Social Cognition in Schizophrenia

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The topic of social cognition has attracted considerable interest in schizophrenia over the last several years. This construct generally refers to the detection, processing, and utilization of social information and, within the field of schizophrenia, includes several skills such as recognizing emotion, understanding the thoughts and intentions of others, and interpreting social cues. Individuals with schizophrenia show significant impairments in social cognition, and these impairments are strongly related to functional outcome. Treating social cognition yields significant improvements in real-world outcomes, including social functioning and social skill. Importantly, social cognitive abilities are linked to specific neural circuits that have been shown to be abnormal in individuals with schizophrenia. Investigations of these neural networks in patients have also demonstrated that brain activation is significantly correlated with social functioning, which suggests that abnormal activation in social cognitive networks may serve as a mechanism for social dysfunction in schizophrenia. Among the many challenges in this area is the issue of measurement. There is disagreement about which tasks best measure social cognition and many existing measures show poor psychometric properties. A recent project, called the Social Cognition Psychometric Evaluation (SCOPE) study, aims to address these problems by providing the field with a well-validated battery of social cognitive tasks that can be used in treatment outcome trials. Research is honing in on the potential mechanisms of social cognitive impairment in patients, and with improved measurement, there is promise for optimizing behavioral and pharmacologic interventions and remediation strategies.

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Social cognition has attracted considerable interest in schizophrenia research over the last 2 decades and generally refers to how people detect, process, and utilize social information. The term originated within social psychology approximately 50 years ago but is now used broadly across disciplines, including psychiatry, clinical psychology, developmental psychology, and neuroscience. While the overall construct addresses how individuals think and behave in social contexts, its application and emphasis vary across disciplines. In schizophrenia, a recent National Institute of Mental Health workshop defined *social cognition* as "the mental operations that underlie social interactions, including perceiving, interpreting, and generating responses to the intentions, dispositions, and behaviors of others."[1] This definition highlights the important link between social cognition and social behavior and indicates that social cognition may be critical for understanding the social impairments that are among the defining features of schizophrenia.

The current review will outline the social cognitive domains and abilities that are considered to be of key importance in schizophrenia. Next, the focus will shift to why social cognition has gained so much attention, with a final discussion on the links between social cognition and functioning and the potential neural substrates of these relations.

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THE 4 DOMAINS OF SOCIAL COGNITION

There is a consensus in schizophrenia research that social cognition is a multidimensional construct comprising several domains; however, the domains to be given priority and how these domains should be defined have not been decided. In an attempt to address this problem, an extensive 2-step survey of expert researchers in the fields of schizophrenia, social psychology, and autism was conducted.[2] This process identified 4 core domains of social cognition for the study of schizophrenia: (1) emotion processing, (2) social perception, (3) theory of mind/mental state attribution (ToM), and (4) attributional style/bias.

Emotion processing is broadly defined as perceiving and using emotional information.[1] This domain encompasses both simple and complex subprocesses. At the lower level is the perception and recognition of emotions (eg, associating a smile with happiness), and at the higher level are the complex skills that enable individuals to manage and regulate emotions.

Social perception is defined as decoding and interpreting social cues in others. It includes the ability to integrate contextual information and social knowledge into judgments about others' behaviors.[3–6] An example would be seeing 2 unfamiliar people and knowing, without interacting with them, that they are in a romantic relationship or in a hierarchical relationship such as boss and employee.

The next domain, ToM, refers to the ability to represent human mental states and/or make inferences about others' intentions and beliefs.[7,8] This domain encompasses skills such as understanding false beliefs, interpreting hints, intentions, deception, metaphor, and irony, and also discerning faux pas. A common example of ToM skills would be understanding that your partner's statement about how delicious your

piece of pie looks most likely means that they would like you to share with them. Other terms that are commonly used interchangeably with ToM include mentalizing, mental state attribution, or cognitive empathy.[9]

The final domain of social cognition in schizophrenia is attributional style. This domain refers to the manner in which individuals interpret, explain, or make sense of the positive and negative social events encountered in life and is thought to have a significant impact on behaviors.[1,8] For example, if a friend does not return your call, you could assume that your friend is purposely ignoring your call, or you could think that your friend may not have gotten your message. The explanation adopted will quite likely determine your emotional and behavioral reaction to this event.

SOCIAL COGNITION AND SCHIZOPHRENIA

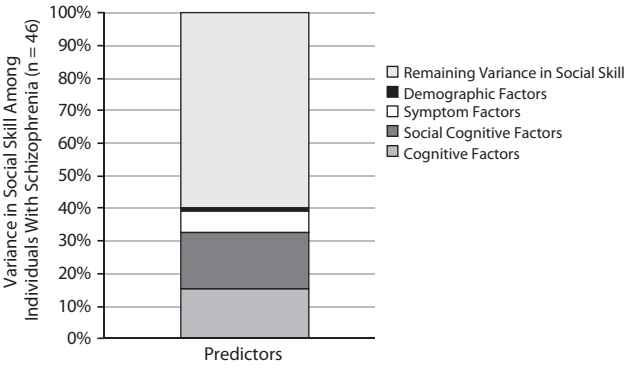
After defining the term social cognition and its components, the next logical question is why is this construct important? What do we know about social cognition in schizophrenia? First, we know that social cognition represents a significant area of impairment for individuals with schizophrenia. The literature documenting these deficits is extensively reviewed elsewhere but can be broadly summarized as follows.[10–13] Individuals with schizophrenia exhibit large deficits in emotion recognition and appear to have greatest difficulty processing negative emotions such as anger and fear. Social cue perception is also problematic, and these impairments are most pronounced for abstract cues. For ToM, patients display impairments in all of the skills comprising this domain, and these deficits become worse during periods of acute symptom exacerbation but are present even during periods of symptom remission. Additionally, when explaining negative social outcomes, individuals with schizophrenia, and particularly those with persecutory delusions, tend to blame others, rather than external situational factors.

Second, social cognitive impairments are evident early in the course of the disorder and are stable over time.[13–16] Importantly, advances in the early identification of individuals who are at either clinical or genetic risk for developing schizophrenia have shown that these impairments are present even in the prodromal phase of the illness.[17–19]

Third, although there is clearly overlap between social cognition and neurocognitive abilities such as memory, attention, and executive function, a growing body of literature indicates that social cognition is largely independent from neurocognition. Evidence from correlational studies,[20] factor analyses,[21–24] and differential deficit designs[25] all support the conclusion that social cognitive impairments are not due solely to the cognitive impairments that are commonly seen in the disorder.

Finally, and perhaps most importantly, social cognitive abilities contribute to real-world outcomes for individuals with schizophrenia, and the impact of social cognition on outcome is greater than that of neurocognitive abilities. For example, in a study that examined how well cognitive and social cognitive factors could explain variance in social skill among patients, cognitive factors including overall intellectual ability and executive function predicted approximately 15% of the variance (Figure 1). However, when social cognitive abilities (ie, emotion recognition, theory of mind, and social knowledge) were added to the regression model, an additional 26% of the variance was explained.[26]

Figure 1. Social Cognition Predicts Social Skill in Patients[a]



[a]Based on data from Pinkham and Penn.[26]

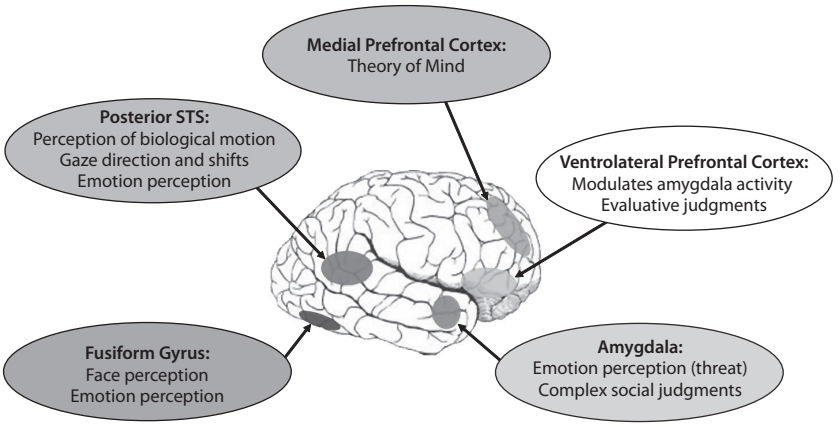
This finding has since been bolstered by a meta-analysis confirming that social cognition has a greater influence on outcomes than neurocognition, which also suggests that improving social cognitive abilities may lead to improved daily functioning for patients.[27] Initial remediation efforts have indicated that this is in fact the case, and social cognitive treatments have been linked to improved outcomes,[28,29] including social adjustment,[30] social functioning,[31–33] social relationships,[34,35] social skills[36] and reductions in numbers of aggressive incidents.[34]

Neural Basis for Social Cognition

The strong links between social cognition and functioning have prompted a good deal of work that seeks to understand the underlying mechanisms of social cognitive impairment. One area that has received increased attention is the neural basis of these abilities. Figure 2 provides an illustration of the key neural structures and areas that have been implicated in social cognition and that have been most commonly investigated in schizophrenia. We will first describe these regions and the social cognitive abilities that are linked to their function and then turn our attention to what is known about the functioning of these regions in individuals with schizophrenia.

To provide a very broad summary, the first 2 areas, the fusiform gyrus (FG) and superior temporal sulcus (STS), have both been implicated in face processing, with the FG responding most strongly to tasks focusing on facial identity and the STS responding most consistently to the changeable aspects of the face such as movements of the eyes or mouth.[37–42] Since facial expressions are often used to communicate emotion, both of these regions have also been linked to emotion processing. Further, since one's movements (eg, changes in eye gaze or facial expression) are often informative for understanding mental states, the STS is also linked to ToM via its role in detecting biological

Figure 2. A Neural Basis of Social Cognition[a]



[a]Based on data from Pinkham et al.[64]
Abbreviation: STS = superior temporal sulcus.

motion.[43–45] The amygdala appears to be responsible for directing attention to salient stimuli[46] and has been specifically linked to processing threat and negative emotions.[46–53] The fourth region, the medial prefrontal cortex (MPFC) is one of a number of cortical regions implicated in ToM[54,55] and may specifically be related to self-reflection.[56,57] Finally, the ventrolateral prefrontal cortex (VLPFC), has been found to modulate activity of the amygdala during the process of making evaluative judgments of facial stimuli.[58–60]

While it may be tempting to think of these regions as each performing a specific task, it is important to note that they evidence reciprocal connections and interact with each other.[61,62] Thus, the neural basis of social cognition may be best thought of as a collective network of neural regions that underlies the processing of social stimuli rather than specific regions that subserve specific skills.

SOCIAL COGNITIVE NEURAL NETWORK IN SCHIZOPHRENIA

A large body of literature has accrued to demonstrate that individuals with schizophrenia show both structural and functional abnormalities to brain regions comprising the social cognitive network.[63,64] Briefly, patients show reduced activation in FG during facial identity processing[65,66] and emotion recognition.[67–69] Findings regarding the STS have been mixed, with several studies failing to show differences in activation relative to controls[70–72] and one study showing increased activation of the STS as compared to controls.[73] Importantly however, increased activation of this region could be indicative of greater compensatory efforts and could therefore still be interpreted as the result of impairment.

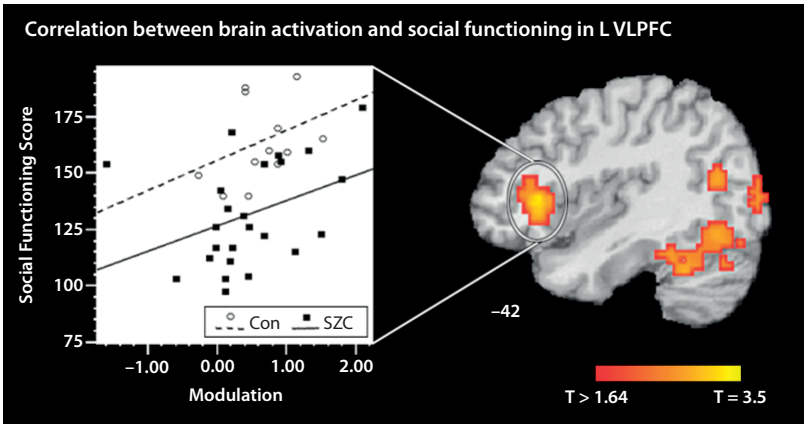
Results concerning amygdala functioning in schizophrenia are also complex, but current work indicates that patients demonstrate increased amygdala responses to neutral, rather than emotional, stimuli[74] and that amygdala functioning may differ between patients based on the presence of paranoid symptoms[69,72,75] and flat affect.[76,77] Finally, reduced MPFC activation in patients has recently been reported across a variety of nonverbal ToM tasks,[78–81] as have reductions in VLPFC activation.[72] On the whole, these studies provide strong support for the hypothesis that abnormal functioning of this network is a mechanism for social cognitive impairment in schizophrenia.

In addition, a few recent studies have also demonstrated that activation of this network is predictive of functional outcome. For example, in Pinkham et al,[72] participants were asked to view a series of faces and to identify each face as either trustworthy or untrustworthy. Previous work had shown that these relatively simple dichotomous judgments evoked differential activation in the network such that ratings of untrustworthy were accompanied by increased activation of social cognitive areas relative to ratings of trustworthy.[82,83] In our work, we also found this pattern of greater activation for trust (relative to untrust) ratings, and it was more pronounced in healthy controls as compared to individuals with schizophrenia. Critically, greater modulation of the neural response was significantly correlated with improved social functioning in both healthy and schizophrenia groups (Figure 3), and this pattern was repeated across each of the regions outlined as part of the social cognitive neural network.

In another related study that focused more specifically on the amygdala, there was again a relationship between activation and functioning.[84] Here, individuals were asked to identify the emotion expressed on a face that was looking either directly at the participant (ie, direct gaze) or slightly away (ie, averted gaze). As Figure 4 demonstrates, patients showed overall reductions in levels of amygdala activation as compared to healthy controls. Further, as in the previous study, the amount of activation in the amygdala in response to direct gaze expressions of anger was significantly and positively correlated to level of social and occupational functioning.

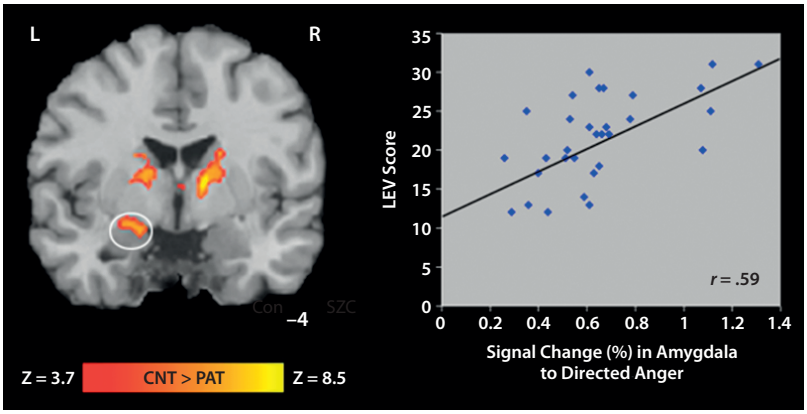
In summary, these sample studies coupled with the data from several others[85] provide a strong argument for the presence of causal links between neural activation, social

Figure 3. Activation in Social Cognitive Neural Networks Predicts Functioning[a,b]



[a]Adapted from Pinkham et al,[72] with permission.
[b]Left: Con: Spearman ρ = .42, $P < .05$; SCZ: Spearman ρ = .44, $P < .01$. Right: Con > SCZ for the contrast of untrustworthy > trustworthy (cluster level corrected at $P < .05$).
Abbreviations: Con = control, L VLPFC = left ventrolateral prefrontal cortex, SZC = schizophrenia.

Figure 4. Further Evidence of Social Cognitive Activation Predicting Functioning[a,b]



[a]Adapted from Pinkham et al,[84] with permission.
[b]Left: cluster-level corrected at $P < .0001$. Right: correlation significant at a Bonferroni-corrected level of $P < .006$.
Abbreviations: CNT = control, LEV = level of function, PAT = patients with schizophrenia.

cognition, and functional outcome. These data also highlight the potential importance of treating social cognitive impairments and pursuing remediation strategies that will normalize these neural processes.

FUTURE IMPLICATIONS IN SOCIAL COGNITION

Despite the obvious promise of social cognition for contributing to our understanding of social impairment in schizophrenia, there are a number of important challenges that must be addressed going forward. Globally, the field is relatively young, particularly when compared with cognition, and as a result, there has been no formal consensus on which domains define the construct of social cognition or which tasks best index these domains until recently. Unfortunately however, the majority of tasks developed to assess social cognition have poor psychometric properties, which raises questions about the reliability of the current findings as well as the utility of these measures for clinical trials. A current project, coined the Social Cognition Psychometric Evaluation (SCOPE) study,[2] aims to address these problems by achieving consensus on key social cognitive domains in schizophrenia and evaluating the psychometric properties of the current best measures. As noted above, the goal of developing a consensus definition of social cognition has been reached, and the project is currently in its third phase. This phase includes an initial psychometric study designed to determine which measures are ready for use in clinical trials and those that require modification prior to use. The fourth phase will modify the tasks that show inadequate characteristics and evaluate the results of those modifications. The fifth and final phase will involve a large validation study, conducted across 3 sites, to determine the psychometric properties of the finalized battery. It is hoped that this program of research will: 1) yield a battery of social cognitive

tasks that can be offered to the field as outcome measures for treatment studies and 2) advance the understanding of social cognitive impairment in schizophrenia by improving our ability to assess the impact of social cognitive abilities on social functioning.

CONCLUSIONS

In summary, social cognition is an exciting and emerging new area of research in schizophrenia. There is clear evidence of stable impairment in schizophrenia patients that is highly related to functioning. Further, clearly defined neural substrates are associated with social cognition. Taken together, these factors lend credence to social cognition as a viable treatment target. With the recently started SCOPE project, there is future promise of improved measures that are optimized for evaluating both behavioral and pharmacologic interventions and remediation strategies.

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