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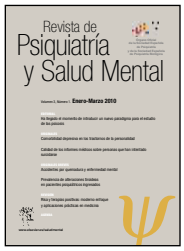

REVIEW

Theory of mind, facial recognition and emotional processing in schizophrenia

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Abstract Social cognition can be understood as "the mental operations underlying social interactions, which include the human ability to perceive the intentions and dispositions of others" (Brothers, 1990). Theory of mind, atributtional style, social perception are involved in social cognition.

It is wellknown that social cognition is impaired in individuals with schizophrenia. Recent investigations for social cognition in schizophrenia has showed that there is a relationship among social cognition, neurocognition and psychosocial functioning. The purpose of this article is to provide a review of social cognition in schizophrenia focusing on the deficit in Theory of mind described by Frith and recent neuroimaging studies. In fact neuroimaging research has demonstrated specific brain regions consistently engaged during theory of mind tasks. We also present some of the instruments avalaible to evaluate social cognition and to review and improve the main intervention programs.

Social cognition may be an important target for pharmacological and psychosocial treatments in the future.

PALABRAS CLAVE
Cognición social;
Esquizofrenia;
Teoría de la mente

Teoría de la mente, reconocimiento facial y procesamiento emocional en la esquizofrenia

Resumen La cognición social se refiere a los procesos que subyacen en las interacciones sociales, en las que se incluye la habilidad humana de percibir las intenciones y estados mentales de los otros (Brothers 2000). La cognición social incluye otras áreas como la Teoría de la mente, el procesamiento emocional, reconocimiento de caras, estilo atribucional y la percepción social. Es un hecho conocido que la cognición social está deteriorada en los individuos con esquizofrenia. Recientes investigaciones sobre cognición social en esquizofrenia han mostrado una relación entre la cognición social, la neurocognición

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y el funcionamiento psicosocial. El propósito de este estudio es ofrecer una revisión de la cognición social en la esquizofrenia, prestando mayor atención al déficit de la Teoría de la Mente propuesto por Frith así como en los recientes estudios de neuroimagen. De hecho investigaciones en neuroimagen han demostrado de forma consistente la relación entre regiones específicas cerebrales mientras se realizan tareas ToM. También presentamos algunos de los instrumentos válidos para evaluar la cognición social y revisar y mejorar los principales programas de intervención. La cognición social podría ser un importante objetivo en el tratamiento farmacológico y psicosocial en el futuro.

Introduction

The study of social cognition became more extensive in the 1990s when Frith, in his book entitled *Cognitive Neuropsychology of Schizophrenia*, put together an explanatory model suggesting that schizophrenic patients have difficulty understanding their behaviour as resulting from their own intentions and may interpret their acts as being under someone else's control. Paralleling this work, other new studies appeared that demonstrated, empirically, the functional importance of social cognition and established it as a mediating variable between neurocognition and the level of social functioning. New interventions also emerged centred on the different components of social cognition (emotional processing, social perception, theory of mind, social knowledge, and attributional style), along with various rehabilitation projects.

Social cognition began to acquire relevance following the April 2003 meeting of the MATRICS project[1] (Measurement and Treatment Research to Improve Cognition in Schizophrenia), where seven critical cognitive domains were identified in which patients with schizophrenia show deficit: speed of processing, attention/vigilance, working memory, learning and verbal memory, learning and visual memory, reasoning and problem solving, and social cognition. The term *social cognition* has not been immune to arguments over its definition; however, Wyer and Skrull[2] were already pointing out that there were numerous definitions for social cognition. One definition would be the human being's ability to perceive the emotions of others, to infer what they are thinking, and to understand and interpret their intentions as well as the norms that govern social interactions.[3,4]

There are numerous studies that have demonstrated the presence of changes in social cognition in schizophrenia. Regarding social perception, Penn et al[5] state that patients disregard the social context when processing social stimuli. Likewise, Nuechterlein and Dawson[6] comment that patients would have problems grasping information that requires abstract reasoning. In this regard, Ziv et al[7] point out that the impaired social cognition in schizophrenia is due to deficiencies in various mechanisms, including the ability to think analytically and to process information relative to emotions and signals. Regarding attributional style, Fenigstein[8] shows that schizophrenics selectively distort the hostile aspects of others. Leonhard and Corrigan[9] point out the problems patients with schizophrenia have in connection with emotional processing and with four basic factors: abstraction, familiarity, the complexity of a situation, and semantic processing. On the other hand, there are studies that show social cognition as a mediating variable between neurocognition and social functioning (Brekke et al[10]); the existence of a specific neuronal substrate for social cognition (Adolphs[11]); and the connection between the deficit in affect perception and cognitive-social problem solving (Spaulding et al[12]). According to this, the conclusion could be that the deficit in social cognition is at the root of the interpersonal problems and the impaired psychosocial functioning seen in schizophrenia. The isolation and blunted affect could be related to this deficit. The isolation that very often precedes the onset of this disease could also be a variable that influences its progression. In fact, some studies maintain that the changes in social behaviour become worse as the disease progresses and contribute to the increasing number of relapses (Pinkham et al[13]). Based on all these factors, social cognition is set forth as a new and promising field for study that may offer a theoretical explanation of the root of the psychopathology in schizophrenia and suggests innovative options for rehabilitation.

The theory of mind concept

The term *theory of mind* (ToM) was first used by Premack and Woodruff; it later gave rise to Brothers's *social brain hypothesis*[14] that described an evolutionary response to changes the brain undergoes in dealing with an increasingly complex social milieu. The initial and most important studies on the ontogeny of ToM were conducted on the autism spectrum disorder (Baron-Cohen et al[15]), but there are also recent studies on patients with fronto-temporal dementia and bipolar disorder, among others. According to this model, children use protodeclarative gestures and simulated games at about 18 months. At 3-4 years, they distinguish between their own beliefs and those of others (first order false belief). Starting at 6-7 years, children gradually learn to comprehend higher order representations, such as irony and metaphor (second order false belief). This continuous model would account for the autism spectrum symptoms that are visible from the earliest developmental years; however, it would not be a satisfactory explanation for schizophrenia because there would be controversy over admitting a continuous model as the sole explanation for

schizophrenia—even when it is true that support for it could be found in the various neurodevelopmental theories. This model is also incomplete in that it rejects the social impact on the development of ToM. Frith[16] (1992) draws an interesting comparison between autism and schizophrenia, explaining the symptoms in terms of a ToM lesion: the schizophrenic knows that mental states exist in other people but loses the ability to figure them out, whereas the autistic child does not know that those other mental states exist because the deficit has been present since the child's birth. There is, then, a dysfunction in metarepresentation or—amounting to the same thing—a cognitive deficit in recognizing the content of one's own mind and that of others' minds, which translates to difficulty describing one's own internal experiences and to a lack of control over the attention process.

Various models to explain theory of mind

Numerous theoretical constructs have emerged attempting to conceptualise the various existing hypotheses on ToM. The difficulties in this are obvious. The most well-known models are listed below.

1. *The modular perspective*. Developed by Fodor,[17] it proposes the existence of an independent ToM. Just as certain cognitive abilities are associated with specific brain functions, it is assumed that ToM is limited to the processing of information with social content only. Arising from this perspective is the so-called *selection processor* that separates the relevant from the irrelevant contextual information. The lack of this processor in schizophrenia could explain the problems with filtering that are seen in these patients. This functional-computational conception of the mind is considered inadequate to explain brain organization.
2. *The theory-theory or "metarepresentational" perspective*. Perner[18] describes a non-modular model suggesting that different levels of representational skills are acquired during childhood. Having authentic metarepresentations enables one to theorize about others' representations.
3. *The Hardy-Baylé model*.[19] This model hypothesizes about a ToM deficit that would be related to an executive deficit. According to this hypothesis, patients with disorganization of thought, speech, and social skills are the ones least able to complete ToM tasks because they are incapable of controlling their own actions. Following this model, ToM deficit would be seen only in patients whose disorganization is predominantly in thought and speech; there are studies that dispute this, however.
4. *Simulation theory*. This model is founded on functional magnetic resonance images of the existence of mirror neurons responsible for empathy that are located primarily in the left inferior frontal cortex and are activated when certain hand or mouth movements are observed in other people (Gallese and Goldman[20]).

Other proposed alternatives are found in Bailey and Abu-akel,[21] who propose a continuity model of ToM deficit and a classification that ranges from the truly impaired ToM to the hyper-ToM that is associated with overattribution of mental states and has been suggested as a causal factor in paranoid states.

Theory of mind and schizophrenia

Frith developed a model based on the existence of possible changes in the ability to metarepresent that, in turn, result in a ToM deficit, and he correlates this fact with the symptomatology of schizophrenia. He points out that schizophrenic patients have difficulty understanding their behaviour as resulting from their own intentions, and they may interpret their actions as under someone else's control. Frith's classifications[22] are disorders of "desired action" (negative symptoms and disorganization), impaired self-monitoring (delusions of external control and hallucinations of voices speaking, and other passivity symptoms), and disorders of thought monitoring. Frith states that there is a failure in monitoring both the action and the intent of the action. Under this model, Frith predicted that patients with a predominance of negative symptoms or disorganization would be the most deficient in performance of ToM tasks. Some psychotic symptoms, such as delusions of control and persecution and disorganization of thought and speech, could be understood in terms of the existence of an inability to explain one's own and others' intentions—in other words, in terms of a ToM impairment. There are studies on ToM in which deficits in this ability are shown to be associated with the symptom typology for the disease. On the basis of this hypothesis, patients with negative symptoms were never able to develop a ToM. Other authors suggest, however, that it is necessary to have an unimpaired ToM to be able, subsequently, to develop delusional ideas of persecution (Walston et al[23]). This hypothesis is based on some studies confirming that patients with paranoid schizophrenia show no symptoms of deficiency in ToM ability. It was suggested that these patients could use their general intelligence to compensate for their ToM deficit. Then again, Brüne[24] points out that the existence of a well developed ToM may mean excessive inferring about the supposed intentions of others, which may trigger erroneous interpretations of the situation. Langdon et al[25] came to the conclusion that ToM impairment could stem from an inability to cognitively inhibit the primary information or from a deficit in the ability to reason about hypothetical states.

Another of the current debates is whether ToM deficit should be considered a trait or a state. According to Frith, psychotic patients in remission and patients who present with only passivity symptoms may show normal performance on ToM tasks. Likewise, Hardy-Baylé postulates that patients with no disorganization symptoms would perform satisfactorily on ToM tasks. This indicates that a ToM deficit would represent a state variable rather than a trait variable. However, what supports the hypothesis of ToM as a state is the fact that it is not only patients with acute exacerbations but also patients with long-standing,

chronic symptoms who perform poorly on ToM tasks (Drury et al[26]).

Functional brain neuroimaging for theory of mind

Based on various neuroimaging studies, there are three key regions involved in the social cognitive deficits seen in schizophrenic patients: the medial prefrontal cortex (MPFC), the amygdala, and the inferior parietal lobe (IPL). Although experiments have proven that many other regions are highly involved in social cognition (the superior temporal sulcus, the temporal poles, IPL, etc.), the importance of the three regions mentioned is based on their well documented participation in the three fundamental aspects of the social deficits seen in schizophrenia: impairment in ToM, perception of emotions, and action monitoring (Brunet-Gouet[27]).

The MPFC's importance stems from the fact that it is one of the most common findings in neuroimaging studies of ToM. It occupies a large section of the frontal lobe and includes many different cortical areas—the anterior cingulate cortex, the paracingulate gyrus, the dorsal MPFC, the ventromedial cortex—that are also involved, in many cases, in social cognition. The "emotional" portion of the medial prefrontal region is activated in almost 90% of the studies on ToM (Steele and Lawrie[28]). Gallagher and Frith[29] determined that the MPFC—specifically, the anterior paracingulate cortex—is the key region for ToM and proposed the "separation mechanism" (initially postulated by Leslie[30]) responsible for distinguishing the mental state itself from reality. Brunet-Gouet[27] established that Theory of Mind tasks often involve co-activations in the MPFC and temporal structures, such as the temporal pole and the superior temporal convolution. This indicates not only that the MPFC mediates reasoning about mental states but also that its function should be understood in terms of interaction between high-level cognition and social and emotional processing. Each subregion of the MPFC the ToM selects may also be involved in maintaining a global contextual representation of one's own mental representations as well as those of others. Some authors propose that ToM is the result of an interaction between domain-general competencies and mechanisms at a more basic level representing social information (Stone and Gerrans[31]).

Abnormalities have also been shown in hemodynamic activation in the MPFC during tasks involved in ToM processes in schizophrenic patients (Brunet-Gouet,[27] Hempel et al[32]). These results suggest that schizophrenic patients fail in processing social information when contextual process, separation mechanism, or response inhibition is required.

Regarding the amygdala, there is no longer any doubt of its involvement in emotional experience and emotional perception. It has been suggested that the amygdala could play a role in ToM development and function (Baron-Cohen;[33] Fine et al[34]). The amygdala is a key component in the emotional process and in "on-line" perception (i.e, during the performance of social content tasks and mentalization) of the basic emotions of others (Blair and Cipolotti[35]), especially when these emotions are negative, as is the case with fear, sadness, disgust, and anger (Adolphs,[36] Calder et al[37]). Its role in epistemic mental states (without affective content) is more controversial. Foci of activation in the amygdala during ToM tasks and the recognition of emotions are found to be scattered, and some of them extend to the hippocampus and temporal pole regions. Thus, the perception of explicit information about other people's emotional states also involves the amygdala. Numerous studies have described the importance of prefrontal modulation of amygdala activity and have shown a strong, top-down influence (Ochsner et al[38,39]). It has been suggested that, although the amygdala could play a role in proper development of ToM, it may not be involved in mentalization per se (Gallagher and Fritz[40]). Other authors point out, however, that schizophrenic patients show deficits in attributing both epistemic mental states and affective mental states and that this is reflected by abnormal prefrontal and amygdala activity (Brunet-Gouet[27]). Therefore, the abnormal prefrontal activity described in schizophrenic patients during social cognition would result from a dysfunction in anterior prefrontal cortex modulation of the amygdala.

One of the first neuroimaging findings on the amygdala's function in face recognition was that of Schneider et al,[41] who demonstrated a hypoactivation of the amygdala when the emotion of sadness was evaluated through viewing photographs in which this emotion was expressed. Those findings were confirmed by Gur et al,[42] who studied this model in depth and demonstrated that left amygdala hypoactivation was specific in emotional discrimination tasks. Takahashi et al[43] and Paradiso et al[44] replicated this relative hypoactivation. However, Kosaka et al[45] and Taylor et al[46] obtained different results. Thus, the findings are dissimilar and include hypoactivation, hyperactivation, and normoactivation, which could denote a dysfunction yet to be determined. Conversely, fear is the expression that produces more intense activation in the amygdala. Studies conducted on the recognition of facial emotions in patients with lesions of the amygdala showed a specific deficit in the identification of fear. However, subsequent studies confirmed that the deficit extended to all negative emotions.

Some studies, in turn, have theorized that the degree to which the amygdala is activated may be related to the allele of the serotonin transporter gene, and short allele carriers of the gene have been associated with amygdala hyperactivation during these tasks.[47] Other studies, such as Salgado-Pineda et al,[48] associate amygdala activation with certain brain neurotransmitters, indicating the key role dopamine plays in amygdala functioning. Other authors have suggested gamma-aminobutyric acid (GABA) as a neurotransmitter that reduces amygdala activity.[49] It is also of interest to point out the existence of connections between GABA projections from the basolateral amygdala over the cingulate and their regulation with the dopaminergic projections coming from the prefrontal cortex. It has been suggested that an imbalance in this network of connections could lead to a hyperactivation of the anterior cingulate.

There are studies of the IPL that have demonstrated the involvement of the right IPL, the posterior cingulate,

and the right frontopolar cortex, as well as other regions, in the shared representations and specific systems for distinguishing between the self and others. This type of monitoring system keeps us from being contaminated by others' thoughts and feelings (Decety and Jackson[50]). Other research has shown that an individual's consciousness of his/her execution of a particular action involves the right IPL. Schizophrenic patients fail when it comes to activating certain components of motor systems involved in these voluntary actions. They also have impaired control over their own actions and over their ability to make self-other distinctions. The hypothesis is that schizophrenic delusions of control appear when deficits in action monitoring are combined with abnormal representations of one's own mental state and that of others (Brunet-Gouet[27]).

Emotion recognition and processing

The brain structures involved in emotion recognition are, primarily, the temporo-occipital cortex, especially the fusiform gyrus, the orbito-frontal and right parietal areas, the amygdala, and the basal ganglia, among others. According to Adolphs,[51] recognition of facial emotions is accomplished via three complementary strategies: perception, analysis, and identification. These consist of activating areas of the visual and motor cortex, which would have the effect of internally representing the observed postures and generating the emotional state corresponding to the one observed. Various studies have found performance in terms of the ability to recognize facial expressions to be inferior to that of the general population, even though the defect has also been found in other diseases. In turn, a connection between the clinical state and the ability to recognize facial emotions has been postulated. Stabilized patients would perform better than those in acute stages. However, difficulty recognizing facial emotions would, in turn, be a permanent feature of schizophrenia—present from the clinical onset of the disease and observable in first degree relatives. It has been suggested that people with schizophrenia would perceive faces as a sum of parts rather than in an integrative manner and that the deficit would be found in this configurational analysis. However, with regard to the fragmented examination of faces, the patients' performance would be similar to that of the general population. These data suggest that the problem would be found at higher levels of processing and integration—in areas of association. Another explanation could be that there are initial errors in the facial features analysis phase. Numerous studies have demonstrated that patients have more difficulty recognizing negative emotions, such as anger and fear, and would erroneously interpret neutral expressions. In terms of how face recognition relates to cognitive functions, a positive association with intelligence quotient, attention, and resistance to interference is appreciated, independent of working memory, fluency, and verbal learning.[52] These data would support the results of Langdon et al[52] who, using picture stories, found a subgroup of patients with a specific impairment for ToM tasks independent of an executive planning deficit, and they confirm face recognition as a parameter that is differentiated with respect to some areas of cognition. Then again, patients who underwent identification of numerous slides made no errors, which supports the hypothesis of an impairment that exists in social cognition areas, specifically. As far as findings on social cognition with respect to gender, Weiss et al[53] discovered a significant difference between the sexes in recognizing neutral expressions. Females erroneously interpreted neutral faces as sad more often than males. In comparison with the females, the males erroneously designated neutral faces as angry.

Regarding the connection between emotion recognition and psychopathology, the results are contradictory. Green and Nuechterlein[54] maintain that there is a strong correlation between negative symptoms, neurocognitive deficit, and visual processing.

A greater complexity is seen in the processing of emotions, where amygdala and MPFC circuits play a key role in processing the emotion of fear, but this circuit, in turn, is modulated by the autonomic activation system that generates feedback between the internal representation of the emotional stimulus and the somatic state. In fact, increased cutaneous reactivity, as a marker of autonomic activity, was correlated with a higher level of persecution and suspicion when fear and anger were encountered. In schizophrenia, the diminished response to fear in the amygdala-MPFC circuit is associated with excessive autonomic activation, especially in patients with paranoid symptomatology. In turn, it has been determined that there are disconnections between autonomic activation and the insula/basal ganglia-MPFC circuit for the emotion of displeasure, and the anterior cingulate cortex-MPFC for the emotion of fear.

Relation between social cognition and basic neurocognition

One of the major and most controversial issues is the possibility that there is a differentiation between social cognition and basic cognition as independent parameters. Various studies favour this distinction,[55-59] and to make it even more consistent, a difference has been seen between schizoaffective patients and schizophrenic patients in the social cognition domain with no changes in the basic cognition domain.[60] Meanwhile, other studies have found that deficits in sustained attention and in ToM are a limiting factor in social cognition, for these would have an impact on functioning at the social level (Bae et al[60]). In this regard, neurocognitive improvement and improved social cognition would mean better psychosocial functioning in schizophrenics and would have implications for treatment in rehabilitation (Brekke et al[61]). On the other hand, there are studies that associate social cognition with changes in attention and information processing. Some authors maintain that attention should not be considered a cognitive function, in essence, but rather a "directional activity" that would facilitate the development of cognitive processes. As an etiopathogenic and evolutionary

model of schizophrenia, Cornblatt and Keilp[62] propose the existence of an attention deficit beginning in infancy that would have an impact on the information processing associated with interpersonal communication. This model would account for the avoidance features seen in some patients. They also postulate that attention disorders would constitute a feature of inheritable disease that is specific and stable over time, regardless of fluctuations in the clinical picture. Alternatively, Calev[63] highlights the difficulty patients with schizophrenia have in selecting the important stimuli, filtering the irrelevant stimuli, and subsequently processing the information to categorize it. Similarly, memory and learning disorders would be related to memory encoding and retrieval disorders. Hemsley[64] picks up this idea, originally proposed by Berze, and suggests that the basic disorder in schizophrenia would be a problem with integrating the current perception with the stored mnestic material.

Visual perception begins, in part, with attention. Subsequently, visual memory will be required to evaluate the affective content of the facial expression, representing a point in common between the primary affective link and cognitive development of the visual process. This connection between social and non-social cognition would be found in the working memory located in the prefrontal lobe, among other areas.[65] The lack of visual attention or limitations in motor execution could be the reason for deficient functioning in the correct reading of facial expressions. Shimizu et al[66] studied eye movements during exposure to different facial expressions. According to their results, patients with schizophrenia analysed facial expressions at shorter intervals than the control groups. They also observed that the antisaccade movements had a longer latency period. Schawrtz and Evans[67] confirmed that, compared to the control group, patients with schizophrenia required a higher level of voluntary control to filter distracting stimuli and showed a reduced ability to filter irrelevant information. These patients would also be impaired in the management of attention-centring. Adolphs et al[68] have observed that patients with bilateral lesions of the amygdala do not recognize fear because they do not look at the eyes during face recognition tests, and this causes a dysfunction in the information gathering system. They suggest that the amygdala does not recognize because it does not primarily search. These results would be related to the altered face-scanning pattern seen in schizophrenics. As for the location of the visual association regions responsible for face recognition, studies of prosopagnosia place them in the occipito-parietal cortex. In research along this line, Lee et al[69] identified a reduction in the volume of the left fusiform temporal gyrus (the area associated with face recognition) in patients having their first psychotic episode. In contrast, Corcoran et al[70] propose an explanatory model that incorporates the patient's ability to remember autobiographical events, maintaining that the interference of others' mental states usually entails the use of previous social interaction experiences. In this context, various models have emerged that attempt to explain the interconnection between neurocognition, social cognition, and social functioning. There is the Vatu, Rüsch, Wirtz, and Corrigan model (2004) showing results that support social cognition as a possible mediator between basic neurocognition and social functioning. On the other hand, the Brekke, Kay, Lee, and Green model (2005) uses social functioning in a global sense as well as functioning in any of its specific aspects—neurocognition, social cognition, social competence, and social support—as predictors of the course of the disease. Finally, the Green and Nuechterlein model (1999) is a complex model that separates out the subcomponents of basic neurocognition, social cognition, and social functioning.

Brain lateralization

Brain lateralization has been proposed as another explanation for the difficulty schizophrenics have with face recognition. Studies conducted in patients with unilateral brain hemisphere damage showed that the non-dominant hemisphere predominated in recognizing faces from visual stimuli. Right-handed patients with unilateral right brain lesion show a marked disability in evaluating facial expressions, though they retain the ability to evaluate emotions expressed through non-facial (bodily) and non-visual (auditory) signals. The prevailing hypothesis asserts that the non-dominant hemisphere is the one primarily responsible for recognizing the emotional aspects of a stimulus. However, both hemispheres process stimuli related to emotion: in right-handed people, the right hemisphere predominantly for perception of negative affects and the left for perception of positive affects. Therefore, in right-handed patients with schizophrenia, errors in decoding facial expressions of emotion would result from a unilateral right hemisphere lesion. Brain lateralization would also account for the speech difficulties.

Evaluation of social cognition

At the present time, there are no instruments available for an overall assessment of social cognition, whereas there is a great variety of instruments that focus on specific aspects of this construct. Among these evaluation instruments are numerous tests that are difficult to use in clinical practice, in most instances, because of how much time the test takes. The majority of them also lack validation and are inadequate because they evaluate mechanisms that are as yet undetermined and because of the obvious difficulty of representing real situations. They may be categorized according to the area they evaluate. We list the evaluation instruments below as well as some empirical studies that attempt to measure ToM (tables 1 and 2).

Cognitive rehabilitation programs

Due to the increased emphasis on social cognition in research, rehabilitation programs have been developed that target the recovery of these cognitive aspects. These programs may be categorized as one of two types: 1) interventions focused on improving emotion

Table 1 Evaluation instruments

1. *Instruments for evaluating face recognition*
   Pictures of facial affect (FEDT) (Ekman, 1976)
   Face Emotion Discrimination Test (FEDT) (Kerr and Neale, 1993). Test designed by Baron-Cohen
   Face Emotion Identification Test (FEIT) (Kerr and Neale, 1993)

2. *Social cognition*
   Task (IPT) created by Archer and Constanzo in 1998
   Mayer-Salovery-Caruso Emotional Intelligence Test (MSCEIT) (Mayer, Salovey and Caruso, 2002) Geopte

3. *Social perception*
   The Schema Compression Sequencing Test - Revised (SCRT-R) (Corrigan and Addis, 1995)
   Situational Feature Recognition Test (SFRT) (Corrigan and Green, 1993)
   Videotape Affect Perception Test (Bellack, Blanchard, and Mueser, 1996)

4. *Theory of Mind*
   The story of Sally and Anne (Wimmer and Perner, 1983) and the story of Cigarettes (Happé, 1994), the story of the ice cream van store (Baron-Cohen, 1989) and the story of the Burglar Store (Happé and Frith, 1994)
   The Hinting Task (Corcoran, Mercer, and Frith, 1995)

Table 2 The most significant empirical studies, methodology and results

Corcoran et al, 1995.[71] Tests of insinuation, deduction of intentions using the indirect style. IQ not controlled. They find ToM deficit in patients with negative symptoms, disorders of thought, or delusional ideas of persecution

Frith et al, 1996.[72] First and second order vignettes. IQ and psychoactive drug therapy controlled. Most severe ToM impairment in patients with negative symptoms or disorders of thought. ToM deficit: state variable

Corcoran et al, 1997.[73] Humorous vignettes. Greatest impairment in patients with negative symptoms or disorders of thought. No impairment in asymptomatic individuals

Drury et al, 1998.[26] Second order false belief test and metaphor test to evaluate first order beliefs. IQ controlled. Impairment on test performance in the most seriously ill patients and those with more intense symptoms. Variable of state

Pickup et al, 2001.[74] First and second order false belief tasks. IQ controlled. Schizophrenics with delusions have ToM impairment but lower IQ. Schizophrenics with impaired behaviour have more difficulty performing ToM tasks but less than in autism

Pollice et al,[75] 2002. First and second order false belief tasks. Measurement of social function via AD and DAS. IQ controlled. ToM tasks may be of use in developing programs to reduce social impairment

ToM: Theory of Mind.

recognition, such as Training of Affect Recognition in which patients learn to identify and differentiate the key facial expressions of the six basic emotions, and 2) more complex intervention programs, such as Emotion Management Training. Objectives for this program include improvement in emotional perception deficits and in the patient's poor social adjustment, as well as instruction in effective confrontation strategies. Alternatively, Integrated Psychological Therapy is a comprehensive program for improving both cognitive functioning and social functioning. It encompasses five programs: cognitive differentiation, social perception, verbal communication, social skills, and interpersonal problem solving. The last of the programs that have emerged in this sphere is Social Cognition and Interaction Training. This program also divides intervention into two phases: comprehension of emotions and social cognition style. The ultimate objective of integration is to consolidate skills and generalize them to daily living.

Conclusions

Since the 1990s, there has been emphasis on the need to develop a cognitive model that would provide a general

theory of schizophrenia, be consistent with the diversity of symptoms, and validate the use of a number of convergent technologies to assess symptoms in human beings. In 2003, social cognition was accepted as one of the seven crucial cognitive domains for improving cognition in schizophrenia. In short, social cognition is understood as "the ability to perceive others' emotions, infer what they are thinking, understand and interpret their intentions, and the norms that govern social interaction," even though a precise and operative definition of the term is still needed. Social cognition is a cognitive domain to bear in mind in schizophrenia, for it has significant implications for prognosis and treatment in rehabilitation. There are various controversies surrounding the changes in social cognition seen in schizophrenia. Whether it is an impaired neurodevelopmental process (as is proposed in autism) or a neurodegenerative process is still subject to debate. Then again, in the studies reviewed, there are contradictory results as to whether the ToM alterations observed are given features of the disease, still present in remission of the psychotic symptoms, or whether they are a factor of state, depending on the course of the disease. Moreover, no one has managed to define whether social cognition is a domain that is independent of basic cognition or is influenced by it. These controversies would serve as a substrate for future research.

There are key neuroanatomical regions significantly involved in social cognitive processes: the MPFC, the amygdala, and the IPL. The previous hypothetical concept of the existence of a specific module responsible for social cognition would give way to other perspectives in which different areas of the brain are involved in carrying out the process of social information. It has also been proposed that impairment in the different social cognition functions and areas of the brain involved may have a significant impact not only on the individual's social functioning but also in the development of some psychotic symptoms, such as delusions, changes in behaviour and speech, and anomalous experiences seen in schizophrenia. It would also be interesting to clarify whether, from a neurocognitive standpoint, there are differences in the various schizophrenia subtypes.

Given the complexity of this field of study, there are currently no properly validated evaluation instruments that integrate all aspects—or at least the most important ones—for a global assessment of basic social cognition processes. The hypothetical models of ToM are theoretical constructs pending scientific validation. In this regard, it would be important to develop validated tools with a view to research. Future research could move toward acknowledgement of the importance of social cognition as a prognostic factor in schizophrenia and toward finding the connection between social cognition, basic neurocognition, and the patient's psychosocial development over the course of the disease.

The Kraepelinian standard, under which cognitive impairment was thought to be a core symptom of schizophrenia, and the Bleuler vision, in which special emphasis was placed on impairment in the affective sphere, have been revived in recent years, and interesting approximations have been made to the aetiology and understanding of schizophrenia.

Conflict of interest

The authors declare that they have no conflict of interest.

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