

Insight in Psychosis: Relationship With Neurocognition, Social Cognition and Clinical Symptoms Depends on Phase of Illness

Piotr J. Quee^{*1,2}, Lisette van der Meer^{2,3}, Richard Bruggeman¹, Lieuwe de Haan⁴, Lydia Krabbendam⁵, Wiepke Cahn⁶, Niels C.L. Mulder⁷, Durk Wiersma¹, and André Aleman^{2,3}

¹Department of Psychiatry & Rob Giel Research Center, University Medical Center Groningen, Groningen, The Netherlands; ²School of Behavioral and Cognitive Neuroscience, University of Groningen, Groningen, The Netherlands; ³Neuroimaging Center Groningen, University Medical Center Groningen, Groningen, The Netherlands; ⁴Department of Psychiatry, Academic Medical Center Amsterdam, Amsterdam, The Netherlands; ⁵Faculty of Psychology and Education, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands; ⁶Department of Psychiatry, University Medical Center Utrecht, Utrecht, The Netherlands; ⁷Erasmus Medical Center & Bavo Europoort Rotterdam, Rotterdam, The Netherlands

*To whom correspondence should be addressed; e-mail: p.j.quee@med.umcg.nl

Reduced insight has been reported in a majority of patients with a psychotic disorder. Most studies have focused on associations with neurocognition, neglecting relations with social cognition. Two hundred seventy patients with nonaffective psychosis participated in this study, which was part of the GROUP (Genetic Risk and Outcome of Psychosis)-project. Linear regression analyses were performed to investigate the predictive value of composite measures of neurocognition, social cognition, and clinical symptoms. The moderating effect of phase of illness was also investigated. Insight was measured with a composite measure, based on the insight item on the Positive And Negative Syndrome Scale (PANSS) and the Birchwood Insight Scale (BIS). Insight on the BIS and the PANSS correlated significantly ($r = .406$). All independent variables correlated with the insight composite measure. The additional effect of social cognition and clinical symptoms were both significant. Phase of illness was a moderating variable: In patients with recent-onset psychosis (ROP), none of the independent variables explained variance. In patients with multiple episode or chronic psychosis, both social cognition and clinical symptoms had additional effects and explained insight, along with neurocognition, together explaining 20% of the variance. These findings indicate that multiple factors are associated with insight in psychosis. Specifically, associations of insight with social cognitive and clinical symptom measures were observed, over and above a contribution of neurocognition. This supports theories that imply a role for deficient emotion recognition and mentalizing in reduced insight. Further studies need to investigate insight in ROP into more detail.

Key words: insight/awareness/schizophrenia/neuropsychology/GROUP

Introduction

Reduced insight (or unawareness of illness) has been reported in a majority of patients with a nonaffective psychotic disorder.¹ Insight can be studied as a set of descriptive beliefs and as a personal narrative.² Most studies investigating the neurocognitive correlates of insight treat the concept as a set of descriptive beliefs, mostly to formalize the concept of insight and thus enabling the subject for quantitative research. Even though studying insight as a personal narrative is of great importance to understand the individual differences with regard to insight, this approach is of a highly subjective nature, making it very hard to study the concept in a quantitative manner. In the current study, consistent with previous studies that investigated insight in psychosis and cognitive function, we focused on insight as a set of descriptive beliefs for which 3 distinct dimensions have been proposed: (1) the recognition that one has a mental illness, (2) the recognition of the need for treatment, and (3) the ability to relabel unusual mental events (delusions and hallucinations) as pathological.³ The concept of insight is clinically relevant because poor insight is associated with psychosocial dysfunction and poorer treatment adherence, in addition to an increase in the number of hospitalizations.⁴ Therefore, investigating which factors are specifically related to poor insight is of crucial importance for understanding psychotic disorders and for further development of treatment strategies.

Over the past decades, a considerable number of studies have investigated the association between neurocognition and insight. A meta-analysis of these studies found that, although there was a significant relationship, the predictive value of neurocognition was rather

modest.⁵ Furthermore, in schizophrenia, all neurocognitive domains (ie, reasoning and problem solving, verbal learning, and memory) were found to predict reduced insight to a similar degree. Thus, employing a composite measure of several neurocognitive domains⁶ may be adequate and may enhance reliability. It is also possible that other neuropsychological aspects are associated with insight. Indeed, in recent years, there is increasing interest in the concept of social cognitive impairments in psychosis. Social cognition has been referred to as “the ability to construct representations of the relations between oneself and others and to use those representations flexibly to guide social behavior.”⁷ Studies of social cognition in psychosis have mainly focused on emotion perception and theory-of-mind processing.⁸ The combination of impaired social cognition and poor insight has been reported in other populations with brain abnormalities.^{9,10} In addition, some studies have reported a relationship between social cognition and insight in psychosis as well,^{11,12} whereas others have not.¹³ When investigating factors associated with insight, it should be taken into account that social cognition and neurocognition are partially overlapping concepts.¹⁴ However, most of the studies to date have investigated whether social cognition or neurocognition is more related to insight, not whether 1 of these factors is of additional value in explaining insight.

A third group of factors that have been found to be related to insight are clinical symptoms.¹⁵ Relationships with positive symptoms, negative symptoms, and disorganization have been found.¹⁵ Some researchers claim that symptoms have more predictive value on insight as compared with neurocognition,^{6,16,17,18} whereas others suggest that these factors may not be mutually exclusive.¹⁹ As with social cognition, no studies have been done to determine whether clinical symptoms really have additional predictive value.

Insight is a complex and multi-dimensional concept and may be related to neuropsychological aspects as well as severity of psychopathology. Thereby, insight may be influenced by so-called “trait” and “state” features of psychosis, in which neurocognition has traditionally been associated with the former, and positive symptoms with the latter, whereas the influence of social cognition is not unequivocal.^{20–23} The phase of illness could be differentially related to various insight-related factors. Tranulis *et al.*²⁴ studied insight in patients in recent-onset psychosis (ROP) and found that self-report and interview-based insight scales were not correlated with one another in this population, which was attributed to the nature of their relatively unstable and evolving period.

The current study was part of the large-scale Genetic Risk and Outcome of Psychosis (GROUP) study. Data were obtained from a sample of relatively young patients, including those with ROP. A neuropsychological assess-

ment was administered in line with the cognitive dimensions used in Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS),²⁵ including social cognition. Composite measures were created for insight, neurocognition, social cognition, and clinical symptoms. The insight composite measure was based on an interview and a self-report questionnaire. It was expected that insight would be better explained by a model that included neurocognition as well as social cognition, as compared to a model that only encompassed neurocognition.^{19,27} Similarly, clinical symptoms, social cognition, and neurocognition were expected to predict insight better, particularly when as compared to a model that included social cognition and neurocognition only.

Methods

Participants

Two hundred seventy patients with psychotic disorders were included in this study. This was a subsample of the patient population participating in the GROUP project. Two out of 4 centers participated in the insight project (Amsterdam and Utrecht). The GROUP project is a large-scale multi-center study that investigates the vulnerability and protective factors for (1) the development of a psychotic disorder and (2) the variation in the course of illness. Diagnoses were confirmed using the Comprehensive Assessment of Symptoms and History.²⁷ The procedure of recruitment, criteria of inclusion and exclusion, informed consent, assessment instruments, approval by the accredited Medical Ethics Review Committee, and population characteristics have been described in a previous report on the GROUP study (N. Korver, P. J. Quee, H. B. M. Boos, C. J. P. Simons, GROUP, unpublished data, 2010).

Eligible patients had to fulfill the following criteria: (1) age: between 18 and 50 (extremes included), (2) meeting Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria for a nonaffective psychotic disorder (schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, psychotic disorder NOS), (3) fluent in Dutch, (4) able and willing to give written informed consent, and (5) the willingness of at least 1 family member to participate in the project.

Table 1 shows the demographic and clinical data for the patient group. Educational degree was adapted from Verhage.²⁹ Global Assessment of Functioning (GAF) scores, adapted from the DSM-IV, were obtained to measure global symptoms and disability.³⁰ Level of intelligence was estimated with the Wechsler Adult Intelligence Scale-III (WAIS-III) short form.^{29,32} ROP was defined as follows: 1 psychotic episode in the year prior to the assessment. The other patients had an illness

Table 1. Demographical and Clinical Data

Variable	Patients (<i>N</i> =270)	
	Mean	SD
Age (years)	27.7	6.5
Gender (<i>N</i>), male/female	222/48	
Educational degree (score), Verhage	4.2	2.1
Ethnicity (<i>N</i>), Dutch/other/unknown	209/52/9	
WAIS-III estimated IQ (short form) ²⁸	95.1	16.1
Duration of illness (years)	4.7	4.6
Psychotic episodes (number)	1.7	1.1
Age of onset psychosis (years)	22.5	6.2
Diagnostic (<i>N</i>)		
Schizophrenia, paranoid type	143	
Schizoaffective disorder	39	
Psychotic disorder NOS	25	
Schizophrenia, undifferentiated	16	
Schizophrenia, disorganized	13	
Other	21	
Phase of illness (<i>N</i>)		
Recent onset psychosis	57	
Multiepisode or chronic psychosis	210	
Unknown	3	
Hospitalizations (number)	2.2	2.1
Global assessment of functioning		
Symptoms	53.5	15.6
Disability	51.7	15.1
PANSS (score)		
Positive	1.9	0.8
Negative	2.2	0.9
General	1.8	0.5
Insight (score)		
Interview based (G12)	2.3	1.4
Self-report (IS)	8.8	2.8

duration of longer than 1 year, or had experienced multiple psychotic episodes and were therefore characterized as having “multiple episode or chronic psychosis” (MECP).

Assessment of Insight

Insight was assessed by means of a semi-structured interview, the Positive And Negative Syndrome Scale (PANSS),³² as well as a self-report scale, the Birchwood Insight Scale (BIS).²⁶ The PANSS provides a single item on insight (G12), based on the patient’s ability to describe and acknowledge symptoms and their psychiatric disorder, the ability to recognize the necessity of treatment, and the ability to describe future plans. Researchers were not always blind to the other outcome measures (eg, cognitive task performance): It has been suggested that this did not influence their knowledge of the patients’ cognitive functioning while judging insight. The BIS is a short questionnaire that consists of 8 questions addressing the 3 components of insight (Need for Treatment, Awareness of Illness, and Relabeling of Symptoms). Each of these com-

ponents is rated on a scale of 0–4: a higher score implies better insight. A composite measure was created, based on both the patients’ *z*-score on the BIS and PANSS, with the latter being negatively recoded. Higher scores on the composite measure indicated better insight. The procedure for translation of all raw scores into *z*-scores is described in the “Statistical Analysis” section.

Neurocognition

All patients were assessed with a neurocognitive task battery containing the following 6 tasks (intended neurocognitive domains of focus are placed between brackets): WAIS-III Digit Symbol Coding (processing speed), Continuous Performance Test-HQ (attention/vigilance),³¹ Word Learning Task (verbal learning and memory),³³ WAIS-III Arithmetic (working memory), WAIS-III Block Design (reasoning and problem solving), Response Set Shifting (set-shifting), WAIS-III Information (verbal comprehension),²⁸ and educational degree.²⁹ *z*-Scores were calculated for each domain, with *d*’ indicating a final score for attention/vigilance.³⁴ For verbal learning & memory, the final *z*-score was based on the mean value of 2 distinct *z*-scores: immediate recall and delayed recall. For set-shifting, the *z*-score was based on the decrement in accuracy performance from an imitation response condition to reversal response, using the Response Set Shifting task, a modified version of the Competing Programs Task.³⁵ Due to its high correlations with neurocognition, a *z*-score for educational degree was also created.³⁶ A final composite measure of neurocognition was based on the mean of the *z*-scores from the 7 outcome variables, while allowing for 4 missing values.

Social Cognition

In addition to the neurocognitive battery, patients were assessed with 2 social cognitive tasks, concerning emotion perception and theory of mind. The degraded facial affect recognition task was used as a measure of emotion perception.³⁷ Sixty-four trials were presented, consisting of 16 face presentations in each of 4 conditions: angry, happy, fearful, and neutral. Patients were asked to label each expression with the appropriate emotion. Theory of mind (or mentalizing) was assessed using the Hinting Task.³⁸ This task tests the ability of subjects to infer the real intentions behind indirect speech utterances. The task comprises 10 short passages presenting an interaction between 2 characters. All passages end with 1 of the characters dropping a hint. For instance, following a long and exhausting journey, Peter enters Ann’s office. Ann immediately starts to update him on a number of business developments. Peter interrupts Ann by saying “Gosh, that really was a long, exhausting journey.” The participant is asked to describe what he thinks Peter is implying with this comment. A composite measure for social cognition was based on the *z*-score on both tasks, allowing for 1 missing value.

Clinical Symptoms

In the GROUP project, current symptom severity was measured with the PANSS, which consists of 30 items. Each item is scored on a scale ranging from 1 (absent) to 7 (extreme), with item rating incorporating the behavioral effect of symptoms as well as their severity. Originally, 3 domains or factors were described for the PANSS. Later, a 5-factor structure was developed.³⁹ A more universally used method is that of remission, for which a number of items (measuring positive, negative, or disorganization symptoms) on the PANSS have been selected that also appear in other symptom rating scales. For remission, a score of 3 or lower for a period of 6 months on the following items is required (PANSS items are placed between brackets): delusions (P1), unusual thought content (G9), hallucinatory behavior (P3), conceptual disorganization (P2), mannerisms/posturing (G5), blunted affect (N1), social withdrawal (N4), and lack of spontaneity (N6).⁴⁰ Because the current study did not emphasize symptom dimensions but rather the concept of clinical symptoms in relation to insight, we decided to calculate the mean (cross-sectional) score of the z-transformed remission items, allowing for 3 missing values.

Statistical Analysis

Subject scores exceeding 2 SD from the mean patient group score were replaced by the recalculated mean group score ± 2 SD. All variables measuring insight (PANSS G12 and BIS), neurocognition, social cognition, and clinical symptoms (see “Assessment of Insight, Neurocognition, Social Cognition, and Clinical Symptoms” sections) were checked for normal distribution of residuals from regression analysis⁴¹ and transformed into z-scores using the mean and SD of the patient group. Otherwise, test scores were transformed to approximate normality by logarithmic, square root, or reciprocal transformations. Higher scores on the neurocognitive and social cognitive domains indicated better performance, whereas higher scores on the clinical symptom dimension indicated more severe symptomatology. Missing values were replaced by the average z-score, which is zero (BIS and PANSS G12: both 11 cases; neurocognition and social cognition: both 6 cases; and clinical symptoms: 11 cases).

First, bivariate correlation analyses were performed to separately investigate relations between insight and neurocognition, social cognition, and clinical symptoms. Second, we investigated the additional explained variance using multiple regression analysis. Neurocognition, social cognition, and clinical symptoms were entered blockwise. This enabled us to investigate the explained variance of each factor in 1 model, as well as the additional explained variance of social cognition as well as the additional explained variance of clinical symptoms.

Age, gender, and phase of illness (recent-onset/multiple episode or chronic psychosis) were entered as covariates into the first block.

Next, we investigated the moderating role of phase of illness. This was done by repeating the first regression analyses, but with the addition of 3 interaction terms: neurocognition \times recent onset yes/no, social cognition \times recent onset yes/no, and clinical variables \times recent onset yes/no.

Finally, 2 separate regression analyses were performed for ROP and MECP with only those predictors that showed a significant interaction with recent onset yes/no in the previous regression analysis. All analyses were performed with 1-tailed hypothesis testing with $\alpha = .05$. Statistical analyses were performed using SPSS 16.0. For descriptive purposes, correlations between all variables are displayed in supplementary table S1.

Results

Self-Reported Insight and Interview-Based Insight

Self-reported insight was significantly correlated with interview-based insight ($r = .406$, $P < 0.001$). Score on the PANSS and BIS correlated significantly with the composite measure of insight ($r = -.837$, $P < 0.001$; $r = .846$, $P < 0.001$, respectively), as with the BIS subscales Relabeling of Symptoms ($r = .615$, $P < 0.001$), Awareness of Illness ($r = .672$, $P < 0.001$), and Need for Treatment ($r = .682$, $P < 0.001$). On the raw PANSS G12 scores, 43.1% of the patients ($n = 132$) had no impairment of insight, 17.0% had “minimal” impairment, 15.7% had “mild impairment” ($n = 48$), 11.8% had “moderate impairment” ($n = 35$), 5.1% had “moderately severe impairment” ($n = 15$), 4.1% had “severe impairment” ($n = 12$), and 0.7% had “extreme impairment” ($n = 2$). On the raw BIS scores, 50.7% had a score in the “no-mild impairment” range (9.1–12; $n = 103$), 30.3% had a score in the “mild-moderate impairment” range (6.1–9; $n = 93$), 14.1% had a score in the “moderate-severe impairment” range (3.1–6; $n = 43$), and 4.9% had a score in the “severe-extreme impairment” range (0–3; $n = 15$).

Relationships With Insight

Bivariate correlation analyses revealed that all independent factors significantly correlated with insight (neurocognition: $r = .249$; $P < .001$; social cognition: $r = .248$, $P < .001$; and clinical symptoms: $r = -.290$, $P < .001$). In addition, neurocognition was significantly correlated with social cognition ($r = .454$, $P < .001$), and both neurocognition and social cognition were inversely correlated with clinical symptoms ($r = -.341$, $P < .001$; $r = -.228$, $P < .001$). Results of the multiple regression analyses are displayed in table 2. Neurocognition significantly predicted insight scores. Social cognition, as an additional predictor variable, significantly increased the explained variance ($R^2_{\text{change}} = .020$; $P_{\text{change}} = .018$). The contribution of both neurocognition and social cognition was

Table 2. Relationships With Insight for Patients Overall, Patients With ROP and Patients With Multiple Episode Psychosis (MECP)

Patient Group/Model	<i>df</i>	Insight Composite Measure									
		$\beta_{\text{Neurocognition}}$	$\beta_{\text{Social Cognition}}$	$\beta_{\text{Clinical Symptoms}}$	<i>P</i>	<i>F</i>	<i>R</i>	<i>R</i> ²	<i>P</i> _{change}	<i>F</i> _{change}	<i>R</i> ² _{change}
Overall											
Neurocognition	4,262	.250 ^b	—	—	<.001	5.540	.279	.078	—	—	—
Social cognition	5,261	.177 ^b	.159 ^a	—	<.001	5.642	.312	.098	.018	5.655	.020
Clinical symptoms	6,260	.108	.140 ^a	-.225 ^b	<.001	7.110	.375	.141	<.001	13.141	.043
ROP patients											
Neurocognition	3,53	.011	—	—	.942	.129	.085	.007	—	—	—
Social cognition	4,52	-.019	.051	—	.975	.118	.095	.009	.763	.092	.002
Clinical symptoms	5,51	-.038	.057	-.059	.986	.127	.111	.012	.684	.168	.003
MECP patients											
Neurocognition	3,206	.315 ^b	—	—	<.001	8.621	.334	.112	—	—	—
Social cognition	4,205	.229 ^b	.203 ^b	—	<.001	8.671	.380	.145	.005	7.950	.033
Clinical symptoms	5,204	.148 ^a	.169 ^a	-.258 ^b	<.001	10.216	.447	.200	<.001	14.165	.056

Note: β = standardized beta coefficient, *P*_{change}, *F*_{change}, and *R*_{change} refer to the statistical significance of the model as compared with its preceding model. Included covariates are gender, age, and phase of illness.

^aCorrelation significant at the 0.05 level.

^bCorrelation significant at the 0.01 level.

significant. Finally, clinical symptoms showed a significant additional increase in the explained variance. More specifically, clinical symptoms explained insight significantly, while the explained variance of neurocognition was nonsignificant.

The next step was to determine whether the phase of illness moderated the effect of the independent factors on insight. When scores of the groups ROP and MECP were compared, these did not differ with respect to insight (*P* = .228), neurocognition (*P* = .308), social cognition (*P* = .655) nor clinical symptoms (*P* = .105). However, when the interaction term neurocognition was added to a regression model containing neurocognition and the covariates (age, gender, and phase of illness), the interaction term was significant (β = .549, *P* = .037). The same moderating effect of the phase of illness was found in regression analyses that included social cognition (β = .483, *P* = .052) and clinical symptoms (β = -.582, *P* = .025). These results indicated that separate analyses for ROP and MECP groups were justified.

Therefore, final regression analyses were performed separately for ROP patients and MECP patients. In ROP patients, neither the neurocognition nor the additional effect of social cognition (*R*²_{change} < .002; *P*_{change} = .763) showed a significant effect. In addition, when clinical symptoms were also investigated, the explained variance was again nonsignificant. In MECP patients, it was demonstrated that neurocognition did significantly explain insight. When social cognition was added to the equation, the explained variance increased significantly (*R*²_{change} = .033; *P*_{change} = .005). Both neurocognition and social cognition significantly explained insight in MECP patients. With the addition of the variable clinical symptoms to

the above-mentioned equation, the explained variance increased once again significantly. Clinical symptoms explained insight significantly, as did neurocognition and social cognition. In total, the predictors explained insight for 20%.

Discussion

The current study investigated the relation of insight in psychosis with neurocognition, social cognition, and clinical symptoms. Results can be summarized as follows: When investigated separately, neurocognition, social cognition, and symptom dimensions were all associated with insight. Phase of illness was found to moderate the relation between insight and the studied predictors. In patients with MECP, both social cognition and clinical symptoms had additional effects and explained insight, along with neurocognition. In patients with ROP, none of the factors were found to be associated with insight.

To our best knowledge, this is the first study that has investigated the unique contribution of neurocognition, social cognition, and clinical symptoms in relation to insight in psychosis. To demonstrate this, not only the predictive value of each factor within 1 model was investigated but also the “additional” predictive value of the factor. In addition, most of the studies to date have focused on subfunctions of neurocognition (eg, working memory) or social cognition (eg, mentalizing) in relation to insight. The current study used composite measures for each factor. This implies that multiple tests have been used to measure the construct, which may represent a more reliable estimate. Furthermore, the analysis is more parsimonious because there is no large number of measures that compromises degrees of freedom.

Although this approach may lead to a certain loss of the fine-grained interpretation of cognitive subfunctions indexed by individual tests, the results show that each of the cognitive outcome variables is related to insight when analyses were restricted to the MECP population (see supplementary table S1). In fact, the association of neurocognition with insight within this group ($r = .312$, $P < .001$) is even higher as compared with the results of a meta-analysis of the previously published literature,⁵ in which mean weighted effect sizes were reported ranging from $r = .14$ to $.28$.

Social cognition, highly correlating with neurocognition, explained additional variance on insight. Social cognition has only been investigated in a few studies in relationship to awareness of illness in psychoses.^{11,12,43} Our results support previous findings of a significant association. For example, Lysaker *et al.*⁴² found that schizophrenia patients with “superficial” insight not only had poorer executive function but also poorer emotion recognition ability and capacity for social relationships than a patient group with “full awareness.” With regard to mentalizing, Langdon and Ward⁴³ reported an association between deficient Theory of Mind performance (measured with tasks of picture sequencing and joke appreciation) and poor insight. Our findings go beyond such previous studies because we show that the concept of social cognition explains “additional” variance when added to a regression model that already contains a wide range of neurocognitive variables. Similar results have been found in studies that investigated the predictive value of these concepts on distinct functional outcomes, such as social behavior,⁴⁴ vocational outcome,⁴⁵ and interpersonal skills.⁴⁶ The finding that social cognition uniquely contributes to insight is supported by theories that imply a role for deficient mentalizing in reduced insight in one’s illness.^{12,43,47} That there is a general difficulty in adopting other mental perspectives, ie, with “seeing the world as others do”, may contribute to deficient awareness of illness over and above general cognitive problems and next to clinical symptoms. A limitation of the current study was that a third described social cognitive domain, namely attributional style, was not included. In addition, it is possible that the additional explained variance of social cognition represents a general capacity to think about thinking, rather than a specific ability to infer other people’s mental state. This general capacity has been referred to as “metacognition,” which also includes the knowledge of one’s own mental state.^{48,49} Indeed, a study on metacognitive decisions in a test of mental flexibility (the Wisconsin Card Sorting Test) found this to be significantly correlated with insight in psychosis.⁵⁰

In ROP patients, neither neurocognition nor social cognition and clinical symptoms were significantly related to insight, although their mean scores were not dissimilar from those with MECP. This is in contrast with

a study by Keshavan *et al.*⁶, which did find a linear trend of a composite measure of neurocognition with PANSS G12 score in ROP patients. An explanation for this discrepancy may be that the correlation between the measures of insight was modest in the current population ($r = -.257$, $P = .032$). Tranulis *et al.*²⁴ have suggested that, in patients with ROP, specific factors may contribute to the measurement of insight. Particularly, recent-onset patients find themselves in a relatively unstable and evolving period. They may be aware of their distress but not yet attribute it to a mental disorder. In addition, different perspectives between the patient who has recently become ill and the interviewer may be responsible for discrepant findings. A recent study by Parellada *et al.*⁵¹ investigated insight in patients with ROP longitudinally using interview-based measures. The authors suggested that, during the acute phase, severity of clinical symptoms might overrule the relationship of “trait”-related features with insight, which in turn becomes more apparent after symptom stabilization. Viewing the supplementary table S1, it cannot be ruled out that clinical symptoms are related to insight in ROP. Thereby, the current study results are partly in agreement with those of Parellada *et al.* Future GROUP studies should be able to confirm this using longitudinal data of the ROP patients.

In line with previous findings, the current study showed medium relationships between the insight measures and a relation of its composite score with important outcome variables such as GAF-scores and number of hospitalizations.⁵² Scores on the PANSS and BIS reflected good insight in 43% of our sample. Although 1 influential study showed that poor insight affected up to 81% of patients with schizophrenia,⁵³ the numbers in the present study are consistent with comparable studies that were published over the last decades.^{27,54,55} Indeed, 1 of the largest studies to date that included a comprehensive assessment of 412 patients found that 41% of schizophrenia patients were aware that they suffered from a mental disorder.⁵⁶ Treatment factors, such as the widespread use of psychoeducation in the Netherlands, might contribute to the relatively high number of unimpaired patients in our sample. Alternatively, our brief measures of insight might have overseen some insight problems. The use of more comprehensive interview-based measures such as the Schedule of Assessment of Insight-Expanded version⁵⁷ and the Scale to Assess Unawareness of Mental Disorder⁵⁸ could yield a higher sensitivity in that regard. Utilization of such scales may also allow for a deeper and more comprehensive exploration of the several domains of insight described in the “Introduction” section. In addition, it has recently been suggested that insight should be studied not only as a belief that leads to treatment adherence, or the possession of specific knowledge, but also as a facet of a larger understanding of an individual life, also referred to as an inextricable part of a personal narrative.^{42,59}

Another drawback of the current study was the use of the PANSS for both the independent measure (clinical symptoms) and the dependent measure (insight, in part). This may well explain the decrease in explained variance of neurocognition and social cognition when the contribution clinical symptoms were taken into account as well. Finally, correlation does not imply causation. Whereas neurocognitive and social cognitive abilities may partly underlie the patients' level of insight, the direction of the effect is less straightforward for clinical symptoms, such as delusions and hallucinations. For instance, a deluded person without insight in his/her psychotic beliefs may receive higher positive symptom ratings as compared with a similar person with insight. Thus, it may not be that stronger symptoms cause poor insight but that poor insight results in higher symptom ratings.

In conclusion, the results of the current study indicate that insight in psychosis is associated with multiple factors and that a distinction between patients in different phases of their illness may lead to a better understanding of this concept. Lack of insight in MECP patients may require not only traditional cognitive aspects (eg, learning, attending, information processing, remembering) but also successful perception and interpretation of social-emotional information. In this, the former may be necessary to understand the world around us as an individual, whereas the latter may be more important to understand others and oneself; it should be noted that the border between these concepts is, in reality, more diffuse. In addition to neurocognition and social cognition, a certain presence of behavior and emotion and absence of deviant perceptions (clinical symptoms) may be requirements for full insight. It should be noted that the nature of insight is paradoxical. Patients with full insight have been found to experience more depressive symptoms and hopelessness,⁶⁰ and the stigma associated with having a schizophrenia diagnosis has been suggested to be a moderating factor.⁶¹ However, increasing insight through treatment does not lead to lasting increases in depression. Indeed, improved insight has been shown to be associated with decreased suicidality.⁶² Indeed, interventions with positive effects on neuropsychological aspects, clinical symptoms as well as well-being and functional outcome⁶³ may hypothetically increase insight as a by-product while preventing the occurrence of negative effects on depressive symptoms.⁶⁰ Such interventions should also address stigma sensitivity and may benefit from using a broader concept of insight, which incorporates the personal narrative. How to increase insight in ROP needs further investigation.

Supplementary Material

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

Funding

The GROUP study was supported by the Geestkracht programme of the Dutch Health Research Council (ZON-MW, grant number 10-000-1002).

Acknowledgments

The authors would like to thank René Kahn, Don Linszen, Jim van Os, Inez Germeys, Carin Meijer, Philippe Delespaul, Peter de Jonge, Dick Smid, Nikie Korver, Agna Bartels, Heleen Boos, Joyce van Baaren, Leo Swart, Erwin Veermans, Truda Driesen, all other involved staff members participating in the GROUP project, the reviewers for their comments and suggestions, Mary Wheeler for correcting the final version of the manuscript, and all participants for their time and co-operation.

The Authors have declared that there are no conflicts of interest in relation to the subject of this study.

References

1. Amador XF, Gorman JM. Psychopathologic domains and insight in schizophrenia. *Psychiatr Clin North Am.* 1998;21:27–42.
2. Roe D, Kravetz S. Different ways of being aware of psychiatric disability: a multifunctional narrative approach to insight into mental disorder. *J Nerv Ment Dis.* 2003;191:417–424.
3. David AS. Insight and psychosis. *Br J Psychiatry.* 1990;156:798–808.
4. Amador XF, David AS. *Insight and Psychosis: Awareness of Illness in Schizophrenia and Related Disorders.* 2nd ed. New York, NY: Oxford University Press; 2004.
5. Aleman A, Agrawal N, Morgan KD, David AS. Insight in psychosis and neuropsychological function: meta-analysis. *Br J Psychiatry.* 2006;189:204–212.
6. Keshavan MS, Rabinowitz J, DeSmedt G, Harvey PD, Schooler N. Correlates of insight in first episode psychosis. *Schizophr Res.* 2004;70:187–194.
7. Adolphs R. The neurobiology of social cognition. *Curr Opin Neurobiol.* 2001;11:231–239.
8. Pinkham AE, Penn DL, Perkins DO, Lieberman J. Implications for the neural basis of social cognition for the study of schizophrenia. *Am J Psychiatry.* 2003;160:815–824.
9. Kipps CM, Hodges JR. Theory of mind in frontotemporal dementia. *Soc Neurosci.* 2006;1:235–244.
10. Bach LJ, David AS. Self-awareness after acquired and traumatic brain injury. *Neuropsychol Rehabil.* 2006;16:397–414.
11. Bora E, Sehitoglu G, Aslier M, Atabay I, Veznedaroglu B. Theory of mind and unawareness of illness in schizophrenia: is poor insight a mentalizing deficit? *Eur Arch Psychiatry Clin Neurosci.* 2007;257:104–111.
12. Lysaker PH, Carcione A, Dimaggio G, et al. Metacognition amidst narratives of self and illness in schizophrenia: associations with neurocognition, symptoms, insight and quality of life. *Acta Psychiatr Scand.* 2005;112:64–71.
13. Drake RJ, Lewis SW. Insight and neurocognition in schizophrenia. *Schizophr Res.* 2003;62:165–173.

14. Sergi MJ, Rassovsky Y, Widmark C, et al. Social cognition in schizophrenia: relationships with neurocognition and negative symptoms. *Schizophr Res.* 2007;90:316–324.
15. Mintz AR, Dobson KS, Romney DM. Insight in schizophrenia: a meta-analysis. *Schizophr Res.* 2003;61:75–88.
16. Buchy L, Malla A, Joober R, Lepage M. Delusions are associated with low self-reflectiveness in first-episode psychosis. *Schizophr Res.* 2009;112:187–191.
17. Simon V, De Hert M, Wampers M, Peuskens J, van Winkel R. The relation between neurocognitive dysfunction and impaired insight in patients with schizophrenia. *Eur Psychiatry.* 2009;24:239–243.
18. De Hert MAF, Simon V, Vidovic D, et al. Evaluation of the association between insight and symptoms in a large sample of patients with schizophrenia. *Eur Psychiatry.* 2009;24:507–512.
19. Osatuke K, Ciesla J, Kasckow JW, Zisook S, Mohamed S. Insight in schizophrenia: a review of etiological models and supporting research. *Compr Psychiatry.* 2008;49:70–77.
20. de Gracia Dominguez M, Viechtbauer W, Simons CJP, van Os J, Krabbendam L. Are psychotic psychopathology and neurocognition orthogonal? A systematic review of their associations. *Psychol Bull.* 2009;135:157–171.
21. Pousa E, Ruiz AI, David AS. Mentalising impairment as a trait marker of schizophrenia? *Br J Psychiatry.* 2008;192.
22. Sprong M, Schothorst P, Vos E, Hox J, van Engeland H. Theory of mind in schizophrenia. *Br J Psychiatry.* 2007;191:5–13.
23. Koelkebeck K, Pedersen A, Suslow T, Kueppers KA, Arolt V, Ohrmann P. Theory of mind in first-episode schizophrenia patients: correlations with cognition and personality traits. *Schizophr Res.* 2010;119:115–123.
24. Tranulis C, Lepage M, Malla A. Insight in first episode psychosis: who is measuring what? *Early Interv Psychiatry.* 2008;2:34–41.
25. Nuechterlein KH, Barch DM, Gold JM, Goldberg TE, Green MF, Heaton RK. Identification of separable cognitive factors in schizophrenia. *Schizophr Res.* 2004;72:29–39.
26. Birchwood M, Smith J, Drury V, Healy J. A self-report insight scale for psychosis: reliability, validity and sensitivity to change. *Acta Psychiatr Scand.* 1994;89:62–67.
27. Andreasen NC, Flaum MC, Arndt S. The Comprehensive Assessment of Symptoms and History (CASH): an instrument for assessing diagnosis and psychopathology. *Arch Gen Psychiatry.* 1992;49:615–623.
28. Blyler CR, Gold JM, Iannone VN, Buchanan RW. Short form of the WAIS-III for use with patients with schizophrenia. *Schizophr Res.* 2000;46:209–215.
29. Verhage F. *Intelligentie en leeftijd: onderzoek bij Nederlanders van twaalf tot zeventenzeventig jaar.* Assen, The Netherlands: Van Gorcum; 1964.
30. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders 4th ed.* Washington, DC: American Psychiatric Publishing, Inc.; 1994.
31. Stinissen J, Willems PJ, Coetsier O, Hulsman WLL. *Manual of the Dutch Edition of the WAIS.* Lisse, The Netherlands: Swets & Zeitlinger; 1970.
32. Kay SR, Fliszbein A, Opfer LA. The Positive and Negative Syndrome Scale (PANSS) for Schizophrenia. *Schizophr Bull.* 1987;13:261–276.
33. Brand N, Jolles J. Learning and retrieval rate of words presented auditorily and visually. *J Gen Psychol.* 1985;112:201–210.
34. Davies DR, Parasuraman R. *The Psychology of Vigilance.* London, UK: Academic Press; 1982.
35. Bilder RM, Turkel E, Lipschutz-Broch L, Lieberman JA. Antipsychotic medication effects on neuropsychological functions. *Psychopharmacol Bull.* 1992;28:353–366.
36. Le Carret N, Lafont S, Mayo W, Fabrigoule C. The effect of education on cognitive performances and its implication for the constitution of the cognitive reserve. *Dev Neuropsychol.* 2003;23:317–337.
37. van't Wout M, Aleman A, Kessels RPC, Larøi F, Kahn RS. Emotional processing in a non-clinical psychosis-prone sample. *Schizophr Res.* 2004;68:271–281.
38. Corcoran R, Mercer G, Frith CD. Schizophrenia, symptomatology and social influence: investigating 'theory of mind' in people with schizophrenia. *Schizophr Res.* 1995;17:5–13.
39. Lançon C, Auquier P, Nayt G, Reine G. Stability of the five-factor structure of the Positive and Negative Syndrome Scale (PANSS). *Schizophr Res.* 2000;42:231–239.
40. Andreasen NC, Carpenter WT, Jr, Kane JM, Lasser RA, Marder SR, Weinberger DR. Remission in Schizophrenia: proposed criteria and rationale for consensus. *Am J Psychiatry.* 2005;162:441–449.
41. Field A. *Discovering Statistics Using SPSS.* 2nd ed. Thousand Oaks, CA: Sage Publications, Inc; 2005.
42. Lysaker PH, Tsai J, Maulucci AM, Stanghellini G. Narrative accounts of illness in schizophrenia: association of different forms of awareness with neurocognition and social function over time. *Conscious Cogn.* 2008;17:1143–1151.
43. Langdon R, Ward P. Taking the perspective of the other contributes to awareness of illness in schizophrenia. *Schizophr Bull.* 2009;35:1003–1011.
44. Brüne M. Emotion recognition, 'theory of mind,' and social behavior in schizophrenia. *Psychiatry Res.* 2005;133:135–147.
45. Bell M, Tsang HWH, Greig TC, Bryson GJ. Neurocognition, social cognition, perceived social discomfort, and vocational outcomes in schizophrenia. *Schizophr Bull.* 2009;35:738–747.
46. Pinkham AE, Penn DL. Neurocognitive and social cognitive predictors of interpersonal skill in schizophrenia. *Psychiatry Res.* 2006;143:167–178.
47. David AS. 'To see ourselves as others see us': Aubrey Lewis's insight. *Br J Psychiatry.* 1999;175:210–216.
48. Semerari A, Carcione A, Dimaggio G, et al. How to evaluate metacognitive functioning in psychotherapy? The Metacognition Assessment Scale and its applications. *Clin Psychol Psychother.* 2003;10:238–261.
49. Lysaker PH, Dimaggio G, Buck KD, et al. Poor insight in schizophrenia: links between different forms of metacognition with awareness of symptoms, treatment need, and consequences of illness [published online September 2, 2010]. *Compr Psychiatry.* doi:10.1093/molbev/msg013.
50. Koren D, Seidman LJ, Poyurovsky M, et al. The neuropsychological basis of insight in first-episode schizophrenia: a pilot metacognitive study. *Schizophr Res.* 2004;70:195–202.
51. Parellada M, Fraguas D, Bombin I, et al. Insight correlates in child- and adolescent-onset first episodes of psychosis: results from the CAFEPS study. *Psychol Med.* 2009;39:1433–1445.
52. Lincoln TM, Lüllmann E, Rief W. Correlates and long-term consequences of poor insight in patients with schizophrenia. A systematic review. *Schizophr Bull.* 2007;33:1324–1342.
53. Wilson WH, Ban TA, Guy W. Flexible system criteria in chronic schizophrenia. *Compr Psychiatry.* 1986;27:259–265.

54. Karow A, Pajonk FG, Reimer J, et al. The dilemma of insight into illness in schizophrenia: self- and expert-rated insight and quality of life. *Eur Arch Psychiatry Clin Neurosci.* 2008;258:152–159.
55. Jensen J, Nilsson L-L, Levander S. Neurocognitive and psychopathological correlates of self-monitoring ability in schizophrenia. *Eur Arch Psychiatry Clin Neurosci.* 2004;254:312–317.
56. Amador XF, Flaum M, Andreasen NC, Strauss DH. Awareness of illness in schizophrenia and schizoaffective and mood disorders. *Arch Gen Psychiatry.* 1994;51:826–836.
57. Kemp R, David A. Psychological predictors of insight and compliance in psychotic patients. *Br J Psychiatry.* 1996;169:444–450.
58. Amador XF, Strauss DH, Yale SA, Flaum MM. Assessment of insight in psychosis. *Am J Psychiatry.* 1993;150:873–879.
59. Lysaker PH, Clements CA, Plascak-Hallberg CD, Knipscheer SJ, Wright DE. Insight and personal narratives of illness in schizophrenia. *Psychiatry: Interpers Biol Process.* 2002;65:197–206.
60. Buchy L, Bodnar M, Malla A, Joobar R, Lepage MA. 12-month outcome study of insight and symptom change in first-episode psychosis. *Early Interv Psychiatry.* 2010;4:79–88.
61. Staring ABP, Van der Gaag M, Van den Berge M, Duivenvoorden HJ, Mulder CL. Stigma moderates the associations of insight with depressed mood, low self-esteem, and low quality of life in patients with schizophrenia spectrum disorders. *Schizophr Res.* 2009;115:363–369.
62. Bourgeois M, Swendsen J, Young F, et al. Awareness of disorder and suicide risk in the treatment of schizophrenia: results of the international suicide prevention trial. *Am J Psychiatry.* 2004;161:1494–1496.
63. Roder V, Muelle DR, Mueser KT, Brenner HD. Integrated Psychological Therapy (IPT) for Schizophrenia: is it effective? *Schizophr Bull.* 2006;32(Suppl 1):S81–S93.