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Metacognition, symptoms and premorbid functioning in a First Episode Psychosis sample.

Angus MacBeth, PhD *†; Andrew Gumley, PhD,‡; Matthias Schwannauer, PhD §; Antonino Carcione, MD ‖; Rebecca Fisher, PhD §; Hamish J. McLeod, PhD‡; Giancarlo Dimaggio, MD¶.

*University of Aberdeen, Scotland, UK
†NHS Grampian, Scotland, UK
‡University of Glasgow, Scotland, UK
§University of Edinburgh, Edinburgh, Scotland, UK
‖ Terzo Centro di Psicoterapia Cognitiva, Rome, Italy
¶ Centre for Metacognitive Interpersonal Therapy, Rome, Italy.

Correspondence to: Dr Angus MacBeth
Pluscarden Clinic
Dr Gray’s Hospital
Elgin
Moray
Scotland
United Kingdom
IV36 1LR

Tel: (+44) 343 567499
Fax: (+44) 343 567 645
e-mail: angus.macbeth@abdn.ac.uk
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Abstract

Significant metacognitive impairments are observed in chronic psychosis samples but metacognition is less understood in first episode psychosis (FEP). The current study explored correlations between metacognition, symptoms and premorbid functioning in an FEP sample. In a cross-sectional cohort study, individuals in the first 12 months of treatment metacognition was assessed with the Metacognition Assessment Scale – Revised version (MAS-R). Psychotic symptomatology, premorbid adjustment, and clinician rated service engagement were also measured. Lower scores for metacognitive understanding of other’s minds were significantly correlated with greater negative symptoms, poorer early adolescent social adjustment and poorer clinician rated help-seeking. Our findings suggest that FEP individuals with difficulties in understanding other’s minds have more social deficits and may be less able to make effective use of treatment.

Keywords: Schizophrenia, psychosis, metacognition, premorbid adjustment
Title: Metacognition in first episode psychosis: associations with symptoms and functioning.

1. Introduction

Individuals with psychotic disorders experience significant difficulties in reflecting upon their own mental states, mental states of others, and in using mental state information to solve problems [1]. These difficulties can be understood as semi-independent capacities, associated with, but not reducible to functional or neurocognitive deficits [2]. Difficulties in mental state capacities have been variously referred to as theory of mind (ToM), social cognition or metacognition. There is evidence of significant metacognitive impairments in chronic psychosis samples [e.g. 1], related to social function, negative symptoms, cognitive disorganization and work performance. The most consistent finding is the association between poor metacognition and greater negative symptoms [3]. Indeed deficits in metacognition are risk factors for suboptimal vocational and functional outcomes, linked to social cognitions and communication difficulties [4-7].

However, there is accumulating evidence to suggest that there are differences between discrete forms of social cognition such as ToM that refer to an individual’s capacity to make judgements regarding one aspect of a given social situation (e.g. presence or absence of sadness), as opposed to synthetic metacognitive processes that refer to the ability to organise complex social information in such a way as to enable the individual to understand and reflect upon the other’s mental state and use this information to cope with distressing experiences and guide the individual’s own actions in a given situation [8-10]. Similarly, there are nuanced differences between ToM’s focus on using mental state information to inform understanding of the
physical world, or to develop cognitive understanding of other’s beliefs; as compared to the greater emphasis in synthetic metacognition on the interplay of cognition, affect and meaning [11]. As evidence suggests that social cognition influences outcome in psychotic disorders [4-7], thus delineation of the boundaries and areas of overlap between discrete and synthetic aspects of metacognition, as they relate to specific outcome domains in psychotic disorders, can enable better matching of specific interventions to specific metacognitive profiles.

Impairments in social cognition and function precede the onset of a first episode of psychosis (FEP), and are associated with poorer outcome in the early stages of the disorder [12,13] however the profile of metacognitive impairment is poorly understood in FEP. Evidence has accumulated suggesting deficits in discrete metacognitive processes, such as Theory of Mind, are present in FEP [e.g. 14,15]. Evidence also suggests that greater degrees of mentalization (a psychological construct related to metacognition and similarly predicated on individuals’ capacity for understanding of self and others’ behaviour in terms of mental states) are not associated with symptoms [16]. In this sample higher levels of metacognition were associated with better engagement with treatment. Furthermore, associations between premorbid factors and synthetic metacognition have not been examined in this population. Therefore, the current study sought to establish the magnitude of associations between synthetic metacognition and other variables, specifically symptoms and premorbid functioning in a FEP sample. Specifically, we hypothesized that lower metacognition scores would be associated with greater negative symptoms, poorer premorbid adjustment and poorer engagement with treatment.
2. Method

Participants were 20 males and 14 females presenting to Early Intervention for Psychosis services in two Scottish cities. Mean age (SD) of participants was 23.3 years (7.6 years) and the median duration of untreated psychosis was 20.5 weeks (range = 1-520). The majority of participants were prescribed antipsychotic medication. Individuals were eligible if they were in the first 12 months of treatment for first episode psychosis. This was defined as presentation to clinical services with psychotic symptoms for the first time, with positive psychotic symptoms of sufficient severity and/or distress to require antipsychotic medication; meeting DSM criteria for an affective or non-affective psychotic disorder [17]; substance misuse, head injury or organic disorder not judged to be the primary cause of psychotic symptoms; and retaining capacity to consent. Identification of participants was facilitated through collaboration with clinicians. The study received review and ethical approval from Greater Glasgow and Lothian Research Ethics Committees (REC: 04/S0703/91), and received managerial approval from the local Research and Development Departments in Lothian and Glasgow. All participants gave voluntary and informed consent to participate in the study.

2.1. Measures

The PANSS [18] is a 30 item semi-structured interview of psychotic symptomatology. We adapted a five factor scoring model, yielding scores for: positive symptoms, negative symptoms, cognitive disorganization, excitement and emotional distress [19]. Each item is scored on a Likert scale from minimal (1) to extreme (7). Inter-rater reliability estimates for PANSS subscales were adequate (all intra-class correlation coefficients >.82).
Duration of untreated psychosis (DUP) was measured using an unstructured interview protocol adapted from Beiser and colleagues’ [20] methodology. Information regarding the circumstances of onset and development of psychotic symptomatology was collected from the individual and (where a clear DUP could not be estimated) a carer or loved one, cross-referenced with clinical case notes, and discussed with the individual’s clinician. The DUP interview was conducted when patients were no longer floridly psychotic. Date of onset of psychosis was calculated to the nearest week and transition to psychosis was indicated by presence of one or more symptoms on the positive symptom scale of the PANSS, rated as 4 or greater (indicating significant impairment). Where the exact date of onset was unclear, the date was taken as the 1st day of the month for which symptoms rated above threshold. The endpoint of the DUP was considered to be the date at which antipsychotic medication was prescribed and/or multi-disciplinary team involvement initiated; and where compliance with the treatment plan could be ascertained at one month after initiation of treatment. DUP was established via a consensual judgement of the information gathered. This was facilitated through monthly consensus meetings between the authors. Failure to reach consensus triggered further assessment of DUP with individuals and key informants, usually family and friends. Test-retest reliability for this method of determining DUP from Larsen et al. [21] is reported as good (intraclass coefficient $r = .96, p<0.01$).

Premorbid Adjustment was measured using the Premorbid Adjustment Scale [PAS; 22] a semi-structured interview that retrospectively measures level of functioning prior to onset of psychosis. The measurement period is from birth till adulthood; sub-divided into four age periods – childhood, early adolescence, late adolescence and adulthood. Given the potential overlap between adult adjustment and DUP we
follow the convention of only reporting data pertaining to the first three time periods [23]. Scores are calculated for academic and social functioning components [22]. Service Engagement was measured using the Service Engagement Scale [SES; 24] a 14-item, clinician-completed scale to assess overall engagement with services. Items assess four subscales including availability, collaboration, help-seeking and treatment adherence. The scale has good reliability and discriminant validity [Cronbach’s alpha = 0.76 – 0.90 for sub-scales; 25].

Metacognition was assessed using the MAS-R [26]. This is a modified version of the MAS [9]. Scores are generated on 3 subscales designed to tap into metacognitive capacities - Understanding Ones’ Own mind (UM), Understanding of Others’ Minds (UOM) and Mastery (M). Understanding Ones’ Own mind refers to the individual’s comprehension of one’s own mental states. Understanding of Others’ Minds (UOM measures the comprehension of other individuals’ mental states. Mastery represents the ability to use knowledge of mental states to intentionally manage conflicts and subjective distress. Lower scores on each subscale reflect greater difficulties in that domain of metacognitive ability. The MAS-R has previously been successfully applied to assessing individuals with schizophrenia under forensic care [27]. Narratives were derived from participant Adult Attachment Interviews’ [AAI; 28]. These were recorded, transcribed and anonymised before MAS-R coding. Further details of the AAI in this sample are available elsewhere [Reference Removed for anonymity]. MAS-R coding was completed by XX and XX, both of whom were blind to any other details regarding the sample. Demographics and treatment data were completed 12 months after initiation of treatment, based on information from case notes and key-workers corroborative report.
2.2. Procedures

A cross sectional cohort design was used. Interview measures were conducted by XX, XX and trained research assistants. The research team were not involved in participants’ clinical care. Symptomatology was measured at the first session after consent, DUP at the second session, and premorbid adjustment thereafter. The SES was completed by the patient’s keyworker or psychiatrist, independently of the researcher.

2.3. Data Analysis

Data were analysed using SPSS version 16. All variables were checked for normality using the Kolmogorov-Smirnov test. Associations between variables were examined using Pearson correlations and t-tests for significant differences between groups. Cohen's criteria for interpreting the strength of correlations were used, whereby $r=0.1-0.3$ is considered a small effect, $r=0.3-0.5$ a moderate effect, and $r=>0.5$ is a large effect. Associations between categorical variables were investigated using Chi-Square tests. DUP was transformed to its natural logarithm to improve normality. This is an accepted method for handling this variable [29]. As the analyses were conducted on a relatively small sample the significance level (alpha) was set at 0.05.

3. Results

Means and standard deviations for key variables are listed in Table 1. Associations between metacognition and symptoms are displayed in Table 2. Metacognitive understanding of one's own mind was not significantly related to symptom variables. Greater metacognitive Understanding of Others' minds' was significantly correlated with fewer negative symptoms ($r=-.437$, $p=.023$), but metacognition was
not significantly correlated with positive psychotic symptoms, cognitive disorganisation, excitement or emotional distress. Mastery was unrelated to psychotic symptoms.

Associations between metacognition, premorbid adjustment and DUP are displayed in Table 2. Metacognitive UM was unrelated to academic or social premorbid adjustment at any developmental point. However, the association between poorer early adolescent premorbid adjustment and poorer UM approached significance (r=-.359; p=.051). Poorer early adolescent social adjustment was significantly associated with poorer UOM (r=-.40; p=.03). UOM was not related to premorbid academic adjustment, or to childhood social adjustment. Mastery was unrelated to premorbid adjustment. Duration of untreated psychosis and metacognition were not significantly associated.

Metacognition was unrelated to overall scores on the SES. Metacognition was also unrelated to the availability, collaboration and treatment adherence sub-scales of the SES. However, there was a significant correlation between greater UM scores and better SES help-seeking (r=-.52; p=.01). Higher scores for UOM were also significantly correlated with better SES help-seeking (r=-.61, p=.002 respectively). Mastery was not related to SES scores.

4. Discussion
This study is the first to explore metacognition, measured using the Metacognition Assessment Scale in a first episode psychosis sample. Our findings demonstrate a specific pattern of associations in FEP, namely between poorer awareness of other’s mind and negative symptoms; between poorer awareness of other’s minds and early
adolescent premorbid social functioning; and between poorer awareness of other’s minds and help-seeking. Our findings with regard to negative symptoms and UoM are consistent with previous studies of the MAS in chronic samples [e.g. 2,3,27]. However, in contrast to studies of metacognition in chronic psychosis samples we did not find associations between negative symptoms and Understanding of ones’ own mind [e.g. 2,3]. This suggests that associations between UoM and negative symptoms are evident in both first and multi-episode groups. This is consistent with the emergence of negative symptoms as a marker of potential chronicity. However, although synthetic metacognition could potentially mediate or moderate the relationship between symptoms and outcome, it may be the case that in the early stages of illness observed difficulties in metacognition are specific to the understanding of other’s mental states. This would be consistent with the observation that social cognition deficits are associated with smaller social networks and engagement in vocational activities [e.g. 30]. However, perhaps as a function of the low power in the study it is disappointing to note that we did not record any significant results for correlations between symptoms and mastery in this sample as in chronic samples where mastery was related to poor functional competence [7], which would have enabled us to comment on the capacity of individuals to use mental state understanding to problem solve. It may be the case that the association between mastery and negative symptoms becomes stronger over time as a function of increased chronicity, thus would be less prominent in an FEP sample.

However, we note significant associations with early adolescent premorbid social adjustment and poorer UoM suggesting that metacognitive difficulties may reflect psychodevelopmental factors. Taken with the aforementioned association between UoM and negative symptoms, this is consistent with review evidence suggesting that
premorbid social and academic deficits are linked to remission of symptoms and poorer quality of life in FEP [13, 31]. Therefore, deficits in metacognitive awareness, similar to social cognition difficulties [10] may represent trait level risk markers for a range of complex psychopathologies [32]. Though associations between metacognition, symptoms and functioning are less evident in FEP than in chronic samples the current data suggest that these patterns of association are already present in first episode. It may be the case that individuals in thier first episode already have premorbid difficulties in comprehending their own and other’s mental states, leading to a process of social withdrawal, exacerbating the emergent interpersonal difficulty. Consistent with the critical period hypothesis [33] it is likely that if these difficulties are not effectively addressed the toxic effect of impaired metacognition grows. This creates a vicious cycle of a lower threshold for the activation of distress in relation to interpersonal situations (e.g. work, relationships), decreased capacity to effectively engage with interpersonal stressors, and increased social withdrawal as a coping strategy, and an atrophying of metacognitive capacity. It is also of note that the current sample had a young mean age, therefore may have little experience of living independently or without family support, further limiting their developmental opportunities to acquire coping skills. Furthermore, although factor analysis using samples with chronic schizophrenia suggests psychometric differences between discrete and synthetic forms of metacognition [10] future research into the validity of this distinction in FEP samples would be valuable.

Our findings also suggest that individuals with difficulties in understanding others’ minds have greater social deficits and may be less able to make effective use of treatment services (e.g. early intervention). We did not find associations between symptoms and UM though it may be the case that different patterns of self-other
metacognitive functioning relate to different clinical presentations. For instance, Lysaker and colleagues [30] reported that individuals with difficulties in accessing both their own and other’s mental states had greater disorganization symptoms and neurocognitive deficits, whereas individuals with awareness of self and other’s mental states had less symptoms and neurocognitive deficits, but reported a greater history of childhood abuse. It is of note that this study did not measure premorbid function. It has been suggested [6] that for some individuals with psychosis, the diminished capacity to form complex and integrated representations of oneself and others leads to difficulties in engaging with the social world, itself leading to the entrenchment of negative symptoms such as social withdrawal and reduction in the experience of affect and volition.

There are methodological limitations in the study. The sample size was small and cross sectional, and we acknowledge possible Type II errors in the results. However, the aim of the study was to generate correlational effect size estimates for replication with a larger sample, which would address these issues. The current data also generate useful hypotheses for further exploration of mediating and moderating factors between premorbid function, metacognition and the development or forestalling of recovery trajectories in FEP. Participants were in a non-acute phase of illness and responsive to an integrated early intervention treatment program. Furthermore, this care is embedded in a public health service setting. Thus, compared with other cohorts our participants may represent a higher functioning group of individuals, with corresponding reduction of variance in the DUP and lower levels of positive and negative symptoms. Equally, our findings may not generalise to other systems of health care provision. However, it is of note that participants were by no means asymptomatic.
These data are also consistent with emerging theoretical and empirical data on the potential for integrating metacognition into psychological interventions for psychotic disorders [e.g. 34-37]. Metacognition is also potentially applicable to psychological therapies specifically targeted at negative symptoms of schizophrenia, such as cognitive remediation therapy [38]. Furthermore, current CBT based approaches to the treatment of negative symptoms emphasise the identification and challenging of defeatist cognitions, and the use of behavioural activation [39-41] However, these strategies may be difficult to acquire for patients who have restricted abilities to understand their own mind.

Role of Funding Source.

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Conflicts of interest

No conflicts of interest are declared.

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Table 1: Demographics and mean symptom, premorbid adjustment and metacognition scores (n=34).

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Descriptive Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenia</td>
<td>11 (32%)</td>
</tr>
<tr>
<td>Schizophreniform Disorder</td>
<td>3 (9%)</td>
</tr>
<tr>
<td>Schizoaffective disorder</td>
<td>4 (12%)</td>
</tr>
<tr>
<td>Persistent Delusional Disorder</td>
<td>2 (6%)</td>
</tr>
<tr>
<td>Bipolar Disorder</td>
<td>11 (32%)</td>
</tr>
<tr>
<td>Mania with psychotic symptoms</td>
<td>1 (3%)</td>
</tr>
<tr>
<td>Recurrent depression with psychotic symptoms</td>
<td>2 (6%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Educational Attainment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Left school before age 16</td>
<td>7 (10.9)</td>
</tr>
<tr>
<td>Left school at age 16 -18</td>
<td>32 (50)</td>
</tr>
<tr>
<td>Completed College course</td>
<td>10 (15.6)</td>
</tr>
<tr>
<td>Completed University degree</td>
<td>8 (12.5)</td>
</tr>
<tr>
<td>Did not complete college/University course</td>
<td>4 (6.3)</td>
</tr>
<tr>
<td>PAS Childhood Academic (Mean; s.d.)</td>
<td>.21 (.18)</td>
</tr>
<tr>
<td>PAS Childhood Social (Mean; s.d.)</td>
<td>.19 (.21)</td>
</tr>
<tr>
<td>PAS Early Adolescence Academic (Mean; s.d.)</td>
<td>.37 (.24)</td>
</tr>
<tr>
<td>PAS Early Adolescence Social (Mean; s.d.)</td>
<td>.18 (.19)</td>
</tr>
<tr>
<td>PANSS items</td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>10.11 (5.6)</td>
</tr>
<tr>
<td>Negative</td>
<td>12.54 (5.1)</td>
</tr>
<tr>
<td>Cognitive Disorganisation</td>
<td>14.14 (6.6)</td>
</tr>
<tr>
<td>Excitement</td>
<td>5.43 (3.4)</td>
</tr>
<tr>
<td>Emotional Distress</td>
<td>9.21 (4.2)</td>
</tr>
<tr>
<td>SES scale Total Score</td>
<td>7.22 (6.6)</td>
</tr>
<tr>
<td>SES Availability</td>
<td>0.79 (1.1)</td>
</tr>
<tr>
<td>SES Collaboration</td>
<td>1.87 (2.3)</td>
</tr>
<tr>
<td>SES Help-seeking</td>
<td>3.48 (2.9)</td>
</tr>
<tr>
<td>SES Treatment Adherence</td>
<td>1.00 (1.8)</td>
</tr>
<tr>
<td>MAS Scales</td>
<td></td>
</tr>
<tr>
<td>Understanding one’s own mind</td>
<td>2.34 (.82)</td>
</tr>
<tr>
<td>Understanding other’s minds</td>
<td>1.87 (.76)</td>
</tr>
<tr>
<td>Mastery</td>
<td>1.60 (.59)</td>
</tr>
</tbody>
</table>

Categorical Data are listed in the form n; (% of total sample). Continuous data are listed as mean (standard deviation). PAS = Premorbid Adjustment Scale; PANSS = Positive and Negative Syndrome Scale; SES = Service Engagement Scale; MAS = Metacognition Assessment Scale.
Table 2: Correlations between Metacognition, symptoms, premorbid adjustment and service engagement.

<table>
<thead>
<tr>
<th></th>
<th>Understanding one’s own mind</th>
<th>Understanding Other’s minds</th>
<th>Mastery</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANSS Positive</td>
<td>-.072</td>
<td>-.180</td>
<td>-.040</td>
</tr>
<tr>
<td>PANSS Negative</td>
<td>-.377</td>
<td>-.437*</td>
<td>-.307</td>
</tr>
<tr>
<td>PANSS Cognitive Disorganisation</td>
<td>-.058</td>
<td>-.097</td>
<td>-.183</td>
</tr>
<tr>
<td>PANSS Excitement</td>
<td>.117</td>
<td>.159</td>
<td>-.015</td>
</tr>
<tr>
<td>PANSS Emotional Distress</td>
<td>-.077</td>
<td>-.222</td>
<td>-.066</td>
</tr>
<tr>
<td>SES scale Total Score</td>
<td>-.299</td>
<td>-.393*</td>
<td>-.132</td>
</tr>
<tr>
<td>SES Availability</td>
<td>-.299</td>
<td>.192</td>
<td>.209</td>
</tr>
<tr>
<td>SES Collaboration</td>
<td>-.251</td>
<td>-.322</td>
<td>-.127</td>
</tr>
<tr>
<td>SES Help-seeking</td>
<td>-.522**</td>
<td>-.614***</td>
<td>-.284</td>
</tr>
<tr>
<td>SES Treatment Adherence</td>
<td>-.052</td>
<td>-.186</td>
<td>-.025</td>
</tr>
<tr>
<td>PAS Childhood Social</td>
<td>-.190</td>
<td>-.092</td>
<td>.019</td>
</tr>
<tr>
<td>PAS Early Adolescence Social</td>
<td>-.359*</td>
<td>-.402**</td>
<td>-.146</td>
</tr>
<tr>
<td>PAS Childhood Academic</td>
<td>-.173</td>
<td>-.069</td>
<td>-.080</td>
</tr>
<tr>
<td>PAS Early Adolescence Academic</td>
<td>-.102</td>
<td>-.092</td>
<td>-.162</td>
</tr>
<tr>
<td>Duration of Untreated Psychosis</td>
<td>-.049</td>
<td>-.042</td>
<td>-.109</td>
</tr>
</tbody>
</table>

*Note: All correlations Pearson’s r; * Transformed to natural logarithm; *p≤.10 (2-tailed); **p≤.05 (2-tailed); ***p≤.01 (2-tailed); PANSS—Positive and Negative Syndrome Scale; SES - Service Engagement Scale; PAS – Premorbid Adjustment Scale.