Social Cognition in Schizophrenia: An NIMH Workshop on Definitions, Assessment, and Research Opportunities

Michael F. Green[1–3], David L. Penn[4], Richard Bentall[5], William T. Carpenter[6,7], Wolfgang Gaebel[8], Ruben C. Gur[9], Ann M. Kring[10], Sohee Park[11], Steven M. Silverstein[12], and Robert Heinssen[13]

[2]Department of Psychiatry and Biobehavioral Sciences, Geffen School of Medicine at UCLA, Los Angeles, CA; [3]VA Greater Los Angeles Healthcare System, Los Angeles, CA; [4]Department of Psychology, University of North Carolina-Chapel Hill, NC; [5]School of Psychology, University of Wales, Bangor, UK; [6]Maryland Psychiatric Research Center, Department of Psychiatry, University of Maryland School of Medicine, Baltimore, MD; [7]VISN 5 Mental Illness Research, Education, and Clinical Center, Veterans Administration, Baltimore, MD; [8]Department of Psychiatry, Heinrich-Heine-University Düsseldorf, Düsseldorf, Germany; [9]University of Pennsylvania School of Medicine, Philadelphia, PA; [10]University of California, Berkeley, CA; [11]Departments of Psychology and Psychiatry, Vanderbilt University, Nashville, TN; [12]University Behavioral HealthCare and Robert Wood Johnson Medical School, University of Medicine and Dentistry of New Jersey, Piscataway, NJ; [13]Division of Adult Translational Research and Treatment Development, National Institute of Mental Health, Bethesda, MD

Social cognition has become a high priority area for the study of schizophrenia. However, despite developments in this area, progress remains limited by inconsistent terminology and differences in the way social cognition is measured. To address these obstacles, a consensus-building meeting on social cognition in schizophrenia was held at the National Institute of Mental Health in March 2006. Agreement was reached on several points, including definitions of terms, the significance of social cognition for schizophrenia research, and suggestions for future research directions. The importance of translational interdisciplinary research teams was emphasized. The current article presents a summary of these discussions.

Key words: social cognition/schizophrenia/NIMH

The term social cognition is defined in various ways, but generally refers to the mental operations that underlie social interactions, including perceiving, interpreting, and generating responses to the intentions, dispositions, and behaviors of others.[1–4] In humans, social cognition means people thinking and forming impressions about people. Social cognitive processes are how we draw inferences about other people's beliefs and intentions and how we weigh social situational factors in making these inferences. Over the past 15 years, clinical investigators and behavioral scientists have increasingly employed social cognitive constructs to explore the symptoms and interpersonal deficits that characterize schizophrenia.[5–7] Indeed, social cognition has emerged as a high priority topic within schizophrenia research as evidenced by a burgeoning empirical literature and increased attention in scientific meetings.[8]

The impetus for the present meeting on social cognition in schizophrenia arose from 2 events. First, social cognition was seen as a key domain for consideration during the first meeting of the NIMH-sponsored Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) Initiative. Social cognition was ultimately included as 1 of the 7 domains represented in the MATRICS Consensus Cognitive Battery for clinical trials in schizophrenia.[9,10] Second, social cognition was a specific topic of discussion at the New Approaches Conference (NAC), which was the final meeting of the MATRICS conference series. Although there was general agreement among NAC participants that social cognition is a valuable construct for understanding the nature and disability of schizophrenia, a number of potential obstacles were identified that could impede progress in this area, including the lack of agreement on terms, definitions, and measurement approaches.[8]

This article summarizes a subsequent meeting sponsored by the National Institute of Mental Health (NIMH) to address issues raised in the NAC. The meeting, "Social Cognition in Schizophrenia: Basic Definitions, Methods of Assessment, and Research Opportunities" was organized by Drs Green, Penn, and Heinssen and took place in March 28–29, 2006. Participants included the authors on this article as well as additional extramural program staff from NIMH. The main topics and discussion questions that structured the meeting are included in table 1. The goals of the meeting were to reach

[1]To whom correspondence should be addressed; UCLA Semel Institute for Neuroscience and Human Behavior, 300 Medical Plaza, Room 2263, Los Angeles, CA 90095-6968; tel: 310-794-1993, fax: 310-825-6626, e-mail: mgreen@ucla.edu.

Table 1. Topics and Questions at the NIMH Workshop on Social Cognition in Schizophrenia

Defining social cognition in schizophrenia
1. What domains of social cognition should be considered? Is there empirical support for these domains?
2. What is the factor structure of social cognition in schizophrenia? Are there data on factor structures in normal populations?
3. What is the relationship of social cognition to neurocognition?
4. Can work from basic neuroscience or social psychology inform the definitions and boundaries of social cognition in schizophrenia?
Establishing the significance of social cognition in schizophrenia: relationships to clinical symptoms and functional outcome
1. What is social cognition's relationship with positive symptoms (eg, paranoia), negative symptoms, and disorganization?
2. What is the functional significance of social cognition? Does it behave as a mediator?
3. Social cognition and the course of schizophrenia: does it predate the illness or occur early in the illness? Are the impairments trait like?
Measuring social cognition in schizophrenia
1. What are the psychometric problems with current measures when used with schizophrenia patients?
2. Should measures developed for other clinical populations (eg, brain injury, autism) guide measurement development for schizophrenia?
Identifying obstacles to research progress
1. What conceptual or methodological barriers must be overcome to advance social cognition research in schizophrenia?
2. How might meaningful collaborations be developed between social cognition researchers in schizophrenia, basic social scientists, and cognitive neuroscientists?

agreement, to the extent possible, on the definitions of terms in this area and on the significance of social cognition in schizophrenia research for understanding clinical symptoms and outcome and to suggest promising research directions. The remainder of this article summarizes the discussion of these topics.

Defining Social Cognition in Schizophrenia

Because definitions of social cognition vary, it was deemed important to devote the first part of the meeting to defining terms that are commonly used in schizophrenia research. Ambiguous and inconsistent terminology can complicate communication among researchers and make it more difficult to draw inferences across studies. As a starting point for the discussion, participants reviewed recent publications that suggested research in social cognition in schizophrenia fits into the following 5 areas.[8,11]

1. Theory of mind. Theory of mind (also called mental state attribution) typically involves the ability to infer intentions, dispositions, and beliefs of others.[12,13] Much of initial interest in this area focused on studies of children and how theory of mind is acquired in normal and abnormal development. Hence, many measures in this area were initially developed for use with children. Theory of mind has been extended to schizophrenia, partly due to similarity between aspects of social dysfunction in autism and a subgroup of patients with schizophrenia, and following suggestions that abnormalities in this process can account for the formation of clinical symptoms.[13–16]
2. Social perception. Tests of social perception assess one's ability to identify social roles, societal rules, and social context.[17–19] In social perception tasks, participants must process nonverbal, paraverbal, and/or verbal cues to make inferences about complex or ambiguous social situations. Individuals may be asked to identify interpersonal features in a situation such as intimacy, status, mood state, and veracity. This area can also include "relationship perception," which refers to perception of the nature of relationships between people.[20] Social perception is one of the social cognitive areas related to community functioning in schizophrenia, and hence, may be a rational focus for interventions designed to enhance functional improvements.[21]
3. Social knowledge. This area refers to awareness of the roles, rules, and goals that characterize social situations and guide social interactions.[22–24] Social knowledge (also called social schema) can be measured with paper and pencil tests that assess one's awareness of what is socially expected in different situations (eg, in a doctor's office vs in a restaurant). It has been studied somewhat less than the other areas in schizophrenia, and it overlaps with social perception; successful social knowledge requires awareness of which cues occur typically in specific social situations (ie, social perception) and how one is supposed to respond to them. Social knowledge is viewed as an initial step and prerequisite for adequate social competence[25] and has been targeted for intervention in some social skills training programs for schizophrenia.[26]
4. Attributional bias. Attributions are causal statements; ie, statements that either include or imply the word "because." They are a very frequent type of verbal behavior and are found in every hundred words or so of speech.[27] Unlike mental state attribution (theory of mind), attribution bias or style reflects how people typically infer the causes of particular positive and negative events. Attributions can be measured by questionnaires[28] or rated from transcripts of interactions.[29] In research involving both psychiatric and nonpsychiatric samples, key distinctions are typically made between external personal attributions (ie, causes attributed to other people), external situational attributions (ie, causes attributed to situational factors), and internal attributions (ie, causes due to oneself). Application of these categories to clinical samples reveals that individuals with persecutory delusions often attribute negative outcomes to others,

rather than situations. This is known as a personalizing bias.[30,31] Research involving persons with schizophrenia has also focused on hostile attributional biases or the tendency to attribute hostile intentions to others' actions.[32]

5. Emotional processing. Emotional processing refers broadly to perceiving and using emotions.[33] One influential model of emotional processing defines emotional intelligence as a set of 4 components, including identifying emotions, facilitating emotions, understanding emotions, and managing emotions.[34,35] This model includes affect perception, a domain of emotion processing that is frequently measured in schizophrenia research.[36–38] Measures of emotion processing vary broadly and include ratings of emotions that are displayed in faces or voices or ratings from brief vignettes of how individuals manage, regulate, or facilitate emotion.

Although there was general agreement among meeting participants that these 5 areas of social cognition capture much of the research in schizophrenia, 2 important caveats were mentioned. First, it was noted that boundaries between the categories listed above are not absolute and there is considerable overlap among the terms. For example, identifying emotions is clearly a component of emotional processing but is sometimes considered to be an aspect of theory of mind. Likewise, social knowledge overlaps with social perception. Second, participants recognized that studies of social cognition in nonpsychiatric samples would not be organized the same way and would include a larger set of research topics. For example, emotion processing, which is often a component of social cognition in schizophrenia research, may be treated as a separate domain from social cognition in nonclinical studies. Other topics that are included in studies of nonclinical social cognition include self-perception, prejudice and stereotyping, empathy, hindsight bias, and counterfactual thinking, among others. For the most part, these topics have not yet been adequately addressed (or only addressed in a preliminary way) in schizophrenia research, although they may have importance for understanding the clinical phenomenology and outcome of the illness. Despite these caveats, this listing of topics provided a reasonable organizing framework for subsequent discussions.

How to Refine and Select Domains

Given the wide range of definitions for social cognitive domains, how does one select domains of social cognition that are of highest priority for future research? The group believed that the database is early in its development and too limited to provide firm answers at this point. However, we identified a process that will help to organize and parse the social cognitive landscape in schizophrenia research. First, if a study includes a sufficient number of measures and an adequate number of participants, the researchers could conduct exploratory analyses of the factor structure of social cognition in schizophrenia. Such analyses are not common because few studies include a range of social cognitive measures, but they are valuable in determining the degree of commonality among measures. Second, after exploratory analyses it will be possible to test theoretical models with confirmatory analyses. Such approaches provide a strong way to test proposed models of social cognition, illness features, and interpersonal functioning, but they require large samples and a clearly articulated theoretical framework as a starting point. Third, the division of social cognitive constructs in schizophrenia will be informed by social and affective neuroscience that can parse social cognitive domains based on overlapping and distinct neural circuits. Identification of separate circuits supporting social cognition will allow clinical researchers to determine which ones are intact or aberrant in schizophrenia. Fourth, it will be ultimately important to develop new measures that assess social cognitive constructs more narrowly and precisely, that are appropriate for clinical populations, and that can be used repeatedly in intervention studies. Such efforts are currently underway with the NIMH Initiative, Cognitive Neuroscience for Treatment Research to Improve Cognition in Schizophrenia (CNTRICS) that is mentioned below.

Establishing the Significance of Social Cognition in Schizophrenia: Relationships to Clinical Symptoms and Functional Outcome

It was emphasized that social cognition in schizophrenia is studied for a variety of different goals using very different frameworks. Hence, the added value of social cognition varies depending on the scientific question of a research program. Four types of goals were mentioned. One was the use of social cognition for understanding the development of particular clinical symptoms of schizophrenia. A second was social cognition's role in explaining functional outcome differences in schizophrenia. A third was determining whether social cognitive impairments are stable traits in schizophrenia or fluctuate over time. Each of these 3 goals will be discussed in this article. A fourth goal in cognitive neuroscience studies in schizophrenia is to use social cognitive constructs to identify neural substrates that are potentially distinct from those of nonsocial cognitive domains.[39–45] As mentioned below, the extent to which these processes are separate is not clear. Empirical (correlational) relationships have been demonstrated among social cognition (theory of mind) impairments, impaired perceptual organization, and disorganized thinking.[46–48] These relationships might be taken to suggest the existence of a common cortical processing algorithm involving dynamic,

context-dependent, binding process and its disruption in schizophrenia. Such disruption may stem from underlying abnormalities in the neural circuitry and neurotransmitter systems known to be involved in modulatory (excitatory and inhibitory) interactions.[49] The topic of neural substrates of social cognition was viewed as very important for schizophrenia research but was not a main focus of this meeting.

Using Social Cognitive Constructs to Deconstruct Psychotic Symptoms in Schizophrenia

One goal is to understand how social cognition is involved in the formation of specific clinical symptoms, such as paranoia or thought control.[13,30] From this perspective, abnormalities in social cognition would be viewed as close (proximal) causes of clinical phenomenology. For example, the tendency of individuals with paranoia or persecutory delusions to blame others, rather than situations for negative outcomes, may result from a jumping to conclusions reasoning bias and difficulties in theory of mind processes,[31] which in turn may be a function of a strong need for closure and cognitive rigidity.[50–52]

Social Cognition and Negative Symptoms in Schizophrenia

Relationships among social cognitive constructs and negative symptoms are less clear. Participants acknowledged that although some overlap exists between negative symptoms and social cognition in schizophrenia, it is unwise to combine the constructs at this point in time. Indeed, some of this overlap may reflect the structure of current negative symptom rating scales that include conceptually distinct areas (such as social cognition) that are not necessarily part of the negative symptom construct.[53] The consensus of the meeting was that it is more informative to study negative symptoms and social cognition separately and to analyze relationships between them until we know more about areas of convergence and divergence. Confirmatory factor analyses may be helpful in testing various structural models, and initial efforts support the distinctiveness of social cognition and negative symptoms.[54] In addition, social cognitive constructs may have value in deconstructing specific negative symptoms such as avolition and flat affect, similar to what has been done with paranoia, although these studies have yet to be conducted.

Along these lines, there was considerable discussion about social motivation/social drive. Social motivation/drive is sometimes viewed as part of a broader definition of social cognition, but it is typically viewed as a negative symptom of schizophrenia (eg, asociality and avolition). At present we cannot determine whether asociality and avolition are by-products of impaired social cognition, or whether these deficits impact social cognitive performance adversely. For the time being, the participants suggested that it is better to continue to view social motivation/drive as part of the negative symptom complex, but that exploring relationships with social cognitive constructs is an important topic for future research. Efforts are currently underway to validate a new negative symptom rating scale that arose from the NIMH-MATRICS consensus development conference on negative symptoms.[55] This measure, the Negative Symptom Rating Scale, carefully evaluates asociality and avolition, so that studies can examine the relationship between these negative symptoms that are associated with social drive and social cognition.

Social Cognition and Functional Outcome

Social cognition may help to explain heterogeneity of functional outcome in schizophrenia.[56] Social cognitive deficits appear to be key determinants of daily functioning in schizophrenia, including instrumental actions, interpersonal functioning, and vocational achievement.[57–62] A recent review of the literature found consistent patterns of association with community functioning for 2 aspects of social cognition (emotion perception and social perception) and preliminary trends in 2 others (theory of mind and attributional style).[61] At an intuitive level, it is reasonable to assume that social cognitive abilities enable subjects to interact effectively with their social environment, and that deficits in social cognition could lead to social misperceptions, resulting in inappropriate interpersonal reactions or social withdrawal.[5,63] Beyond the replicated bivariate associations, there is increasing support from studies that have used exploratory and confirmatory analytic approaches that suggest that social cognitive processes act as key mediators between basic (nonsocial) cognition and functional outcome.[21,64–66] These studies of mediation show that social cognition has significant relationships to basic cognition on the one hand, and to community functioning on the other, and that the direct relationships between cognition and outcome are reduced (and sometimes eliminated) when social cognition is added to the model. In addition, social cognition contributes variance to models of functional outcomes that is independent of nonsocial cognition.[60,61,67,68]

Based on the studies mentioned above, it is clear that social cognitive measures have value for understanding outcome, above and beyond basic (nonsocial) cognition alone. However, the meeting participants acknowledged that there is still considerable debate regarding whether social cognitive processes are mainly unique or are fully overlapping with basic cognitive processes. It is safe to conclude there is some overlap between social and non-social cognition, so the argument is about the degree of overlap. A confirmatory factor analysis showed that although social and basic cognition were closely connected, separating these 2 domains provided significantly

better model fit compared with when they were combined.[54] Similarly, a structural equation modeling study found that the data fit the model well when social cognition and basic cognition were separated but not when the 2 domains were combined.[65] A third study recently used confirmatory factor analysis to evaluate the structure of IQ subtests in schizophrenia.[69] The results supported a separate factor consisting of subtests with social content (labeled a social cognition factor).

Consistent with these confirmatory analyses, a recent study found that schizophrenia and schizoaffective patients differed on a social cognitive measure but not basic cognitive measures.[70] In addition, interesting dissociations have been observed in Williams syndrome, where social cognition is relatively preserved, whereas nonsocial cognition is not, although the opposite pattern may be found in individuals with high functioning autism.[44] Data from neurologically impaired populations also support the idea of some independence of social and nonsocial cognition. For example, Parkinson's disease patients have demonstrated impairments in emotion perception, making emotional faces, and emotional facial imagery, while demonstrating intact object imagery.[71] Dissociations between emotion perception and facial identification have also been observed in cases of neurodegenerative disease.[72] Affective and social neuroscience have a similar discussion about degree of overlap in nonclinical samples based on neuroimaging results. Some investigators argue for unique neural circuits associated with social and emotional processes by subtracting out comparison tasks that control for the nonsocial components.[42,73–76] However, these conclusions are actively debated.[77–79] Future studies with and without clinical samples will continue to clarify neural circuitry that is unique to social cognition versus circuits shared with other aspects of cognition.

State vs Trait Characteristics

There is little direct evidence on the question of whether the impairments in social cognition can be viewed as state or trait like. Attributional bias is viewed as a proximal determinant of psychotic symptoms, and hence, tends to be viewed as state related. At the same time, it would be possible for stable low-level attributional bias in at-risk individuals to contribute to vulnerability to later psychosis. Other social cognitive constructs such as emotion and social perception may be more trait like because they have been observed in first-degree relatives of patients with schizophrenia (although the findings are not entirely consistent[80,81]) and appear to be stable across different stages of schizophrenia and in longitudinal studies[82,83] (see Penn et al[11] for review of state vs trait issues for emotion perception and theory of mind). In addition, emerging social cognitive data on the prodromal phase of schizophrenia will help to address whether impairment in social cognitive processes precedes the onset of psychotic symptoms.[84,85] Overall, the state vs trait aspects of social cognition was seen as a basic question that remains largely unanswered, but it seemed likely to the participants that the social cognitive subdomains will vary in this respect.

Measuring and Treating Social Cognition in Schizophrenia

Factor Structure

Participants thought it unreasonable to assume that social cognition could be represented as a single construct. Few data exist, however, concerning the factor structure of various aspects of social cognition in schizophrenia. So far this has not been a large problem as most studies in schizophrenia include only one measure of social cognition, although there are exceptions.[54,67] However, this will become a bigger challenge for the field as more studies include multiple measures, making it necessary to indicate which subdomains are being represented. Given the current paucity of data to address this question, the group thought there is a risk in oversimplifying the constructs and, for the time being, it may be safer to examine measures separately and not make strong assumptions about common factors.

Psychometric Characteristics of Existing Measures

There was agreement that the psychometric properties of current social cognitive measures for schizophrenia are generally inadequate or unknown. Measurement problems are a consideration for all social cognitive domains but appear to be especially prominent for measures of theory of mind and attributional style. One reason for these difficulties is the common practice of taking measures developed for a nonpsychotic population and applying them directly to schizophrenia (such as measures of theory of mind from autism). This process tends to result in scaling problems (eg, ceiling effects), scoring dilemmas, and difficulties in interpreting the meaning of scores. For attributional style, the Internal Personal Situational Attributions Questionnaire (IPSAQ[28]) was developed to help address psychometric problems in other measures. Although the IPSAQ has shown adequate psychometric properties, questions remain on how to best quantify performance and whether it is better to use ratings from subjects or rater codings of responses as the most relevant index of performance.[86] A further difficulty in assessment of attributional style is that the underlying mechanisms may be affected by situational factors, which would make them less reliable and more state like. For example, activation of situation-relevant self-schemas may increase the availability of internal explanations for negative events, resulting in a pessimistic shift in attributional style that may be more pronounced in clinical groups compared with controls.[87] Finally, it is unclear whether problems in attributional style are present across

all social situations or only those in which intention is ambiguous. This issue has implications for treatment.[32] Thus, further work is required to investigate the mechanisms involved in making causal judgments in both healthy and clinical groups.

The group noted that good psychometrics is desirable in any type of research, but that the requirement for strong psychometrics in measures increases with certain types of studies. As one starts to measure social cognition in studies that use correlational analyses (including regression analysis, path analysis, and structural equation modeling), it is critical for measures to have adequate range and distributional properties. Perhaps, even higher standards apply when tests are used as outcome measures in clinical trials, as described in the next section.

Social Cognition as an Intervention Target

Although social cognition has not been commonly used as an endpoint for intervention studies, it is increasingly viewed as a treatment target for both pharmacological and nonpharmacological (psychosocial) interventions. Social cognitive measures were included in some clinical trials with second-generation antipsychotic medications, and the results have not been overly encouraging.[88,89] Social cognition will now be examined more consistently with novel compounds for cognition enhancement due to its inclusion as one of the domains of the MATRICS Consensus Cognitive Battery. Regarding nonpharmacological interventions, a variety of studies have examined short-term intervention probes for social cognition, and longer term social cognitive training programs are starting to emerge internationally.[90–94] The validation studies for these programs indicate that social cognitive interventions are well tolerated and generally enjoyed by patients over extended periods (typically months). The initial results from these studies, although preliminary, strongly suggest that social cognitive interventions can improve performance on social cognitive outcome measures.[95] In addition, some studies have found that social cognitive interventions lead to improvements in aspects of adaptive functioning.[90,91]

When social cognition is used as an endpoint for pharmacological or psychosocial interventions, it is essential that the measures are standardized, have excellent test–retest reliability, and possess good utility as a repeated measure. In the absence of these psychometric qualities, it will be difficult (or require very large samples) to detect change with treatment,[9] and to interpret the clinical significance or results. Challenges in adapting measures from cognitive and affective neuroscience for use in clinical trials of schizophrenia are currently being addressed in an ongoing series of meetings, CNTRICS, with summaries of anticipated problems and possible solutions posted on the CNTRICS Web site (http://cntrics.ucdavis.edu/index.shtml).

Identifying Obstacles to Progress in this Area

Three general obstacles to research progress were identified: (1) psychometrics and measurement, (2) maturity of the field, and (3) a lack of interdisciplinary bridges between clinical and basic researchers. The first issue (psychometrics and measurement) was mentioned above, so here we briefly mention the other 2.

Maturity of the Field

The study of social cognition in schizophrenia is far from new.[56,96,97] However, there is a large disparity between the relatively limited data-based literature on social cognition in schizophrenia and the very extensive and well-established literature on nonsocial cognition. Hence, there is still a need for a critical mass of data on relationships between social cognition and symptoms or functional outcome. Similarly, there is a need for a critical mass of researchers in this area. Despite several established research programs in social cognition in schizophrenia, it is not currently viewed as critical for social cognitive measures to be included in performance assessments or for study findings to be interpreted in social cognitive terms. The participants at this meeting expect that social cognitive considerations will become increasingly common, if certain interdisciplinary bridges can be built.

Lack of Bridges to Basic Scientists

The single most problematic obstacle identified was the lack of bridges connecting schizophrenia investigators to other scientists, including social psychologists, social and affective neuroscientists, preclinical scientists, and clinical trial researchers. These translational bridges are hard to build and typically take many years to become firmly established. It is common to find that paradigms developed by basic behavioral scientists are not applicable to schizophrenia samples (too long, too complex) or to clinical trials (not easily repeated, not highly reliable). It is also common for clinical scientists to use older experimental paradigms until newer measures are tried and adequately adapted for use in psychopathology research. Another difficulty is that basic scientists often have more interest in discovering patterns and principles that apply broadly as opposed to those that are limited to specific disease states such as schizophrenia. Clearly, it is important to find ways to improve the communication between basic researchers and psychopathologists.

To address obstacles such as these, NIMH cosponsors 3 research announcements that foster translational partnerships among scientists who study basic behavioral processes and those who study the etiology, diagnosis, treatment, and prevention of mental and behavioral disorders. PAR-06-355 (http://grants.nih.gov/grants/guide/pa-files/PAR-06-355.html) addresses the needs of investigators in the formative stages of the collaborative

research process, who are just beginning to explore translational research questions and designs. PAR-06-357 (http://grants.nih.gov/grants/guide/pa-files/PAR-06-357.html) is designed for investigators who have already initiated some collaborative partnerships but need to build additional translational research infrastructure. Finally, PAR-07-155 (http://grants.nih.gov/grants/guide/pa-files/PAR-07-155.html) supports studies that address a specific translational research question and possess sufficient pilot data to warrant an R01 research project grant. Grant support mechanisms such as these have fostered several successful collaborations between basic behavioral scientists and clinical schizophrenia researchers in other research areas.[98,99] Because productive translational partnerships exist in the areas of attention, memory, and perception, participants expressed hope that similar collaborations are possible between clinical and basic science researchers with common interests in social cognitive phenomena.

Identifying Key Research Topics

Social cognition in schizophrenia was confidently viewed as an area that offers substantial prospects for discovery. Over the course of the meeting, key research topics were identified that were partly, or largely, unexplored. Here is a partial listing:

- Alternative approaches to measuring social cognition in schizophrenia, including reaction time measures that would help resolve scaling problems, experience sampling methods in which subjects report their activities and emotions in daily life, role plays of generated social situations with confederates, and use of filmed (as opposed to written) vignettes of social interactions.
- The factor structure within social cognition and between social and basic cognition.
- The degree to which social cognition impairments are state related or trait related.
- The timing of social cognitive impairment relative to the development of functional impairment and onset of clinical symptoms (eg, social cognition during the prodromal phase).
- The extent to which the social cognitive impairments observed in schizophrenia occur in other psychiatric diagnoses (eg, bipolar disorder) or in schizoaffective disorder.
- The connection between social cognition and neural circuits that are known to be dysfunctional in schizophrenia.

To stimulate a translational research approach to address these questions, the group envisioned a series of meetings or conferences along the lines of MATRICS and CNTRICS that would facilitate collaborations between clinical researchers and basic scientists. However, participants believed that meetings by themselves are not sufficient to accomplish meaningful translational research in this area. The group encouraged the adoption of a new framework for constructing the relationships among scientists that includes such elements as extended meeting time (eg, workshops or institutes), finding ways to encourage pilot collaborative projects, and, if successful, launching subsequent investigator initiated grant applications of translational research.

Concluding Comments

This consensus-building meeting generated several conclusions. First, there was agreement on definitions of terms used in social cognition research in schizophrenia. Participants also noted that many potentially important aspects of social cognition that are studied in nonclinical samples remain understudied in schizophrenia. Second, there was agreement on the added value of social cognition for deconstructing clinical symptoms and for explaining functional outcome in schizophrenia. For these reasons, social cognition is starting to be viewed as a reasonable treatment target. Third, obstacles to development in this area were identified, including the relative lack of maturity compared with research on basic neurocognition and difficulty in building bridges among scientists from related disciplines. Fourth, the participants produced a listing of potentially informative research directions.

The meeting also clarified important steps that can be taken to stimulate advances in this area. The participants suggested ways to improve assessment of key constructs and to achieve the necessary psychometric qualities that will allow tests to be useful across a range of samples and research questions, including clinical trials. Although not a focus of discussion at this meeting, subsequent meetings were envisioned that will explore interfaces between social cognition in schizophrenia and cognitive and affective neuroscience, including human functional neuroimaging studies and animal models of social cognition. Finally, and perhaps most importantly, the participants emphasized that it takes time, energy, and resources to identify and build bridges between scientists of different disciplines. As reflected by the goals of the NIMH mechanisms and programs, a key step to achieving traction on these questions will be close collaborations among social scientists, neuroscientists, and clinical researchers. If such interdisciplinary bridges can be constructed, social cognition is well poised to explain many features of schizophrenia, from neural circuits, to clinical symptoms, to community functioning.

Acknowledgments

This article presents a summary of the discussion from a workshop supported by the NIMH. The views expressed are those of the authors and do not

necessarily reflect the official views of the NIMH, the National Institutes of Health, or any other branch of the US Department of Health and Human Services.

References

1. Brothers L. The neural basis of primate social communication. *Motiv Emot.* 1990;14:81–91.
2. Kunda Z. *Social Cognition: Making Sense of People.* Cambridge, MA: MIT Press; 1999.
3. Fiske ST, Taylor SE. *Social Cognition.* 2nd ed. New York, NY: McGraw-Hill Book Company; 1991.
4. Ostrom TM. The sovereignty of social cognition. In: Wyer RS, Srull TK, eds. *Handbook of Social Cognition.* Hillsdale, NJ: Erlbaum; Vol 1: 1984;1–37.
5. Penn DL, Corrigan PW, Bentall RP, Racenstein JM, Newman L. Social cognition in schizophrenia. *Psychol Bull.* 1997;121:114–132.
6. Bentall RP, Kaney S. Abnormalities of self-representation and persecutory delusions: a test of a cognitive model of paranoia. *Psychol Med.* 1996;26:1231–1237.
7. Heimberg C, Gur RE, Erwin RJ, Shtasel DL, Gur RC. Facial emotion discrimination: III. Behavioral findings in schizophrenia. *Psychiatry Res.* 1992;42:253–265.
8. Green MF, Olivier B, Crawley JN, Penn DL, Silverstein S. Social cognition in schizophrenia: recommendations from the MATRICS New Approaches Conference. *Schizophr Bull.* 2005;31:882–887.
9. Green MF, Nuechterlein KH, Gold JM, et al. Approaching a consensus cognitive battery for clinical trials in schizophrenia: the NIMH-MATRICS conference to select cognitive domains and test criteria. *Biol. Psychiatry*. 2004;56:301–307.
10. Nuechterlein KH, Barch DM, Gold JM, Goldberg TE, Green MF, Heaton RK. Identification of separable cognitive factors in schizophrenia. *Schizophr Res.* 2004;72:29–39.
11. Penn DL, Addington J, Pinkham A. Social cognitive impairments. In: Lieberman JA, Stroup TS, Perkins DO, eds. *American Psychiatric Association Textbook of Schizophrenia.* Arlington, VA: American Psychiatric Publishing Press, Inc.; 2006:261–274.
12. Baron-Cohen S, Wheelwright S, Hill J, Raste Y, Plumb I. The "Reading the mind in the eyes" test revised version: a study with normal adults, and adults with Asperger syndrome or high-functioning autism. *J Child Psychol Psychiatry*. 2001;42:241–251.
13. Frith CD. *The Cognitive Neuropsychology of Schizophrenia.* Hove, UK: Lawrence Erlbaum Associates; 1992.
14. Corcoran R. Theory of mind and schizophrenia. In: Corrigan PW, Penn DL, eds. *Social Cognition and Schizophrenia.* Washington, DC: American Psychological Association; 2001:149–174.
15. Brune M. "Theory of mind" in schizophrenia: a review of the literature. *Schizophr Bull.* 2005;31:21–42.
16. Leitman DI, Ziwich R, Pasternak R, Javitt DC. Theory of mind (ToM) and counterfactuality deficits in schizophrenia: misperception or misinterpretation? *Psychol Med.* 2006;36:1075–1083.
17. Toomey R, Schuldberg D, Corrigan PW, Green MF. Nonverbal social perception and symptomatology in schizophrenia. *Schizophr Res.* 2002;53:83–91.
18. Sergi MJ, Green MF. Social perception and early visual processing in schizophrenia. *Schizophr Res.* 2002;59:233–241.
19. Penn DL, Ritchie M, Francis J, Combs D, Martin J. Social perception in schizophrenia: the role of context. *Psychiatry Res.* 2002;109:149–159.
20. Fiske AP. The four elementary forms of sociality: framework for a unified theory of social relations. *Psychol Rev.* 1992;99:689–723.
21. Sergi MJ, Rassovsky Y, Nuechterlein KH, Green MF. Social perception as a mediator of the influence of early visual processing on functional status in schizophrenia. *Am J Psychiatry*. 2006;163:448–454.
22. Corrigan PW, Green MF. Schizophrenic patients' sensitivity to social cues: the role of abstraction. *Am J Psychiatry*. 1993;150:589–594.
23. Corrigan PW, Wallace CJ, Green MF. Deficits in social schemata in schizophrenia. *Schizophr Res.* 1992;8:129–135.
24. Subotnik KL, Nuechterlein KH, Green MF, et al. Neurocognitive and social cognitive correlates of formal thought disorder in schizophrenia patients. *Schizophr Res.* 2006;85:84–95.
25. Bellack AS, Sayers M, Mueser K, Bennett M. Evaluation of social problem solving in schizophrenia. *J Abnorm Psychol.* 1994;103:371–378.
26. Hansen DJ, St. Lawrence JS, Christoff KA. Effects of interpersonal problem-solving training with chronic aftercare patients on problem-solving component skills and effectiveness of solutions. *J Consult Clin Psychol.* 1985;53:167–174.
27. Zullow HM, Oettingen G, Peterson C, Seligman MEP. Pessimistic explanatory style in the historical record: CAVing. LBJ, Presidential candidates, and East versus West Berlin. *Am Psychol.* 1988;43:673–682.
28. Kinderman P, Bentall RP. A new measure of causal locus: the internal, personal, and situational attributions questionnaire. *Pers Individ Dif.* 1996;20:261–264.
29. Lee DA, Randall F, Beattie G, Bentall RP. Delusional discourse: an investigation comparing the spontaneous causal attributions of paranoid and non-paranoid individuals. *Psychol Psychother.* 2004;77(Pt 4):525–540.
30. Bentall RP, Corcoran R, Howard R, Blackwood N, Kinderman P. Persecutory delusions: a review and theoretical integration. *Clin Psychol Rev.* 2001;21:1143–1192.
31. Garety PA, Freeman D. Cognitive approaches to delusions: a critical review of theories and evidence. *Br J Clin Psychol.* 1999;38:(Pt 2):113–154.
32. Combs DR, Penn DL, Wicher M, Waldheter E. The Ambiguous Intentions Hostility Questionnaire (AIHQ): a new measure for evaluating hostile social-cognitive biases in paranoia. *Cogn Neuropsychiatry*. 2007;12(2):128–143.
33. Feldman-Barrett L, Salovey P. *The Wisdom in Feeling: Psychological Processes in Emotional Intelligence.* New York, NY: Guilford Press; 2002.
34. Mayer JD, Salovey P, Caruso DR, Sitarenios G. Emotional intelligence as a standard intelligence. *Emotion*. 2001;1:232–242.
35. Salovey P, Sluyter DJ. *Emotional Development and Emotional Intelligence.* New York, NY: Basic Books; 1997.
36. Edwards J, Jackson HJ, Pattison PE. Emotion recognition via facial expression and affective prosody in schizophrenia: a methodological review. *Clin Psychol Rev.* 2002;22:789–832.
37. Kohler CG, Bilker W, Hagendoorn M, Gur RE, Gur RC. Emotion recognition deficit in schizophrenia: association with symptomatology and cognition. *Biol Psychiatry*. 2000;48(2):127–136.
38. Salem JE, Kring AM, Kerr SL. More evidence for generalized poor performance in facial emotion perception in schizophrenia. *J Abnorm Psychol.* 1996;105:480–483.

39. Hariri AR, Bookheimer SY, Mazziotta JC. Modulating emotional responses: effects of a neocortical network on the limbic system. *Neuroreport*. 2000;11(1):43–48.
40. Whalen PJ, Rauch SL, Etcoff NL, McInerney SC, Lee MB, Jenike MB. Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *J Neurosci.* 1998;18:411–418.
41. Adolphs R. How do we know the minds of others? Domain-specificity, simulation, and enactive social cognition. *Brain Res.* 2006;1079:25–35.
42. Saxe R. Why and how to study theory of mind with fMRI. *Brain Res.* 2006;1079:57–65.
43. Phillips ML, Drevets WC, Rauch SL, Lane R. Neurobiology of emotion perception II: implications for major psychiatric disorders. *Biol Psychiatry*. 2003;54:515–528.
44. Pinkham AE, Penn DL, Perkins DO, Lieberman JA. Implications of a neural basis for social cognition for the study of schizophrenia. *Am J Psychiatry*. 2003;160:815–824.
45. Gur RE, McGrath C, Chan RM, et al. An fMRI study of facial emotion processing in patients with schizophrenia. *Am J Psychiatry*. 2002;159:1992–1999.
46. Uhlhaas PJ, Phillips WA, Schenkel LS, Silverstein SM. Theory of mind and perceptual context-processing in schizophrenia. *Cogn Neuropsychiatry*. 2006;11:416–436.
47. Schenkel LS, Spaulding WD, Silverstein SM. Poor premorbid social functioning and theory of mind deficit in schizophrenia: evidence of reduced context processing? *J Psychiatr Res.* 2005;39:499–508.
48. Phillips WA, Singer W. In search of common foundations for cortical computation. *Behav Brain Sci.* 1997;20:657–683; discussion 683–722.
49. Phillips WA, Silverstein SM. Convergence of biological and psychological perspectives on cognitive coordination in schizophrenia. *Behav Brain Sci.* 2003;26:65–82; discussion 82–137.
50. Garety PA, Freeman D, Jolley S, et al. Reasoning, emotions, and delusional conviction in psychosis. *J Abnorm Psychol.* 2005;114:373–384.
51. Bentall RP, Swarbrick R. The best laid schemas of paranoid patients: autonomy, sociotropy and need for closure. *Psychol Psychother.* 2003;76(Pt 2):163–171.
52. Randall F, Corcoran R, Day JC, Bentall RP. Attention, theory of mind, and causal attributions in people with persecutory delusions: a preliminary investigation. *Cogn Neuropsychiatry*. 2003;8:287–294.
53. Horan WP, Kring AM, Blanchard JJ. Anhedonia in schizophrenia: a review of assessment strategies. *Schizophr Bull.* 2006;32:259–273.
54. Sergi MJ, Rassovsky Y, Widmark C, et al. Social cognition in schizophrenia: relationships with neurocognition and negative symptoms. *Schizophr Res.* 2007;90:316–324.
55. Kirkpatrick B, Fenton W, Carpenter WT, Marder SR. The NIMH-MATRICS consensus statement on negative symptoms. *Schizophr Bull.* 2006;32:296–303.
56. Penn DL. Cognitive rehabilitation of social deficits in schizophrenia: a direction of promise or following a primrose path? *Psychosoc Rehabil J.* 1991;15:27–41.
57. Kee KS, Green MF, Mintz J, Brekke JS. Is emotional processing a predictor of functional outcome in schizophrenia? *Schizophr Bull.* 2003;29:487–497.
58. Corrigan PW, Toomey R. Interpersonal problem solving and information processing in schizophrenia. *Schizophr Bull.* 1995;21:395–403.
59. Mueser KT, Doonan B, Penn DL, et al. Emotion recognition and social competence in chronic schizophrenia. *J Abnorm Psychol.* 1996;105:271–275.
60. Penn DL, Spaulding WD, Reed D, Sullivan M. The relationship of social cognition to ward behavior in chronic schizophrenia. *Schizophr Res.* 1996;20:327–335.
61. Couture SM, Penn DL, Roberts DL. The functional significance of social cognition in schizophrenia: a review. *Schizophr Bull.* 2006;32:Suppl 1:S44–S63.
62. Silverstein SM. Information processing, social cognition, and psychiatric rehabilitation of schizophrenia. *Psychiatry*. 1997;60:327–340.
63. Green MF, Nuechterlein KH. Should schizophrenia be treated as a neurocognitive disorder? *Schizophr Bull.* 1999;25:309–319.
64. Brekke JS, Kay DD, Kee KS, Green MF. Biosocial pathways to functional outcome in schizophrenia. *Schizophr Res.* 2005;80:213–225.
65. Vauth R, Rusch N, Wirtz M, Corrigan PW. Does social cognition influence the relation between neurocognitive deficits and vocational functioning in schizophrenia? *Psychiatry Res.* 2004;128:155–165.
66. Addington J, Saeedi H, Addington D. Facial affect recognition: a mediator between cognitive and social functioning in schizophrenia? *Schizophr Res.* 2006;85:142–150.
67. Pinkham AE, Penn DL. Neurocognitive and social cognitive predictors of interpersonal skill in schizophrenia. *Psychiatry Res.* 2006;143:167–178.
68. Gur RE, Kohler CG, Ragland JD, et al. Flat affect in schizophrenia: relation to emotion processing and neurocognitive measures. *Schizophr Bull.* 2006;32:279–287.
69. Allen DN, Strauss GP, Donohue B, van Kammen DP. Factor analytic support for social cognition as a separable cognitive domain in schizophrenia. *Schizophr Res.* 2007;93:325–333.
70. Fiszdon JM, Richardson R, Greig T, Bell MD. A comparison of basic and social cognition between schizophrenia and schizoaffective disorder. *Schizophr Res.* 2007;91:117–121.
71. Jacobs DH, Shuren J, Bowers D, Heilman K. Emotional facial imagery, perception, and expression in Parkinson's disease. *Neurology*. 1995;45:1696–1702.
72. Adolphs R, Tranel D, Damasio H, Damasio A. Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*. 1994;372:669–672.
73. Mitchell JP, Banaji MR, Macrae CN. General and specific contributions of the medial prefrontal cortex to knowledge about mental states. *Neuroimage*. 2005;28:757–762.
74. Mitchell JP, Neil Macrae C, Banaji MR. Forming impressions of people versus inanimate objects: social-cognitive processing in the medial prefrontal cortex. *Neuroimage*. 2005;26:251–257.
75. Saxe R, Powell LJ. It's the thought that counts: specific brain regions for one component of theory of mind. *Psychol Sci.* 2006;17:692–699.
76. Gur RC, Schroeder L, Turner T, et al. Brain activation during facial emotion processing. *Neuroimage*. 2002;16:651–662.
77. Beer JS, Ochsner KN. Social cognition: a multi-level analysis. *Brain Res.* 2006;1079:98–105.
78. Macrae CN, Quinn KA, Mason MF, Quadflieg S. Understanding others: the face and person construal. *J Pers Soc Psychol.* 2005;89:686–695.
79. Cunningham WA, Zelazo PD. Attitudes and evaluations: a social cognitive neuroscience perspective. *Trends Cogn Sci.* 2007;11(3):97–104.

80. Toomey R, Seidman LJ, Lyons MJ, Faraone SV, Tsuang MT. Poor perception of nonverbal social-emotional cues in relatives of schizophrenic patients. *Schizophr Res.* 1999;40:121–130.
81. Kee KS, Horan WP, JM, Green MF. Do the siblings of schizophrenia patients demonstrate affect perception deficits? *Schizophr Res.* 2004;67:87–94.
82. Addington J, Addington D. Facial affect recognition and information processing in schizophrenia and bipolar disorder. *Schizophr Res.* 1998;32:171–181.
83. Wolwer W, Streit M, Polzer U, Gaebel W. Facial affect recognition in the course of schizophrenia. *Eur Arch Psychiatry Clin Neurosci.* 1996;246(3):165–170.
84. Pinkham AE, Penn DL, Perkins DO, Graham K, Siegel M. Emotion perception and the course of psychosis: a comparison of individuals at risk, and early and chronic schizophrenia spectrum illness. *Cogn Neuropsychiatry*. 2007;12:198–212.
85. Addington J, Penn DL, Woods SW, Addington D, Perkins D. Facial affect recognition in individuals at clinical high risk for psychosis. *Br J Psychiatry*. In press.
86. Martin JA, Penn DL. Attributional style in schizophrenia: an investigation in outpatients with and without persecutory delusions. *Schizophr Bull.* 2002;28:131–141.
87. Bentall RP, Kaney S. Attributional lability in depression and paranoia. *Br J Clin Psychol.* 2005;44(Pt 4):475–488.
88. Harvey PD, Patterson TL, Potter LS, Zhong K, Brecher M. Improvement in social competence with short-term atypical antipsychotic treatment: a randomized, double-blind comparison of quetiapine versus risperidone for social competence, social cognition, and neuropsychological functioning. *Am J Psychiatry*. 2006;163:1918–1925.
89. Sergi MJ, Green MF, Widmark C, et al. Social cognition and neurocognition: effects of risperidone, olanzapine, and haloperidol. *Am J Psychiatry*. 2007;164:1585–1592.
90. Combs DR, Adams SD, Penn DL, Roberts D, Tiegreen J, Stem P. Social Cognition and Interaction Training (SCIT) for inpatients with schizophrenia spectrum disorders: preliminary findings. *Schizophr Res.* 2007;91: 112–116.
91. Roncone R, Mazza M, Frangou I, et al. Rehabilitation of theory of mind deficit in schizophrenia: a pilot study of metacognitive strategies in group treatment. *Neuropsychol Rehabil.* 2004;14:421–435.
92. Hogarty GE, Flesher S, Ulrich R, et al. Cognitive enhancement therapy for schizophrenia. *Arch Gen Psychiatry*. 2004;61:866–876.
93. Wolwer W, Frommann N, Haufmann S, Piaszek A, Streit M, Gaebel W. Remediation of impairments in facial affect recognition in schizophrenia: efficacy and specificity of a new training program. *Schizophr Res.* 2005;80:295–303.
94. Penn DL, Roberts DL, Combs D, Sterne A. Best practices: the development of the social cognition and interaction training program for schizophrenia spectrum disorders. *Psychiatr Serv.* 2007;58:449–451.
95. Horan WP, Kern RS, Penn DL, Green MF. Social cognition training for individuals with schizophrenia: emerging evidence. *Am J Psychiatr Rehabil*. In press.
96. Morrison RL, Bellack AS, Mueser KT. Deficits in facial-affect recognition and schizophrenia. *Schizophr Bull.* 1988;14:67–83.
97. Bentall RP, Kaney S, Dewey ME. Paranoia and social reasoning: an attribution theory analysis. *Br J Clin Psychol.* 1991;30(Pt 1):13–23.
98. Luck SJ, Fuller RL, Braun EL, Robinson B, Summerfelt A, Gold JM. The speed of visual attention in schizophrenia: electrophysiological and behavioral evidence. *Schizophr Res.* 2006;85:174–195.
99. Nuechterlein KH, Pashler HE, Subotnik KL. Translating basic attentional paradigms to schizophrenia research: reconsidering the nature of the deficits. *Dev Psychopathol.* 2006;18:831–851.