

# Social Cognition and Its Neural Correlates in Schizophrenia and Autism

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## FOCUS POINTS

- Patients with schizophrenia frequently show impaired performance in social cognition tasks utilising emotion perception and theory of mind skills. Whether these deficits are specific to the domain of social cognition or are part of more generalized impairments is not yet clear.
- Impairments in a variety of emotion perception tasks are consistently observed in those with autism. Deficits in theory of mind skills however, appear to apply to perhaps only the most severely affected individuals.
- In both schizophrenia and autism, the fusiform gyrus and amygdala are consistently implicated during emotion perception tasks. Theory of mind tasks appear to involve the frontal cortex, in particular the medial frontal and medial prefrontal regions.

## ABSTRACT

The study of social cognition in psychiatric disorders has become increasingly popular in recent years. This is due to its proposed link to social functioning and the inability of general neurocognitive skills to explain the spectrum of impairments observed in patients. This article reviews research into two of the processes thought to underlie social cognition (emotion perception and theory of mind) in schizophrenia and autism. This is followed by a look at neuroimaging studies and their efforts to localize the neural correlates of emotion perception and theory of mind in the two disorders. We concluded that while a specific impairment in emotion perception and theory of mind skills cannot be generalized to all individuals with autism and schizophrenia, there are subpopulations that have lingering deficits of social cognition tasks. Neuroimaging work consistently points to the involvement of the fusiform

gyrus and amygdala in emotion processing, while the medial prefrontal and frontal cortex are implicated in tasks invoking theory of mind. We propose that deficits of social cognition may benefit from cognitive remediation therapy and pharmacological cognitive enhancers.

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## INTRODUCTION

Impairment in social functioning is the hallmark of most psychiatric disorders and is found in the defining diagnostic criteria for most conditions (eg, *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [DSM-IV]*<sup>1</sup>). In recent years, there has been a surge of interest in attempting to clarify the processes that may underlie social dysfunction. This has led to the concept of social cognition, considered to be directly related to social functioning skills and the search for its neural correlates. In this review, we will concentrate on two of the most studied processes considered to be involved in social cognition (emotion perception and theory of mind), their functional significance, and neural correlates in schizophrenia and autism.

## Definitions of Social Cognition

Social cognition has been defined as “the way in which people make sense of other people and themselves”<sup>2</sup> and “the ability to construct representations of the relation between oneself and others and to use those representations flexibly to guide social behavior.”<sup>3</sup> For the purposes of this article, social cognition will be viewed as an innate, internal ability to perceive and understand social information in order to attain goals that can be classified under “successful” social function, such as the ability to initiate and maintain rewarding interpersonal relationships.

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### **Why Study Social Cognition?**

Social cognition has been linked to eventual functional outcome. For example, there is evidence that, in schizophrenia, impairments in social functioning are linked to the rate of relapse.<sup>4</sup> Despite findings that social functioning is linked to neurocognitive abilities,<sup>5</sup> this alone is unable to explain the observed deficits.<sup>6</sup> Recent literature points to the idea of social cognition being an independent module of intelligence separate from generalized cognitive abilities.<sup>7,8</sup> This paves the way for the suggestion that it is an impairment in the domain of social cognition that may best explain social dysfunction. More importantly, if the neural basis of social cognition can be pinpointed, then attempts at remedying its deficits can begin.

### **Emotion Processing and Theory of Mind Skills: Aspects of Social Cognition**

It has been postulated, that the ability to correctly identify the emotional state of an individual from social cues, such as facial expressions, is a crucial aspect of successful interpersonal communication and hence social competence.<sup>9</sup>

Emotion perception has mainly been evaluated through studies investigating subject's ability to correctly identify and discriminate facial or vocal emotions, relative to their performance on a non-affective control task, such as identity, gender, or object recognition. A non-affective vocal task may involve pitch or tone identification or the ability to match a sound to an object.

The theory of mind hypothesis, first coined by Premack and Woodruff,<sup>10</sup> suggests that in order to predict and explain the behavior of others, we need the innate ability of attributing mental states to others. Impairments in theory of mind skills have been one of the most researched theoretical accounts for social dysfunction to date.

As well as involving representations of others' mind and beliefs, theory of mind also involves the ability to understand that it is possible for an individual to hold false beliefs.<sup>11</sup> First-order and second-order false-belief tasks have been commonly used in the investigation of theory of mind skills in psychiatric disorders. First-order false-belief tasks assess the ability of subjects to understand that a story character holds a false belief about the world. Second-order false-belief tasks, investigate the ability of an individual to appreciate scenarios where one story character holds a false belief about the beliefs of another character.<sup>12</sup>

### **Social Function in Schizophrenia**

Since the introduction of the term schizophrenia by Eugen Bleuler in 1911,<sup>13</sup> attempts at classifica-

tion have been a matter of heated debate due to the heterogeneity of the symptom profiles observed in those diagnosed and the overlap of symptoms with other disorders. Following the suggestion of Crow,<sup>14</sup> however, the majority of modern literature agrees that schizophrenia encompasses positive and negative symptoms. Positive symptoms include hallucinations, delusions, and thought disorder. Negative symptoms include flat affect, poverty of speech, and loss of volition.

In addition to the foregoing symptoms, one of the most universally agreed upon characteristics of those with schizophrenia is an impairment of their social functioning. Compromised social relationships, social isolation, and withdrawal have been observed throughout the whole of the schizophrenia disease course, from the premorbid stage,<sup>15</sup> through to first episode,<sup>16</sup> during subsequent relapses,<sup>17</sup> to presentation as a deficit state during remitted periods,<sup>18</sup> and have been shown to be present despite treatment with antipsychotics.<sup>17</sup> This is echoed by the presence of social dysfunction as part of the diagnostic criteria in the *DSM-IV*.<sup>1</sup> The presence of reported premorbid social function deficits in schizophrenia patients,<sup>15</sup> suggests that their social dysfunction may not simply be a consequence of the often-accompanying hospitalization and social isolation. Hence, in recent years, there have been attempts to explain this social impairment in terms of a specific deficit in social cognition skills.

### **Emotion Processing in Schizophrenia**

Impairments in emotion processing using facial and vocal stimuli have been shown to be present in patients with schizophrenia. Most studies have found that compared with controls, the performance of schizophrenia patients is impaired in both affective and non-affective facial<sup>19,21</sup> and vocal<sup>19</sup> perception tasks and that these impairments are not related to age, gender, or medication status and dose.<sup>21-23</sup> There is also evidence of a specific impairment in processing of facial<sup>24</sup> and vocal<sup>25</sup> emotional stimuli, while others have found no task differences between patients and controls at all.<sup>26</sup>

The inconsistency of the aforementioned findings could be due to large variation in symptom profiles typically observed between those with the diagnosis of schizophrenia. Therefore, attempts have been made to relate emotion perception deficits with specific symptoms. Impairments in affect perception have been associated with specific positive symptoms, such as thought disorder<sup>20</sup> and bizarre behavior,<sup>22</sup> negative symptoms, such as alogia,<sup>20</sup> and overall positive<sup>27</sup> and negative symptoms.<sup>22,27</sup> Others, however, have failed

to find any relationship between affect perception deficits and symptomatology.<sup>21</sup>

There also seems to be an interaction between affect perception and the type of emotion, with the consistent finding that patients with schizophrenia have particular problems with negative affects,<sup>24,28</sup> especially fear<sup>29</sup> and sadness.<sup>26</sup>

While most evidence points to impairments in affect perception being a stable deficit present across all phases of illness,<sup>30,31</sup> it is also the case that some schizophrenia patients in remission and later stages of the acute phase have been found to perform better on facial affect recognition tasks than those in the acute stage.<sup>32,33</sup> Finally, there have been suggestions that facial emotion recognition is positively correlated with intelligence.<sup>22</sup>

### **Theory of Mind in Schizophrenia**

Patients with schizophrenia have shown impaired performance on a variety of theory of mind tasks relative to psychiatric and normal controls. Among others, impairments have been shown in tasks requiring the ability to infer intention behind indirect speech,<sup>34</sup> comprehension of irony,<sup>35</sup> first- and second-order false-belief tasks<sup>12,36,37</sup> and second-order false-belief tasks alone.<sup>38</sup>

Evidence with regards to whether impairment in theory of mind is a specific deficit or is part of generalized cognitive abilities is ambiguous. Some studies<sup>12,36,39</sup> have found differences in theory of mind performance to be present when intelligence quotients (IQ) is covaried out suggesting that theory of mind impairments are specific, rather than related to general cognitive abilities. Others,<sup>38,40</sup> however have shown links between IQ and performance in theory of mind tasks, which could suggest impairment in theory of mind tasks to be indicative of a deficit in more generalized cognitive abilities.

Similar to the face processing literature, studies show that the ability of schizophrenia patients to accurately attribute mental states to others can be linked to specific symptoms. Theory of mind has been linked to behavioral symptoms (positive and negative) and paranoid symptoms,<sup>34,36,38,39</sup> to negative symptoms only,<sup>12,36</sup> and to thought and speech disorganization.<sup>41</sup> In addition, Using Liddle's<sup>42</sup> three-dimensional model, which differentiates between schizophrenia patients on the basis of psychomotor poverty, reality distortion, and disorganization, Mazza and colleagues<sup>12</sup> showed task-specific variations on theory of mind performance between the three schizophrenia subgroups. Schizophrenia patients with psychomotor retardation performed worse than the other two on

first-order false-belief tasks. While the psychomotor retardation group had the worst performance on one of the second-order false-belief tasks, reality distortion and disorganization groups had significantly worse performance on the longer and more complex second-order false-belief task.

Research into the relationship between illness phase and theory of mind so far, mainly suggests that impairments in theory of mind are state dependent.<sup>34,37,43</sup> Langdon and Coltheart,<sup>44</sup> however, have suggested that there is evidence for theory of mind deficits to be viewed as a trait characteristic.

### **Functional Significance of Emotion Perception and Theory of Mind Deficits in Schizophrenia**

There is evidence that emotion perception as measured by face perception is associated with impairments in social function in both acutely ill and remitted schizophrenia patients.

Both vocal and facial affect recognition in schizophrenia outpatients has been linked to interpersonal relationships, as measured by the Quality of Life scale,<sup>45</sup> and to social function, as measured by the Social Dysfunction Index.<sup>46</sup> Mueser and colleagues<sup>47</sup> found that overall face perception, both affective and non-affective, was associated with social competence measured by the Conversation Probe and the Social Behavior Schedule<sup>47</sup> in a group of chronic schizophrenia inpatients. Specifically, they found a correlation between overall face perception, both affective and non-affective, and social functioning indicated by the Social Behavior Schedule, while only facial recognition impairments were associated with the conversation probe. The researchers suggested that this finding could be explained by the fact that the Social Behavior Schedule looked at social function in a variety of settings, while the conversation probe only looked at social function over the duration of two 3-minute structured interviews.

Finally, Penn and colleagues<sup>48</sup> showed that facial affect recognition deficits were associated with reduced social competence, social interest, and hygiene in a group of inpatients even after controlling for other cognitive abilities. Contrary to the aforementioned, Ihnen and colleagues<sup>49</sup> found only a weak association between facial affect recognition and social skills.

Due to a lack of work to date on the relationship of impairments in theory of mind with social competence, there is only indirect evidence of a possible link for a functional significance between the two. It has been argued that appropriate use of conversational rules, leading onto successful communication, is ultimately linked to an intact theory of mind.<sup>50</sup> Therefore, stud-

ies showing that schizophrenia patients have impairments in their conversational skills could be taken as indirect evidence for a functional significance between theory of mind and schizophrenia.<sup>51,52</sup>

Cutting and Murphy<sup>53</sup> suggested that at least a subgroup of schizophrenia patients may have a social naïveté related to their impairment in a social knowledge task relative to a non-social task. The social task consisted of participants being presented with multiple-choice questions asking how people would behave in various social situations. It could be said that success in this task would necessitate intact theory of mind skills.

### **Social Function in Autism**

In 1943, Kanner<sup>54</sup> suggested that autistic children “have come into the world with innate ability to form the usual, biologically provided affective contact with people.” Childhood autism is characterized by a profound lack of interest in the social world, accompanied by impairments in verbal and non-verbal communication.<sup>55</sup> As such, social dysfunction is included as part of the diagnostic criteria<sup>1</sup> of autism. Other symptoms include mental retardation, specific islets of ability, lack of pretend play, and insistence on sameness.<sup>55</sup> Although some symptoms of autism may be attributable to below average IQ levels,<sup>56</sup> social dysfunction seems to be a specific impairment, independent of generalized cognitive abilities.<sup>57</sup> A more detailed analysis of the nature of this specific dysfunction is therefore warranted.

### **Emotion Perception in Autism**

It is widely accepted that individuals with autism have problems in processing facial displays of emotion, and have been described as being “face inexperienced.”<sup>58</sup> Children with autism have shown impairments in their ability to match photographs and drawings of facial emotions,<sup>59</sup> matching drawings of postures to facial expressions on video tape,<sup>60</sup> labeling and identifying emotions using photographs,<sup>61</sup> and in their ability to sort photographs of faces on the basis of emotional expression.<sup>62</sup> While the aforementioned studies have found differential facial emotion perception deficits relative to intact performance in tasks employing objects, there is also evidence for a more generalized deficit in overall face perception.<sup>61,63</sup>

### **Theory of Mind in Autism**

Impairment in theory of mind abilities has been one of the most investigated theories seeking to explain the social dysfunction observed in persons with autism. Children with autism have been found

to be impaired on various versions of false-belief tasks.<sup>55,64,65</sup> It is also the case that some children with autism pass first-order false-belief tasks.<sup>55,66</sup> This effect is reduced when using second-order false-belief tasks. For example, Baron-Cohen<sup>67</sup> found that out of children who successfully completed first-order tasks, none passed the second-order false-belief task, whereas 90% of the younger normal children and 60% of the mentally retarded children were successful. Contrary to such evidence, other studies<sup>68,69</sup> present high-functioning individuals with autism who can also pass second-order false-belief tasks. It must be remembered that while some individuals with autism may be successful at theory of mind tasks, they are still, by the definition of their diagnosis, significantly impaired in their social functioning.<sup>55</sup> From the above findings, it would seem that perhaps there are limitations in the applications of theory of mind impairments to autism.<sup>68</sup> In support of such a limitation, a significant correlation has been observed between overall IQ, verbal IQ, and performance on both first- and second-order tasks.<sup>70</sup> Additionally, Buitelaar and colleagues<sup>71</sup> found that verbal memory, performance IQ, age, and gender were the best predictors of performance in theory of mind tasks in a group of children with autism. Finally, Dahlgren and Trillingsgaard<sup>68</sup> have presented a relationship between the ability to succeed at false-belief tasks and verbal intelligence in their sample of high-functioning subjects although this was non-significant. Concluding that the inability to ascribe false belief to others may be secondary to intellectual levels rather than a primary deficit in autism, they suggest that impairments in false-belief tasks are perhaps mainly present in the more severely affected children.

### **Functional Significance of Emotion Perception and Theory of Mind Deficits in Autism**

To our knowledge, there appears to be a lack of research designed specifically to address the issue of the functional significance of emotion perception and theory of mind in autism. However, studies have presented such a relationship with reference to pervasive developmental disorder (PDD). Since autism is often described as a PDD, we feel such findings may also be generalized to autism.

An inability to read facial emotions could lead to the lack of eye contact, the tendency to treat objects and human alike, and lack of physical contact. Specifically, Hobson<sup>59</sup> presented evidence that performance levels in an emotion task were related to everyday social functioning as measured by the Handicaps, Behavior and Skills Schedule.<sup>72</sup> Evidence

can also be drawn from finding that face and affect matching but not object matching was significantly correlated with social behavior and play in children with PDD.<sup>73</sup>

An impairment in the ability to appreciate that people have beliefs independent of each other and such beliefs may be false, would lead to immense difficulties in being able to interpret why people act as observed, hence, causing a chaotic and perhaps disturbing world.<sup>74</sup> Impairment in theory of mind skills, therefore, could be used to explain some of the often-observed symptoms in those with autism, such as the insistence on sameness and routine<sup>73</sup> and lack of pretend play.<sup>55</sup> Fein and colleagues<sup>75</sup> found a significant impairment in the ability to match a social context with its appropriate emotion in a group of children with PPD to be associated with decreased social skills levels.

### **NEURAL BASIS OF SOCIAL COGNITION**

A network of neural regions consisting of the orbitofrontal cortex, the superior temporal sulcus, and the amygdala has been proposed by Brothers<sup>76</sup> to underlie social cognition, comprising the “Social Brain.”<sup>77</sup>

Among the areas consistently linked to facial and emotional perception are: the lateral fusiform gyrus,<sup>78,79</sup> the superior temporal sulcus,<sup>80</sup> and the amygdala.<sup>81,82</sup> Imaging studies have found activation of particular frontal cortical regions during theory of mind tasks, specifically the medial frontal cortex,<sup>83,84</sup> the medial prefrontal cortex,<sup>85,86</sup> and the orbitofrontal cortex.<sup>87</sup>

### **Neuroimaging Studies of Emotion Perception in Schizophrenia**

Reductions in the left fusiform gyrus volume in patients with schizophrenia have been demonstrated relative to healthy controls.<sup>88,89</sup> Recently, Lee and colleagues<sup>90</sup> also found a bilateral reduction in the fusiform gyrus of a group of first episode patients with schizophrenia in comparison with healthy persons suggesting that neural abnormalities are present at the early stages of the illness. An functional magnetic resonance imaging (fMRI) study also found a reduction in blood flow in the right lateral fusiform gyrus during both facial identity and emotional face-processing tasks compared with healthy controls.<sup>91</sup>

There also seems to be a reduction in the size of the amygdala associated with schizophrenia. A meta-analysis<sup>92</sup> has shown that individuals with schizophrenia have an amygdala, on average 94% of the volume of that of controls. This is paralleled by studies presenting abnormal amygdalar activation in schizophrenia patients during affect perception tasks.<sup>93-95</sup>

Using an emotional intensity judgement task where subjects had to discriminate between emotive (both positive and negative) and neutral faces, Kosaka and colleagues<sup>93</sup> found a general increase in amygdalar activations of the schizophrenia group compared with the controls, relative to no differences in task accuracy between the two groups. However, this increase was non-significant in the negative discrimination task. The authors suggested that this increased activation in the amygdala of schizophrenia patients could reflect an impairment in the gating of emotive sensory input. Using a similar task, Gur and colleagues<sup>94</sup> found that patients had a significant reduction in cerebral blood flow to the left amygdala and bilateral hippocampus relative to controls, again in the context of no difference in accuracy between the two groups. It was postulated that, while such a reduction in limbic activation didn't impair the patient's performance, it could do so in a more complex task. Furthermore, a significant decrease in the bilateral activation of the amygdala-hippocampal complex has also been observed during a facial affect labeling task.<sup>95</sup>

### **Neuroimaging Studies of Theory of Mind in Schizophrenia**

Using fMRI, Russell and colleagues<sup>96</sup> found that the impaired ability of schizophrenia patients in a mental attribution task requiring the inference of emotional states from eyes alone, was correlated with left medial frontal cortex underactivation.

More recently, a positron emission tomography (PET) scan<sup>97</sup> revealed that while healthy controls had significant activation of the right prefrontal cortex during a task involving non-verbal attribution of intention, patients with schizophrenia were lacking in such activity. Such results are in line with other structural and functional studies<sup>98,99</sup> indicating frontal dysfunction in schizophrenia patients during non-theory of mind tasks.

### **Neuroimaging studies of Emotion Perception in Autism**

Using PET scanning, Halland and colleagues<sup>100</sup> have demonstrated lower regional blood flow in the fusiform cortex of a group of high functioning autistic men during an emotion recognition task where the emotive salience of the facial stimuli had been enhanced by adding matched prosodic voices. This study supports the findings—within the context of no difference in task performance to controls—of, first, Pierce and colleagues<sup>58</sup> who found a significant reduction of fusiform gyrus activation in a group of adults

with autism<sup>58</sup> and, secondly, Shultz and colleagues<sup>101</sup> who made a similar finding in a group of adults with autism and Asperger's syndrome.<sup>101</sup> (Asperger's syndrome is a milder<sup>102</sup> member of the autistic spectrum disorders.) Pierce and colleagues<sup>58</sup> also observed that while each control activated the fusiform "face area," those with autism activated individual specific areas (eg, frontal cortex, primary visual cortex). This was associated with a non-significant reduction in the volume of the fusiform gyrus.

Amygdalar abnormalities have also been reported in autism. Pierce and colleagues<sup>58</sup> found a significant reduction in activation in the left amygdala paralleled by a significant reduction in volume supporting previous findings.<sup>103,104</sup> Using fMRI, Ogai and colleagues<sup>105</sup> have also shown that high functioning persons with autism have different patterns of corticolimbic activity relative to controls in a task involving recognition of facial expressions of fear, disgust, and happiness.

### **Neuroimaging Studies of Theory of Mind in Autism**

In a PET study, Happé and colleagues<sup>102</sup> investigated a group of individuals with Asperger's syndrome using a task in which subjects were required to read stories with or without mental state elements. The pattern of activation between the two groups was found to be very similar, however, while the control group showed activation of Brodmann area (BA) 8/9 in the medial prefrontal cortex during the theory of mind task no such activity was found in the Asperger's syndrome group. Instead, they showed activation of the BA 9/10 that was not present in the control group. This finding was in the context of a significant impairment in task accuracy in the theory of mind task.

It is thought that BA 9 and BA 10 are involved in general problem-solving tasks and their involvement in theory of mind tasks in Asperger's syndrome may be due to such individuals using a more general non-face dedicated cognitive strategy to solve a social problem, whereas healthy controls may utilize a dedicated area of the BA 8 for social cognition.<sup>106</sup>

There is also evidence that the amygdala may be involved in theory of mind skills. Baron-Cohen and colleagues<sup>77,107</sup> have shown that in a task requiring inference of mental state from eyes alone, there was a lack of amygdalar activity in those with autism relative to controls. The authors suggested this observation provides evidence for the amygdala theory of autism. It was interesting that the identical task used by Russell and colleagues<sup>96</sup> did not demonstrate any difference in amygdala activity between patients with schizophrenia and controls

### **CONCLUSION**

This article aimed to provide an overview of the current research attempting to explain the social functioning problems observed in individuals with schizophrenia and autism in terms of impairments in their social cognition abilities. Social cognition was evaluated as a function of individuals' abilities to perceive social cues, such as facial expressions, affective prosody to a smaller extent, and mental state attribution to others. Attempts were made to link social cognition deficits to everyday social functioning, and finally the possible neural mechanisms underlying social cognition relative to the clinical groups in question were looked at.

Evidence seems to point to impairments in emotion perception abilities of schizophrenia patients but whether this is a specific problem with reading facial or vocal emotion or can be attributed to more generalized deficits remains undecided. Both affective and non-affective face processing seems to be sensitive to the heterogeneity of schizophrenia, patients seem to have a special difficulty with negative emotions and while most research points to face emotion processing as being a stable factor there is also evidence that performance improves with remission of symptoms.

The theory of mind data suggests that patients with schizophrenia are impaired in a variety of tasks requiring the ability to infer the mental states of others, but again, whether this is a specific deficit or is part of more generalized abilities, is ambiguous. Theory of mind skills also seem to be sensitive to the different symptom profiles of patients and are generally state dependent. Overall face processing, both affective and non-affective, vocal affect recognition, and theory of mind, seem to be related to social competence observed in schizophrenia patients.

Facial emotion perception deficits seem to be consistently observed in those with autism, but there is also some evidence that this may be part of more general face perception impairments. The theory of mind data, however, is less clear.

Individuals with autism show impairments in their theory of mind skills and while some can pass first order false-belief tasks, most fail second-order false-belief tasks. There are, however, still some high-functioning individuals that pass even the more difficult second-order false-belief tasks. Such findings suggest that an impairment in theory of mind skills cannot be generalized to all those with autism. Additionally, evidence that theory of mind skills, IQ, and verbal abilities are related suggests that theory of mind, may not be independent of generalized cognitive abilities. Finally, indirect evidence from children with PDD

suggests that emotion perception and theory of mind skills are linked to functional outcome.

A promising start is shown by convergence of what little evidence there is for the neural basis of facial emotion perception and theory of mind in the autism and schizophrenia populations. Impairments in facial emotion perception tasks in both autism and schizophrenia consistently point to abnormalities within the fusiform gyrus and amygdala, while theory of mind deficits implicate the frontal cortex, in particular, the medial frontal and medial prefrontal regions. This suggests that the social cognition deficits observed in these two groups may have the same biological basis. With deficits in these structures identified and the notion of neuronal plasticity, the hope that these regions may have increased activation following treatment with cognitive enhancers and cognitive remediation therapy, may not be so distant. Additionally, the finding that neural abnormalities may be present at the very early stage of illness (eg, in first episode schizophrenia patients<sup>89</sup>) highlights the need for early intervention strategies with the aim of halting further degeneration.

It is clear that social cognition deficits are observed in many individuals with autism and schizophrenia. On the other hand, the ability of some individuals to perform normally on emotion perception and theory of mind tasks, while still being significantly impaired in their social life, suggests that the theory of a specific deficit in social cognition skills cannot alone explain the social function deficits of all those with schizophrenia and autism and perhaps other factors such as intellectual capabilities, especially with regards to theory of mind abilities may be at play.

Methodological issues that plague this area of research may account for inconsistent results between studies. These include the issue of medication, heterogeneity of the samples, matched performance, unequal task demands between control and experimental conditions, the generally small sample sizes used and the large variation in types of tasks used.

We conclude that while the notion of social cognition is relatively new, and a lot more research is required, there may be subgroups of patients with schizophrenia and autism characterized by their specific deficit in social cognition. Such individuals would especially benefit from possible rehabilitation programs, both psychological and pharmacologic tailored to meet their social cognition deficits, thereby leading to a significant improvement in their everyday social functioning. **CNS**

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