COGNITIVE CAPACITY SIMILARLY PREDICTS INSIGHT INTO SYMPTOMS IN FIRST- AND MULTIPLE-EPISODE PSYCHOSIS

Running title: Insight across stages of psychosis

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Postprint - Sauvé, G., Kline, R.B., Shah, J.L., Joober, R., Malla, A. et Lepage, M. (2019). Cognitive capacity similarly predicts insight into symptoms in first- and multiple-episode psychosis. *Schizophrenia Research*, 206, 236-243. doi: 10.1016/j.schres.2018.11.013

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ABSTRACT

Background: Lack of insight is a frequent characteristic of psychotic disorders, both in patients who recently experienced a first episode of psychosis (FEP) and those who experience recurrent multiple episodes (MEP). Insight is a multifaceted construct: its clinical form notably includes the unawareness of being ill, of symptoms, and of the need for treatment. Cognitive capacity is among the key determinants of insight into symptoms, but less is known about whether stage of illness (FEP vs. MEP) moderates this association. *Methods*: Our aim is to evaluate the association between cognitive capacity and symptom unawareness using structural equation modeling and moderated multiple regression. A total of 193 FEP and MEP patients were assessed using the CogState battery and the Scale to Assess Unawareness of Mental Disorder. *Results*: Analyses suggest that cognitive capacity accounts for a relatively small proportion of the total variation in symptom unawareness (6.4%). There was no evidence to suggest a moderating effect of stage of illness on this association. *Conclusions*: The effect of general cognitive capacity on symptom unawareness is relatively small, and this basic relation was unrelated to stage of illness. It is possible that stage of illness could moderate this association only for certain facets of insight not assessed in this study (e.g., unawareness of the need for treatment).

KEYWORDS: schizophrenia; awareness; cognition; first-episode; multi-episode chronic; enduring

1. INTRODUCTION

Experiencing a first episode of psychosis evolves into a recurrent disabling condition for some patients (Keefe et al., 2016; McGorry et al., 2010). Positive and negative symptoms, cognitive impairment, and unawareness of being ill are intertwined characteristics of psychotic disorders, including schizophrenia (Owen et al., 2017; Vohs et al., 2016). There is a relatively large literature on the *insight deficit*, or the unawareness of various facets of one's illness, a condition observed in about 50–80% of psychosis patients (Lincoln et al., 2007; Thompson et al., 2001). Yet, our understanding of its determinants remains limited (Poyraz et al., 2016).

Some distinctions in terms are warranted. *Cognitive insight* is distinguished from *clinical insight* (Van Camp et al., 2017). The former concerns the evaluation of incorrect beliefs or interpretations (Beck et al., 2004, p.321). Aspects include (1) self-reflectiveness, or the ability to consider alternative explanations; and (2) self-certainty, or overconfidence in one's judgment. The latter (clinical insight) is the failure to acknowledge illness signs (Amador and Kronengold, 2004; Vohs et al., 2016). Facets of clinical insight include the unawareness of being ill, of symptoms (positive or negative), of the need for treatment, and of the social consequences of illness (Bouroubi et al., 2016). The terms "insight," "unawareness," and "poor awareness" are generally used interchangeably in the literature (e.g., Gilleen et al., 2016; Pousa et al., 2017). An exception is "insight into symptoms", which includes both the unawareness of and the misattribution of symptoms to something other than the psychotic disorder (Amador et al., 1993). It has been challenging to operationalize clinical insight due to the absence of a clear consensus definition or use of measures comprised of a single item (Amador and Kronengold, 2004; Lincoln et al., 2007).

While various predictors of clinical insight, such as metacognition deficits (Vohs et al., 2016), have been studied, each appears to account for only a relatively small, albeit statistically significant, portion of the variation in clinical insight. For instance, results of a meta-analysis by Mintz et al. (2003) indicate that symptom severity explains about 7% of the total variation in clinical insight. Nonetheless, the largest source of variance in clinical insight is still generally unknown and most likely consists of the cumulative contribution of a number of variables (Beland and Lepage, 2017; Ritsner and Blumenkrantz, 2007).

Another possible factor is cognitive capacity. Results of some recent meta-analytic studies indicate relatively small, but statistically significant, relations between cognitive capacity and the general construct of clinical insight (e.g., about 3% explained variation; Aleman et al., 2006; Nair et al., 2014). Results of some primary studies also indicate relatively low associations between these variables (Quee et al., 2011; Wiffen et al., 2012), yet several authors have reported perhaps more robust relations with prefrontally-mediated cognitive functions (e.g., conceptual flexibility, abstract thinking) (e.g., Mingrone et al., 2013; Simon et al., 2009). Morgan and David (2004) suggested that evaluation of more specific facets of clinical insight, such as insight into symptoms, instead of global constructs (e.g., clinical insight per se) could be beneficial. Certain results are consistent with this view (e.g., Cuesta et al., 2006; Gilleen et al., 2011; Mohamed et al., 1999). For example, Wiffen et al. (2012) found that verbal memory was the best predictor of insight into symptoms.

Stage of illness, as described in the clinical staging model of McGorry et al. (2010), has been studied as a possible moderator of the relation between cognitive capacity and insight into symptoms. Specifically, the experience of psychotic symptoms may change the association between these two variables compared with the experience of more enduring or chronic episodes

(Gerretsen et al., 2014). For example, cognitive level among some patients deteriorates as the illness progresses, and this change could alter the role that cognitive capacity plays in insight into symptoms (Oie et al., 2010; Zhang et al., 2015).

Recent results by Quee et al. (2011) are consistent with the hypothesis just mentioned: in this study, cognitive capacity, social reasoning, and symptom severity explained about 20% of the variation in clinical insight among MEP patients but failed to appreciably predict the same criterion among FEP patients. Other results are mixed. For example, verbal memory, executive functions, and working memory were found to be associated with insight into symptoms among FEP samples (Drake and Lewis, 2003; Morgan et al., 2010; Mutsatsa et al., 2006; Subotnik et al., 2005; Wiffen et al., 2012), but only executive functions were reported to have similar predictive validity in MEP samples (Monteiro et al., 2008; Nakano et al., 2004; Smith et al., 2000; Young et al., 1998). And some authors have reported a lack of association between cognitive capacity and insight into symptoms (Freudenreich et al., 2004; McCabe et al., 2002). A limitation of all the works just cited is the failure to take explicit account of measurement error in indicators of cognitive capacity and insight into symptoms. In regression analysis, the failure to explicitly control for measurement error can seriously bias the results (e.g., Cole and Preacher, 2014; Westfall and Yarkoni, 2016). Other limitations of previous studies that could partly explain these mixed findings include small sample sizes and the use of different sets of instruments to assess clinical insight.

In the present study, the technique of structural equation modeling (SEM) was applied in order to estimate the association between cognitive capacity and insight into symptoms while controlling for measurement error and addressing some of the limitations of previous studies. This means that both cognitive capacity and insight into symptoms were analyzed as latent

variables, not as manifest variables subject to measurement error (Kline, 2016; Nachtigall et al., 2003). Error terms are therefore estimated based on the collected data and included in the model. Next, factor scores for cognitive capacity were derived for patients with a psychotic disorder classified as either FEP or MEP. We hypothesized that stage of illness would appreciably moderate the association between cognitive capacity and insight into symptoms, and this prediction was evaluated using moderated multiple regression. The magnitudes of effects in both analyses were of key interest.

2. MATERIALS AND METHODS

2.1 Participants

The total sample consisted of 193 non-affective psychotic disorder patients. A total of 61 were classified as FEP. These patients attended the Prevention and Early Intervention Program for Psychosis (PEPP) clinic at the Douglas Mental Health University Institute in Montréal, Canada (Iyer et al., 2015). Consecutively-admitted patients were invited to participate in a longitudinal study about cognitive and clinical outcomes in FEP. All participants signed a consent form approved by the institutional ethics committee. Briefly, FEP patients were 17–35 years old and generally had not taken antipsychotic medication for more than one month prior to their entry into the clinic. Diagnoses of schizophrenia and schizoaffective disorder established with the Structural Clinical Interview for DSM-IV (First et al., 1998) were verified through consensus between two senior psychiatrists (R.J. & A.M.). Exclusion criteria were (1) diagnosis of affective psychosis (i.e., bipolar disorder, major depressive disorder with psychotic features), (2) IQ score below 70, and (3) incomplete cognitive testing or insight assessment results.

A total of 131 MEP patients were also included in the sample. Their age range was 18–50 years, and all had received psychiatric treatment for ≥ 4 years as inpatients or outpatients. We

considered the latter as an indication that they had experienced more than one psychotic episode. They were recruited as part of a larger cross-sectional study on psychological and neuronal determinants of insight in schizophrenia (Emami et al., 2016). Each patient signed a consent form approved by local ethics committees. This patient group could also be designated as "prolonged treatment" among other terms, but we chose the term "MEP" to avoid stigmatizing terminology (Lesage and Morissette, 2002), and to underscore the parallel between the concept of FEP and the detrimental effects of illness chronicity. The term MEP is also used in the latest version of the DSM (American Psychiatric Association, 2013) and provides a more hopeful perspective of the illness by adopting a recovery philosophical standpoint.

Summarized in the top part of **Table 1** are characteristics of the FEP and MEP patients. Also reported in the table are values of effect sizes for differences between the two groups, standardized mean differences (i.e., d) or the phi coefficient (i.e., ϕ). Table 1 also includes descriptive statistics by stage of illness for measures of positive symptoms (Scale for the Assessment of Positive Symptoms; Andreasen, 1984), negative symptoms (Scale for the Assessment of Negative Symptoms; Andreasen, 1983), depression (Calgary Depression Scale for Schizophrenia; Addington et al., 1990), and anxiety (Hamilton Anxiety Rating Scale; Hamilton, 1959). A more detailed description of the sample is provided in Supplementary Methods.

2.2 Insight into symptoms

Insight into symptoms was assessed using an abbreviated (11-item) version of the Scale to Assess Unawareness of Mental Disorder (SUMD, V.2/14/99; Amador et al., 1993; Dumas et al., 2013). Scores on short versions of the SUMD are reasonably reliable (e.g., Amador et al., 1994; Michel et al., 2013; Raffard et al., 2010), and evidence for convergent and discriminant validity is generally positive (e.g., Dumas et al., 2013). Patient unawareness and misattributions

of four symptoms—hallucinations, delusions, flat affect, and asociality—were rated on a 6-point Likert scale. For each item, a score of "0" indicated the absence of the corresponding symptom (i.e., awareness could not be rated); these responses were treated as missing data. A score of "1" means that the patient is aware of the symptom, a score of "3" means the patient is somewhat aware, and a score of "5" means that the patient is unaware of the symptom. Items scores of "2" and "4" refer to intermediate levels of awareness. Thus, higher scores on SUMD items indicate greater unawareness (i.e., less awareness).

Symptom attribution items on the SUMD are administered only if the score on the corresponding symptom awareness item were "3" or lower (i.e., the patient has at least some level of awareness), which results in a higher rate of missing data for attribution items. Consequently, only total scores over the symptom unawareness items were analyzed; specifically, for each patient, awareness scores for the aforementioned symptom items (2 positive, 2 negative symptoms) were averaged. Values of descriptive statistics for symptom unawareness total scores are reported in Table 1 for the FEP and MEP patients. The group means differ by less than 10% of a standard deviation with FEP patients showing marginally more symptom unawareness than MEP patients.

2.3 Cognitive Capacity

When stable enough to meaningfully assess their cognitive capacity, patients were administered the CogState Schizophrenia Battery (Pietrzak et al., 2009), a computerized test that measures the seven domains within the scope of the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS; Horan et al., 2011). These domains include processing speed, attention/vigilance, working memory, verbal learning and memory, visual learning and memory, reasoning and problem solving, and social cognition. Correlations between

198 composite scores from the CogState and MATRICS batteries among schizophrenia patients
199 generally range from .70–.80 (Pietrzak et al., 2009).
200 The 12 CogState tasks described by Benoit et al. (2015) were administered to all patients.
201 Because the estimation method for the SEM analyses described later assumes multivariate
202 normality, normalizing transformations were applied to the scores of eight CogState tasks—see
203 Table 1 and Supplementary Methods for more details. Next, scores on three tasks, Groton Maze
204 Learning, Groton Maze Learning Delayed Recall, and Detection, were reflected so that higher
205 scores indicate better performance, just as for all other CogState tasks. Reported at the bottom of
206 Table 1 are descriptive statistics by group.
207
208 2.4 Statistical Analyses
209 The techniques of SEM and moderated multiple regression require large samples for
210 statistical power to be reasonably high (Aguinis et al., 2011; Wolf et al., 2013), but the sample
211 size in the present study is not large. A problem when studying disorders with relatively low base
212 rates, such as schizophrenia, is that it may be practically impossible in a primary study to collect
213 samples large enough for adequate statistical power. There is a similar challenge when studying
214 effects of smaller, but still meaningful, magnitude (Gagne et al., 2014).
215 In the present study, we report the outcomes of significance testing in tables, but dealt
216 with the consequences of low power by de-emphasizing the role of *p*-values in the analysis.
217 Instead, we relied on best practice recommendations for conducting SEM or moderated multiple
218 regression (e.g., Aguinis et al., 2011; Kline, 2016, chap. 18) including the estimation of effect
219 sizes. The latter is consistent with the conclusion of the International Committee of Medical
220 Journal Editors (2016, p.15) that *p*-values do not directly reflect effect size. We are also mindful

that significance testing has been banned in some journals (Trafimow and Marks, 2015).

The method of SEM was applied over two steps. The question evaluated in the first step with confirmatory factor analysis (CFA) is whether the CogState tasks listed in Table 1 measure a common cognitive capacity factor. Details about modifications to the initial measurement model are provided in the Supplementary Methods.

Analyzed in the second step was the structural regression model (SR) presented in **Figure 1**. The cognitive factor with its CogState task indicators are from the final CFA measurement model in the first analysis step. The other factor in the figure is symptom unawareness, which has a single indicator, the average score on the SUMD awareness items. A method for analyzing a single indicator with an error term that represents the reliability of its scores was used (Kline, 2016, pp. 214-217). This method does not affect model fit, but measurement error in the single indicator is controlled. Of key interest in the analysis of the model in Figure 1 was the magnitude of the coefficient for regressing symptom unawareness on cognitive capacity.

Model fit was evaluated using best practice recommendations for SEM in Kline (2016, chap. 12) and Schumacker and Lomax (2016, chap.16). This means that the outcome of the chi-square test was taken seriously; the use of now-discredited thresholds, or cutting points, for values of certain global fit statistics that purportedly indicate “good” model fit, such as CFI > .95, was avoided (see also Hayduk et al., 2007); and residuals were inspected before making any decision about whether to retain a model.

The final analyses concerned whether stage of illness moderates the association between cognitive capacity and symptom unawareness. Although this question could be addressed in a multiple-groups SEM analysis where the relation between the cognitive capacity and symptom unawareness is estimated separately for FEP versus MEP patients, the group sizes in this sample

244 are too small (Meade and Bauer, 2007). An alternative is moderated multiple regression
245 conducted with manifest variables only, which may require smaller sample sizes compared with
246 latent variable analysis (i.e., SEM).

247 After the SEM analyses, we calculated a composite score for each case based on values
248 of the unstandardized pattern coefficients for CogState indicators of the cognitive capacity factor
249 in the SR model of Figure 1. These composites are factor scores. Next, the total score on the
250 SUMD measure of symptom unawareness was regressed on three predictors, the cognitive
251 composite, group membership (i.e., FEP vs. MEP), and the product of the two variables just
252 mentioned. The product term represents the interactive effect of stage of illness and cognitive
253 capacity in predicting the degree of symptom unawareness. Additional detail on this moderated
254 multiple regression analysis is provided in Supplementary Methods.

255 **3. RESULTS**

256 **Table 1** presents the sociodemographic, clinical and cognitive characteristics of the
257 sample. Age ($r = -.07$, $p = .36$) and duration of illness ($r = .06$, $p = .38$) did not significantly
258 correlate with insight into symptoms and were not controlled for in our analyses. All patients
259 were taking antipsychotic medication at the time of their assessments. Medication was also not
260 controlled for in our analyses because it did not significantly correlate with symptom
261 unawareness in the whole sample ($r = -.09$, $p = .24$) and in each group separately (FEP: $r = .02$,
262 $p = .91$; MEP: $r = -.14$, $p = .12$).

263 Correlations between the 12 CogState tasks and symptoms unawareness scores for FEP
264 and MEP patients separately revealed that only the GML and ONB tasks were significantly
265 associated (Bonferroni correction $n = 12$) with insight into symptoms for each group
266 respectively. The values of all correlations for FEP and MEP separately are provided in

Supplementary Material. The effect sizes of the association between cognitive composite scores and unawareness of symptoms were similar for FEP ($r = -.22$, $p = .08$) and MEP patients ($r = -.19$, $p < .05$).

Reported in Table 2 are the descriptive statistics for the measure of symptom unawareness and the 12 CogState tasks calculated for the total sample ($N = 193$). These data in summary form were analyzed in the lavaan package for SEM (Roseel, 2012) in R (R Core Team, 2016). The estimation method is maximum likelihood applied to the covariance matrix assembled from the summary statistics in **Table 2**. This method assumes multivariate normality, which implies that all univariate distributions should be approximately normal in shape. Reported at the bottom of Table 2 are values of the skew and kurtosis indices for all variables. None of these results indicate severe non-normality (Kline, 2016). All solutions in the analysis were admissible; that is, there were no indications of problems among the estimates, such as Heywood cases. The R syntax and output for all analyses described next are provided in Supplementary Material.

A total of five single-factor measurement models were analyzed in CFA. These models concerned the CogState tasks as indicators of a common cognitive factor. Values of selected fit statistics for all five CFA models are reported in **Table 3**. Additional information on these different models and the criteria used to select the final one are provided in Supplementary Results. Next, the SR model in Figure 1 was analyzed. The error variance for the single indicator of the symptom unawareness factor, SUMD, is fixed to equal the constant .360. Details on the calculations are provided in Supplementary Results. Fixing the error variance for the single indicator is also necessary in order to identify the SR model in Figure 1.

Reported in Table 3 are values of selected fit statistics after fitting the model in Figure 1 to the data in Table 2. The model passes the chi-square test, $\chi^2(12) = 9.885$, $p > .05$; values of other fit statistics do not suggest an obvious problem; no absolute correlation between residuals exceeded .10; and no standardized residuals were significant. The solution was admissible. Given all these results, the model in Figure 1 was retained. An equivalent version of Figure 1 is a two-factor CFA model with a covariance between the factors. For the model just described, $\chi^2(12) = 9.885$, which equals the same result for Figure 1.

Parameter estimates for the model in Figure 1 are reported in **Table 4**. More details on these results are provided in Supplementary Results. The standardized error variance for the symptom unawareness factor is .936. This means that the cognitive capacity factor explains a total of 6.4% of the variance in the symptom unawareness factor (i.e., $R^2 = .064$).

Next, scores on a cognitive composite were calculated for each case by applying the unstandardized pattern coefficients in Table 4 for the cognitive factor as follows:

Comp. = .555*ISL + .428*ISLR + .029*OCL + .038*CPAL + .261*GMR

Scores on the composite were centered, and both the cognitive composite and group membership (FEP vs. MEP) were entered as predictors of observed SUMD scores about symptom unawareness at the first step in a hierarchical multiple regression analysis. Results are reported in **Table 5**, for which additional details are provided in Supplementary Results. The overall R^2 at step 2 with the product term, or .033, is the same result at three-decimal accuracy at step 1 without the product term, or $\Delta R^2 = 0$. That is, estimating interaction fails to increase the overall proportion of explained variation. None of the individual regression coefficients are significant at step 2. The power of this analysis is probably quite low, but the miniscule effect size makes it apparent that stage of illness does not appreciably moderate the relation between cognitive

capacity and symptom unawareness.

4. DISCUSSION

These results suggest that patients with a psychotic disorder who show greater cognitive capacity as measured with computer-administered tasks have better symptom awareness. The tasks assess verbal memory, executive functions, and visual memory, all of which have been associated with insight into symptoms (Monteiro et al., 2008; Morgan et al., 2010; Wiffen et al., 2012). Yet, the magnitude of this relation is, although statistically significant, relatively small. In latent variable analyses, cognitive capacity accounted for about 6.4% of the variation in symptom unawareness while controlling for measurement error. This finding is consistent with other results by Freudenreich et al. (2004) and McCabe et al. (2002) that cognitive capacity and symptom unawareness are not strongly related.

Results of manifest variable analyses in the present study also indicate that illness stage did not appreciably moderate the association between symptom unawareness and cognitive capacity. Perhaps episode recurrence does not have a great impact on the relation between symptom unawareness and cognitive capacity because cognitive capacity per se explains only a relatively small proportion of the variance.

One possibility is that the determinants of some facets of insight could remain stable across the stages of illness, while predictors of other dimensions of insight may fluctuate over time (Ayesa-Arriola et al., 2011; Gilleen et al., 2014). For example, Cuesta et al. (2011) reported that insight into illness, another facet of clinical insight, among FEP patients is determined over time by somewhat different predictors compared with MEP patients. Other results about the relation between duration of untreated psychosis and clinical insight are more mixed (Buchy et

al., 2010a; Compton et al., 2011; Drake et al., 2000; Gumley et al., 2014; Hui et al., 2015; O'Donoghue et al., 2014), so the status of stage of illness as a moderator is unclear. Age and the duration of illness were not significantly correlated with the level of symptom unawareness in our dataset. Having acquired more knowledge and vocabulary about psychosis through more extended care in MEP may therefore not explain the qualitative differences in insight into symptoms that are thought to exist between these two stages (Gerretsen et al., 2014; Koren et al., 2013). Studies comparing FEP and MEP patients have found that the former group presents more severe deficits of clinical insight (Koren et al., 2013; Schennach et al., 2012; Thompson et al., 2001). Some of the explanations for this observation include better illness acceptance or longer time undergoing treatment in MEP; and greater psychological defensiveness or lack of knowledge about psychosis in FEP patients (Gerretsen et al., 2014). However, all of the above remains to be empirically verified.

Perhaps *cognitive insight* indirectly affects insight into symptoms. Some authors have suggested that cognitive insight is a prerequisite for good clinical insight (Beck et al., 2004; De Vos et al., 2015; Nair et al., 2014; Riggs et al., 2012; Van Camp et al., 2017). Yet, cognitive insight is reportedly associated with insight into symptoms as well as cognitive capacity in both FEP and MEP samples, which could suggest a mediating role for this variable or at least a more complex interaction between the three constructs (Buchy et al., 2010b; Cooke et al., 2010; Gilleen et al., 2011; Lepage et al., 2008; Pedros Rosello, 2018).

An integrative approach that addresses multiple determinants of clinical insight may be promising. This is because individual pharmacological treatments and psychosocial interventions appear to only modestly improve clinical insight among both FEP and MEP patients (Kobayashi et al., 2009; Misiak et al., 2016; Pijnenborg et al., 2015; Pijnenborg et al., 2013). Results of a

recent study by Lalova et al. (2013) indicate that insight into symptoms was amenable to treatment among MEP through cognitive remediation therapy, which involves the teaching of strategies and exercises practice (Fisher et al., 2013). Given our results, cognitive remediation could be an interesting therapeutic avenue for FEP patients, too. Perhaps more comprehensive forms of cognitive interventions—such as cognitive enhancement therapy, which integrates remediation of social and nonsocial cognitive skills—could enhance clinical insight by influencing some of its cortical underpinnings (Buchy et al., 2017; Eack et al., 2010; Shad and Keshavan, 2015).

Strengths of the present study include applying SEM analyses within well-defined samples of both FEP and MEP patients while controlling for measurement error. Limitations include a relatively small sample size considering the type of statistical analyses used and the corresponding need to replicate our results. The measure of symptom unawareness in our study dealt with only four symptoms. Nonetheless, these four symptoms likely represent the most prevalent ones (Sauvé et al., in press). Insight into a broader range of symptoms and other facets of clinical insight, such as unawareness of the need for treatment, should be studied. In a related fashion, specific associations between individual cognitive domains and symptoms could exist. We opted for a parsimonious approach and conceptualized our cognitive capacity variable as latent in part because we had averaged the awareness scores of different symptoms together, and also because our sample was too small to perform such specific comparisons.

Responding to computer-administered tasks may have been more challenging for the MEP patients in our sample, who were somewhat older than FEP patients. The relatively limited age range of our sample could have also masked some of the effects of aging on cognitive capacity for instance and its influence on symptom awareness. Future studies investigating the

relation of cognitive capacity and insight in older adults would therefore be interesting.

The design of the present study is cross-sectional. Ideally, the same patients would be longitudinally evaluated at different stages of their illness trajectory to minimize the influence of personal characteristics, experience of the illness and treatment, among other variables, and to appreciate the effect of possible cognitive changes or IQ decline (cf. Bergh et al., 2016; Rund et al., 2016). Yet, the cross-sectional comparison of illness stages is in line with the clinical staging framework proposed by McGorry et al. (2014; 2010) and represents an interesting preliminary step in identifying important variables and relations that would merit further attention in more costly longitudinal studies. Finally, additional predictors of symptom unawareness mentioned earlier could be included in a more complete statistical model of clinical insight.

In summary, our results suggest that cognitive capacity predicts a relatively small portion of variation in symptom unawareness among non-affective psychotic disorder patients. Stage of illness did not moderate this association. Future studies may benefit from separately analyzing insight into positive and negative symptoms as the cognitive determinants of each may differ (Gilleen et al., 2011). Given an expected association between insight deficit and functional outcome, more studies taking the influence of episode recurrence into account may help to better define or develop improved stage-specific interventions.

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FIGURE LEGENDS

Figure 1. Final structural regression model of cognitive capacity and symptom unawareness. Values of identifying constraints are shown, including scaling constants for factors or error terms (1) and the error variance for the single indicator of symptom unawareness (.360). ISL, International Shopping List; ISLR, International Shopping List Delayed Recall; GML, Groton Maze Learning task; GMR, Groton Maze Learning task Delayed Recall; OCL, One-Card Learning task; CPAL, Continuous Paired Associate Learning task; SUMD, Scale to Assess Unawareness of Mental Disorder.

Table 1. *Sample demographic, clinical, and cognitive characteristics*

Variable	First-episode psychosis	Multiple-episode psychosis	Effect size[a]
n	62	131	—
Percent men	61.3	73.3	.12
Age (yrs.)[b]	24.1 (4.6)[c]	35.4 (7.8)	−1.62
Duration of illness (yrs.) [b]	.2 (.1)	12.9 (7.6)	−2.03
Education (yrs.)	12.4 (2.8)	11.4 (2.5)	.38
Antipsychotics[b, d]	168.1 (123.9)	805.3 (865.3)	−.88
IQ (WASI)[b]	103.1 (13.4)	95.9 (13.1)	.54
Positive symptoms (SAPS)			
Hallucinations	1.8 (1.8)	2.2 (1.9)	−.21
Delusions	2.8 (1.6)	2.2 (1.7)	.36
Bizarre behavior[b]	2.0 (1.4)	1.1 (1.2)	.71
Thought disorder	1.5 (1.5)	1.3 (1.4)	.14
Negative symptoms (SANS)			
Affective flattening	2.1 (1.3)	2.4 (1.2)	−.24
Alogia	1.8 (1.5)	1.4 (1.2)	.31
Avolition-apathy	3.0 (1.2)	2.7 (1.2)	.25
Anhedonia-asociality	2.8 (1.2)	2.7 (1.3)	.08
Affective symptoms			
Depression (CDSS)	3.8 (4.0)	2.9 (3.0)	.27
Anxiety (HARS)	8.3 (6.6)	7.0 (5.2)	.23

Symptom unawareness			
SUMD (items 3a-6a)	3.0 (1.1)	2.9 (1.3)	.08
Symptom misattribution			
SUMD (items 3b-6b) [b,f]	3.5 (1.2)	2.7 (1.6)	.54
CogState tasks[e]			
ISL[b]	24.6 (4.0)	21.0 (4.6)	.81
ISLR[b]	8.4 (2.5)	6.5 (2.5)	.76
ONB[b]	1.2 (.1)	1.1 (.1)	1.00
TWOB	1.1 (.1)	1.1 (.1)	0
GML[b]	6.8 (1.6)	5.7 (1.6)	.69
SETS	.5 (.03)	.5 (.03)	0
DET	1.1 (.03)	1.1 (.03)	0
GMCT[b]	1.7 (.3)	1.1 (.4)	1.61
OCL[b]	1.0 (.1)	0.9 (.1)	1.00
CPAL[b]	1.3 (.1)	1.2 (.1)	1.00
GMR[b]	4.7 (1.0)	4.2 (1.0)	.50
IDN	.6 (.1)	.6 (.1)	0

[a]Standardized mean differences except for percent men, for which the phi coefficient is reported.
[b]$p < .05$ after Bonferroni correction for multiple comparisons (30).
[c]*M* (*SD*).
[d]Chlorpromazine equivalent, based on $n = 51$ for FEP and $n = 124$ for MEP. For the FEP group the value represents the cumulative dose since their entry into the first-episode clinic, while the value refers to the current dose for the MEP group.
[e]Normalizing transformations, ONB, $1 - \log_{10}(2 - X)$; TWOB, $2 - (3 - X)^{1/2}$; GML, $(X - 3)^{1/2}$; SETS, $1/(3 - X)$; DET, $\log_{10}(X - 1)$; CPAL, $\log_{10}(X)$; GMR, $(X + 1)^{1/2}$; IDN, $1/(3 - X)$. Next, scores on four tasks were reversed so that high scores indicate better performance. Original scores were multiplied by −1, and then a constant was added so that lowest score is 1 (GML, 13.49; DET, 1.31; GMR, 7.73, CPAL, 1.40)
Note. WASI, Wechsler Abbreviated Scale of Intelligence; SAPS, Scale for the Assessment of Positive Symptoms; SANS, Scale for the Assessment of Negative Symptoms; CDSS, Calgary Depression Scale for Schizophrenia; HARS, Hamilton Anxiety Rating Scale; SUMD, Scale to Assess Unawareness of Mental Disorder; ISL, International Shopping List; ISLR, International Shopping List Delayed Recall; ONB, One-Back task; TWOB, Two-Back task; GML, Groton Maze Learning task; SETS, Set-Shifting task; DET, Detection task; GMCT, Groton Maze Chase task; OCL, One-Card Learning task; CPAL, Continuous Paired Associate Learning task;

GMR, Groton Maze Learning task Delayed Recall; IDN, Identification task.
[f] Based on *n* = 60 for FEP group and *n* = 110 for MEP group

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Table 2. *Summary Statistics (Correlations, Means, Standard Deviations) for Symptom Unawareness and Cognitive Tasks*

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. SUMD	—												
2. ISL	−.144	—											
3. ISLR	−.155	.753	—										
4. ONB	−.184	.238	.236	—									
5. TWOB	−.096	.290	.248	.460	—								
6. GML	−.215	.329	.334	.397	.442	—							
7. SETS	−.190	.275	.218	.351	.474	.352	—						
8. DET	.005	.289	.277	.309	.362	.193	.128	—					
9. GMCT	−.080	.424	.398	.291	.327	.314	.127	.559	—				
10. OCL	−.146	.398	.347	.474	.369	.411	.325	.203	.326	—			
11. CPAL	−.143	.430	.439	.301	.416	.526	.296	.294	.422	.470	—		
12. GMR	−.108	.316	.307	.398	.362	.672	.312	.257	.307	.451	.532	—	
13. IDN	−.132	.239	.282	.390	.281	.332	.305	.027	.147	.304	.203	.298	
M	2.933	22.166	7.083	1.155	1.073	6.073	.543	1.113	1.319	.951	1.241	4.370	.627
SD	1.225	4.713	2.670	.122	.124	1.664	.032	.036	.436	.116	.121	1.009	.064
Skew	.031	−.347	−.246	.086	.427	−.334	−.536	−.470	−.018	−.076	.382	−.200	−.327
Kurtosis	−.850	−.153	−.228	−.044	3.623	1.339	−.574	.048	−.458	−.491	−.475	.311	−.587

Note. N = 193. SUMD, Scale to Assess Unawareness of Mental Disorder; ISL, International Shopping List; ISLR, International Shopping List Delayed Recall; ONB, One-Back task; TWOB, Two-Back task; GML, Groton Maze Learning task; SETS, Set-Shifting task; DET, Detection task; GMCT, Groton Maze Chase task; OCL, One-Card Learning task; CPAL, Continuous Paired Associate Learning task; GMR, Groton Maze Learning task Delayed Recall; IDN, Identification task.

Table 3. *Values of Fit Statistics for Measurement Models of Cognitive Capacity and a Structural Regression Model of Cognitive Capacity and Symptom Unawareness*

Model	Retained?	χ^2	*df*	RMSEA [90% CI]	CFI	SRMR
One-factor CFA						
1. Twelve cognitive tasks[a]	N	276.860	54	.146 [.129, .164]	.734	.090
2. Nine tasks (SETS, DET, IDN out)	N	173.101	27	.167 [.144, .192]	.776	.086
3. Seven tasks (ONB, GCMT out)	N	135.897	14	.212 [.181, .246]	.766	.093
4. Six tasks (TWOB out)	N	129.942	9	.264 [.225, .305]	.741	.104
5. Six tasks, two error correlations[b]	Y	5.220	7	0 [0, .073]	1.000	.023
Two-factor SR	Y	9.885	12	0 [0, .062]	1.000	.027

[a]ISL, International Shopping List; ISLR, International Shopping List Delayed Recall; ONB, One-Back task; TWOB, Two-Back task; GML, Groton Maze Learning task; SETS, Set-Shifting task; DET, Detection task; GMCT, Groton Maze Chase task; OCL, One-Card Learning task; CPAL, Continuous Paired Associate Learning task; GMR, Groton Maze Learning task Delayed Recall; IDN, Identification task.
[b]ISL, ISLR, GML, OCL, CPAL, GMR, ISL ↔ ISLR, GML ↔ GMR.
Note. $p < .05$ for chi-square, Models 1–4 only. RMSEA, Steiger-Lind root mean square error of approximation; CI, confidence interval; CFI, Bentler comparative fit index; SRMR, standardized root mean squared residual; CFA, confirmatory factor analysis; SR, structural regression.

Table 4. *Maximum Likelihood Parameter Estimates for a Structural Regression Model of Cognitive Capacity and Symptom Unawareness*

Parameter	Unstandardized	*SE*	Standardized
Pattern coefficients			
SUMD	1.0	—	.871
ISL	1.0	—	.546
ISLR	.555	.059	.535
GML	.428	.070	.663
OCL	.029	.005	.634
CPAL	.037	.006	.778
GMR	.261	.042	.667
Error variances and covariances			
SUMD	.360	—	.241
ISL	15.498	1.784	.701
ISLR	5.059	.578	.713
GML	1.543	.206	.560
OCL	.008	.001	.598
CPAL	.006	.001	.395
GMR	.562	.076	.555
ISL ↔ ISLR	5.764	.873	.651
GML ↔ GMR	.383	.102	.412
Factor or disturbance variance			
Cognitive Capacity	6.599	1.806	1.0
Symptom Unawareness	1.060	.146	.936
Factor regression coefficient			
Cognitive → Unawareness	−.105	.040	−.254

Note. $p < .05$ for all unstandardized estimates with standard errors. SUMD, Scale to Assess Unawareness of Mental Disorder; ISL, International Shopping List; ISLR, International Shopping List Delayed Recall; ONB, One-Back task; TWOB, Two-Back task; GML, Groton Maze Learning task; SETS, Set-Shifting task; DET, Detection task; GMCT, Groton Maze Chase task; OCL, One-Card Learning task; CPAL, Continuous Paired Associate Learning task; GMR, Groton Maze Learning task Delayed Recall; IDN, Identification task.

Table 5. *Moderated multiple regression results for predicting symptom unawareness from cognitive capacity and first-episode versus multiple-episode psychosis*

	Step 1			Step 2		
Predictors	*B*	*SE*	*b*	*B*	*SE*	*b*
Group	−.237	.201	−.090	−.241	.214	−.092
Cognitive composite	−.065[a]	.026	−.194	−.068	.051	−.202
Group × Cognitive	—	—	—	.004	.059	.009
R^2		.033[a]			.033	
ΔR^2		—			0	

[a]$p < .05$.
Note. *B*, *SE*, *b* refer to, respectively, unstandardized coefficient, standard error, standardized coefficient. Scores on the cognitive capacity composite are centered. For the group variable, 0 = first-episode psychosis, 1 = multiple-episode psychosis. The constant for step 1 is 3.093 (*SE* = .162), and for step 2 the constant is 3.099 (*SE* = .184)

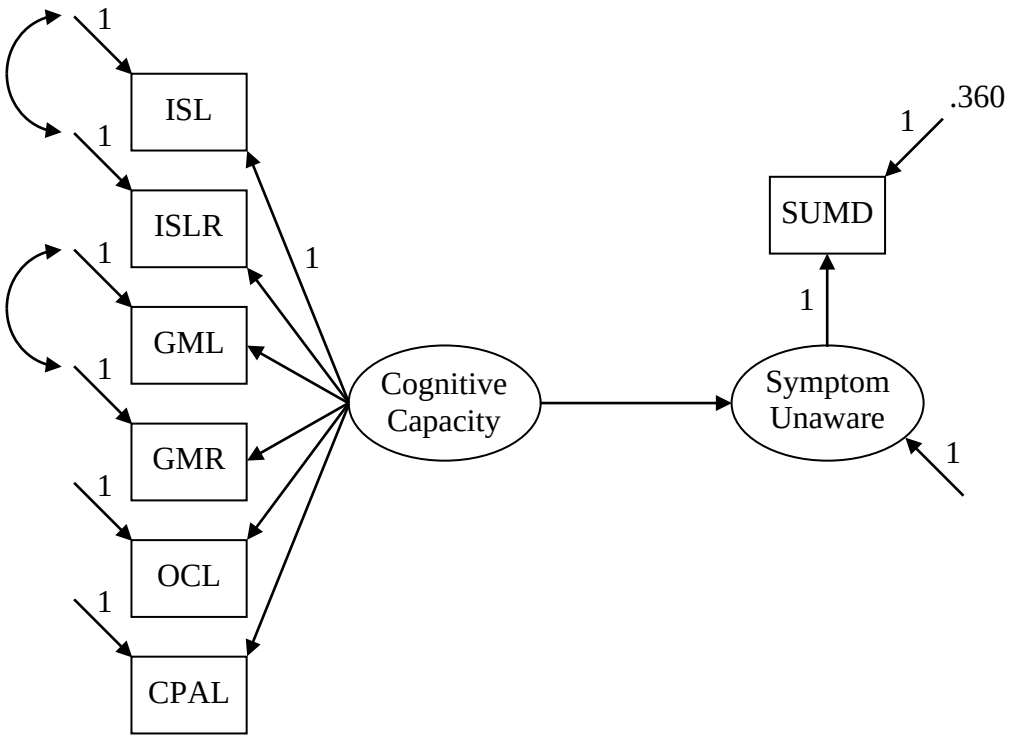

Figure 1. Final structural regression model of cognitive capacity and symptom unawareness.