Social Cognition in Schizophrenia: An Overview

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The purpose of this column is to provide an overview of social cognition in schizophrenia. The column begins with a short introduction to social cognition. Then, we describe the application of social cognition to the study of schizophrenia, with an emphasis on key domains (i.e., emotion perception, Theory of Mind, and attributional style). We conclude the column by discussing the relationship of social cognition to neurocognition, negative symptoms, and functioning, with an eye toward strategies for improving social cognition in schizophrenia.

Key words: social cognition/attributions/emotion perception/theory of mind/functional outcome

What Is Social Cognition?

Social cognition refers to how people think about themselves and others in the social world. The term originated within social psychology during the general “cognitive revolution”1 of the late 1960s and early 1970s. The social cognition construct provides a broad theoretical perspective that focuses on how people process information within social contexts. It includes person perception, causal attributions concerning self and others, and bringing social judgments to decision making, among other elements.2–5

Social cognition research shares 4 common features.3 The first is an unabashed mentalism, a focus on mental representations such as “schemas.” These are organized sets of ideas, such as a person’s conceptualization of oneself, attitudes toward racial groups, or notions about the physical properties of the external world. Once schemas are activated and accessible, they have far-reaching consequences, such as when inferring whether someone is friendly or when deciding whether to continue a romantic relationship.

Researchers have also shown that “metacognitive” experiences are critical to people’s mental representations. These are subjective experiences that accompany schemas, such as the ease or difficulty of recall or association. For example, if one attempts to retrieve examples supporting another’s trustworthiness but finds this subjectively difficult to accomplish, one may instead infer that the other is untrustworthy. Reliance on metacognitive experience for making judgments is heightened under conditions of relatively limited cognitive resources (e.g., distraction, load, or working memory deficits)7 that are common in schizophrenia. Thus, this process has paradoxical implications for the common technique of “generating alternatives” used in cognitive therapy for psychosis. For example, an individual may report that he saw a black car in front of his house and is therefore certain that the Central Intelligence Agency is after him. This will lead the therapist to suggest that the individual think of other possible reasons why the car may be parked there. However, cognitive deficits may make this such an effortful process for the individual that, even if he can generate other possibilities, he may conclude that the belief must be true (because the other reasons do not readily come to mind), further entrenching his delusion. Thus, research on metacognition suggests that clinicians may need to strike a balance between asking them to think flexibly about situations but not to the extent that the process becomes effortful, aversive, and countertherapeutic.

A second common feature is that social cognition is process oriented. Researchers attempt to understand the precise causal mechanisms intervening between initial interactions with stimulus persons and product behaviors. For example, upon being asked for money by a panhandler, a person’s response may differ greatly depending on whether an intervening attribution engendered annoyance or sympathy. Social cognition researchers have used increasingly sophisticated referential methods ranging from reaction timing to brain imaging to assess intervening processes.8 For example, because of potential limits to insight or honesty, social cognition researchers have developed a variety of “implicit” measures involving reaction timing to examine the automatic association between attitudes and behaviors.9

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Third, social cognition is characterized by interdisciplinary approaches and cross-fertilization of ideas. Not only is there an obvious melding of social with traditional cognitive psychology but also a melding with other fields such as developmental psychology, clinical psychology, and neuroscience. For example, neuroscientific approaches have found that specific brain regions are activated in response to social decision making. These regions, like the striatum, which are active for basic rewards, also appear to encode more abstract social rewards such as positive feelings produced by mutual cooperation, commonalities that would not have been uncovered without an interdisciplinary approach.

Finally, social cognition researchers are concerned with real-world applications. One interesting example is that social cognition research has been used to enlighten legal decisions of the US Supreme Court. Researchers have been called as expert witnesses to testify to the antecedent conditions, indicators, consequences, and remedies of stereotyping. This has influenced decisions about prejudice and discrimination cases ranging from trials and appeals courts to the Supreme Court’s reviews.

Social Cognition in Schizophrenia

What do we know about social cognition in schizophrenia? There appear to be 3 primary domains of inquiry: emotion perception, Theory of Mind (ToM), and attributional style. In regard to emotion perception (eg, identifying emotion displayed in various facial expressions or tone of voice), the following conclusions can be drawn (reviewed by Edwards et al, Hellewell and Whittaker, Kohler and Brennan, and Mandal et al). First, individuals with schizophrenia display deficits compared with nonclinical control participants. Second, these deficits are more severe relative to individuals with other psychiatric disorders such as depressive disorder (unless psychotic features are present). Third, the greatest deficits are evident in the perception of negative emotions (compared with positive emotions). Fourth, the deficit in emotion perception is stable over time, although evidence suggests that individuals in remission may outperform individuals in an acute phase of the disorder. Fifth, individuals with schizophrenia perform worse trying to “read between the lines” (ie, identifying what a given individual is thinking or feeling) but are less impaired on more concrete social judgments (ie, identifying what a person is wearing or doing). Sixth, many individuals with schizophrenia display restricted visual scanning and spend less time examining salient facial features during emotion perception tasks. Finally, impairments in emotion perception are present early in the course of illness.

ToM refers to the ability to represent human mental states and/or to make inferences about another’s intentions. It includes understanding false beliefs, hints, intentions, deception, metaphor, irony, and faux pas. Over 30 studies have been conducted on ToM in schizophrenia, leading to the following conclusions (reviewed in Brune and Harrington). In general, individuals with schizophrenia exhibit deficits in ToM relative to nonill and psychiatric controls. The bulk of research supports the conclusion that this impairment in schizophrenia is a trait deficit. First-degree relatives of individuals with schizophrenia who also score high on schizotypy have impaired ToM, lending support for ToM as a potential endophenotype for schizophrenia. ToM deficits are present in both inpatient and outpaient samples, are not accounted for by deficits in general cognitive functioning, and are not uniquely associated with any specific symptom type (eg, paranoia). The etiology of ToM deficits in schizophrenia remains unclear, in part because the genesis of normal ToM is still unknown (eg, Leslie and Harris).

Attributional style refers to explanations people generate regarding the causes of positive and negative events in their lives. Normally, people attribute responsibility for positive events to themselves and responsibility for negative events to others. The majority of work in schizophrenia has focused on attributional style in individuals with paranoia or persecutory delusions. Such individuals tend to blame others rather than situations for negative events, an attributional style known as a “personalizing bias.” This becomes a dynamic mechanism for regulating self-esteem because attributing negative intentions to others maintains a positive self-image. However, it comes at the cost of increasingly negative perceptions of others. Such negative attributions to others are not corrected in paranoia, even when one obtains subsequent disconfirming information about the other’s culpability. According to Bentall et al, 2 factors prevent individuals with persecutory delusions from correcting their bias in the face of disconfirming informational: a strong need for closure (ie, an intolerance of ambiguity) and impairments in ToM. Indeed, preliminary research shows an association between need for closure and persecutory delusions and between deficits in ToM and personalizing attributions. Individuals with persecutory delusions may of course have other social cognitive biases such as the tendency to “jump to conclusions” and to demonstrate a “confirmation bias” (ie, seeking confirmatory evidence for a belief rather than disconfirmatory evidence; reviewed in Freeman). In all, a variety of social cognitive deficits and biases may underlie paranoia.

Relationship of Social Cognition to Cognition and Negative Symptoms

How does social cognition relate to other domains, most notably neurocognition (eg, attention, memory, executive function) and negative symptoms? Conceptually,
social cognition involves the interface of emotional and cognitive processing, whereas neurocognitive processing is relatively affect-neutral. In contrast, negative symptoms could spring from a similar affective processing dysfunction as social cognitive impairments. For example, diminished social reward salience found in negative symptoms (eg, anhedonia and amotivation) may contribute to subtypes of social cognitive dysfunction. Alternatively, failed empathy or mental simulation of others’ cognitive-affect states (a putatively social cognitive phenomenon) may underlie ToM deficits and also foster the extinction of social reinforcement, leading to increased negative symptoms.

Empirically, studies using statistical modeling techniques and matched task designs have concluded that social cognition is best understood as related to, but distinct from, neurocognition and negative symptoms. This distinction is also observed at the neural level because activation circuitry for social cognition vis-à-vis neurocognition and negative symptoms are relatively independent.

Treatment Implications

Recent enthusiasm for social cognition in schizophrenia has followed upon research showing that it has a relationship with functional outcomes (eg, social skills, community functioning). This, in turn, has inspired researchers to examine whether social cognition can be improved (reviewed by Horan et al) because social cognition may be an important target for pharmacological and psychosocial treatments. Interestingly, there has been little support for atypical medications improving social cognition in schizophrenia because the one large adequately powered study found that neither quetiapine nor risperidone resulted in improved emotion perception among 289 individuals with schizophrenia. Thus, there has been growing interest in psychosocial treatments as a means of improving social cognition.

Psychosocial treatment programs use a variety of techniques to ameliorate social cognitive deficits, from “targeted” interventions that focus on a specific skill (eg, asking clients to imitate others’ facial expressions to improve emotion perception) to those that target integrative social cognitive abilities via viewing videotapes and role-playing. While there is growing evidence that social cognition can be improved, future research needs to determine whether improvements in social cognition generalize to other social cognitive domains as well as to behaviors. Initial data from our own laboratory on the Social Cognition and Interaction Training program suggest that targeting social cognition may result in changes in real-world behavior, although these findings are preliminary and in need of replication.

It might be the case that social cognition training cannot be done in isolation but must be linked with broader based skills training in a manner consistent with the most effective cognitive remediation studies (reviewed in McGurk et al). Thus, one can imagine tailoring treatment (cognitive remediation, social cognitive training, cognitive behavioral therapy, etc.) to the needs of the client with schizophrenia, rather than hoping that a “one-size intervention” will fit all, an unrealistic expectation given the heterogeneity of the disorder (and its changes over time). Clearly, however, social cognition training has promise as an addition to the armamentarium of the treatments for schizophrenia.

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