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Social cognition in schizophrenia: Relationships with neurocognition and negative symptoms

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Abstract

Despite the growing importance of social cognition in schizophrenia, fundamental issues concerning the nature of social cognition in schizophrenia remain unanswered. One issue concerns the strength of the relationships between social cognition and key features of the disorder such as neurocognitive deficits and negative symptoms. The current study employed structural equation modeling to examine three key questions regarding the nature of social cognition in schizophrenia: 1) Are social cognition and neurocognition in schizophrenia better modeled as one or two separate constructs? 2) Are social cognition and negative symptoms in schizophrenia better modeled as one or two separate constructs?, and 3) When social cognition, neurocognition, and negative symptoms are included in a single model, is social cognition more closely related to neurocognition or to negative symptoms? In this cross sectional study, one hundred outpatients with schizophrenia or schizoaffective disorder were administered measures of social cognition, neurocognition, and negative symptoms. A two-factor model that represented social cognition and neurocognition as separate constructs fit the data significantly better than a one-factor model, suggesting that social cognition and neurocognition are distinct, yet highly related, constructs. Likewise, a two-factor model that represented social cognition and negative symptoms as separate constructs fit the data significantly better than a one-factor model, suggesting that social cognition and negative symptoms are distinct constructs. A three-factor model revealed that the relationship between social cognition and neurocognition was stronger than the relationship between social cognition and negative symptoms. The current findings start to provide insights into the structure of social cognition, neurocognition, and negative symptoms in schizophrenia. © 2006 Elsevier B.V. All rights reserved.

Keywords: Schizophrenia; Social cognition; Neurocognition; Negative symptoms; Social perception; Emotion perception

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1. Introduction

Social cognition, the ability to construct mental representations about others, oneself, and relations between others and oneself, facilitates skillful social

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interactions (Adolphs, 2001; Brothers, 1990). Relative to healthy persons, schizophrenia patients display impairments in domains of social cognition such as emotion processing (Archer et al., 1994; Pollard et al., 1995), social perception (Corrigan and Green, 1993; Toomey et al., 2002), theory of mind (Greig et al., 2004; Roncone et al., 2002), and social knowledge (Corrigan and Addis, 1995; Penn et al., 2002). Correlational and structural equation modeling analyses strongly suggest that social cognition serves as a mediating link between neurocognition and community functioning in schizophrenia (e.g., Brekke et al., 2005; Sergi et al., 2006; Vauth et al., 2004). To the extent that social cognition acts as a mediator, it may be more closely related to community functioning than neurocognition, and hence a logical target for psychosocial and pharmacological interventions. As an indication of social cognition's increasing visibility in schizophrenia research, the investigators of the NIMH Initiative Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) identified social cognition as one of seven domains that should be routinely assessed in intervention studies of schizophrenia (Green et al., 2004).

Despite the growing focus on social cognition in schizophrenia, fundamental issues concerning the nature of social cognition in schizophrenia remain unanswered (Green et al., 2005). One unresolved issue concerns the relationship between social cognition and key features of the disorder such as neurocognitive deficits and negative symptoms.

The current study's sample size (100 persons with schizophrenia or schizoaffective disorder) and diverse range of assessments enabled us to employ structural equation modeling to examine three research questions regarding the nature of social cognition in schizophrenia: 1) Are social cognition and neurocognition in schizophrenia better modeled as one or two separate constructs?, 2) Are social cognition and negative symptoms in schizophrenia better modeled as one or two separate constructs?, and 3) When social cognition, neurocognition, and negative symptoms are included in a single model, is social cognition more closely related to neurocognition or to negative symptoms?

For the first question, we anticipated that structural equation modeling would show that social cognition and neurocognition are better represented as two separate constructs for two reasons. First, social cognition involves emotional and social processing while neurocognition typically does not. Second, recent studies suggest that social cognition contributes variance to functional outcome in schizophrenia beyond the vari-

ance explained by neurocognition (e.g., Brekke et al., 2005; Sergi et al., 2006). We hypothesized that a two-factor model that represented social cognition and neurocognition as separate constructs would fit the data significantly better than a one-factor model that represented both domains as a single construct. As few studies have examined relations between social cognition and negative symptoms in schizophrenia, we did not have a strong prediction about whether social cognition and negative symptoms are better represented as a single construct or as separate constructs. For the same reason, we did not have a strong prediction about whether social cognition is more closely related to neurocognition or to negative symptoms.

2. Method

2.1. Participants

One hundred outpatients with schizophrenia or schizoaffective disorder (91 males and 9 females) completed the measures included in the current analyses as part of their baseline assessment in a Veterans Affairs study of the therapeutic and cognitive effects of atypical and conventional antipsychotic medications (MF Green, P.I.). All participants provided written informed consent after complete description of the medication study. This study was carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans. The consent and recruitment procedures employed were approved by the Institutional Review Boards of the Veterans Affairs Greater Los Angeles Healthcare System (VAGLAHS), the VA Long Beach Healthcare System (VALBHS), and the VA San Diego Healthcare System (VASDHS). The participants were recruited from treatment clinics in the three VA healthcare systems as well as local board-and-care facilities. All participants met criteria for schizophrenia or schizoaffective disorder based upon interview with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First et al., 1997). All SCID interviewers were trained to administer the SCID in the Treatment Unit of the VA Mental Illness Research Education Clinical Center (MIRECC) and demonstrated agreement between their ratings and the consensus ratings of the MIRECC's expert diagnosticians (minimum Kappa coefficient of .80). Psychiatric symptoms were rated with the Brief Psychiatric Rating Scale (BPRS; Ventura et al., 1993). All BPRS raters were trained to minimum intraclass correlation coefficients of 0.80 for the BPRS, based on their agreement with consensus ratings of the

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