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Commentary: Social Cognitive Functioning in Prodromal Psychosis: A Meta-Analysis

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Schizophrenia is a chronic, severe and debilitating mental disorder, with the lifetime prevalence of approximately 1% of the population. It is characterized by deficits in thought processes, perceptions, and emotional responsiveness. It thus causes significant disruption in social behavior, daily activity, and decreased quality of life from its earliest stages through its chronic course. The detection of early indicators of vulnerability and development of early intervention before the onset of an overt psychotic symptom has been raised as an important issue over recent decades. In this regard, the concept of 'clinical-high risk' (CHR) for psychosis, also called 'ultra-high risk' (UHR) has been developed to reveal the pathophysiological mechanisms of schizophrenia and to come up with optimal intervention strategies in this population[1,2]. In schizophrenia, impairment in social cognition is one of the most common findings. Social cognition refers to how people perceive, encode, store, and apply information about other people and ourselves in social interaction contexts. Therefore, impaired social cognitive processes include difficulties in perceiving social cues, identifying and inferring emotions and thoughts of others, sharing experience and reacting appropriately to others in social situations[3]. Such deficits prevent patients with schizophrenia from forming social connections and are critical determinants of poor daily functioning[4]. Fett et al. performed a meta-analysis of the relationship between neurocognition and social cognition with functional outcomes in schizophrenia and they found that social cognition was more strongly associated with community functioning than neurocognition[5]. The National Institute of Mental Health (NIMH) consensus statement on social cognition in schizophrenia identified five relevant domains: theory of mind (ToM), social perception, social knowledge, attributional bias, and emotion processing. A recent meta-analysis including 112 studies examined the average magnitude of differences between schizophrenia patients and normal controls across multiple domains of these social cognitive domains[6]. This analysis observed that patients with schizophrenia performed worse than normal controls across all domains, with large effects on social perception, ToM, and emotion processing.

More importantly, social cognitive deficits have trait-like qualities that precede the onset of illness and are thus candidate endophenotypes for schizophrenia[3]. Also, these deficits have been found to appear in the prodromal phase before the onset of full-

blown schizophrenia[7,8]. In this regard, understanding the characteristics of impaired social cognition in prodromal phase may not only identify specific risk factors for schizophrenia, but also provide important insights into pathophysiological mechanisms associated with the development of schizophrenia. However, findings have been inconsistent in each domain in CHR population work, which interferes with identification of specific social cognitive domains for better targeted treatments.

In order to provide the most impaired domain in social cognition in individuals at CHR,[9] performed a domain-by-domain quantitative analysis of social cognition in CHR group, which was published in Schizophrenia Research. The authors included four social cognitive domains based on the Social Cognition Psychometric Evaluation (SCOPE) initiative of the NIMH, which were as follows: ToM, social perception, attributional bias, and emotion processing. Twenty studies including 1,229 individuals at CHR and 825 healthy controls met the inclusion criteria. The authors found that individuals at CHR showed significant impairments in all domains of social cognition, compared to healthy controls, and the largest effect size was observed in the domain of attributional bias. Given such findings, attributional bias might have indicated a possible trait marker of schizophrenia. However, the authors also found that CHR non-converters exhibited an increased trend of personalized attribution style, compared to CHR individuals who later developed psychosis, when they divided the population into the groups of converters and non-converters. Instead, CHR converters exhibited increased impairment of ToM compared to the CHR non-converters, consistent with a prior meta-analysis for ToM[10]. However, recent longitudinal study of social cognition in CHR individuals did find no differences in social cognition, including ToM, between those who made the transition to psychosis and those who did not[11], although CHR individuals as a group showed persistent ToM impairment compared to healthy controls. More studies are needed to explore the associations between the nature and trajectories of social cognition and their relations to outcomes in CHR individuals.

The authors have suggested the possibility that inclusion of a high proportion of false-positives in CHR population may contribute to the largest effect size observed in the attribution bias. Especially, the high comorbidity of depression in CHR patients has been suggested, based on the evidence that high attribution style is characteristic of patients with depression. Indeed, recent work of CHR individuals has highlighted the decreasing rate of transition to psychosis and the inclusion of a considerable number of false positives in CHR studies[12,13,14]. Furthermore, prior work in healthy individuals has suggested that psychotic-like experiences are common in the general population[15]. In this regard, the inclusion of a large number of false-positive individuals is likely to conceal the exact nature and magnitude of social cognitive deficits associated with conversion to schizophrenia. Accordingly, findings by Lee et al. highlight the need to screen for false-positives who will not develop schizophrenia and to reassess the inclusion criteria for CHR. Indeed, the current CHR criteria, which are mainly based on positive symptoms, have led to the inclusion of a considerable number of false positives in CHR studies.

Furthermore, the authors have found a more severe deficit in the CHR converters in ToM domain, compared to the CHR non-converters, suggesting that ToM may serve as a more promising predictor for the development of schizophrenia. This notion is supported by findings that impaired ToM was one of the most severe social cognitive deficits in patients with schizophrenia, whereas they exhibited relatively small impairment in attribution bias across multiple domains[6]. Furthermore, ToM deficits in schizophrenia had stronger associations with poor community functioning than other social cognitive domains[5]. A recent meta-analysis also found significant ToM deficits in CHR individuals, highlighting the possible predictive value of ToM for the transition into schizophrenia[8].

Overall, the study by[9] provides important clinical and research implications. From a clinical perspective, there is a clear need for eliminating the false-positives that may mask the characteristics of increased risk factors for schizophrenia. More alternative inclusion criteria and better prediction models for true-positives are required. Given, also, that CHR individuals have a high prevalence of *comorbidity including anxiety and depression[16], the impact of comorbid psychiatric disorders on the associations between social cognitive deficits and clinical outcomes is needed to be investigated via longitudinal studies. This may* help to identify a subgroup of subjects that would benefit the most from preventive interventions. From a research perspective, this study indicates that *differences in clinical information and IQ contribute to the performances in social cognition. Thus, the challenge in the future CHR research will be to determine whether* potential moderators (e.g., age, sex, duration of illness, age at onset, or IQ) are relevant to the social cognition-outcome relationship. The inclusion of various moderators may help to account for the heterogeneity in the associations between social cognition and clinical status.

The study by[9] has some methodological limitations that should be addressed in future studies. First, different tasks that assess each social cognitive domain were pooled together. This may constitute a major conceptual problem. Moreover, the domain of attribution bias, compared to the number of individuals included for other three social cognitive domains,

was comprised of the smallest number of CHR individuals. This may allow undetected study population biases that may lead to resulting in relatively stronger effects. Second, the authors included a study for the attribution bias that did not report demographic data for the control group, leading to large effect of the whole meta-analysis.

In summary, this study is the first meta-analysis to investigate the domain-by-domain performances of social cognition in individuals at CHR. Their findings suggest careful characterization of heterogeneous CHR samples to increase the role of social cognition in the prediction of psychosis. More longitudinal studies are needed to explore the course of social cognitive changes and their relations to outcomes. Such attempts may yield specific social cognitive domains and help to elucidate the pathophysiological changes that occur early in schizophrenia for improved targeted treatments.

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