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Lower- and Higher-Level Social Cognitive Factors Across Individuals With Schizophrenia Spectrum Disorders and Healthy Controls: Relationship With Neurocognition and Functional Outcome

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Background: **Schizophrenia spectrum disorders (SSDs) often feature social cognitive deficits. However, little work has focused on the factor structure of social cognition, and results have been inconsistent in schizophrenia. This study aimed to elucidate the factor structure of social cognition across people with SSDs and healthy controls. It was hypothesized that a 2-factor model, including lower-level "simulation" and higher-level "mentalizing" factors, would demonstrate the best fit across participants.** ***Methods:*** **Participants with SSDs (N = 164) and healthy controls (N = 102) completed social cognitive tasks ranging from emotion recognition to complex mental state inference, as well as clinical and functional outcome, and neurocognitive measures. Structural equation modeling was used to test social cognitive models, models of social cognition and neurocognition, measurement invariance between cases and controls, and relationships with outcome measures.** ***Results:*** **A 2-factor (simulation and mentalizing) model fit the social cognitive data best across participants and showed adequate measurement invariance in both SSD and control groups. Patients showed lower simulation and mentalizing scores than controls, but only mentalizing was significantly associated with negative symptoms and functional outcome. Social cognition also mediated the relationship between neurocognition and both negative symptoms and functional outcome.** ***Conclusions:*** **These results uniquely indicate that distinct lower- and higher-level aspects of social cognition exist across SSDs and healthy controls. Further, mentalizing may be particularly linked to negative symptoms and functional outcome. This informs future studies of the neural circuitry underlying social cognition and the development of targeted treatment options for improving functional outcome.**

Key words: social cognition/mentalizing/theory of mind/simulation/emotion recognition/negative symptoms/functional outcome/neurocognition/schizophrenia

Introduction

Individuals with schizophrenia spectrum disorders (SSDs) often exhibit social cognitive deficits, including impaired emotion recognition, social perception, and theory of mind.[1,2] These impairments persist over time and are highly debilitating, as they interfere with interpersonal interactions and predict social functioning and quality of life.[3–5] Notably, meta-analytic results have shown a stronger relationship between community functioning and social cognition than neurocognition.[6] Social cognition is believed by many to include lower- and higher-level processes subserved by at least partially dissociable neural networks.[7–10] Lower-level social cognition, or "simulation," is thought to rely on embodied simulation of others' experiences, encompassing first-order mental representation, emotional empathy, and basic emotion detection. In contrast, higher-level "mentalizing" involves intention attribution and complex mental state representation. Despite evidence supporting the distinction of these constructs, little work has examined the factor structure of social cognition. Delineating the components of social cognition in SSDs is critical, given

its relationship with functioning and the need to identify treatment targets.

Several investigations in SSDs have focused on social cognition vs neurocognition, demonstrating that these are separable but related constructs.[11–13] Conversely, few studies have examined the factor structure of social cognition in SSDs, particularly using larger sample sizes and a range of social cognitive tasks. Specifically, a 3-factor model including "lower-level social cue detection" and "higher-level inferential and regulatory processing" factors was identified in outpatients with psychosis using exploratory factor analysis.[14] Similarly, "socio-emotional processing" and "social-inferential ability" components of social cognition have been extracted in both people with schizophrenia and healthy controls.[15] In contrast, a 1-factor model of social cognition recently showed good fit across schizophrenia and control groups using confirmatory factor analysis (CFA), although a 2-factor model was not tested.[16]

Thus, it remains unclear whether social cognition includes lower- and higher-level factors or is a unidimensional construct in SSDs, and whether a similar structure exists in healthy controls. Critically, few investigations have also included healthy individuals and tested for measurement invariance across SSD and control groups, consistent with a Research Domain Criteria (RDoC) model. It is also uncertain how these factors relate to functional outcome and psychopathology, and whether they may differentially mediate the relationship between neurocognition and functional outcome.[17]

Our objective was to elucidate the factor structure of social cognition across a large group of people with SSDs and healthy controls. Further, we hoped to determine whether different social cognitive factors were related to negative symptoms and functional outcome, which may be particularly important for informing and evaluating treatment options. We used a range of recommended social cognitive tasks[18,19] and structural equation modeling (SEM), including CFAs to test models of social cognition, and multiple regression to identify relationships with outcome measures of interest. We hypothesized that a 2-factor model, including simulation and mentalizing factors, would demonstrate the best fit across participants. We also expected social cognitive and neurocognitive factors to load on separate respective higher-order factors, and social cognition to mediate the relationship between neurocognition and clinical and functional outcome measures.

Methods

Participants

Participants were recruited for the ongoing National Institute of Mental Health–funded "Social Processes Initiative in the Neurobiology of the Schizophrenia(s) (SPINS)," a multicenter RDoC study (Centre for Addiction and Mental Health [CAMH], Toronto; Zucker Hillside Hospital [ZHH], New York; Maryland Psychiatric Research Center [MPRC], Maryland) of social cognition in SSDs. Of the 266 participants who completed all study visits and met eligibility requirements throughout, 164 had SSDs (108 males and 56 females) and 102 were healthy individuals (52 males and 50 females; cases and controls by site—CAMH: 63, 38; MPRC: 56, 39; ZHH: 45, 25). Participants with SSDs met DSM-5 (*Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition) diagnostic criteria for schizophrenia ($n = 113$), schizoaffective disorder ($n = 38$), schizophreniform disorder ($n = 3$), delusional disorder ($n = 0$), or psychotic disorder not otherwise specified ($n = 10$), assessed using the Structured Clinical Interview for DSM (SCID-IV-TR), and had no change in antipsychotic medication or decrement in functioning/support level in the 30 days before enrollment. Controls did not have a current or past Axis I psychiatric disorder, excepting adjustment disorder, phobic disorder, past major depressive disorder (over 2 years prior; presently unmedicated), or a first-degree relative with a history of psychotic mental disorder. Additional exclusion criteria included a history of head trauma resulting in unconsciousness, a substance use disorder (confirmed by urine toxicology screening), intellectual disability, debilitating or unstable medical illness, or other neurological diseases. All participants provided written informed consent before any research procedures. The protocol was approved by the respective research ethics boards and institutional review boards. All research was conducted in accordance with the Declaration of Helsinki.

Procedure

Data collection occurred across 3 visits (visit 1: consent, screening, clinical scales; visit 2: magnetic resonance imaging; visit 3: neurocognitive and social cognitive testing). All participants completed the Wechsler Test of Adult Reading[20] as a measure of premorbid intelligence quotient. Social cognitive measures were selected based on the findings from the Social Cognition Psychometric Evaluation study[19] and the Social Cognition and Functioning in Schizophrenia project,[18] which sought to identify psychometrically sound measures of social cognition.

Clinical and Functional Outcome Measures

Psychiatric symptoms were evaluated in the SSD sample using the Brief Psychiatric Rating Scale (BPRS)[21] and the Scale for the Assessment of Negative Symptoms (SANS).[22] The Quality of Life Scale (QLS)[23] was also administered to assess functioning. In SSD and control groups, the Birchwood Social Functioning Scale (BSFS)[24] was administered to evaluate social functioning. Total

scores on the BPRS, SANS (excluding Attention), QLS, and BSFS (omitting Employment) are reported.

Neurocognitive and Social Cognitive Measures

Neurocognition was evaluated using the MATRICS (Measurement and Treatment Research to Improve Cognition in Schizophrenia) Consensus Cognitive Battery (MCCB),[25] which includes tests of processing speed, reasoning and problem-solving, attention/vigilance, working memory, and verbal and visual learning. Social cognitive tasks ranged from basic emotion recognition to complex mental state inference. These included the Penn Emotion Recognition Test (ER40),[26] which assesses basic emotion recognition from static images; the Reading the Mind in the Eyes Test (RMET),[27] involving mental state inference from the eye region of faces; and the Empathic Accuracy (EA) task,[18,28] which was performed during functional magnetic resonance imaging and entails rating how positive or negative someone is feeling throughout an emotional video. Participants also completed The Awareness of Social Inference Test–Revised (TASIT),[29] which involves viewing social video clips and includes 3 subtests (TASIT 1: identifying emotions; TASIT 2 and 3: social inference, including detection of lies and sarcasm). Detailed task descriptions appear in supplementary material.

The simulation factor in our proposed 2-factor model of social cognition included ER40, RMET, EA, and TASIT 3 lies, whereas our mentalizing factor included TASIT 2 simple sarcasm, TASIT 2 paradoxical sarcasm, and TASIT 3 sarcasm. The specification of indicators onto lower-level simulation and higher-level mentalizing factors was determined based on the nature of the tasks (eg, level of complexity, from basic emotion or valence identification to more complex mental state inference), a preliminary exploratory factor analysis (supplementary table S1), and empirical evidence from the literature. In particular, TASIT 3 lies exchanges are less complex than the sarcastic exchanges and have loaded onto a lower-level factor in prior work.[14] TASIT 1 was excluded from the CFAs due to the preliminary exploratory factor analysis demonstrating separation of the indicators as expected into 2 factors (loadings > 0.4),[30] aside from a modest cross-loading for TASIT 1 on both. Sincere exchanges from TASIT 2 was also excluded, as it has been used as a control condition for basic task demands.[31,32] Accordingly, participants with SSDs performed worse than controls across tasks, aside from the TASIT 2 sincere exchanges (supplementary table S2).

Data Analysis

Data were analyzed using RStudio Version 1.0.143[33] and the lavaan package.[34] Outliers were detected and removed from all variables using the adjusted boxplot method.[35] All variables then underwent Yeo-Johnson power transformations due to skewed distributions,[36] other than the MCCB subtests, which showed relatively normal distributions according to visual inspection and Q-Q plots (supplementary table S2). Variables were then *z*-transformed. Preliminary exploratory factor analysis with maximum likelihood estimation and oblique (promax) rotation was used to optimize social cognitive factor structure. CFAs were conducted using full information maximum likelihood estimation with robust standard errors, as some distributions remained non-normal following transformation. This method uses partial information where there are missing data, increasing efficiency and consistency of parameter estimates.[37] Indicators were freely estimated and latent factor variances were set to 1 for model identification.

For social cognitive variables, 2-factor (simulation and mentalizing) and 1-factor models were tested. The fit of a higher-order model of social cognition and neurocognition was also evaluated and compared to 2- and 1-factor models to determine whether our data would corroborate the distinction of these constructs (higher order refers to the model specification here (ie, a hierarchical model), whereas lower- and higher-level social cognition refer to the specification of the constructs rather than the model itself). The higher-order model included 2 first-order factors of social cognition (simulation and mentalizing) based on the results of our social cognitive CFAs, and 3 first-order factors of neurocognition (processing speed, attention/working memory, and learning) based on demonstrations of good fit for this model in individuals with SSDs[38] and severe mental illness.[39] Nested model comparisons were made using the chi-square difference test. Measurement invariance of the models with the best fit was also evaluated across participants with SSDs and controls using a series of increasingly constrained CFAs to confirm that the same constructs were being evaluated in both groups.[40] Model fit was first tested using separate CFAs in the SSD and the control groups. Following this, multigroup CFAs were used to test for configural invariance (equal factor structures across groups), metric invariance (equal factor loadings across groups), and scalar invariance (equal indicator intercepts across groups). Latent mean comparisons were then conducted between SSD and control groups. Measurement invariance testing was also conducted across genders, given some evidence for differential social cognitive processing in females and males.[41] Demonstrating configural and at least partial metric and scalar invariance is necessary to compare latent factor mean differences between groups.[42]

SEM was also used to elucidate the relationship between identified factors and clinical and functional outcome measures. We tested models regressing our clinical (SANS total) and functional outcome (BSFS total and QLS total) measures of interest on the latent

simulation and mentalizing factors in our 2-factor model of social cognition. We focused on the SANS given evidence for the association of negative symptoms with social cognition.[11,43]

Mediation analyses were performed using the higher-order social cognition and neurocognition model, including multiple regressions with the higher-order neurocognition factor as a predictor of the clinical and functional outcome measures of interest, and the higher-order social cognition factor as mediator. Due to the cross-sectional data, we also tested mediation models with social cognition as the predictor and neurocognition as the mediator. Further, models including both simulation and mentalizing factors as mediators between neurocognition and outcome measures were tested. Mediation models were tested using full information maximum likelihood estimation. Indirect effects were determined using nonparametric bootstrapping with 5000 resamples, where the effect is considered significant ($P < .05$) if the bias-corrected and accelerated 95% CI does not include 0.[44] We also tested models regressing our clinical and functional outcome measures of interest on the latent neurocognition, simulation, and mentalizing factors. Standardized coefficients are reported.

Results

Participant demographic and clinical characteristics are presented in table 1. Results from the preliminary exploratory factor analysis appear in supplementary table S1. Social cognitive and neurocognitive task performance data appear in supplementary table S2. Supplementary table S3 shows correlations between social cognitive and neurocognitive indices included in the CFAs.

Confirmatory Factor Analyses for Models of Social Cognition Across Participants

Model fit was assessed using ranges of acceptable fit values outlined by Hu and Bentler,[45] including comparative fit index (CFI) ≥ 0.95, Tucker-Lewis index (TLI) ≥ 0.95, root mean square error of approximation (RMSEA) ≤ 0.06, and standardized root mean square residual (SRMR) ≤ 0.08 to suggest that the hypothesized model fits the observed data relatively well. The chi-square statistic is also a metric of absolute fit, but it is sensitive to sample size. Accordingly, the proposed 2-factor model, including simulation and mentalizing factors, fits the social cognitive data very well across participants with SSDs and healthy controls ($\chi^2 = 12.18$, $df = 13$, $P = .513$, CFI = 1.00, TLI = 1.00, RMSEA = 0.00, SRMR = 0.021; table 2). All indicators also loaded highly onto their respective factors (all $P < .001$). The fit indices were poorer for a 1-factor model of social cognition (table 2). Indeed, the 2-factor model fits the data significantly better than the 1-factor model (table 2). Factor models and loadings are depicted in figure 1.

Separate CFAs confirmed that the 2-factor model fits the social cognitive data well in both the SSD and control groups. The 2-factor model also showed configural, metric, and partial scalar invariance, as demonstrated using a series of increasingly constrained multigroup CFAs (table 3; details in supplementary material). Latent means comparisons revealed that healthy controls scored 0.451 and 0.912 units higher on the simulation and mentalizing factors, respectively, than participants with SSDs (both $P < .001$). Measurement invariance for the 2-factor model was also demonstrated across female and male participants, with no significant differences between simulation and mentalizing latent means between genders (supplementary table S4; details in supplementary material).

Table 1. Participant Demographic and Clinical Characteristics

	SSD ($N = 164$)		Control ($N = 102$)		
	n	%	*n*	%	*P*
Gender (male)	108	65.9	52	51	.023
	Mean	SD	Mean	SD	*P*
Age	32.54	9.92	31.25	9.99	.308
Education (highest grade)	13.49	2.19	15.64	1.91	<.001
WTAR (standard score)	106.08	14.73	112.33	11.94	.005
BPRS total	31.69	8.17	—	—	—
SANS total	24.73	12.71	—	—	—
QLS total	75.30	21.34	—	—	—
BSFS total	136.12	24.51	176.02	19.00	<.001

Note: SSD, schizophrenia spectrum disorder; WTAR, Wechsler Test of Adult Reading; BPRS, Brief Psychiatric Rating Scale; SANS, Scale for the Assessment of Negative Symptoms; QLS, Quality of Life Scale; BSFS, Birchwood Social Functioning Scale.

Table 2. Fit Indices for Models of Social Cognition, and Social Cognition and Neurocognition

	Model Fit							Nested Model Comparisons			
Model	χ^2	*df*	*P*	CFI	TLI	RMSEA	SRMR	Model comparison	$\Delta\chi^2$	Δdf	*P*
Social cognition											
Two-factor	12.18	13	.513	1.00	1.00	0.000	0.021				
One-factor	51.40	14	<.001	0.942	0.913	0.103	0.049	Two- vs one-factor	49.13	1	<.001
Social cognition and neurocognition											
Higher-order	153.61	98	<.001	0.971	0.965	0.046	0.040				
Two-factor	228.08	103	<.001	0.935	0.925	0.067	0.049	Higher-order vs two-factor	71.92	5	<.001
One-factor	394.74	104	<.001	0.850	0.827	0.102	0.063	Higher order vs one-factor	254.31	6	<.001

Note: Reported fit indices are robust. *df*, degrees of freedom; CFI, comparative fit index; TLI, Tucker-Lewis index; RMSEA, root mean square error of approximation; SRMR, standardized root mean square residual.

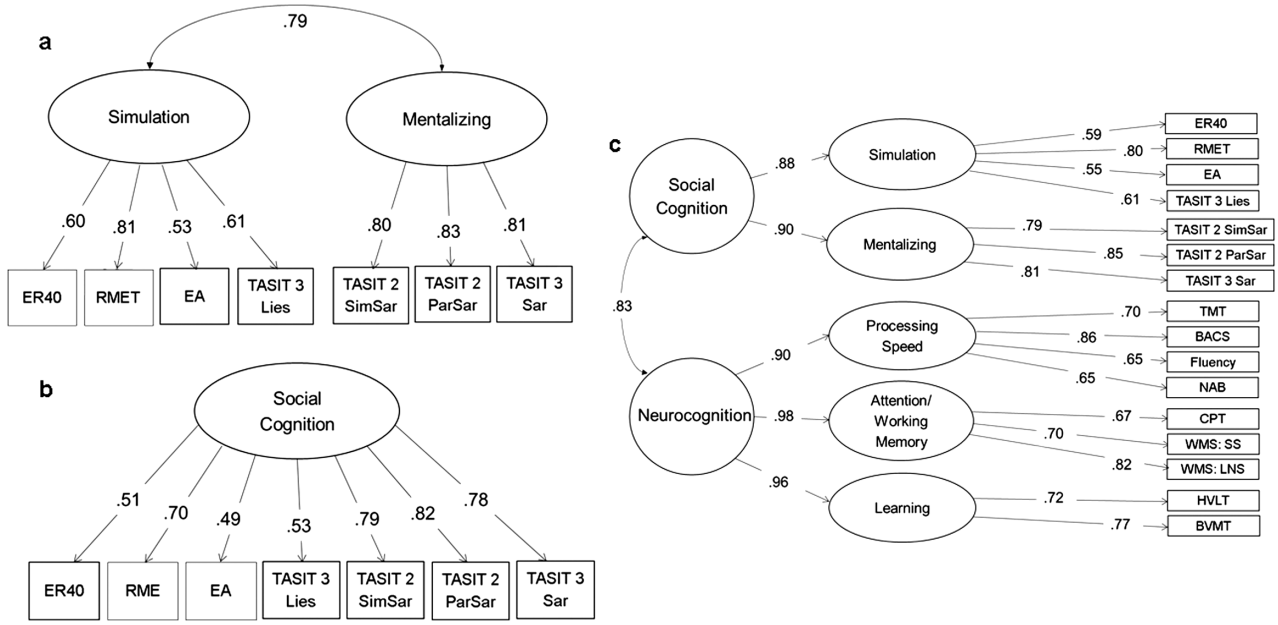

Fig. 1. Factor structure and standardized loadings for the (a) two-factor and (b) one-factor models of social cognition, and the (c) higher-order model of social cognition and neurocognition. ER40, Penn Emotion Recognition Test; RMET, Reading the Mind in the Eyes Test; EA, Empathic Accuracy task; TASIT, The Awareness of Social Inference Test–Revised; SimSar, Simple Sarcasm; ParSar, Paradoxical Sarcasm; Sar, Sarcasm; TMT, Trail Making Test: Part A; BACS, Brief Assessment of Cognition in Schizophrenia: Symbol Coding; Fluency, Category Fluency: Animals; NAB, Neuropsychological Assessment Battery: Mazes; CPT, Continuous Performance Test: Identical Pairs; WMS, Wechsler Memory Scale; SS, Spatial Span; LNS, Letter-Number Span; HVLT, Hopkins Verbal Learning Test–Revised; BVMT, Brief Visuospatial Memory Test–Revised.

Structural Equation Modeling

The mentalizing factor (β = .416, *P* = .001), but not the simulation (β = .084, *P* = .528) factor, was significantly associated with BSFS total score across SSD and healthy groups (r^2 = 23.5). In the SSD group, mentalizing was significantly related to both SANS (β = −.302, *P* = .022) and QLS total scores (β = .272, *P* = .039), whereas simulation was not significantly associated with either (β = −.073, *P* = .596; β = .119, *P* = .368, respectively). This model accounted for 12.9% of the variance in SANS total score and 13.6% of the variance in QLS total score. These models both demonstrated good fit (supplementary table S5).

Confirmatory Factor Analyses for Models of Social Cognition and Neurocognition

The hypothesized higher-order 2-factor (social cognition and neurocognition) model fits the data well across participants with SSDs and healthy controls (χ^2 = 153.61, *df* = 98, *P* < .001, CFI = 0.971, TLI = 0.965, RMSEA = 0.046, SRMR = 0.040; figure 1c). Two-factor

Table 3. Measurement Invariance Testing Across SSD and Healthy Control Groups for the Models of Best Fit

	Model Fit							Nested Model Comparisons			
Model	χ^2	*df*	*P*	CFI	TLI	RMSEA	SRMR	Model Comparison	$\Delta\chi^2$	Δdf	*P*
Social cognition											
Two-factor											
SSD	8.15	13	.834	1.00	1.02	0.000	0.024				
Control	13.93	13	.379	0.987	0.980	0.026	0.049				
Configural invariance	21.62	26	.709	1.00	1.02	0.000	0.033				
Metric invariance	29.33	31	.552	1.00	1.00	0.000	0.045	Metric vs configural	8.10	5	.151
Scalar invariance	48.69	36	.077	0.973	0.968	0.051	0.073	Scalar vs metric	19.03	5	.002
Partial scalar invariance	29.70	34	1.00	1.00	1.01	0.000	0.045	Partial scalar vs metric	0.50	3	.920
Social cognition and neurocognition											
Higher-order											
SSD	121.47	98	.054	0.978	0.974	0.038	0.050				
Control	134.79	98	.008	0.873	0.845	0.058	0.071				
Configural invariance	260.53	196	.001	0.955	0.945	0.048	0.058				
Metric invariance	271.71	210	.003	0.956	0.949	0.046	0.065	Metric vs configural	12.98	14	.529
Scalar invariance	308.29	219	<.001	0.935	0.929	0.055	0.076	Scalar vs metric	31.21	9	<.001
Partial scalar invariance	281.15	216	.002	0.953	0.948	0.047	0.068	Partial scalar vs metric	8.96	6	.176

Note: Empathic Accuracy and The Awareness of Social Inference Test–Revised (TASIT) 3 lies indicators are freely estimated in the Social Cognition 2-Factor Partial Scalar Invariance model. Empathic Accuracy, TASIT 3 lies, and Brief Assessment of Cognition in Schizophrenia (BACS) indicators are freely estimated in the Higher-Order Social Cognition and Neurocognition Partial Scalar Invariance model. Reported fit indices are robust. Abbreviations are explained in the footnote to table 2; SSD, schizophrenia spectrum disorder.

(social cognition and neurocognition) and 1-factor models were also tested across all participants using CFAs, both of which showed deterioration in model fit indices (table 2). The higher-order model of social cognition and neurocognition showed significantly better fit than both the 2- and 1-factor models (table 2).

Results from measurement invariance testing of the higher-order factor model using separate CFAs for the SSD and healthy control groups followed by multigroup CFAs are presented in table 3. This model demonstrated configural, metric, and partial scalar invariance (details in supplementary material). Latent means comparisons showed that healthy controls scored 0.418 units higher on the higher-order social cognition factor and 0.571 units higher on the neurocognition factor than participants with SSDs (both $P < .001$).

Mediation Analyses

First, SEM models regressing social functioning (across groups), quality of life (SSD only), and negative symptom (SSD only) scores on higher-order neurocognition revealed that there was a significant direct effect of neurocognition on BSFS ($\beta = .503$, $P < .001$), SANS ($\beta = -.323$, $P < .001$), and QLS ($\beta = .305$, $P < .001$) total scores. This confirmed the association between neurocognition and our outcome variables of interest before conducting mediation analyses. Social cognition was found to mediate the relationship between neurocognition and BSFS total score ($r^2 = 27.2\%$), neurocognition and SANS total score ($r^2 = 15.8\%$), and neurocognition and QLS total score ($r^2 = 16.6\%$; figure 2). Switching the predictor and mediator revealed that neurocognition did not mediate the relationship between social cognition and BSFS, SANS, or QLS total scores (supplementary figure S1). In models including both simulation and mentalizing factors as mediators of the relationship between neurocognition and our 3 outcome measures of interest, neither the simulation nor the mentalizing factors showed a significant indirect effect on BSFS, SANS, or QLS total scores (supplementary table S6).

In models regressing outcome measures of interest on neurocognition, simulation, and mentalizing factors, mentalizing ($\beta = .281$, $P = .020$) and neurocognition ($\beta = .273$, $P = .013$), but not the simulation factor ($\beta = -.006$, $P = .964$), were significantly associated with

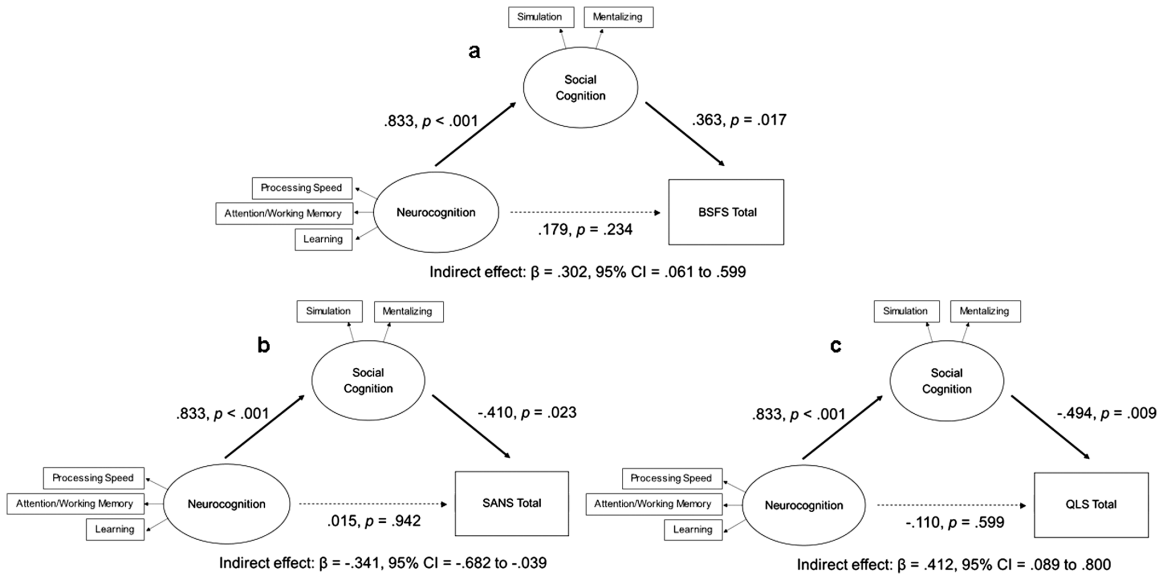

Fig. 2. Mediation analyses. The association between neurocognition and (a) Birchwood Social Functioning Scale (BSFS), (b) Scale for the Assessment of Negative Symptoms (SANS), and (c) Quality of Life Scale (QLS) total scores, mediated by social cognition. The relationships between neurocognition and the outcome measures of interest reflect those once social cognition has been added as a mediator (values prior to this are reported in the "Results" section).

BSFS total score across groups (r^2 = 26.5%). The mentalizing factor was also significantly related to SANS and QLS total scores (β = −.270, P = .044; β = .271, P = .043, respectively) in the SSD group, whereas neurocognition (β = −.096, P = .475; β = .014, P = .917, respectively) and simulation (β = −.032, P = .820; β = .111, P = .438, respectively) were not (r^2 = 13.4%, 13.7%, respectively). These mediation and regression models all demonstrated good fit (supplementary table S5).

Discussion

In this study, a 2-factor social cognitive model, including lower-level "simulation" and higher-level "mentalizing" factors, showed the best fit across participants. Further, this model fits the social cognitive data well in both SSD and healthy control groups. Though individuals with SSDs showed lower scores than healthy controls on both the simulation and mentalizing factors, only the mentalizing factor was found to be significantly related to negative symptoms and functional outcome. Further, social cognition mediated the relationship between neurocognition and both clinical and functional outcome measures. Our results indicate that distinct lower- and higher-level aspects of social cognition exist in both individuals with SSDs and healthy controls. Further, mentalizing may be particularly linked to negative symptoms and functional outcomes.

Lower- and Higher-Level Social Cognition in SSDs and Healthy Controls

Results from studies investigating the factor structure of social cognition in schizophrenia have been mixed,[46–53] likely due to the limited range of included tasks, variability in analytic approaches, and inadequate sample sizes.[13] The 2-factor model presently identified, including simulation and mentalizing, aligns well with factors identified in 2 prior investigations that used larger sample sizes and a range of social cognitive tasks.[14,15] However, unlike prior investigations suggesting the existence of these factors, this study uniquely included a large group of participants with SSDs and healthy controls, tested multiple models, and used SEM, allowing for between-group comparisons in factor structure and the assessment of associations between multiple latent and observed variables, accounting for measurement error.[54] Notably, this 2-factor model showed significantly better fit than a 1-factor model. The distinction of these lower- and higher-level aspects of social cognition is also supported by neuroimaging[55] and lesion data.[10] Further, these factors align with constructs identified as best positioned for future translational work regarding socioemotional deficits in schizophrenia and other psychiatric disorders.[56]

In addition, we uniquely demonstrated that the 2-factor model fits well in SSD and healthy control groups, suggesting that lower- and higher-level social cognitive constructs exist in both. A 2-factor solution including emotion perception and theory of mind factors has previously been identified in healthy individuals[57] and a transdiagnostic group including individuals with schizophrenia and bipolar disorder.[58] Taken together, this provides justification for the between-group comparison of simulation and mentalizing constructs, and dimensional approaches interrogating these within the RDoC framework.

Relationship Between Factors, Negative Symptoms, and Functional Outcome

Individuals with SSDs showed significantly lower latent means for both simulation and mentalizing, consistent with prior evidence.[1,2] However, we found that mentalizing, but not simulation, was significantly associated with social functioning across participants with SSDs and healthy controls, as well as negative symptoms and quality of life in those with SSDs. Thus, these higher-level inferential abilities appear to be particularly linked to negative symptoms and functional outcomes. This aligns with results from a meta-analysis focused on the relationship between functional outcome measures and social cognitive and neurocognitive domains, revealing that theory of mind was most strongly associated with functional outcomes.[6] Further, it is consistent with findings from Mehta et al.,[15] who found that social-inferential ability, but not socio-emotional processing, was significantly related to negative symptoms and motivational impairments in schizophrenia.

Social Cognition, Neurocognition, and Outcome Measures

In relation to neurocognition,[13] our results build on prior work by demonstrating that our 2-factor model of social cognition also fits well within a broader model including separate higher-order factors of social cognition and neurocognition, in both SSD and healthy control groups. Consistent with a meta-analysis on the mediational role of social cognition between neurocognition and functional outcome in schizophrenia,[17] our higher-order social cognition factor mediated the relationship between neurocognition and negative symptoms, quality of life, and social functioning. However, neither simulation nor mentalizing factors exhibited a significant indirect effect individually. Although more studies have investigated and confirmed the role of lower-level social cognition (vs higher-level), as a mediator between neurocognition and functional outcome,[17,59] theory of mind has been identified as a mediator in individuals with schizophrenia[59] and nonpsychotic disorders.[60] In our data, mentalizing was found to be significantly associated with social functioning across SSDs and control groups, and the only significant predictor of negative symptoms and quality of life in individuals with SSDs, when simulation and neurocognition were included. This further suggests that mentalizing is particularly linked to negative symptoms and functional outcomes.

Limitations

As with all factor analyses, our results are inherently limited by the measures used in our models. However, we did include a comprehensive range of recommended social and neurocognitive tasks to optimize the generalizability and applicability of our findings. Notably, we did not include any measures of attributional style given our interest in lower- vs higher-level social cognition, as it has been shown to load separately.[14] Score distributions also appeared similar across our social cognitive measures, and the SSD group showed intact performance on TASIT 2 sincere exchanges reflecting task comprehension (supplementary table S2), suggesting that task difficulty did not underlie factor separation. However, it should be noted that the mentalizing factor was composed of measures exclusively from the TASIT (though different subtests, and not all TASIT indices). In addition, the use of SANS total scores may conflate subdomains of negative symptoms, given that negative symptoms appear to have a multifactorial structure, as well as negative symptoms and functional outcome, as some SANS items tap both.[61] Nevertheless, our results regarding the association between higher-level social cognition and negative symptoms align with previous findings using the Positive and Negative Syndrome Scale.[15] Further, though we did not control for medication or duration of illness, the finding that our models fit well across SSD and control groups suggests that these variables were likely not driving our effects. Finally, longitudinal studies should be conducted to examine the stability and ecological validity of our findings. In particular, the use of cross-sectional data for mediation analyses weakens causal inference. Nonetheless, in sensitivity analyses, switching the predictor and mediator in our mediation models demonstrated that social cognition, but not neurocognition, exhibited a significant indirect effect on negative symptoms and functional outcomes.

Conclusions

The present findings provide novel evidence for a similar structure of social cognition, including lower- and higher-level factors, across individuals with SSDs and healthy controls. This provides justification for the between-group comparison of lower- and higher-level social cognitive constructs, and transdiagnostic dimensional approaches interrogating these within the RDoC framework. Further, they indicate that mentalizing may be particularly linked to negative symptoms and functioning. These results also confirm the importance of social cognition beyond neurocognition as it relates to negative symptoms and functional outcomes, and thereby as a potential treatment target in SSDs. Elucidating the factor structure of social cognition has important implications for future investigations of its underlying neural circuitry and the development of targeted treatments for improving functional outcome. More specifically, identifying neural circuits subserving higher-level social cognition may be of particular interest and may also serve as outcome measures in future investigations.

Supplementary Material

Supplementary data are available at *Schizophrenia Bulletin* online.

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