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Is it possible to have impaired neurocognition but good social cognition in schizophrenia?

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Abstract

Social cognitive impairment in schizophrenia is common and associated with poor functional outcome. While correlations in the moderate range suggest that social cognition and neurocognition are separate but overlapping domains, less is known about whether intact neurocognition represents a “necessary but not sufficient” condition for intact social cognition, as has been suggested. In the present study we examined the following in a sample of 119 psychiatrically stable outpatients with schizophrenia: 1) correlations between multiple social cognitive measures and neurocognition, 2) the predictive value of neurocognitive domains in explaining social cognitive performance, and 3) the co-occurrence of social cognitive and neurocognitive impairment within participants. While ¼ of participants showed intact overall neurocognition and impaired overall social cognition, only one participant had normal-range social cognition and impaired neurocognition. Results support the notion that normal range neurocognition is a necessary though not necessarily sufficient building block for good social cognitive performance.

Keywords

Social Cognition; Neurocognition; Schizophrenia; Theory of Mind; Affect Recognition

1. Introduction

Social cognition represents the mental processes of perceiving, interpreting, and responding to social information (Penn et al., 1997) and comprises several facets including theory of mind, social perception, social knowledge, attributional bias, and emotional processing (Green et al., 2008). In recent years, social cognition has garnered attention in schizophrenia

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research because of its relationship to functioning (Couture et al., 2006) and its potential as a proximal target for treatment aimed at improving social functioning (Horan et al., 2008; Kurtz, 2011).

Existing research indicates that: 1) both neurocognition and social cognition are impaired in schizophrenia and predict level of functioning (Brekke et al., 2007; Fett et al., 2011; Pinkham & Penn, 2006); 2) social cognitive impairments appear to be present in schizophrenia across different phases of illness (Green et al., 2011; Horan et al., 2011); 3) neurocognition and social cognition correlate in the medium-range (Bryson et al., 1997; Penn et al., 1993); and 4) social cognition appears to mediate the relationship between neurocognition and functioning (Addington et al., 2006; Bell et al. 2009; Schmidt et al., 2011; Sergi et al., 2006). It has been suggested that neurocognition may be a necessary but not sufficient condition for social cognition (Ostrom, 1984; Penn et al., 1997), but this notion has not adequately been addressed in studies to date. Results of a recent meta-analysis indicate that social cognitive and neurocognitive domains generally correlate in the .22 to .33 range (Ventura et al., 2011). This translates to approximately 5 - 11% of the variance in social cognition that is explained by neurocognition. While this suggests that neurocognitive performance has a moderate impact on social cognitive ability, it does not directly address the question of the potential hierarchical nature of these two domains. In other words, it does not tell us whether normal-range neurocognitive ability is a necessary precursor for adequate social cognitive performance. This question is significant as it has implications for whether social cognitive rehabilitation programs in schizophrenia should also target neurocognitive functioning.

In the current analyses, we sought to better understand the relationship between social cognitive and neurocognitive functioning in psychiatrically stable outpatients with a schizophrenia-spectrum diagnosis. Specifically, we examined 1) correlations between social cognitive and neurocognitive task performance (to replicate previous studies); 2) the amount of variance in social cognition measures accounted for by neurocognition; and 3) the co-occurrence of social cognitive and neurocognitive impairment within study participants. Of particular interest was whether it is possible to have impaired neurocognition but normal-range social cognition, which would suggest that adequate neurocognitive performance is not a pre-requisite for normal-range social cognition.

2. Methods

2.1. Participants

The present analyses used baseline data from an existing dataset of 119 individuals (77 men and 42 women) diagnosed with schizophrenia ($n = 85$) or schizoaffective disorder ($n = 34$) enrolled in one of two IRB-approved vocational rehabilitation programs at VA Connecticut Healthcare System or Connecticut Mental Health Center. All subjects were psychiatrically stable (no psychiatric hospitalizations, medication changes, or housing changes in the previous 30 days, and no current substance abuse or dependence). In order to be eligible for the study, participants had to be aged 18-65, be proficient in English, have a GAF score above 30, and have no auditory or visual impairment, traumatic brain injury, neurological disease, or developmental disability. Diagnoses were confirmed using the Structured Clinical Interview for DSM-IV (SCID; First et al., 1996).

2.2. Measures

2.2.1. Social Cognitive Measures—The Hinting Task (Corcoran, et al., 1995; Greig et al., 2004) is a measure of Theory of Mind (ToM) consisting of 10 short vignettes of social interactions, each ending with one of the characters giving a hint to the other character about

what they would like that character to do. Based on normative data (Corcoran et al., 1995), a cut-score of more than 1 SD below the healthy control mean (17 or less) was used as a marker of impairment. One subject had missing data for the Hinting Task. The Egocentricity subscale of the Bell Object Relations and Reality Testing Inventory (BORRTI; Bell, 1995) is a self-report measure that assesses a more autistic self-experience in which others are viewed principally as need gratifying or need frustrating objects (for example “I believe a good mother should always please her children”) and has been found to correlate with other measures of social cognition. Higher scorers reflect greater impairment. A T-score greater than or equal to 60 was used as a cut-off for impairment. The Bell Lysaker Emotion Recognition Task (BLERT; Bryson, et al., 1997; Bell et al., 1997) measures affect recognition. Participants must identify the emotion portrayed by an actor reciting a neutral script. An empirically-derived (Fiszdon & Johannesen, 2010) cut score of less than 16 was used to indicate impairment. The Mayer-Salovey-Caruso Emotional Intelligence Test, Managing Emotions subscale (MSCEIT-ME; Mayer et al., 2002) is a measure of emotion processing and awareness of emotion regulation skills. A T-score lower than 40 reflects impairment. Participants were also dichotomized into impaired versus intact on overall social cognitive ability based on whether they scored in the normal range on at least three of four social cognitive measures.

2.2.2. Neurocognitive Measures—The MATRICS Consensus Cognitive Battery (MCCB; Nuechterlein & Green, 2006) is the gold standard neurocognitive battery for schizophrenia research trials. For the current analyses, we examined the following MCCB indices: processing speed, attention/vigilance, working memory, verbal learning, visual learning, and reasoning/problem-solving. In addition, we computed an overall neurocognitive composite score based on the average of T-scores for the six domains. For all MCCB variables, T-scores less than 40 were indicative of impairment.

3. Results

The mean age of the sample was 44.95 years ($SD = 11.04$). Participants were 59% Black 37% White, and 4% other and had an average of 12.89 years of education ($SD = 2.32$). Twenty-three percent were married at least once in their lifetime. Mean Positive and Negative Syndrome Scale (Kay et al., 1987) total score was 63.88 ($sd = 14.39$), indicating mild to moderate psychiatric symptoms.

Overall, neurocognitive domains showed correlations with social cognitive domains in the medium range (Table 1), consistent with previous reports. Working memory, in particular, was significantly associated with all social cognitive domains. Affect recognition was significantly correlated with all neurocognitive domains except reasoning/problem-solving, which was a statistical trend ($p < .06$).

In order to examine the variance in social cognition explained by neurocognition, separate backward deletion regressions (probability of F to remove ≥ 0.15 ; probability of F to enter < 0.10) were conducted for each social cognition task with the six neurocognitive domain scores entered in the first step as predictor variables and then removed in a conditional stepwise manner. This approach allowed us to 1) estimate the total variance in social cognition accounted for by the six neurocognitive domains used in this study and 2) model which neurocognitive domains were significant unique predictors of social cognition and the magnitudes of these relationships. R^2 for the full model reflects the proportion of variance explained when all six predictors are entered into the model in the first step. R^2 for the final model reflects the proportion of variance explained by the predictors retained after stepwise removal. Neurocognitive functioning explained a significant portion of variance in all four social cognition measures. Affect recognition relied most heavily on neurocognition

(BLERT; R^2 full model = .34, $F(6, 112) = 9.79$, $p < .001$; R^2 final model = .33, $F(3, 115) = 18.95$, $p < .001$), followed by ToM (Hinting Task; R^2 full model = .19, $F(6, 111) = 4.34$, $p < .01$; R^2 final model = .15, $F(1, 116) = 20.44$, $p < .001$), emotional awareness (MSCEIT-ME; R^2 full model = .14, $F(6, 112) = 3.10$, $p < .01$; R^2 final model = .11, $F(1, 117) = 14.76$, $p < .001$), and social schema (BORRTI; R^2 full model = .12, $F(6, 112) = 2.57$, $p < .05$; R^2 final model = .11, $F(2, 116) = 6.89$, $p < .01$). Affect recognition was uniquely predicted by working memory ($\beta = .31$, $t = 3.13$, $p < .01$) and attention/vigilance ($\beta = .27$, $t = 2.87$, $p < .01$). ToM was uniquely predicted by processing speed ($\beta = .39$, $t = 4.52$, $p < .001$) and emotional awareness was uniquely predicted by working memory ($\beta = .34$, $t = 3.84$, $p < .001$). None of the neurocognitive domains uniquely predicted social schema scores, although attention/vigilance was a trend ($\beta = -.20$, $t = -1.94$, $p < .06$).

Finally, Table 2 shows the co-occurrence of impairment between specific social cognitive measures and the neurocognitive composite measure. Across the individual social cognitive tasks approximately half of participants were impaired in both social cognition and overall neurocognition. Of particular interest, more than 2/3 of the sample was impaired in both overall social cognition and overall neurocognition, while only one participant had normal-range social cognition and impaired overall neurocognition. Conversely, 1/4 of participants showed intact overall neurocognition and impaired overall social cognition. Significant Chi-square analyses indicate that the distributions of impairments across social cognitive and neurocognitive domains were not random.

4. Discussion

Consistent with earlier studies, we found that social cognition measures were moderately correlated with neurocognitive domains. Neurocognition generally explained 10-20% of the variance in social cognition, with the exception being affect recognition, which depended more heavily on neurocognitive ability (34%). In examining the overlap of neurocognitive and social cognitive impairment, a majority of participants in the sample (68%) were impaired in both domains. In spite of the relatively small amount of variance in social cognition explained by neurocognition, it was very rare (<1%) for a participant to show intact social cognition while having impaired neurocognition. In contrast, it was not uncommon (25%) for participants to have impaired social cognition in the presence of intact neurocognition.

Penn and colleagues (1997) suggested that neurocognitive ability may represent a “necessary but not sufficient” prerequisite for social cognitive ability ($p. 115$). The theory behind this idea is logical- that social cognition, which would appear to be more complex than neurocognition, cannot be intact if the latter is impaired (e.g., the “building block model” of social cognition; Ostrom, 1984). However, while correlation and regression analyses suggest that neurocognition is indeed not sufficient for social cognition, they do not tell us how “necessary” neurocognition is to social cognition. Having found that good social cognition is extremely rare in the presence of poor neurocognition, and that the reverse is far more common, we can be more confident that neurocognition really is a “necessary” basis of social cognition.

Our results have implications for treatment studies targeting social cognition. Namely, researchers conducting interventions for social cognition should consider the nature and severity of concomitant neurocognitive deficits and design the treatments in a way that reduces cognitive load or provide them in the context of neurocognitive remediation. Differential emphasis may need to be placed on remediating specific neurocognitive performance deficits depending on the domain of social cognition targeted by an intervention.

Finally, while remediating or compensating for neurocognitive dysfunction is an important consideration in the refinement or design of future interventions targeting social cognition, ameliorating neurocognitive deficits in and of itself may not be enough to improve social cognitive performance. Continued efforts in designing and evaluating social cognitive interventions are very much needed, as are further analyses of non-cognitive variables that may be related to social cognitive impairments in schizophrenia.

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Table 1

Correlations between MCCB neurocognitive domains and social cognition measures (N=119)

	Processing Speed	Attention/Vigilance	Working Memory	Verbal Learning	Visual Learning	Reasoning/Problem-Solving	Composite Neurocognition ^b
Hinting Task ^a	.39***	.13	.24**	.23*	.12	.13	.28**
BORRTI	-.11	-.29**	-.28**	-.14	-.20*	-.12	-.28**
BLERT	.33***	.49***	.50***	.35***	.32***	.17 [†]	.51***
MSCEIT	.25**	.26**	.34***	.16	.06	.13	.28**

BORRTI = Bell Object Relations and Reality Testing Inventory, Egocentricity scale; BLERT = Bell Lysaker Emotion Recognition Task, MSCEIT = Mayer Salovey Caruso Emotion Intelligence Test, Managing Emotions subscale.

* $p < .05$

** $p < .01$

*** $p < .001$

[†] $p < .06$.

^a $N = 118$

^b The mean of the six MATRICS Consensus Cognitive Battery (MCCB) neurocognitive T-scores.

Table 2

Frequency of impaired neurocognition and impaired social cognition by measure and overall

	Neurocognition			
	Impaired n (%)	Normal Range n (%)	χ^2 (df)	
Hinting Task ^a				
Impaired	53 (45%)	16 (13%)	5.15 (1)	*
Normal Range	28 (24%)	21 (18%)		
BORRTI				
Impaired	58 (49%)	16 (13%)	8.19 (1)	**
Normal Range	24 (20%)	21 (18%)		
BLERT				
Impaired	66 (55%)	20 (17%)	8.89 (1)	**
Normal Range	16 (13%)	17 (14%)		
MSCEIT				
Impaired	55 (46%)	16 (13%)	6.01 (1)	*
Normal Range	27 (23%)	21 (18%)		
Composite ^{a,b}				
Impaired	80 (68%)	29 (25%)	12.23 (1)	***
Normal Range	1 (1%)	8 (7%)		

Neurocognition = mean of the six MCCB neurocognitive domain T-scores, BORRTI = Bell Object Relations and Reality Testing Inventory, BLERT = Bell Lysaker Emotion Recognition Task, MSCEIT = Mayer Salovey Caruso Emotion Intelligence Test, Managing Emotions subscale. Composite = summed performance on all social cognitive task ("Normal range" = normal range scores on at least 3 of the 4 tasks).

* $p < .05$

** $p < .01$

^a $N = 118$

^b Yates correction applied to Pearson's χ^2 test.