" ability. *Schizophrenia Research*, 24(3), 319-327.
- Corcoran, R., Mercer, G., & Frith, C.D. (1995). Schizophrenia, symptomatology and social inference: investigating "theory of mind" in people with schizophrenia. *Schizophrenia Research*, 17, 5-13.
- Corrigan, P.W., & Penn, D.L. (2001). *Social cognition and schizophrenia*. Washington DC: American Psychological Association.
- Couture, S.M., Penn, D.L., Addington, J., Woods, S.W., & Perkins, D.O. (2008). Assessment of social judgments and complex mental states in early phases of psychosis. *Schizophrenia Research*, 100, 237-241.
- Crow, T.J. (1990). The continuum of psychosis and its genetic origins: The sixty-fifth Maudsley lecture. *British Journal of Psychiatry*, 156, 788-797.
- Eysenck, H.J. (1960). Classification and the problem of diagnosis. En H.J. Eysenck (Ed.), *Handbook of Abnormal Psychology*. London: Pitman.
- Fine, C., Lumsden, J., & Blair, R.J.R. (2001). Dissociation between 'theory of mind' and executive functions in a patient with early left amygdala damage. *Brain*, 124, 287-298.
- Fletcher, P.C., Happé, F., Frith, U., Baker, S.C., Dolan, R.J., Frackowiak, R.S.J., et al. (1995). Other minds in the brain: a functional imaging study of "theory of mind" in story comprehension. *Cognition*, 57, 109-128.
- Frith, C.D. (1987). The positive and negative symptoms of schizophrenia reflect impairment in the perception and initiation of action. *Psychological Medicine*, 17, 631-648.
- Frith, C.D. (1992). *The cognitive neuropsychology of schizophrenia*. Hove: Lawrence Erlbaum.
- Frith, C.D. (1994). Theory of mind in schizophrenia. En A.S. David & J.C. Cutting (Eds.), *The Neuropsychology of Schizophrenia*. Hove: Psychology Press.
- Frith, C.D., & Corcoran, R. (1996). Exploring "theory of mind" in people with schizophrenia. *Psychological Medicine*, 26(3), 521-530.
- Frith, C.D., & Frith, U. (1991). Elective affinities in schizophrenia and childhood autism. En *Social psychiatry: Theory, methodology and practice*. P. Bebbington (Ed.). New Brunswick, New Jersey: Transactions Press.
- Gavilán, J.M., & García-Albea, J.E. (2011). Theory of mind and language comprehension in schizophrenia: Poor mindreading affects figurative language comprehension beyond intelligence deficits. *Journal of Neurolinguistics*, 24, 54-69.
- Golden, C.J. (1978). *Stroop Color and Word Test. A manual for clinical and experimental uses*. Wood Dale, Illinois: Stoelting Co.
- Green, M.F., Kern, R.S., Braff, D.L., & Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: Are we measuring the "right stuff"? *Schizophrenia Bulletin*, 26, 119-136.
- Happé, F.G. (1994). An advanced text of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally

- handicapped and normal children and adults. *Journal of Autism and Developmental Disorders*, 24, 129-154.
- Harrington, L., Siegert, R.J., & McClure, J. (2005). Theory of mind in schizophrenia: A critical review. *Cognitive Neuropsychiatry*, 10, 249-286.
- Herold, R., Tényi, T., Lénárd, K., et al. (2002). Theory of mind deficit in people with schizophrenia during remission. *Psychological Medicine*, 32, 1125-1129.
- Janssen, I., Krabbendam, L., Jolles, J., et al. (2003). Alterations in theory of mind in patients with schizophrenia and non-psychotic relatives. *Acta Psychiatrica Scandinavica*, 108, 110-117.
- Joanette, Y., Ska, B., & Coté, H. (2004). *Batterie Montréal d'Évaluation de la Communication*. Isbergues: Ortho-Édition.
- Langdon, R., & Coltheart, M. (2004). Recognition of metaphor and irony in young adults: The impact of schizotypal personality traits. *Psychiatry Research*, 125, 9.
- Langdon, R., & Coltheart, M., (1999). Mentalising, schizotypy, and schizophrenia. *Cognition*, 71, 43-71.
- Langdon, R., Coltheart, M., Ward, P.B., & Catts, S.V. (2001). Mentalising, executive planning and disengagement in schizophrenia. *Cognitive Neuropsychiatry*, 6(2), 81-108.
- Langdon, R., Coltheart, M., Ward, P.B., & Catts, S.V. (2002b). Disturbed communication in schizophrenia: The role of poor pragmatics and poor mind-reading. *Psychological Medicine*, 32, 1273-1284.
- Langdon, R., Davies, M., & Coltheart, M. (2002a). Understanding minds and understanding communicated meanings in schizophrenia. *Mind & Language*, 17, 68-104.
- Langdon, R., Michie, P.T., Ward, P.B., McConaghy, N., Catts, S.V., & Coltheart, M. (1997). Defective self and/or other mentalizing in schizophrenia: A cognitive neuropsychological approach. *Cognitive Neuropsychiatry*, 2, 167-193.
- Leslie, A.M. (1987). Pretense and representation - the origins of theory of mind. *Psychological Review*, 94(4), 412-426.
- Mazza, M., DeRisio, A., Surian, L., Roncone, R., & Casacchia, M. (2001). Selective impairments of theory of mind in people with schizophrenia. *Schizophrenia Research*, 47, 299-308.
- Meyer, J., & Shean, G. (2006). Social-cognitive functioning and schizotypal characteristics. *Journal of Psychology*, 140, 199-207.
- Perner, J. (1991). *Understanding the representational mind*. Cambridge, MA: MIT Press.
- Pousa, E., Duñó, R., Brébion, G., David, A.S., Ruiz, A., & Obiols, J. (2008). Theory of mind deficits in chronic schizophrenia: Evidence for state dependence. *Psychiatry Research*, 158, 1-10.
- Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a "theory of mind"? *Behavioural and Brain Sciences*, 4, 515-526.
- Reitan, R.M. (1958). Validity of the Trail Making Test as an indicator of organic brain damage. *Perceptual and Motor Skills*, 8, 271-276.
- Spreen, O., & Benton, A. (1977). *Neurosensory center comprehensive examination for aphasia* (2nd ed.). Victoria, Canada: University of Victoria.
- Sprong, M., Schothorst, P., Vos, E., Hox, J., & van Engeland, H. (2007). Theory of mind in schizophrenia: Meta-analysis. *British Journal of Psychiatry*, 191, 5-13.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11-30.