

Social cognition and neurocognition as independent domains in psychosis

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Abstract

Patients with psychosis display alterations in social cognition as well as in the realm of neurocognition. It is unclear, however, to what degree these cognitive domains represent two separate dimensions of liability or the pleiotropic expression of a single deficit. The purpose of the present study was to investigate (i) to what extent alterations in social cognition represent an independent area of vulnerability to psychosis, separate from neurocognitive deficits and (ii) whether social cognition is one construct or can be divided into several subcomponents. Five social cognition and three neurocognitive tasks were completed by 186 participants with different levels of vulnerability for psychosis: 44 patients with psychotic disorder; 47 subjects at familial risk; 41 subjects at psychometric risk and 54 control subjects. The social cognition tasks covered important basic subcomponents of social cognition, i.e. mentalisation (or theory of mind), data gathering bias (jumping to conclusions), source monitoring and attribution style. Neurocognitive tasks assessed speed of information processing, inhibition, cognitive shifting and strategy-driven retrieval from semantic memory. The results of factor analysis suggested that neurocognition and social cognition are two separate areas of vulnerability in psychosis. Furthermore, the social cognition measures lacked significant overlap, suggesting a multidimensional construct. Cognitive liabilities to psychosis are manifold, and include key processes underlying basic person–environment interactions in daily life, independent of cognition quantified by neuropsychological tests.

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1. Introduction

Individuals with psychosis not only display neurocognitive deficits, but also experience alterations in the processing of social information. Alterations in areas of social cognition, including processing of emotions, social perception, mentalisation and social knowledge,

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may have important effects on symptom formation (Bentall et al., 1991; Frith and Corcoran, 1996; Garety et al., 2001) as well as on interpersonal relationships and community functioning (Vauth et al., 2004; Pinkham and Penn, 2006).

It has been hypothesised that social cognition may serve as a mediating link between neurocognition and community functioning (Vauth et al., 2004), suggesting that social cognition and neurocognition are related domains acting on the same pathway. Several studies have presented associations between social cognition and specific cognitive domains, such as attention (Addington and Addington, 1998), early visual processing (Sergi et al., 2006), executive functioning (Bryson et al., 1997) and verbal memory (Greig et al., 2004). For example, Vauth et al. (2004) analysed data from 133 inpatients with a diagnosis of schizophrenia showing that neurocognition accounted for 83% of the variance in social cognition. However, there is also evidence that the domains of social cognition and neurocognition are not overlapping, including evidence pointing to separate neural pathways for neurocognition and social cognition (Adolphs, 2001; Pinkham et al., 2003). One study, analysing data pertaining to 100 outpatients, showed that a two-factor model, representing social cognition and neurocognition, fitted the data better than a one-factor model (Sergi et al., 2007). In conclusion, it remains unclear whether these cognitive domains represent two separate areas of liability or whether there is one overlapping factor underlying both deficiencies.

Part of the reason for the diversity of findings is likely related to the fact that studies generally treat social cognition as a unidimensional construct, represented by, for example, a single measure of emotion recognition. However, social cognition refers to the ability to construct representations about others, oneself, and relations between others and oneself (Adolphs, 2006). It includes an array of abilities, such as theory of mind, emotional processing, emotion perception, source monitoring, styles of attribution and data gathering. In the current study, therefore, not a single but five measures of social cognition, covering the areas of mentalisation, data gathering bias, source monitoring and attributional style, were examined to address two questions. The first question was to what degree the measures used to operationalise social cognition in psychosis represent a single construct or can be divided into several subcomponents. The second question was to what degree alterations in social cognition are associated with neurocognitive deficits, not only in patients with psychotic symptoms exposed to antipsychotic medication and other possible confounds, but also in individuals at risk.

2. Materials and methods

2.1. Subjects

The subjects were all participants of the 'Cognitive functioning in Psychosis' (CoP) study. This study consisted of four groups at variable risk for psychotic disorder: (i) patients with a lifetime history of a period of non-affective psychosis in clear consciousness, (ii) first degree relatives of patients with non-affective psychosis, (iii) persons scoring high (>75th percentile) on the positive dimension of psychosis-proneness measured by the Community Assessment of Psychic Experience (CAPE; (Stefanis et al., 2002), a self-report trait questionnaire to assess dimensions of the subclinical psychosis phenotype, validated in previously (Konings et al., 2006) and (iv) 'healthy controls' i.e. participants scoring in the average range on the CAPE positive psychosis dimension (i.e. between 40th and 60th percentile). All participants were between the ages of 18 to 55 years, fluent in Dutch, and without a history of neurological disorders such as epilepsy and concussion with loss of consciousness. All participants signed an informed consent conforming to the local ethic's committee guidelines.

Patients were recruited from the catchment areas of the Community Mental Health Centre (source population: 350 000) and Psychiatric Hospital in the South of the Netherlands. Inclusion criteria for patients were the lifetime prevalence of a period of psychosis of at least 2 weeks in clear consciousness according to the RDC (Research Diagnosis Criteria: (Spitzer et al., 1978) and being sufficiently stable to allow psychological testing.

Relatives were all free from a lifetime history of psychosis. This group was sampled through participating patients or through associations for relatives of patients with psychotic symptoms. Subjects with average and high levels of psychotic experiences were recruited from an earlier longitudinal family study in the general population conducted in the city of Sittard (Continuum of Mental Disorders study, COMED; (Hanssen et al., 2003)). All participants of the COMED study completed the CAPE. The subjects with mean and a high score on the CAPE positive psychosis dimensions were invited to participate in the CoP study.

The present study included 44 patients with psychosis, 47 non-psychotic first degree relatives, 41 subjects with a high level of psychotic experiences and 54 healthy controls with an average level of psychotic experiences.

All patients were screened for symptoms listed in the operational Criteria Checklist for Psychotic Disorder (OCCPI; (McGuffin and Farmer, 2001)). Where

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