

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.

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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

MIRECC's expert diagnosticians. The symptom rating subscales of the BPRS indicate that the outpatients were clinically stable at the time of testing (positive symptoms assessed with the thinking disturbance index: mean=2.7, SD=1.5; negative symptoms assessed with withdrawal/retardation index: mean=2.2, SD=1.0; BPRS ratings range from 1 to 7). The mean age of the participants was 49.0 years (SD=7.1). The mean education of the participants was 12.6 years (SD=2.1). Participants were individually administered measures of social cognition, neurocognition, and negative symptoms as part of an assessment battery that lasted about 3 h.

2.2. Measures

2.2.1. Social cognition

Participants were administered two measures of emotion perception: the Facial Emotion Identification Test (FEIT; Kerr and Neale, 1993) and the Voice Emotion Identification Test (VEIT; Kerr and Neale, 1993). The FEIT consists of 19 black and white still photographs that are presented on videotape. The photographs, developed by Izard (1971) and Ekman (1976), were presented for approximately 15 s each, with an interval of 10 s between photographs. After each photograph, the subjects were asked to circle on an answer sheet the one emotion of six basic emotions (i.e., happy, angry, afraid, sad, surprised, and ashamed) that best described the emotion expressed by the face in the photograph. The number of items correct on the FEIT was used as an indicator in the structural equation modeling (SEM) analyses. The VEIT consists of 21 audio recordings of verbally presented statements with neutral content (e.g. 'Fish can jump out of the water', 'He tossed the bread to the pigeons'). The voice tone for each statement conveyed one of the same six emotions included in the FEIT. The subjects listened to each sentence and then circled on an answer sheet the one emotion of the six basic emotions that best described the emotion expressed by the speaker's tone of voice. The number of items correct on the VEIT was included as an indicator in the SEM analyses.

Participants were administered two measures of social perception: the Half-Profile of Nonverbal Sensitivity (Half-PONS; Ambady et al., 1995) and the Interpersonal Perception Task-15 (IPT-15; Costanzo and Archer, 1993). The Half-PONS consists of the first 110 scenes of the Profile of Nonverbal Sensitivity (PONS, Rosenthal et al., 1979). Scenes of this videotape-based measure last 2 s and contain the facial expressions, voice intonations, and/or bodily gestures of

a Caucasian female; one to three social cues per scene. After watching each scene, participants were asked to select from two labels (e.g., saying a prayer, talking to a lost child) the label that best described a situation that would give rise to the social cue(s) observed. As in prior studies that have used the PONS in schizophrenia (e.g., Monti and Fingeret, 1987; Toomey et al., 1997), the administration procedure was modified to reduce the measure's demands on sustained attention and reading comprehension. Prior to each scene, the videotape was paused as the experimenter read the two possible labels aloud and the participant read the labels silently from a 4" by 6" index card. To ensure that the participants understood the task, a practice sample of five scenes was randomly selected from the second 110 items of the PONS and administered prior to the scored scenes. The number of items correct on the Half PONS was included as an indicator in the SEM analyses.

The IPT-15 was also used to assess social perception. The scenes of this videotape-based measure are unscripted interpersonal interactions that range from 30 to 90 s in length. Scenes contain multiple social cues (e.g., facial expressions, verbalizations, voice tone/paraverbals, gestures, situational context, proxemics, haptics) displayed by one to four persons. One multiple-choice question is asked about each scene. Questions relate to varied aspects of social perception such as intimacy, status, and veracity. To reduce the measure's demands on sustained attention and reading comprehension, the videotape was paused prior to each scene while the multiple choice question and potential responses were read aloud by the experimenter and read silently by the participant. The number of items correct on the IPT-15 was included as an indicator in the SEM analyses.

2.2.2. Neurocognition

The neurocognitive measures included tests of attention/vigilance, executive functioning/problem solving, speed of processing, motor dexterity, verbal fluency, verbal episodic (secondary) memory, and verbal working memory. The measures used to assess neurocognition are displayed in Table 1.

2.2.3. Negative symptoms

The Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1984) was used to assess negative symptoms. This interview-based rating scale contains anchored items (0=not at all, 5=severe) that lead to global ratings of five negative symptoms: affective flattening, avolition, anhedonia/asociality, avolition/apathy, and inattention. SANS items and global ratings

Table 1
Measures of neurocognition

Measure	Construct	Description	Indicator
California Verbal Learning Test (CVLT; Delis et al., 1983)	Verbal episodic (secondary) memory	This word list learning task asks participants to recall 16 words from four taxonomic categories presented over a series of five trials.	Words correctly recalled in the five trials
Wisconsin Card Sorting Test (WCST; Heaton et al., 1993)	Executive functioning and problem solving skill	Participants use performance feedback to match individually presented stimulus cards to one of four key cards based on the shape, number, or color of the symbols on each card.	Categories completed
Degraded-Stimulus Continuous Performance Test (DS-CPT; Nuechterlein and Asamow, 1992)	Attention/visual vigilance	This computer-based measure presents a series of single digits at a rate of one per second and asks participants to press a response button whenever they see the target number '0'	Sensitivity score (i.e., an index of the ability to discriminate hits from false alarms)
Letter–Number Span Test (LNS; Gold et al., 1997)	Verbal working memory	Participants are verbally presented strings of letters and numbers of increasing length and asked to repeat them back in the same order, or by re-ordering them numbers first in numerical order then letters in alphabetical order	Letter–number strings correct in both test conditions (with and without reordering)
Digit Symbol-Coding subtest of the Wechsler Adult Intelligence Scale - III (DS-C; Wechsler, 1997)	Speed of processing	The participant copies symbols that are paired with numbers. Using a key, the subject draws each symbol under the corresponding number.	Total correct responses in 120 s
The Grooved Pegboard Test (GPT; Matthews and Klove, 1964)	Motor dexterity	The participant inserts grooved metal pegs into a metal template with corresponding grooved slots as quickly as possible.	Time to completion for both hands (left plus right hand)
Controlled Oral Word Association Test (COWAT, Benton and Hamsher, 1989)	Verbal fluency	Allowed 60 s per trial, participants generate as many words as possible that begin with a certain letter of the alphabet (e.g., F, A, S) and then as many animal words as possible.	Total number of correct words generated (F+A+S+animals)

range from 0 (not at all) to 5 (severe). All SANS raters were trained to minimum intraclass correlation coefficients of 0.75, based on their agreement with consensus ratings of the MIRECC's expert diagnosticians. SANS global ratings for affective flattening (SANS-AF), alogia (SANS-AL), anhedonia/asociality (SANS-ANH), and avolition/apathy (SANS-AV) were included as indicators in the SEM analyses (the global rating for inattention was not included because of its potential overlap with performance-based attention).

2.3. Statistical analyses

Structural equation modeling (SEM; Bentler, 1996) was employed to examine the relationships between social cognition, neurocognition, and negative symptoms. SEM uses a combination of confirmatory factor analysis and multiple regression to determine relations among constructs and measured variables (Ullman, 2001). Constructs (i.e. latent variables) are estimated in SEM through a factor analytic strategy using theoretically related measures (i.e. indicator variables). Factor loadings specify the association between an indicator variable and a latent variable. Regression analyses determine the relations between the latent variables.

Associations reported between latent variables are path coefficients, typically presented in a standardized form.

SEM was first used to assess the relationship between social cognition and neurocognition in schizophrenia. A one-factor model (Fig. 1) with one latent variable and eleven indicators that represented social cognition and neurocognition as a single construct was compared to a two-factor model (Fig. 2) with two latent variables that represented social cognition and neurocognition as two separate constructs. The relative fit of the models was tested with chi-square coefficients. We are aware that neurocognition in schizophrenia is often represented in terms of separable dimensions of cognitive deficits (e.g., Nuechterlein et al., 2004). Hence, the grouping of neurocognition as a single construct is somewhat artificial. We did so in the current analysis to reduce the complexity of the models, minimize the number of parameters, and maximize the robustness of the findings.

SEM was also used to assess the relationship between social cognition and negative symptoms in schizophrenia. A one-factor model with one latent variable and eight indicators that represented social cognition and neurocognition as a single construct was compared to a two-factor model with two latent variables, social

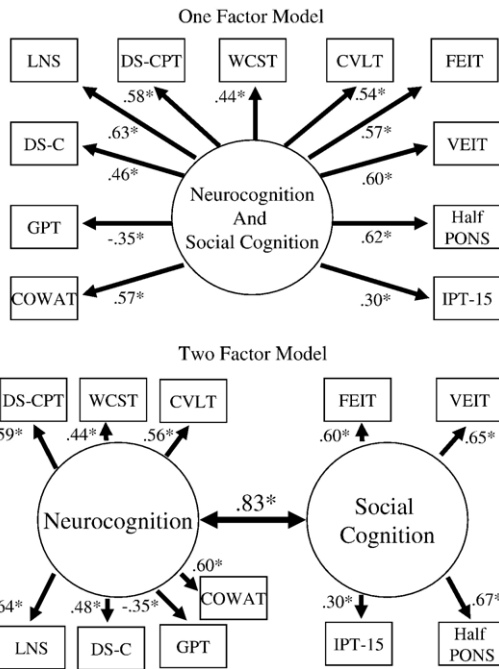


Fig. 1. Panel A: A one-factor SEM model that represents social cognition and neurocognition as a single latent variable. Circle represents the latent variable, rectangles represent measured variables. *=estimated parameters. All coefficients (presented in standardized form) are significant at the .05 level, $\chi^2(44, N=100)=38.58, p=.70$. Panel B: A two-factor SEM model that represents social cognition and neurocognition as two separate latent variables. Circles represent latent variables, rectangles represent measured variables. *=estimated parameters. All coefficients (presented in standardized form) are significant at the .05 level, $\chi^2(43, N=100)=34.39, p=.82$.

cognition and negative symptoms. The relative fit of the models was tested with chi-square coefficients. Similar to neurocognition, negative symptoms can be represented as two separate factors, but was treated as one for the purposes of these analyses (Blanchard and Cohen, 2006). Finally, we used SEM to examine the strength of the association between social cognition and neurocognition relative to the association between social cognition and negative symptoms. A three-factor model that represented social cognition, neurocognition, and negative symptoms as separate constructs was created in order to compare the path coefficient (partial correlation) between social cognition and neurocognition to the path coefficient between social cognition and negative symptoms.

The models were estimated with the EQS Structural Equation Package (Bentler, 1996), using maximum likelihood solution. Because 17 of the 100 participants had missing data on some of the variables, analyses were performed using two methods for handling missing data: listwise deletion (in which cases that do not have

data on all variables are omitted) and pairwise deletion (where a correlation between each pair of variables is calculated from all cases that have valid data on those two variables) (Jamshidian and Bentler, 1999). As the pattern of results from the two computation methods was virtually identical, only the results obtained using the pairwise deletion method are reported.

3. Results

Correlations between the indicator variables are displayed in Table 2. All of the indicator variables were examined for normality. Square root transformations normalized the distributions of the SANS global ratings of affective flattening and alolia as well as the distribution of the sensitivity score of the DS-CPT.

To examine the separateness of social cognition from neurocognition, a one-factor model that represented social cognition and neurocognition as a single latent variable was compared to a two-factor model that

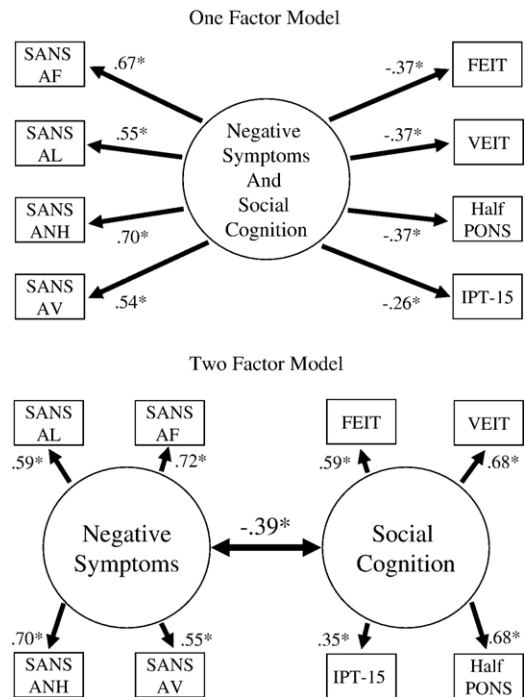


Fig. 2. Panel A: A one-factor SEM model that represents social cognition and negative symptoms as a single latent variable. Circle represents the latent variable, rectangles represent measured variables. *=estimated parameters. All coefficients (presented in standardized form) are significant at the .05 level, $\chi^2(20, N=100)=44.83, p<.01$. Panel B: A two-factor SEM model that represents social cognition and negative symptoms as two separate latent variables. Circles represent latent variables, rectangles represent measured variables. *=estimated parameters. All coefficients (presented in standardized form) are significant at the .05 level, $\chi^2(19, N=100)=12.28, p=.87$.

Table 2
Zero order correlations of the indicator variables

Indicator	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. FEIT	–													
2. VEIT	.36**	–												
3. Half PONS	.36**	.51**	–											
4. IPT-15	.10	.23*	.22*	–										
5. CVLT	.16	.31**	.21*	.24*	–									
6. WCST	.19	.25*	.37**	.17	.25*	–								
7. DS-CPT	.38**	.26*	.41**	.12	.32**	.28**	–							
8. LNS	.34**	.37**	.42**	.23*	.43**	.32**	.28**	–						
9. DS-C	.20	.23*	.20	.14	.28**	.18	.31**	.20	–					
10. GPT	-.30**	-.22*	-.13	.03	-.01	-.08	-.18	-.12	-.23*	–				
11. COWAT	.21*	.30**	.42**	.14	.31**	.33**	.33**	.43**	.38**	-.13	–			
12. SANS-AF	-.26**	-.19	-.08	-.13	-.21	-.19	-.30**	-.16	-.23*	.31**	-.27	–		
13. SANS-AL	-.11	-.17	-.17	-.01	-.20	-.27*	-.15	-.07	.02	.18	-.28	.49**	–	
14. SANS-ANH	-.19	-.19	-.26*	-.18	-.16	-.10	-.23*	-.17	-.11	.22*	-.23	.53**	.34**	–
15. SANS-AV	-.16	-.16	-.17	-.05	-.05	-.25*	-.08	.03	-.06	.13	-.12	.34**	.41**	.43**

* $p < .05$; ** $p < .01$; two-tailed.

represented social cognition and neurocognition as separate latent variables (see Fig. 1, Panels A and B). The independence model, testing whether or not the observed data fit the expected data, was rejected, $\chi^2 (55, N=100) = 197.26, p < .001$. (The chi-square for the independence model should always be significant, indicating that there is a relationship among the variables. The independence model was the same for the two models.) Both the one-factor and two-factor models fit the data well, as indicated by the non-significant chi-squares and the moderate-to-high loadings of all indicators on their respective latent variables (all were significant at the .05 level). As can be seen in Panel B of Fig. 1, a high covariance was found between the latent variables of social cognition and neurocognition (standardized coefficient = .83, $p < .05$). Nonetheless, testing the difference between the chi-squares of the two models revealed that the two-factor model (the model that represented social cognition and neurocognition as separate constructs) fit the data significantly better than the one-factor model, $\chi^2 (1, N=100) = 4.19, p < .05$.

Next, we examined the separateness of social cognition from negative symptoms. A one-factor model that represented social cognition and negative symptoms as a single latent variable was compared to a two-factor model that represented social cognition and negative symptoms as separate latent variables (see Fig. 2, Panels A and B). Again, the independence model, testing whether or not the observed data fit the expected data, was readily rejected, $\chi^2 (28, N=100) = 128.38, p < .001$. Although the indicators all had moderate-to-high loadings on their factor (all were significant at the .05 level), the one-factor model provided a poor fit for

the data, as indicated by the significant chi-squares (see Panel A of Fig. 2). Conversely, the two-factor model provided a good fit for the data, as indicated by the non-significant chi-squares (see Panel B of Fig. 2). Despite the moderate covariance between the latent variables of social cognition and negative symptoms (standardized coefficient = $-.39, p < .05$), the two-factor model fit the data significantly better than the one-factor model, $\chi^2 (1, N=100) = 32.55, p < .001$.

To compare the association between social cognition and neurocognition with the association between social cognition and negative symptoms, a three-factor model that represented social cognition, neurocognition, and negative symptoms as separate latent variables (Fig. 3)

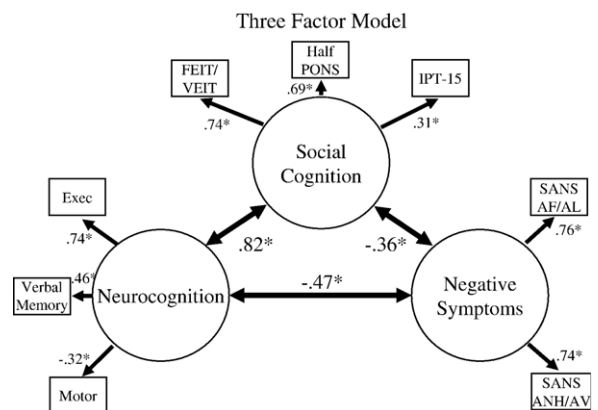


Fig. 3. A three-factor SEM model that represents social cognition, neurocognition, and negative symptoms as three separate latent variables. Circles represent latent variables, rectangles represent measured variables. * = estimated parameters. All coefficients (presented in standardized form) are significant at the .05 level, $\chi^2 (17, N=100) = 16.55, p = .49$.

was examined. Given the large number of parameters to be estimated within the three-factor model relative to the sample size, conceptually related indicators were combined to reduce the number of parameters. For social cognition, the FEIT was combined with the VEIT. For neurocognition, we created three composite scores: a) verbal episodic memory (CVLT), b) executive, working memory and attentional functions (WCST, DS-CPT, LNS, and COWAT), and c) motor speed (DS-C and GPT). For negative symptoms, we combined SANS global ratings based on previous factor analyses (Blanchard and Cohen, 2006): SANS-AF was combined with SANS-AL, and SANS-ANH was combined with SANS-AV. (Notably, the pattern of results remained the same when the three-factor model was examined with all separate indicators.) The independence model was readily rejected, $\chi^2(28, N=100)=155.01, p<.001$. The three-factor model provided a very good fit for the data, $\chi^2(17, N=100)=16.55, p=.49$. All indicators had moderate-to-high loadings on their respective latent variables, and all were significant at the .05 level. As can be seen in Fig. 3, the covariance between social cognition and neurocognition was substantially higher than between these two constructs and negative symptoms (all standardized coefficients were significant at the .05 level).

4. Discussion

Structural equation modeling with data from 100 outpatients with schizophrenia or schizoaffective disorder yielded three main findings. First, a two-factor model that represented social cognition and neurocognition as separate constructs fit the data better than a one-factor model that represented social cognition and neurocognition as a single construct. While the high covariance between social cognition and neurocognition in the two-factor model (.83) is quite high, associations between latent factors in SEM Models are often higher than the zero order correlations between the measured variables. In fact, the zero order correlations between the indicators of social cognition and neurocognition were low to moderate. Second, the two-factor model that represented social cognition and negative symptoms as separate constructs clearly fit the data better than a one-factor model that represented social cognition and neurocognition as a single construct. Third, when social cognition, neurocognition, and negative symptoms were included in the same model, the association between social cognition and negative symptoms, while significant, was weaker than the relationship between social cognition and neurocognition.

Schizophrenia researchers have typically conceptualized and studied social cognition as a construct separate from neurocognition. The present findings support the value of considering social cognition as a separate construct, even though it is closely related to neurocognition. The results also support the inclusion of social cognition in neurocognitive test batteries such as the MATRICS Consensus Cognitive Battery (Nuechterlein and Green, 2006). Recent studies support social cognition's role as a mediator of relations between neurocognition and functioning; and therefore, some separateness and added explanatory value for social cognition (Brekke et al., 2005; Sergi et al., 2006; Vauth et al., 2004). For example, the SEM model of Vauth et al. (2004) displayed a correlation of .91 between social cognition and neurocognition. Likewise, previous studies have found moderate but significant bivariate associations between social cognition (emotion perception, social perception, or theory of mind) and varied domains of neurocognition such as attention/visual vigilance (Addington and Addington, 1998; Bryson et al., 1997), early visual processing (Kee et al., 1998; Sergi and Green, 2003; Sergi et al., 2006), executive functioning (Bryson et al., 1997), sensorimotor gating (Wynn et al., 2005), and verbal memory (Corrigan et al., 1994; Greig et al., 2004).

The moderate relationship observed between social cognition and negative symptoms (−.36), suggests that the connections between these two domains deserves further study. Future studies might address how specific negative symptoms associate with social cognition. One possibility is that negative symptoms that involve reduced emotional experience (i.e., anhedonia) or expression (i.e., affective flattening) may be more associated with social cognition than other negative symptoms. Also, the connection between social motivation and performance on social cognitive measures is largely unexplored.

The current paper attempted to address some key questions about the nature of social cognition in schizophrenia, but others remain. For example, the relative independence of the many domains of social cognition (e.g., emotion processing, social perception, theory of mind, social knowledge) has yet to be carefully examined. Future studies might use factor analysis to determine if the domains of social cognition separate out indicating that they are unique subconstructs or if they load heavily on a single factor indicating that they are redundant terms. Other studies could address the independence of the domains of social cognition by examining their patterns of association with the domains of functional status.

The present study has limitations. The VA-based sample was mainly male, so the present findings are limited to males with schizophrenia and schizoaffective disorder. Moreover, the currently observed relations between social cognition, neurocognition, and negative symptoms, because they were identified from cross-sectional data, may not necessarily represent the longitudinal relationships between these constructs. Also, the assessments tapped only two domains of social cognition and just seven domains of neurocognition. Therefore, the present conclusions do not speak to the whole of social cognition and the whole of neurocognition.

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