

## Social Cognition in Schizophrenia

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**Social cognition in schizophrenia is a rapidly emerging area of study. Because the number and diversity of studies in this area have increased, efforts have been made to better define terms and provide organizing frameworks. A key challenge confronting the study of social cognition in schizophrenia is building bridges between clinical scientists and social neuroscientists. The articles in this theme summarize data-based studies that have attempted to build or strengthen such bridges to better understand the neural bases of social cognitive impairment in schizophrenia.**

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From time to time, certain terms break into the research literature before they are adequately defined. They show up in titles of articles and as key search terms, even before there is a consensus on their meaning. Such terms have *cachet* before they have “precision.” Social cognition in schizophrenia is one of these terms.

Figure 1 shows the rapid increase in the number of published articles that had “social cognition” as a key term linked with “schizophrenia” based on a search by PsycINFO.

As can be readily seen, the use of the linked terms increased dramatically from 2003 to 2006. The increase could be simply due to a shift in labeling, ie, social cognition may have been used to describe activities and paradigms that would have been previously been called something else. However, it is much more likely that the figure reflects a true reorientation of research interest because it coincides with several converging trends. For example, we now have an accumulation of information on the role of social cognition in daily functioning of schizophrenia patients.<sup>1–5</sup> We also have more information on the role of social cognition in formation of par-

ticular symptoms, such as paranoia.<sup>6–8</sup> Finally, the increased interest in social cognition in schizophrenia mirrors a similar pattern within cognitive neuroscience as it has rapidly accommodated social and affective neuroscience. Wherever cognitive science goes, schizophrenia research is sure to follow.

Of course, the study of social cognition in healthy individuals is well established. The topics in normal social cognition are rather broad, and only a subset of the areas has been carried into schizophrenia research. For example, stereotyping and prejudice are key topics for social cognition in healthy people, but their value in understanding schizophrenia is less obvious. Similarly, psychopathology typically lumps emotion processing with social cognition, whereas these topics can form the basis of entirely separate courses in a psychology curriculum.

So what topics do get covered in the social cognition of schizophrenia? This question was considered in a session at the final meeting of the National Institute of Mental Health (NIMH) Initiative, Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS).<sup>9</sup> The definitions of terms were further refined in a subsequent NIMH-sponsored consensus meeting that also identified obstacles to progress and research priorities.<sup>10</sup> These discussions led to an agreement that the research in this area can be roughly divided into 5 partially overlapping domains: emotion processing, social perception, social knowledge, theory of mind, and attributional biases. As emphasized in the published summaries of these meetings, the factor structure of these domains and the relationships among them are not known, so some areas may be gainfully split or clumped.

Another way to approach the organization of social cognitive domains comes from social neuroscience. The NIMH Initiative, Cognitive Neuroscience Treatment Research to Improve Cognition in Schizophrenia (CNTRICS)<sup>11</sup> was launched to consider ways to better transition measures from cognitive and social neuroscience to clinical trials in schizophrenia. At the first CNTRICS meeting, Kevin Ochsner<sup>12</sup> reviewed the functional neuroanatomy of social and emotional processes and, based on that review, proposed 5 constructs that comprise a social-emotional pathway. These domains include (1) acquisition of socio-affective values and responses, (2) recognizing and responding to social-affective stimuli, (3) “embodied” simulation or low-level mental state inference, (4) high-level mental

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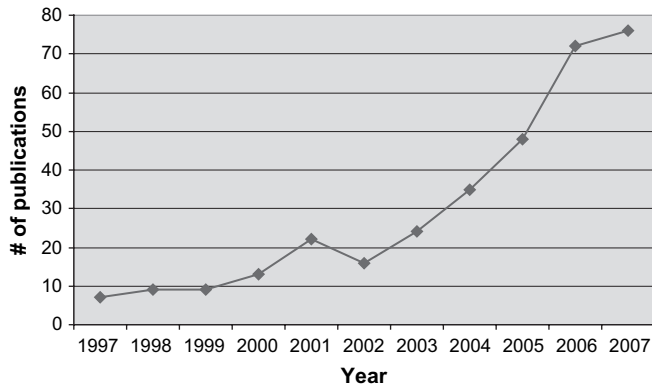


Fig. 1. Social cognition and schizophrenia publications by year.

state/trait inference, and (5) context-sensitive regulation. The first of these areas (ie, acquisition of values and responses) involves the development of reinforcement value and may fit better as part of negative symptoms. The remaining domains, however, are clearly relevant to social cognition in schizophrenia. Hence, the field now has 2 useful organizational schemes, one that is based on a partition of recent work in schizophrenia and the other based on the nonclinical neuroimaging literature. Despite the different origins, the overlap between the 2 schemes is quite good. For example, theory of mind is part of the higher level mental state inference (construct 4) and attributional bias is part of the context-sensitive regulation (construct 5).

A key challenge confronting the study of social cognition in schizophrenia is how to build bridges between basic and clinical scientists. The articles in this theme have all attempted to build or strengthen such bridges. Given the way these articles orient to social neuroscience, it is useful to observe how they map onto the components of Ochsner's model. Indeed, we can sequence the articles according to their placement in the social-emotional processing stream.

Two of the articles examined the neural correlates of construct 2: acquisition of socio-affective values and responses. Leitman et al.<sup>13</sup> examined the functional connectivity of fear detection in faces, using a novel technique (ie, CORANOVA). This technique measures the significance of differences among correlated correlations and can help determine connectivity among neural nodes of the affective appraisal network by testing differences in correlation strengths for time-locked activity between patients and controls. The results, while preliminary, suggest that abnormalities in the sensation and integration of visual information may underlie fear evaluation dysfunction in schizophrenia.

Wynn et al.<sup>14</sup> examined construct 2 using event-related potentials associated with facial affect processing in schizophrenia patients and controls. Using emotional and nonemotional tasks, 3 separate event-related components were measured (P100, N170, N250) that corre-

spond to different stages of visual, facial, or facial emotion processing. In this study, patients showed reduced amplitude for the N250 wave, which is associated with face emotion processing, even though groups did not differ in performance level during the task. The reduced amplitude in patients was noted across emotion and nonemotion tasks, suggesting a general inefficiency in later stage decoding.

Pinkham et al.<sup>15</sup> examined a task that spans construct 2 and construct 4. They used functional magnetic resonance imaging to examine regional brain activity associated with evaluating faces as trustworthy or not trustworthy in 3 groups of subjects: paranoid patients, nonparanoid patients, and healthy controls. The controls and nonparanoid patients showed increased activation in regions of interest for faces deemed untrustworthy, but the paranoid patients did not. Aside from this link between neural activity and an important clinical feature of schizophrenia, this study also found, across groups, that greater activation was associated with better social functioning (thereby linking neural processes and community functioning).

The article by Park et al.<sup>16</sup> examined construct 3 (ie, embodied simulation). These authors demonstrated that patients have significant deficits in their ability to imitate hand and mouth movements, as well as facial gestures. These deficits correlated with negative symptom severity and reduced social functioning. It is suggested that problems in these imitation tasks may reflect an inability to construct or manipulate internal representations of social actions.

Finally, Andreasen et al.<sup>17</sup> focused the fourth construct (ie, high-level mental state inference) and examined the neural substrates of theory of mind with positron emission tomography. This study utilized a valuable sample of patients who were medication naive or had been off of antipsychotic medications for at least 3 weeks. The authors found reduced blood flow in brain areas that have been implicated in previous studies (several cortical regions, thalamus, and cerebellum) and also found increased blood flow primarily in the right hemisphere, suggesting a possible compensatory mechanism for the deficits.

There are many clinical research laboratories working in this area, and the articles in this theme represent a limited sampling. Nonetheless, we hope that these articles convey a sense of the creativity, energy, and relevance in this emerging area of exploration.

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