Social Cognitive Markers of Short-Term Clinical Outcome in First-Episode Psychosis

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Abstract

Objective: In psychotic disorders, impairments in cognition have been associated with both clinical and functional outcome, while deficits in social cognition have been associated with functional outcome. As an extension to a recent report on neurocognition and short-term clinical outcome in first-episode psychosis (FEP), the current study explored whether social cognitive deficits could also identify poor short-term clinical outcome among FEP patients. **Methods:** We defined the social-cognition domain based on the scores from the Hinting Task and the Four Factor Tests of Social Intelligence. Data were collected in 45 FEP patients and 26 healthy controls. The patients were divided into good- and poor-outcome groups based on clinical data at six months following initiation of treatment. Social cognition was compared among 27 poor-outcome, 18 good-outcome, and 26 healthy-control participants. **Results:** Outcome groups significantly differed in the social cognition domain (z-scores: poor outcome=-2.0 [SD=1.4]; good outcome=-1.0 [SD=1.0]; p=0.005), with both groups scoring significantly lower than the control group (p<0.003). Moreover, outcome groups differed significantly only on the Cartoon Predictions subtest (z-scores: poor outcome=-2.7 [SD=2.7]; good outcome=-0.7 [SD=1.8]; p=0.001) among the five subtests used. **Conclusions:** Overall, social cognition appears to be compromised in all FEP patients compared to healthy controls. More interestingly, significant differences in social cognitive impairments exist between good and poor short-term clinical outcome groups, with the largest effect found in the Cartoon Predictions subtest.

Key Words: Social Cognition, Clinical Outcome, First-Episode Psychosis

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Introduction

Individuals who experience first-episode psychosis (FEP) vary in their response to treatment (1, 2), as well as in their cognitive abilities. Furthermore, there has been growing evidence that cognitive deficits are a core feature of schizophrenia (3, 4) and other psychotic disorders, and that cognitive deficits, namely verbal memory, are associated with aspects of functional outcome in chronic patients (5, 6), as well as in those experiencing their first episode (7, 8).

As part of the recent attempt to establish consensus on domains of cognition in schizophrenia (Measurement and Treatment Research to Improve Cognition in Schizophrenia [MATRICS]), social cognition has been added because of

Clinical Implications

Social cognitive deficits have been hypothesized to affect the clinical outcome of patients by delaying the response to treatment or by impairing the client's motivation to adhere to treatment as prescribed (7). Consequently, it would be important to identify a poor outcome earlier on so, as clinicians, we can pay special attention to this specific subgroup and possibly provide more intensive psychosocial interventions and/or introduce alternative antipsychotics earlier on in the treatment process to better benefit a larger proportion of clients. In addition, studies have found evidence between cognitive improvement and better functional outcome, suggesting that cognition should be part of the focus during the treatment of schizophrenia (37). If one operates on the basis that social cognitive deficits are linked to short-term clinical outcome, psychosocial interventions should include elements of psychoeducation about the illness, behavior activation to improve motivation, and cognitive remediation in order to improve overall areas of neurocognition in hope of better outcome.

its relevance for clinical trials and functional outcome (9). Social cognitive abilities are composed of several mental operations such as perception, interpretation and the processing of social information. Social cognition is best defined as "the ability to conceptualize other people's beliefs, thoughts and intentions in order to explain and anticipate their behavior" (10).

Over the past two decades, studies have clearly shown that social cognition is significantly impaired in chronic schizophrenia (11, 12) and in FEP (13-15). More interestingly, these deficits have been related to a poorer functional outcome in both schizophrenia (11, 16, 17) and in FEP (13, 14). The relationship between social cognition performance and symptomatic or short-term clinical outcome has received little attention so far, although associations have been identified. Deficits in social cognition have been explained by either a lack of development of such abilities premorbidly, as in the case of patients with predominantly negative symptoms, or as result of a loss of such abilities consequent to positive symptoms (18). Whether social cognition is associated with an early reduction of symptoms, similar to what has been reported for verbal memory (19), still remains unexplored. If such an association does exist, it will further increase the significance of assessing the domain of social cognition as part of a cognitive battery of tests given the importance of a full syndromal remission early in the treatment of psychosis (20).

With past research relating social cognitive deficits to a poor premorbid adjustment in patients with predominantly negative symptoms or resulting from a loss of abilities linked to positive symptomatology (18), it would be expected that deficits in this domain could significantly affect clinical outcome. Based on this and the above mentioned findings, we hypothesized that all FEP patients would show social cognitive deficits in comparison to matched healthy controls. Furthermore, we hypothesized that deficits in social cognition would be associated with a poor short-term clinical outcome during the initial stages of treatment following an FEP.

Methods

Participants, Treatment Setting and Treatment Protocol

All participants were part of an ongoing, longitudinal behavioral and imaging study being conducted at the Douglas Mental Health University Institute in Montreal, Canada. All FEP patients were recruited and treated through the Prevention and Early Intervention Program for Psychoses (PEPP-Montreal), a specialized early-intervention service with integrated clinical, research, and teaching modules. The program involves a comprehensive approach with intensive medical and psychosocial management. All patients are provided modified assertive case management and interventions to assist in their recovery (for further details on the program, see [21]). Patients aged 14 to 30 years from the local catchment area suffering from either affective or nonaffective psychosis, who had not taken antipsychotic medication for more than one month, were consecutively admitted to the program as either in- or out-patients. There is no competing service and treatment is publicly funded.

From PEPP-Montreal we recruited 48 patients for an imaging study, who had also completed supplementary social cognitive tests: the Hinting Task (18) and the Four Factor Tests of Social Intelligence (22). From our study, three patients were subsequently removed due to a later confirmed diagnosis of substance-induced psychosis. In addition, two poor-outcome patients refused antipsychotic medications as a treatment option. These clients still received the psychosocial interventions. The remaining 45 patients were subsequently separated into good-outcome (n=18) and poor-outcome (n=27) groups based on six-month clinical data. As per an earlier report, good outcome was defined by a rating of 2 or less (mild) on all global subscales of the SAPS and 3 or less (moderate) on all global subscales of the SANS, excluding the subscale of "attention" (23). For the present study, all FEP patients were included, which was comprised of 39 schizophrenia spectrum disorder (poor outcome=24;

Table 1 **Characteristics and Global Symptom Ratings of Poor-Outcome and Good-Outcome Groups. (Number of participants included [n] for each variable where different from sample.)**

	Poor Outcome (n=27)	Good Outcome (n=18)	Analysis		
			Statistic	df	p value
DUP (median, weeks)*	38.3±44.3	93.7±148.2	t=-1.27	43	0.21
DUI (median, weeks)*	277.4±250.2	295.3±260.3	t=-0.24	43	0.81
Antipsychotic at Testing (mg/day)			χ^2=5.31	5	0.38
Olanzapine	11.8±5.0 (n=11)	10.9±8.0 (n=7)	t=0.40	16	0.77
Risperidone	2.3±0.8 (n=9)	1.5±0.8 (n=5)	t=1.80	12	0.10
Quetiapine	350.0±173.2 (n=5)	775 (n=1)	t=-2.24	4	0.90
Risperidone-Injected	(n=0)	25.0 (n=1)	n/a		
Haloperidol	(n=0)	1.5 (n=1)	n/a		
None	(n=2)	(n=3)	n/a		
Medication Adherence†	3.2±1.0	3.5±0.8 (n=16)	t=-1.04	41	0.31
SAPS Total					
Baseline	28.9±16.6	35.1±17.6	t=-1.20	43	0.24
Six Months	12.1±11.8	1.7±2.8	t=3.66	43	0.001‡
Change	16.8±12.4	33.4±17.4	t=-3.75	43	0.001‡
SANS Total					
Baseline	28.3±13.6	27.4±13.3	t=0.22	43	0.83
Six Months	25.3±11.1	14.5±18.7	t=2.44	43	0.02‡
Change	3.0±13.2	12.9±17.4	t=-2.18	43	0.04‡

DUP=duration of untreated psychosis; DUI=duration of untreated illness; SAPS=Scale for the Assessment of Positive Symptoms; SANS=Scale for the Assessment of Negative Symptoms. *DUP and DUI are presented in raw form; however, these were analyzed using transformed data. †Medication adherence average over six months: 0 (never adherent) to 4 (always adherent). ‡Significant at 0.05 level.

good outcome=15), 4 affective psychosis (poor outcome=2; good outcome=2), and 2 psychosis NOS (poor outcome=1; good outcome=1).

Twenty-six healthy controls were recruited through advertisements in local newspapers and took part in social cognitive testing sessions. Controls were included only if they had no current or past history of: 1) any Axis I disorders; 2) any neurological diseases; 3) head trauma causing loss of consciousness; and, 4) a first-degree family member suffering from schizophrenia or related schizophrenia spectrum psychosis. Controls were also chosen on sociodemographic variables such as age (at testing), gender, and parental socioeconomic status during childhood matched to FEP patients who were taking part in a neuroimaging study.

After a comprehensive description of the study, written informed consent was obtained from all participants. The Douglas Mental Health University Institute review board approved the research protocols.

Clinical and Demographic Assessments

Patients were diagnosed according to the *DSM-IV* criteria based on the Structured Clinical Interview for DSM-IV (24) and confirmed through consensus between two senior research psychiatrists (A.M. and R.J.). Positive and negative symptoms were assessed with the Scale for the Assessment of Positive Symptoms (SAPS) (25) and the Scale for the Assessment of Negative Symptoms (SANS) (26), respectively. The baseline interview session was conducted within one month of entry into the program (mean=23.4 days, SD=8.6 days, range=4.8–46.2 days). The symptom ratings covered the previous one month and were repeated monthly until the third month and then again at six months, nine months, and twelve months past baseline as shown in Table 1. Symptom ratings are performed by research assistants (ICC=0.75 for both the SAPS and the SANS) who had received extensive training and supervision with interrater reliability measured at least once a year.

Medication adherence was assessed at each of the aforementioned time points and averaged over the first six-month period to provide an overall adherence score. Medication adherence was based on a 5-point scale ranging from 0 (never adherent) to 4 (fully adherent) on information obtained from patients, family members, case managers, and psychiatrists. Duration of untreated psychosis (DUP) was

Table 2 Sociodemographic Data of Poor-Outcome, Good-Outcome, and Healthy- Control Groups

	Poor Outcome (n=27)	Good Outcome (n=18)	Healthy Controls (n=26)	Analysis		
				Statistic	df	p value
Age (years)	23.5±3.7	23.9±3.0	24.7±3.6	F=0.81	2,68	0.45
Parental SES*	3.8±1.1	2.9±1.4	3.2±1.0	χ^2=6.82	2	0.03‡
Gender (M/F)	19/8	12/6	14/12	χ^2=1.67	2	0.43
Education†	11.6±2.8	12.2±2.5	14.4±1.7	F=10.01	2,68	<0.001‡

*Hollingshead socioeconomic status (1=highest and 5=lowest); data was not available for all patients. Mann-Whitney post hoc analyses revealed: poor>good (p=0.03), poor<control (p=0.03), good=control (p=0.52).
†Tukey HSD post hoc analyses revealed: poor=good (p=0.68), poor<control (p<0.001), good<control (p=0.01).
‡Significant at 0.05 level.

calculated as the time period from onset of psychotic symptoms judged to be at threshold level according to *DSM-IV* criteria until time of adequate treatment with antipsychotics (thirty days of continuous treatment or less if remission of positive symptoms occurred). Duration of untreated illness (DUI) was defined as the time period from onset of any psychiatric symptoms to adequate treatment with antipsychotics (27).

Parental socioeconomic status (SES) was assessed with the Hollingshead socioeconomic status rating scale (28). SES is an estimation that is achieved by considering the occupational status and the highest level of education attained by a parent, including other family assets and resources. Finally, the type and dosage of antipsychotic taken at the time of the social cognitive evaluation were recorded. All interview sessions acquiring the collection and assessment of pertinent data were performed by a trained professional.

Social Cognition Measures

Patients were assessed after the initiation of treatment and only when in a stable, but not necessarily asymptomatic, condition. As presented in Table 1, there was no difference between patient groups with respect to when evaluations took place following entry into the program (poor outcome: mean=19.8 weeks, SD=9.6; good outcome: mean=18.7 weeks, SD=13.0; t=0.33, df=43, p=0.74).

The two social cognitive tests used were the Hinting Task (18) and the Four Factor Tests of Social Intelligence (22). The Hinting Task tests the ability of subjects to infer the real intentions behind indirect statements. Ten short passages are read to the subject one at a time, presenting an interaction between two characters with one of the characters giving a very obvious hint at the end. The examinee must then tell what the character really meant. If the examinee fails, then he is asked what one character wants the other one to do.

The Four Factor Tests of Social Intelligence measures the ability to understand thoughts, feelings, and intentions of other people. There are four different subtests (Cartoon Predictions, Expression Grouping, Social Translation, and Missing Cartoons), of which three use pictorial stimuli (comic strips and drawings) and one employs printed sentences only. Each question is worth one point, yielding four distinct scores (one for each subtest) and a global composite score (by summing up the totals of the subtests) of social cognition.

The Cartoon Predictions Subtest

The Cartoon Predictions subtest is a 14-item task that measures the ability to predict social consequences by showing a cartoon strip where the examinee must be able to anticipate the logical sequence of a given social situation simply by interpreting the cartoon characters' emotional reactions. The examinee must select, from four alternative cartoon frames, the one that most likely follows from an interpersonal situation depicted in the first cartoon frame. A common example involves a strip where a man is holding on to a roof, while a young boy is standing there, watching the scene. The man's facial expression seems to depict fear. The examinee must be able to conceive that the most logical strip would show the boy being helped by a woman who is carrying a ladder with the intention of helping the man come safely down from the roof (29).

The Expression Grouping Subtest

The Expression Grouping subtest is a 15-item task that involves the ability to abstract common attributes from different expressive images. Each item of the test consists of a group of three pictures representing either facial expression, hand gestures or body posture that correspond to a common thought, feeling or intention. To demonstrate a correct understanding of the situation, the participant must select

Table 3 Z-Scores (Mean, SD, and Range) and Between-Group Comparisons of Social Cognitive Tests among Good-Outcome, Poor-Outcome, and Healthy-Control Groups

	Poor Outcome			Good Outcome			Healthy Controls			Fisher's LSD Comparisons		
										Good vs. Poor	Poor vs. Control	Good vs. Control
	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range			
Overall Social Cognition	-2.0	1.4	-5.0–0.8	-1.0	1.0	-3.3–0.4	0.0	1.0	-0.8–1.0	0.005*	<0.001*	0.003*
Hinting Task	-1.9	2.2	-6.8–0.7	-1.5	1.8	-6.1–1.3	0.0	1.0	-2.0–1.3	0.615	0.001†	0.007†
Four Factor Social Intelligence												
Cartoon Predictions	-2.7	2.7	-9.8–1.5	-0.7	1.8	-5.1–1.5	0.0	1.0	-2.2–1.5	0.001†	<0.001†	0.251
Expression Grouping	-1.5	1.8	-5.1–2.2	-0.6	0.8	-2.0–1.0	0.0	1.0	-2.0–2.2	0.036	<0.001†	0.148
Social Translation	-2.7	2.1	-6.5–0.6	-1.7	1.5	-4.9–0.6	0.0	1.0	-1.8–1.4	0.048	<0.001†	0.001†
Missing Cartoons	-1.3	1.3	-3.5–1.9	-0.7	1.6	-3.1–1.9	0.0	1.0	-2.2–1.9	0.122	0.001†	0.097

*p value significant at 0.05. †p value significant at 0.01 (0.05/5—corrected for multiple comparisons).

one picture representing the same emotion from four alternatives.

The Social Translation Subtest

The Social Translation subtest is a 12-item task designed to measure the ability to recognize changes in behavioral meaning. Based on the principle that similar expressional cues can be associated with different meanings as a function of different contexts, the examinee must choose one of three possible sentences having a different meaning from the target sentence.

The Missing Cartoons Subtest

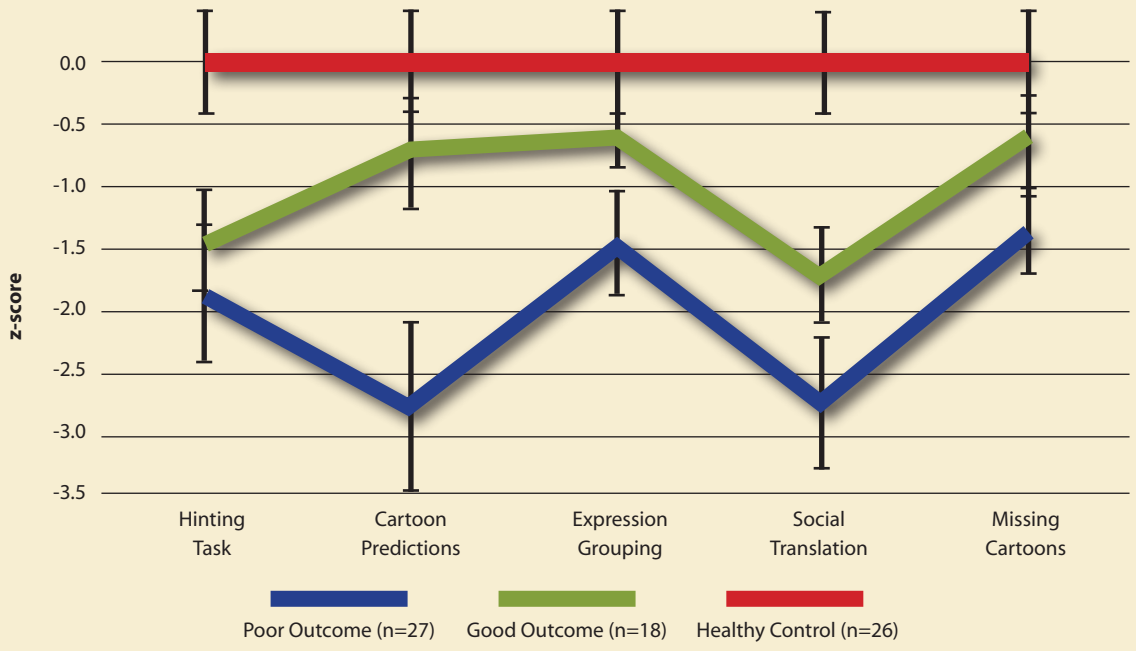
Finally, the Missing Cartoons subtest is a 14-item task measuring the ability to consider social context. Each item consists of an incomplete cartoon strip and, after interpreting each scene, the examinee chooses, from four alternatives, the panel that best completes the cartoon strip, giving the story a logical flow.

Statistical Analysis

All clinical characteristics were normally distributed except for duration of untreated psychosis (DUP) and duration of untreated illness (DUI), which were normalized using logarithmic and square root transformations, respectively. A one-way analysis of variance (ANOVA) was used to examine age (at testing) among the three groups. Independent t-tests were used to compare baseline and six-month total symptom ratings, changes in symptom scores, dosage of antipsychotic medication, medication adherence, DUP, DUI, and Premorbid Adjustment Scale (PAS) scores between the patient outcome groups. Parental SES and education level among the three groups were contrasted using a Kruskal-Wallis one-way ANOVA. Gender and type of antipsychotic medication were compared using cross tabulation and chi-square tests. All of the social cognitive variables were normally distributed. All subtest scores were transformed into standard equivalents (z-scores) using the mean and standard deviation of the healthy-control group.

For the present study, we created a social cognition domain by combining all five of the aforementioned subtests: that is, the Hinting Task and the four subtests of the Four Factor Tests of Social Intelligence. A univariate analysis of covariance (ANCOVA) was used to compare the performance of the social cognition domain among the groups, using group membership (poor outcome, good outcome, and control) as the between-group factor, the global social cognition domain as the within-group factor, and parental SES as a covariate. Post hoc Fisher's LSD was used to identify any group differences. A subsequent and supplementary within-subject multivariate analysis of covariance (MANCOVA) was used to examine the profile of the five subtests among the three groups using group membership as the between-group factor, the five subtests as the within-group factors, and parental SES as a covariate. Post hoc univariate ANCOVAs, along with Fisher's LSD, were used to identify any group differences. The critical p value for this analysis was set to 0.01, following the Bonferroni correction procedure to control for multiple comparisons. This analysis would allow us to observe if there were any differences with-

Figure 1 Social Cognitive Profile of Poor-Outcome, Good-Outcome, and Healthy-Control Groups



Results are displayed in z-scores with healthy controls defined with a mean of 0 and SD of 1. Error bars are equal to SD.

in each of the five subtests (that made up our social cognitive domain) among the three groups. Finally, for the entire sample, Pearson's chi-square and Spearman's rho (ρ) examined the independence and correlations, respectively, between the subtests and symptom levels at the time of the evaluation. Symptom data at the time of social cognitive testing were estimated from the symptom evaluation closest to administration. Additionally, cross tabulation and chi-square tests were used to examine if there was an effect of the heterogeneous sample on the social cognitive profile. All statistical tests were two-tailed with the critical p value set at 0.05 (except for the MANCOVA, as previously noted), and were performed using the Statistical Package for the Social Sciences, version 12 (30).

Results

Clinical and Demographic Data

The statistical analyses did not reveal any significant differences between the poor-outcome, good-outcome and control groups with respect to age and gender. The level of education of participants did not significantly differ among the experimental groups (good versus poor outcome), but these groups both differed from the control group. However, Table 2 shows how parental SES differed between the poor-outcome and good-outcome groups and the poor-outcome and control groups. In light of these differences, this variable was included as a covariate in our analysis. There were no differences in DUP, DUI, overall medication adherence, and the type of antipsychotic taken during social cognitive testing between the outcome groups. Finally, there were no between-group differences in both positive and negative symptoms at baseline. At six months, the poor-outcome group displayed significantly higher negative and positive symptoms, as per design. In addition, over the six-month period, improvements in positive and negative symptoms were significantly better for the good-outcome group as presented in Table 1.

Social Cognition Data

The univariate ANCOVA revealed mean differences in social cognition among the groups ($F=25.51$, $df=2, 67$, $p<0.001$; ES=0.81). Fisher's LSD revealed the poor-outcome group functioned at levels significantly below the good-outcome group, and that both outcome groups functioned significantly below that of the healthy-control group (Table 3). The MANCOVA revealed the social cognitive profiles among the three groups were not parallel as indicated by the significant (group x subtest) interaction ($F=5.13$, $df=10, 126$, $p<0.001$; ES=0.64; Figure 1). There were significant differences among the three groups on all five subtests: Hinting Task ($F=7.35$, $df=2, 67$, $p=0.001$; ES=0.47); Cartoon Predictions ($F=13.06$, $df=2, 67$, $p<0.001$; ES=0.63); Expression Grouping ($F=7.99$, $df=2, 67$, $p=0.001$; ES=0.49); Social

Table 4 Raw Data of Social Cognitive Tests for Poor-Outcome Patients, Good- Outcome Patients, and Healthy-Control Groups

	Poor Outcome			Good Outcome			Healthy Controls		
	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range
Hinting Task (max 20)	15.3	3.3	8–19	15.9	2.6	9–20	18.0	1.5	15–20
Cartoon Predictions (max 14)	9.5	2.8	2–14	11.6	1.8	7–14	12.4	1.1	10–14
Expression Grouping (max 15)	6.9	2.9	1–13	8.3	1.4	6–11	9.4	1.6	6–13
Social Translation (max 12)	6.8	2.7	2–11	8.1	1.9	4–11	10.3	1.3	8–12
Missing Cartoons (max 14)	5.9	3.0	1–13	7.3	3.5	2–13	8.8	2.2	4–13

Translation ($F=17.83$, $df=2, 67$, $p<0.001$; $ES=0.73$); and, Missing Cartoons ($F=6.43$, $df=2, 67$, $p=0.003$; $ES=0.44$). The mean performance of the social cognition subtests reveals these significant differences among the groups (Table 4). The good- and poor-outcome groups differed the least on the Hinting Task subtest (mean=15.9, SD=2.6; mean=15.3, SD=3.3, respectively), while they were most discrepant on the Cartoon Predictions task (mean=11.6, SD=1.8; mean=9.5, SD=2.8, respectively). Fisher's LSD revealed the poor-outcome group performed significantly lower than the good-outcome group in only the Cartoon Predictions subtest. Moreover, compared to the healthy controls, the poor-outcome group displayed significant deficits on all five subtests, while the good-outcome group displayed significant deficits in only the Hinting Task and Social Translation subtest, as shown in Table 3. In our previous study on nonsocial cognitive domains and short-term clinical outcome (23), we had observed significant group differences on verbal memory and working memory. We conducted those analyses again (ANCOVA and MANCOVA) and then examined social cognitive performance while covarying each of these nonsocial cognitive domains, but our results remained unchanged for all three groups. In particular, the largest group difference on nonsocial cognitive domain was observed on the working memory measure and, as can be seen in Table 5, covarying for it did not alter the results.

Finally, the total positive and negative symptoms at the time of testing were independent of all the social cognitive tests (all χ^2 values<356.6, all p values>0.10) except for positive symptoms and Social Translation subtest ($\chi^2=195.3$, $p=0.04$); symptoms were not correlated with any of the tests ($-0.25<\rho<0.07$, all p values>0.10). Chi-square tests revealed no effect of diagnosis on social cognitive tests (all χ^2 values<19.5, all p values>0.53) and diagnosis was independent of outcome ($\chi^2=0.29$, $p=0.87$).

Discussion

The present study identified a deficit in social cognition as a marker of short-term clinical outcome in first-episode psychosis (FEP) patients after six months of treatment. We found a significantly lower performance in the poor-outcome patients compared to the good-outcome patients at baseline, in addition to both outcome groups functioning below that of the control group. This finding adds to our previous report on nonsocial cognitive domains in which we reported poorer verbal memory and working memory performance were associated with a poor short-term clinical outcome in FEP patients (23).

Current trends in research, such as the NIMH-MATRICS, have suggested that, as a seventh domain, social cognition should include multiple measures including: emotional processing, theory of mind, social perception, social knowledge and attributions (31), making it comparable to the other cognitive domains (31). Furthermore, the MATRICS committee has made several recommendations, one of which is to use a single-test evaluation (Mayer-Salovey-Caruso Emotional Intelligence Test [MSCEIT]-Managing Emotions) to evaluate overall social cognitive ability. In contrast, the overall social cognitive domain included multiple measures, and the one-hour evaluation session for this domain is somewhat time consuming. Moreover, if one of the subtests, which can be administered in about twelve minutes, could have the same predictive ability as the overall domain, it would make more sense to have a shorter session if one is solely interested in predicting outcome. As such, we decided to investigate if a single test was having an overall effect on the domain. Our results suggested that the Cartoon Predictions subtest was the driving force behind the overall effect of the domain; subsequently leading to a shortened evaluation if the goal was to predict the clinical outcome. Similarly to the recommendations made by the MATRICS, we are suggest-

Table 5 P Values of the Different Group Comparisons for the Global Measure of Social Cognition and for the Different Subtests

	Good vs. Poor		Poor vs. Control		Good vs. Control	
	Original p	p with WM	Original p	p with WM	Original p	p with WM
Social Cognition Domain (average of 5 subtests)	0.005*	0.007*	<0.001*	<0.001*	0.003*	0.007*
Subtests						
Hinting Task	0.615	0.715	0.001†	0.004†	0.007†	0.017
Cartoon Predictions	0.001†	0.002†	<0.001†	<0.001†	0.251	0.470
Expression Grouping	0.036	0.084	<0.001†	0.002†	0.148	0.198
Social Translation	0.048	0.518	<0.001†	<0.001†	0.001†	0.003†
Missing Cartoons	0.122	0.412	0.001†	0.008†	0.097	0.168

Note: The "original" columns denote the p values observed for the group comparison whereas the "p with WM" denote the p value for the same group comparison after covarying for working memory performance. *p value significant at 0.05. †p value significant at 0.01 (0.05/5—corrected for multiple comparisons).

ing that Cartoon Predictions, which takes no more than ten minutes to administer, could possibly be used for evaluating a global deficit in social cognition in a time- and cost-efficient way.

The observed differences between the good- and poor-outcome groups, with respect to the Cartoon Predictions task, can be partially explained by the concept of "theory of mind." Social cognitive abilities are comprised of various mental operations, which include perception, interpretation, and processing of social information, to name only a few (32). One of the main features of social cognition is "theory of mind" (33), and it is defined as "the ability to conceptualize other people's beliefs, thoughts and intentions in order to explain and anticipate their behavior" (15). It is believed that the Cartoon Predictions task best embodies the concept of "theory of mind," in that this subtest measures the ability to predict social consequences by interpreting the intention and feelings of characters. In fact, severe social cognitive impairments in schizophrenia were found to be the best predictor of illness onset compared to nonsocial cognition and were linked to the duration of the illness and more so to "theory of mind" deficits (34). Considering that our poor-outcome group showed severe social cognitive impairments in the Cartoon Predictions task, one could hypothesize that a deficit in "theory of mind" could lead to an earlier onset of illness which would, in turn, have a negative effect on outcome for a subgroup of patients with a poor prognosis.

Our groups did significantly vary on the level of education. Once we further investigated the relationship amongst groups and level of education we found that there were no significant differences between the good- and poor-outcome groups. However, we did find that these two groups significantly differed from the healthy controls on this variable. Although there is well-established association between low level of education and risk for the development of schizophrenia, this association is orthogonal to our research question. Indeed, the focus of our study was to look mainly at the possible existence of differences in social cognition performance among first-episode psychosis patients who experience short-term good or poor outcome. This association could be further explored, but the focus of our study was to look at existing differences in patient course of illness and social cognition. The poor-outcome group functioned significantly below the healthy-control group on all subtests. Although the good-outcome group functioned at levels below that of the healthy controls, these groups differed significantly only on two of the subtests: the Hinting Task and the Social Translation subtest. This could suggest that those who achieve a quicker and more pronounced resolution of symptoms function better and, in some cases, on par with healthy controls as far as subtests of social cognition are concerned. Although in contrast with our particular result, a previous study demonstrated that people with remitted schizophrenia functioned on par with healthy individuals on the Hinting Task (18). At any rate, when compared to healthy controls, the functional impairments on specific subtests may not be equally compromised for all people suffering from psychosis. Although numerous studies have shown significant social cognitive differences between psychotic and control groups (10, 13, 14, 18), these studies did not account for the heterogeneity of outcome within FEP patients (i.e., good vs. poor outcome). Strangely, this did seem to be the case when examining overall social cognition performance (all subtests included). That is, both good- and poor-outcome groups functioned below that of the healthy controls. How-

ever, we must point out that this overall effect was driven by two tasks in particular: the Hinting Task and the Social Translation subtest. As such, there appears to be a heterogeneity of social cognitive functioning within those suffering from psychosis in relation to short-term clinical outcome with respect to individual subtests. Furthermore, even re-running all of the analyses covarying for the six nonsocial cognitive domains, which included working memory, did not significantly change our results. As such, it appears safe to conclude that nonsocial cognition has no significant effect on social cognitive ability in relation to short-term clinical outcome, which was our main variable of study.

The strengths of our study include a well-characterized sample of first-episode psychosis patients. Consistently, the clinic from which the sample derives is a well-established program which offers a thorough research protocol that includes systematic follow-up assessments and a consistent re-evaluation and validation of diagnosis. Moreover, by using a healthy-comparison group, we controlled for possible demographic differences that may occur with comparisons made to normative data. The heterogeneity of our sample, with respect to diagnosis, provided a more efficient research design for an outcome study (35). This follows from the idea that baseline diagnoses of first-episode patients change rather frequently (36), which could lead to erroneously drawing conclusions toward a specific diagnostic category.

This study has some limitations. From our study, two poor-outcome patients refused antipsychotic medications as a treatment option. These clients still received the psychosocial intervention and support allocated through the PEPP clinic, and the removal of these clients from the sample did not have any effect on our results. Although our size was adequate to detect highly significant group differences, our smaller sample size diminishes the generalization of our results to the general patient population. As such, replication of our results is needed to verify if there is indeed a true effect of social cognition in relation to clinical outcome.

Furthermore, our assessment of cognitive functioning in clinical settings often takes place at times when the patient is in a stable, but not necessarily asymptomatic, condition. A stable condition can sometimes be achieved within one or two month post treatment. Based on these latter findings, having some of the patients tested near the sixth month separation time from entering the program to receiving ongoing treatment for a period of over six months, we can assume that psychotic symptoms should have very little to no overall effect on our results. Nonetheless, we cannot entirely reject the possibility that psychotic symptomatology may have had an effect on performance of the social cognition tasks. Social cognition may need to be further investigated; and, until then, we cannot define the extent of how symptoms, time or clinical stability will affect social cognition.

Conclusions

Both of our studies have indicated that cognition appears to be a reliable marker of short-term clinical outcome following a first episode of psychosis. The present study found that poor social cognition (or more specifically, a deficit in the ability to predict social situations) is a marker of poor short-term clinical outcome after six months of treatment; our previous study identified verbal memory and working memory in the same capacity. Taken together, it would appear that specific impairments in either social cognition or nonsocial cognition, namely verbal memory, may be useful for identifying a poor prognosis early on in the treatment process following FEP.

Social cognitive deficits have been hypothesized to affect the clinical outcome of patients by delaying the response to treatment or by impairing the client's motivation to adhere to treatment as prescribed (7). Consequently, it would be important to identify a poor outcome earlier on so, as clinicians, we can pay special attention to this specific subgroup and possibly provide more intensive psychosocial interventions and/or introduce alternative antipsychotics earlier on in the treatment process to better benefit a larger proportion of clients. In addition, studies have found evidence between cognitive improvement and better functional outcome, suggesting that cognition should be part of the focus during the treatment of schizophrenia (37). If one operates on the basis that social cognitive deficits are linked to short-term clinical outcome, psychosocial interventions should include elements of psychoeducation about the illness, behavior activation to improve motivation, and cognitive remediation in order to improve overall areas of neurocognition in hope of better outcome.

We have attempted to provide evidence that specific deficits in social cognition are possible markers of poor short-term clinical outcome in FEP, and that not all patients show an equal deficit on all social cognitive measures. That is, patients responding to treatment function at levels similar to healthy individuals on particular subtests. Similarly to the MATRICS, the current study identified a single subtest from the social cognition domain—the Cartoon Predictions task—which appears to be useful for identifying a poor outcome in a short, twelve-minute evaluation session. However, additional studies will be needed in the future in order to support the current findings.

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References

1. Lieberman J, Jody D, Geisler S, Alvir J, Loebel A, Szymanski S, et al. Time course and biologic correlates of treatment response in first-episode schizophrenia. Arch Gen Psychiatry 1993;50(5):369-376.
2. Rosen K, Garety P. Predicting recovery from schizophrenia: a retrospective comparison of characteristics at onset of people with single and multiple episodes. Schizophr Bull 2005;31(3):735-750.
3. Goldberg TE, Green MF. Neurocognitive functioning in patients with schizophrenia: an overview. In: Davis KL, Charney D, Coyle JT, Nemeroff C, editors. Neuropsychopharmacology: the fifth generation of progress. Nashville (TN): American College of Neuropsychopharmacology; 2002. p. 657-669.
4. Gold JM, Green MF. Neurocognition in schizophrenia. In: Sadock VA, editor. Kaplan and Sadock's comprehensive textbook of psychiatry. 8th ed. Baltimore: Lippincott, Williams & Wilkins; 2002. p. 1426-1448.
5. Green MF. What are the functional consequences of neurocognitive deficits in schizophrenia? Am J Psychiatry 1996;153(3):321-330.
6. Green MF, Kern RS, Braff DL, Mintz J. Neurocognitive deficits and functional outcome in schizophrenia: are we measuring the "right stuff"? Schizophr Bull 2000;26(1):119-136.
7. Malla AK, Norman RM, Manchanda R, Townsend L. Symptoms, cognition, treatment adherence and functional outcome in first-episode psychosis. Psychol Med 2002;32(6):1109-1119.
8. Addington J, Saeedi H, Addington D. The course of cognitive functioning in first episode psychosis: changes over time and impact on outcome. Schizophr Res 2005;78(1):35-43.
9. Nuechterlein KH, Barch DM, Gold JM, Goldberg TE, Green MF, Heaton RK. Identification of separable cognitive factors in schizophrenia. Schizophr Res 2004;72(1):29-39.
10. Bertrand MC, Achim AM, Harvey PO, Sutton H, Malla AK, Lepage M. Structural neural correlates of impairments in social cognition in first episode psychosis. Soc Neurosci 2008;3(1):79-88.
11. Penn DL, Corrigan PW, Bentall RP, Racenstein JM, Newman L. Social cognition in schizophrenia. Psychol Bull 1997;121(1):114-132.
12. Corrigan PW, Penn DL. Social cognition and schizophrenia. Washington, DC: American Psychological Association; 2004.
13. Addington J, Saeedi H, Addington D. Influence of social perception and social knowledge on cognitive and social functioning in early psychosis. Br J Psychiatry 2006;189:373-378.
14. Williams LM, Whitford TJ, Flynn G, Wong W, Liddell BJ, Silverstein S, et al. General and social cognition in first episode schizophrenia: identification of separable factors and prediction of functional outcome using the IntegNeuro test battery. Schizophr Res 2007;99(1):182-191.
15. Bertrand MC, Sutton H, Achim AM, Malla AK, Lepage M. Social cognitive impairments in first episode psychosis. Schizophr Res 2007;95(1-3):124-133.
16. Brekke J, Kay DD, Lee KS, Green MF. Biosocial pathways to functional outcome in schizophrenia. Schizophr Res 2005;80(2-3):213-225.
17. Couture SM, Penn DL, Roberts DL. The functional significance of social cognition in schizophrenia: a review. Schizophr Bull 2006;32(Suppl 1):S44-63.
18. Corcoran R, Mercer G, Frith CD. Schizophrenia, symptomatology and social inference: investigating "theory of mind" in people with schizophrenia. Schizophr Res 1995;17(1):5-13.
19. Rubinsztein J, Michael A, Paykel ES, Sahakian J. Cognitive impairment in remission in bipolar affective disorder. Psychol Med 2000;30:1025-1036.
20. Cassidy C, Rabinovitch M, Joober R, Malla A. A comparison study of multiple measures of adherence to antipsychotic medication in first episode psychosis. Schizophr Res 2008;98(1):81.
21. Malla A, Norman R, McLean T, Scholten D, Townsend L. A Canadian programme for early intervention in non-affective psychotic disorders. Aust N Z J Psychiatry 2003;37(4):407-413.
22. O'Sullivan M, Guilford JP. Four factor tests of social intelligence (behavioral cognition): manual of instructions and interpretations. Orange (CA): Sheridan Psychological Services, Inc.; 1976.
23. Bodnar M, Malla A, Joober R, Lepage M. Cognitive markers of short-term clinical outcome in first-episode psychosis. Br J Psychiatry 2008;193(4):297-304.
24. First MB, Spitzer RL, Gibbon M, Williams JBW. Structured clinical interview for DSM-IV Axis I disorders, patient edition (SCID-I/P & SCID-I/NP), Version 2. New York: New York Psychiatric Institute, Biometrics Research; 1998.
25. Andreasen NC. Scale for the assessment of positive symptoms (SAPS). Iowa City: University of Iowa; 1984.
26. Andreasen NC. Modified scale for the assessment of negative symptoms (SANS). Iowa City: University of Iowa; 1984.
27. Malla A, Norman R, Schmitz N, Manchanda R, Bechard-Evans L, Takhar J, et al. Predictors of rate and time to remission in first-episode psychosis: a two-year outcome study. Psychol Med 2006;36(5):649-658.
28. Miller DC. Handbook for research design and social measurement. 5th ed. Newbury Park (CA): Sage Publications; 1991.
29. Visser BA, Ashton MC, Vernon PA. Beyond g: putting multiple intelligences theory to the test. Intelligence 2006;34(5):487-502.
30. SPSS. SPSS for Windows, release 12.0.1. Chicago, IL: SPSS; 2003.
31. Green MF, Olivier B, Crawley JN, Penn DL, Silverstein S. Social cognition in schizophrenia: recommendations from the measurement and treatment research to improve cognition in schizophrenia new approaches conference. Schizophr Bull 2005;31(4):882-887.
32. Ostrom TM. The sovereignty of social cognition. In: Srull TK, editor. Handbook of social cognition. L. Erlbaum Associates: Hillsdale (NJ); 1984. p. 1-37.
33. Premack D, Woodruff G. Chimpanzee problem-solving: a test for comprehension. Science 1978;202:532-535.
34. Brüne M. Emotion recognition, 'theory of mind,' and social behavior in schizophrenia. Psychiatry Res 2005;132(2):135-147.
35. Verdoux H, Liraud F, Assens F, Abalan F, van Os J. Social and clinical consequences of cognitive deficits in early psychosis: a two-year follow-up study of first-admitted patients. Schizophr Res 2002;56(1-2):149-159.
36. Schwartz JE, Fennig S, Tanenberg-Karant M, Carlson G, Craig T, Galambos N, et al. Congruence of diagnoses 2 years after a first-admission diagnosis of psychosis. Arch Gen Psychiatry 2000;57(6):593-600.
37. Gold JM. Cognitive deficits as treatment targets in schizophrenia. Schizophr Res 2004;72(1):21-28.