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Social Cognition in Schizophrenia, Part 2: 12-Month Stability and Prediction of Functional Outcome in First-Episode Patients

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This study evaluated the longitudinal stability and functional correlates of social cognition during the early course of schizophrenia. Fifty-five first-episode schizophrenia patients completed baseline and 12-month follow-up assessments of 3 key domains of social cognition (emotional processing, theory of mind, and social/relationship perception), as well as clinical ratings of real-world functioning and symptoms. Scores on all 3 social cognitive tests demonstrated good longitudinal stability with test-retest correlations exceeding .70. Higher baseline and 12-month social cognition scores were both robustly associated with significantly better work functioning, independent living, and social functioning at the 12-month follow-up assessment. Furthermore, cross-lagged panel analyses were consistent with a causal model in which baseline social cognition drove later functional outcome in the domain of work, above and beyond the contribution of symptoms. Social cognitive impairments are relatively stable, functionally relevant features of early schizophrenia. These results extend findings from a companion study, which showed stable impairments across patients in prodromal, first-episode, and chronic phases of illness on the same measures. Social cognitive impairments may serve as useful vulnerability indicators and early clinical intervention targets.

Key words: functional outcome/emotional processing/theory of mind/social perception/cross-lagged panel analyses/longitudinal prediction

Introduction

Individuals with schizophrenia demonstrate substantial and relatively stable impairments in social cognition, which are important determinants of functional outcome.[2,3] However, the stability and functional significance of impairments in social cognition during the early phase of schizophrenia are relatively unexplored. Cross-sectional studies indicate that first-episode or recent-onset patients demonstrate deficits on measures of facial/vocal affect perception,[4–7] theory of mind,[8–11] and social perception.[12] To the best of our knowledge, only one research group examined temporal stability and found that scores on social perception tasks demonstrated good stability over a 12-month period.[12] Two studies examined cross-sectional relations to functioning in recent-onset patients: one found poor affect and social perception correlated with worse subjective quality of life[12] and the other that emotional processing and self-reported social functioning were related.[13] Thus, the limited available evidence suggests that social cognitive disturbances are stable and related to real-world functioning during the early course of schizophrenia.

The current study evaluated the longitudinal stability and functional correlates of social cognition in first-episode schizophrenia. We assessed 3 domains with strong theoretical importance for adaptive social interactions using tasks that have rarely been applied to schizophrenia. Two of the domains, emotional intelligence and theory of mind, were assessed with existing measures that have established psychometric properties and validity in healthy subjects, the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT[14]) and The Awareness

of Social Inference Test (TASIT[15]), respectively. The third domain was relationship perception, which refers to perception of the nature of relationships between people as opposed to perception of individuals acting alone.[16] This was assessed with the Relations Across Domains (RAD[17]) test, a measure recently developed and validated by our group. In chronically ill patients, we found that each of these tests demonstrated good psychometric properties, medium-to-large patient vs healthy control impairments, and small-to-moderate associations with real-world functioning.[17–19] In an accompanying article,[1] we evaluated the performance of patients during prodromal, first-episode, or chronic phases of illness on the same social cognitive measure used in the current study. Each patient group demonstrated similarly large performance deficits compared with matched healthy comparison samples with no evidence of progression or improvement across phase of illness, a pattern consistent with a vulnerability indicator.[1]

To further evaluate the status of these social cognitive measures, this report focuses on the 12-month longitudinal stability and prediction of functional outcome within the first-episode sample. The recent-onset period is particularly informative for evaluating the trait-like quality and functional significance of social cognition; this period is often characterized by greater clinical fluctuations than later stages of illness, providing an excellent opportunity to clarify the extent to which candidate vulnerability indicators represent state or trait phenomena.[20] It also minimizes confounds associated with prior treatment and chronicity, permitting stronger inferences about the validity of vulnerability indicators than with chronically ill patients.[21] We addressed the following research questions: (1) Is performance on the social cognition measures stable over a 12-month period following an initial psychotic episode? (2) Do the social cognition measures demonstrate significant cross-sectional and longitudinal correlations with functioning at the baseline and 12-month follow-up assessment points? Prior research in chronic and recent-onset samples led us to predict that the measures would demonstrate substantial stability and at least moderate relations to outcome across the study period.

Methods

Participants

Participants with first-episode schizophrenia were recruited through the University of California Los Angeles (UCLA) Center for Neurocognition and Emotion in Schizophrenia.[1] A full description of the recruitment procedures and inclusion/exclusion criteria is available in the accompanying article. This report focuses on 55 (42 men and 13 women) of the original 81 first-episode patients who remained in the study and reached the project's 12-month follow-up assessment point. There were no significant differences between participants from the full sample in the accompanying article compared with those in the current study on any demographic, clinical, social cognitive, or functional outcome variable at baseline (all P's $> .05$).

At study entry, the mean age of the participants was 22.3 years (SD = 4.3), the mean level of education was 12.7 years (SD = 2.1), and the mean parental education level was 13.8 (SD = 3.9). In terms of race, 10 were Caucasian, 14 Hispanic, 23 African American, 5 Asian, and 3 other/mixed. Participants had a mean of 1.2 hospitalizations (SD = 0.8), a mean total duration of illness (since time of psychosis onset) of 8.5 months (SD = 6.4), and a mean duration of untreated psychosis of 6.9 months (SD = 6.2).

At the time of the baseline assessment, all participants were clinically stabilized on oral risperidone—this assessment occurred an average of 2.8 months (SD = 2.0) after entry into the project. Participants received clinically determined antipsychotic medications and dosages during the follow-up period. All participants were outpatients at the time of the baseline and follow-up assessments. The study was approved by the UCLA Institutional Review Board, and all participants provided written informed consent.

Measures

Symptom Ratings. Psychiatric symptoms during the previous month were assessed using the expanded 24-item UCLA version of the Brief Psychiatric Rating Scale (BPRS)[22] by a trained rater. This study focused on positive symptoms as assessed by the thinking disturbance factor (mean of unusual thought content, hallucinations, conceptual disorganization), negative symptoms (mean of blunted affect, motor retardation, emotional withdrawal), and general psychopathology (mean of all 24 BPRS items).

Social Cognition. Full descriptions of the following 3 social cognitive measures and their psychometric properties are provided in the companion article[1]:

Mayer-Salovey-Caruso Emotional Intelligence Test 2.0 The MSCEIT is a self-report instrument that consists of 141 items and 8 ability subscales, which assess 4 components (branches) of emotional processing that each includes 2 subscales[23]: (1) Identifying Emotions, (2) Using Emotions (to facilitate cognition), (3) Understanding Emotions, and (4) Managing Emotions. Responses include 5-point Likert ratings with specific anchor points for some items and a 5-item multiple-choice format for others. MSCEIT scores were derived using the general consensus approach based on a large community sample rather than the expert rating approach.

TASIT Part III: Social Inference—Enriched The TASIT[15] (Part III) consists of 16 videoed scenes, each

lasting 15–60 seconds, depicting lies or sarcasm (8 of each presented in a fixed random order). For each scene, participants answer 4 types of forced-choice (yes/no) questions: (1) What one character in the scene is doing to the other; (2) What the character is trying to say to the other person; (3) What the character is thinking; and (4) What the character is feeling. The test provides an overall total score (maximum = 64).

Relationships Across Domains The RAD is a 75-item paper-and-pencil measure of social perception that assesses competence in relationship perception.[16] This measure is unique in its focus on the ability to appreciate different types of relationships between 2 people in a vignette (eg, does one person have more authority than the other; do 2 people share everything as equals). This type of relationship/social perception differs from person perception in which the focus is on detection of discrete social cues displayed by a single person (eg, posture, eye gaze, and hand gestures). The RAD contains 25 vignettes that describe interactions involving a male-female dyad, each followed by 3 statements that describe the dyad's interpersonal behavior in domains of social life different from that of the vignette. Participants are asked to use what they learned about the dyad from the vignette to indicate whether the behaviors described in the 3 statements are likely or unlikely to occur by answering "yes" or "no" (maximum = 75).

Real-World Outcome Measures. General psychosocial functioning was assessed using the Role Functioning Scale (RFS[24]), an interviewer-rated scale that measures 4 major domains of functioning in everyday life. The current study examined 3 of these domains: work (or school) productivity (frequency and quality of engagement in productive vocational activities), independent living (level of self-sufficiency and self-care skills), and social network (number of close friends, frequency of contact, and quality of engagement in interactions), each rated on specific anchor points ranging from 1 (severely limited functioning) to 7 (optimal functioning) (the domain of relationships with family and spouse was not considered due to limited variability). The RFS ratings were completed based on the Community Assessment of Functioning interview[25], a comprehensive semi-structured interview conducted with participants that assesses several aspects of community functioning (collateral informants were not used). The RFS has sound psychometric properties[26] and has been used widely in services outcome studies in schizophrenia.[27]

Data Analysis

Preliminary analyses of the distributional properties of all measures at both assessments indicated that parametric statistical tests were appropriate. At baseline, 1 participant was missing the TASIT and 1 was missing the RAD. At follow-up, 3 participants were missing the MSCEIT, 8 were missing the TASIT, and 8 were missing the RAD. All statistical tests are 2-tailed, using a significance level of $P < .05$.

Primary analyses examined (1) the stability of mean scores and correlations between social cognitive task performance at 0 and 12 months and (2) cross-sectional and longitudinal associations between social cognition and functional outcome using cross-lagged panel analyses. In the stability analyses, performance on the 3 social cognitive tests across occasions was evaluated with paired *t*-tests and Pearson correlations. Given the absence of differential predictions for the 3 social cognitive measures and their relatively high degree of shared variance (*r*'s = .60–.66 at baseline; .65–.73 at follow-up), a composite social cognition score was computed for use in these stability analyses and in the following cross-lagged panel analyses. Composite scores were calculated by converting scores on each social cognition measure to population *z* scores based on a large sample ($n = 174$) of healthy comparison subjects and then computing an average score from the 3 measures for each participant (in case of missing data we required that at least 2 of the 3 social cognitive measures be present to compute a composite score; $n = 46/55$ at follow-up). There were no significant differences at baseline between the 46 participants who did and the 9 participants who did not have sufficient data to compute a follow-up social cognition composite score on any demographic or clinical variables (all *P*'s > .05). For comparative purposes, stability analyses were also conducted for the main symptom and functional outcome variables.

The 2-wave panel design of this study (0 and 12 mo) allowed us to evaluate associations between social cognition and functional outcome using cross-lagged panel analyses,[28] which incorporate (1) cross-sectional correlations between social cognition and outcome at each occasion and (2) longitudinal effects of social cognition at baseline on functional outcome over 12 months. A key benefit of cross-panel analysis over standard zero-order correlations is that it allows investigators to examine hypotheses about causality—ie, whether social cognition affects functional outcome or vice versa by evaluating the temporal order of effects. Causality is traditionally inferred on the basis of (1) correlation (A and B are associated with each other), (2) time precedence (a cause "A" is assumed to temporally precede and affect "B"), and (3) nonspuriousness—ie, effects are not attributable to unmeasured "third variables."[28,29] Cross-lagged panel analyses address not only the correlation criterion but also the time precedence criterion by examining the predictive association between two variables over time, each explicitly controlling for effects at earlier time points.

Following the procedures described by Kenny,[28] we evaluated the "cross-lagged" correlations between each measure at baseline and the other measure at 12 months

(baseline social cognition and 12-mo functioning vs baseline functioning and 12-mo social cognition). These tests were simultaneous, ie, they incorporated information from all other correlations within the same model and take autocorrelations and concurrent correlations into account.[28] A finding that one of these cross-lagged correlations is significantly larger suggests that the direction of causality is from that baseline variable to the other. Importantly, cross-lagged panel analyses do not directly address the nonspuriousness criterion mentioned above, thereby constraining causal inferences. However, the current analyses also considered one key potential third variable, namely, the impact of clinical symptoms.

Results

Stability of Social Cognition, Symptoms, and Functioning

Descriptive data and statistical test results are presented in table 1. For the social cognition measures, the Composite, TASIT, and RAD scores improved across the follow-up period with effect sizes (Cohen's *d*) in the small to medium range. Scores on the MSCEIT did not significantly change, and the effect size was small. Test-retest correlations were in the good to high range[30] exceeding .70 for all the social cognition measures. Thus, despite small-to-medium improvements on 3 of the social cognition measures across the 12-month follow-up period, scores on all measures were quite stable over time.

For symptoms, there was a significant decrease in overall symptom levels from 0 to 12 months, indicating that patients showed general improvements in psychopathology over the study period. However, there was only a small nonsignificant trend for improvement in positive symptoms and no significant change for negative symptoms. Test-retest correlations were notably smaller for symptoms than for scores on the social cognition measures. On the functioning measures, there were significant improvements in work and independent living, which were both in the medium range. Social functioning did not significantly change across assessments. Test-retest correlations for the functional measures were all significant and were medium to large.

Cross-lagged Panel Analyses

Cross-sectional and longitudinal correlations between the social cognition composite score and the 3 functional outcome variables are shown in the cross-lagged panels in figure 1.

Cross-sectional Correlations. For the cross-sectional correlations, presented on the vertical lines of each panel, there was a similar pattern across the 3 outcome domains. At baseline, the correlations between social cognition and functioning were uniformly small and nonsignificant. In contrast, at the 12-month follow-up, the correlations between social cognition and functioning were consistently significant and moderate to large. The correlations were all in the direction of better social cognition relating to better real-world functioning.

Table 1. Social Cognition, Symptoms, and Functioning at Baseline and 12-Month Follow-up Assessments

	Baseline		Follow-up				
	Mean	SD	Mean	SD	*t*	*d*	*r*
Social cognition							
Composite	−1.36	1.21	−1.13	1.30	2.63*	.38	.87***
MSCEIT	87.18	14.45	86.04	14.12	1.11	.16	.87***
TASIT	46.55	7.12	48.82	8.39	2.50*	.36	.71***
RAD	48.67	8.75	51.09	9.64	2.43*	.35	.74***
Symptoms							
Total	1.60	0.39	1.43	0.26	2.79**	.41	.30*
Positive	1.97	1.12	1.72	0.87	1.71	.25	.44**
Negative	2.04	0.79	1.84	0.94	1.47	.21	.33*
Functioning							
Work	3.08	1.72	4.11	2.02	3.89***	.57	.47***
Independent living	4.06	1.51	4.47	1.41	2.55*	.37	.67***
Social	4.34	1.71	4.68	1.71	2.16	.32	.78***

Note: Composite score is a standard score based on a normative sample of 174 healthy comparison subjects. MSCEIT, Mayer-Salovey-Caruso Emotional Intelligence Test; TASIT, The Awareness of Social Inference Test; RAD, Relationships Across Domains test; *d* = effect size for paired-samples *t*-test.
*$P < .05$; **$P < .005$; ***$P < .001$.

Causal Explanatory Models of Longitudinal Relations: Directionality of Causal Effects. The cross-temporal correlations are shown as diagonal lines between baseline measures in one domain and 12-month measures in the other. Across all functional domains, better baseline social cognition significantly predicted better 12-month functioning. However, the reciprocal correlations between baseline functioning and 12-month social cognition were uniformly nonsignificant. Statistical comparisons of the cross-lagged correlations indicated that correlations between baseline social cognition and 12-month functioning were significantly larger than the reciprocal cross-lag correlations (12-month social cognition to baseline functioning) for the domain of work ($Z = 2.22$, $P = .01$), a trend for independent living ($Z = 1.38$, $P = .08$) and nonsignificant for social networks ($Z = 0.42$, $P = .34$). For work, these findings suggest that the direction of causality is from baseline social cognition leading to 12-month functioning. This overall pattern of correlations and cross-lagged correlations for the composite social cognition score generally mirrored the patterns found for each of the 3 individual social cognition tests (see online supplementary material for Figures 1–3).

Role of Symptoms. Additional analyses were conducted to determine whether the association between social

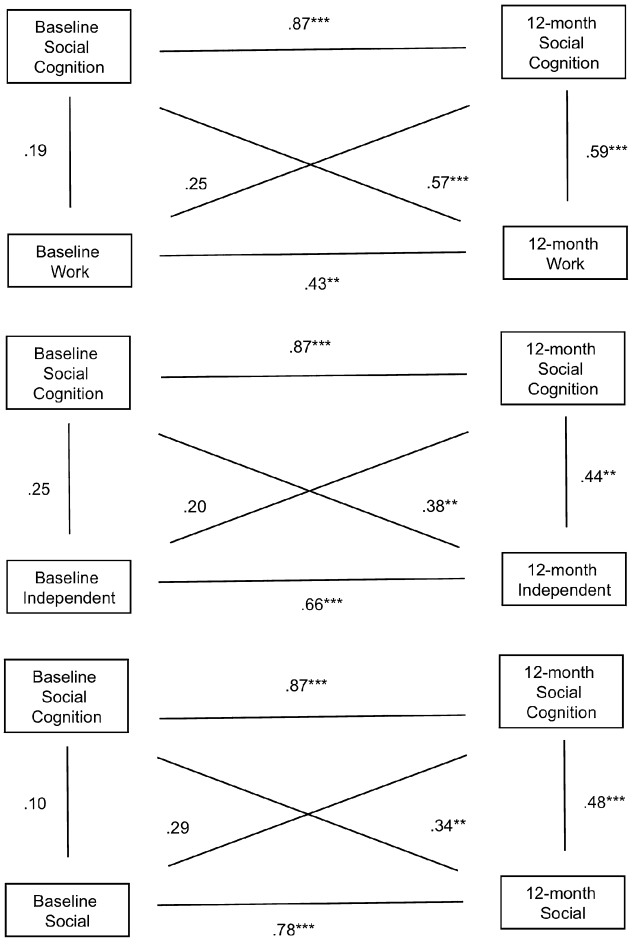

Fig. 1. Cross-lagged panel correlations between social cognition composite score and work productivity, independent living, and social networks.

cognition and later real-world functioning was affected by total, positive, or negative symptoms. Significant correlations between social cognition and symptoms are summarized as follows: Baseline social cognition significantly correlated with total symptoms at baseline ($r = -.36$, $P < .05$) and follow-up ($r = -.41$, $P < .01$), with positive symptoms at baseline ($r = -.33$, $P < .05$) and follow-up ($r = -.31$, $P < .05$), and with negative symptoms at follow-up ($r = -.33$, $P < .05$); follow-up social cognition significantly correlated with total symptoms at follow-up ($r = -.41$, $P < .005$) and with negative symptoms at follow-up ($r = -.49$, $P < .005$).

For these analyses, we controlled for the effect of symptoms on the social cognition and outcome measures by partialing out the symptom ratings. Then, cross-lagged panel correlations were recalculated using these residual scores. For the domain of work, the cross-lagged correlations from baseline social cognition to 12-month work remained significantly larger than the reciprocal cross lags after accounting for total, positive, or negative symptoms (all Z's > 2.0, $P < .05$). For the domains of independent living and social functioning, the differences between cross-lagged correlations were not significant after accounting for total, positive, or negative symptoms.

Discussion

This study evaluated the longitudinal stability and functional correlates of 3 relatively new social cognitive measures in first-episode schizophrenia. All 3 measures demonstrated generally good stability over the 12-month study period. Although social cognition showed minimal cross-sectional relations with functioning at baseline, social cognition scores at the baseline and follow-up assessments were both robustly associated with work, independent living, and social functioning at follow-up. Furthermore, cross-lagged panel analyses suggested that lower levels of baseline social cognition led to poorer work outcome over 1 year. As discussed further below, the longitudinal stability of social cognitive impairments during early schizophrenia extends the results of a companion study,[1] which found stable impairments across patients in different developmental phases of illness on the same social cognitive measures. These converging results have important implications for understanding vulnerability to schizophrenia and for developing clinical interventions that maximize long-term functional recovery.

Stability

Although impairments on the social cognitive tests used in this study and on more traditional tasks have previously been found in cross-sectional studies of early schizophrenia,[1] this study is among the first to examine these impairments longitudinally.[12] Tests of emotional intelligence, theory of mind (detection of sarcasm and deception), and relationship/social perception were highly stable in terms of test-retest correlations, ranging from .71 to .87 across the 12-month study period. These correlations were substantially higher than those found for clinical symptom ratings. This stability is striking in the context of the major lifestyle changes and adjustments to engaging in treatment that typically accompany early schizophrenia. There is emerging evidence that the stable social cognitive impairments found in this recent-onset sample may precede the development of the full syndrome of schizophrenia.[4,6] For example, a companion study using the same measures found large social cognitive deficits across prodromal, first-episode (including those in the current study), and chronically ill patients.[1] The longitudinal stability in the current study converges with evidence of cross-phase stability, as well as mild impairments found in unaffected biological relatives[31–34] and psychometrically defined schizotypy (eg, Phillips and Seidman,[35] Aguirre *et al*,[36] Henry *et al*,[37,38] Langdon and Coltheart,[39] and Meyer and Shean,[40] but see Jahshan and

Sergi[41] and Fernyhough *et al*[42]) to suggest that social cognitive variables play a key role in understanding vulnerability to schizophrenia.

Further research is needed to establish social cognition as a vulnerability factor for schizophrenia, to specify the ways it contributes to vulnerability, and to clarify its association with other putative vulnerability factors. For example, in the current study, there was some evidence that social cognitive impairment is not fully independent of symptom state; impairments showed associations with some clinical symptoms and small-to-medium improvements on 2 of the social cognitive tasks as general clinical symptoms improved across the 12-month follow-up period. These findings suggest that social cognitive impairment may reflect a mediating vulnerability factor (ie, present during acutely symptomatic and relatively remitted periods, but more deviant during symptomatic periods) rather than a stable vulnerability factor (ie, independent of symptom fluctuations and not directly linked to development of symptomatic periods) (see Nuechterlein *et al*[21] and Nuechterlein and Dawson[43] for further discussion of this distinction). The issue of whether social cognitive deficits contribute to vulnerability independently of nonsocial neurocognitive vulnerability factors also requires further attention. However, evidence that social cognition is psychometrically distinguishable from neurocognition, relies on at least semi-independent neural substrates, and is impaired in patients unaffected relatives even after accounting for neurocognition[44–46] suggests that social cognitive characteristics may provide informative new endophenotypes for neurobiological and genetic studies.[47] In addition, developmental models that incorporate social cognitive variables may provide a useful framework for studies of vulnerability and conversion to psychosis, a research direction that we are currently pursuing.

Longitudinal Associations With Functioning

To our knowledge, this is the first study to demonstrate that social cognitive impairments prospectively predict later real-world functioning during the early course of schizophrenia. Although social cognition showed relatively small cross-sectional relations with functioning at baseline, social cognition at baseline and follow-up assessments robustly and broadly predicted functioning at the 12-month follow-up across the domains of work, independent living, and social networks. The magnitude and consistency of these functional correlates are more pronounced than those we have previously found with the same measures in chronically ill patients.[17–19] These findings bolster growing evidence that social cognitive impairments are related to functional outcome,[48] even during the early course of schizophrenia.[12,13]

It is unclear why the cross-sectional relations between social cognition and outcome were significant at the 12-month assessment but not at baseline. Although one might suspect that this reflects a truncated range on the outcome measures at baseline due to poor functioning around the start of treatment, variability on the outcome measures did not dramatically differ across assessments. One possibility is that the sample was closer to a psychotic episode at baseline, and this clinical instability had a larger general impact on functioning than social cognition. As patients continued to stabilize, the relevance of social cognition to functioning emerged by the 12-month follow-up, which may have reflected a more characteristic and stable estimate of the patients' levels of functioning. Another potentially relevant consideration is that the patients were involved in treatment throughout the study period. Perhaps baseline social cognition levels in this sample reflect how much patients are able to benefit from engagement in intensive treatment.

The longitudinal design of this study also enabled us to explore whether the data supported a model in which social cognition has a causal effect on later functional outcome. Cross-lagged panel analyses suggested that the direction of causality is from lower levels of baseline social cognition to worse functional outcome over 1 year. This was particularly true for work functioning over 1 year, where the results held up even after accounting for clinical symptoms. The predictive relations for independent living and social functioning were diminished after accounting for symptoms, suggesting that psychiatric symptoms may have a more pronounced impact on functioning in the early phase of schizophrenia than in chronic samples.[49,50]

One may wonder why social cognition had a more robust impact on outcome in the domain of work functioning than social functioning. The process of successfully obtaining and maintaining work or school activities is often highly saturated with social demands, which rely on one's ability to navigate novel and fluctuating interpersonal interactions. In addition, prior longitudinal studies support linkages between social cognition and work outcome. In a study of chronically ill patients using the same analytic approach,[51] our group found support for a causal relation between baseline affect perception and later work (combined with independent living) functioning. Furthermore, a structural equation modeling study of predictors of work rehabilitation success found that poor social cognition led to social discomfort on the job, which in turn led to poorer rehabilitation success.[52] Thus, social cognitive skills may play a key role in determining how well people interact with others in the work place, ultimately impacting overall work quality and tenure. The less robust findings for the domain of social functioning in the current may partly reflect features of the particular functional outcome measure that was used. The RFS social network rating focuses on the frequency and quality of engagement in close friendships, which may limit the scale's sensitivity to capture adaptive functioning in more general everyday interactions.

Limitations and Clinical Implications

This study has some limitations. First, although social cognition predicted later functioning, we did not evaluate the extent to which this was independent of other known predictors, such as neurocognition. The current study is limited by the absence of a neurocognitive battery, though our group and others have previously found that social cognition accounts for unique variance in outcome beyond basic neurocognition.[2,3] Second, the patients were taking antipsychotic medications at clinically determined dosages and their impact on task performance is unclear. Available data suggest that, if anything, antipsychotic medications may slightly improve social cognition.[3] Third, our functional outcome measures were based on information provided by patients during a semi-structured interview; the use of collateral information could enhance the validity of outcome assessments.[53] Fourth, follow-up data were not collected from healthy controls to directly compare longitudinal stability between groups. Fifth, the model of causation provided by cross-lagged panel correlation analysis is limited.[29] We have shown both association and temporal order of our hypothesized cause-and-effect relationship between social cognition and outcome, but we are not able to exclude the case that an underlying third variable is the cause of both. Causal inferences about social cognition should therefore be interpreted cautiously and further investigated using alternative study designs, such as longitudinal studies of high-risk samples that minimize potential confounds (eg, medication effects) or randomized clinical trials that assess the functional impact of treatments designed to enhance social cognition.

Social cognitive deficits appear to be stable determinants of real-world functioning during even the early phase of schizophrenia. Clinically, these findings highlight the potential value of intervening at the level of social cognition as a means of improving functioning outcome. An emerging body of treatment development research in chronically ill patients suggests that social cognition can be improved through targeted skills training approaches.[54] Initial evidence indicates that implementing such treatments early in the course of illness could provide a potent benefit for promoting recovery and long-term outcome.[55]

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Supplementary Material

Supplementary material is available at http://schizophreniabulletin.oxfordjournals.org.

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