Cluster B personality symptoms in persons at genetic risk for schizophrenia are associated with social competence and activation of the right temporo-parietal junction during emotion processing

Micaela Giuliana Goldschmidt a,b, Mirta Fabiana Villarreal a,c,1, Delfina de Achával a,b,c, Lucas Javier Drucaroff a,b, Elsa Yolanda Costanzo a,b, Mariana Nair Castro a,b,c, Jaime Pahissa a, Joan Camprodon f, Charles Nemeroff e, Salvador Martín Guinjoan a,b,c,d,e,1

a FLENI Cognitive Neurology and Psychiatry, Buenos Aires, Argentina  
b Department of Mental Health (FLENI Teaching Unit), University of Buenos Aires School of Medicine, Buenos Aires, Argentina  
c Argentine National Council of Scientific and Technological Research (CONICET), Mendoza, Argentina  
d Department of Neuropsychology, University of Buenos Aires School of Psychology, Buenos Aires, Argentina  
e Department of Psychiatry and Behavioral Sciences, Leonard M. Miller School of Medicine, University of Miami, Coral Gables, FL, United States  
f Laboratory for Neuropsychiatry & Neuromodulation, Massachusetts General Hospital, Boston, MA, United States

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Personality disorders are common in nonpsychotic siblings of patients with schizophrenia, and some personality traits in this group may be associated with an increased risk for full-blown psychosis. We sought to establish if faulty right-hemisphere activation induced by social cognitive tasks, as previously described in patients with schizophrenia, is associated with specific personality symptoms in their unaffected siblings. We observed that cluster B personality symptoms in this group were inversely related to activation in the right temporo parietal junction (rTPJ, a structure critical in social cognitive processing) in response to a basic emotion processing task and also to social competence, whereas in contrast to our initial hypothesis, cluster A traits were not associated with right hemisphere activation during emotion processing or with social competence. These findings suggest the existence of clinical traits in at-risk individuals which share a common neurobiological substrate with schizophrenia, in regards to social performance.

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1. Introduction

Early epidemiological work on schizophrenia demonstrated that close biological relatives of patients display higher than expected rates of “subsyndromal” disease, in part, in the form of personality disorders (Kety et al., 1971, 1994). This has been confirmed in more recent samples, which found an excess of cluster A personality traits in close relatives of psychotic patients (Braff, 1981; Dickey et al., 1999; Kendler et al., 1993). The presence of other personality traits, particularly cluster B, has also been described (Hogg et al., 1990; Lysaker et al., 2004; Schultz-Z-Lutter et al., 2012). Cluster A traits result in an “odd” or “eccentric” personality pattern, including suspiciousness, eccentric thinking, or even peculiar perceptual experiences, whereas cluster B traits are significant for “dramatic” manifestations such as attention-seeking behavior, deceitfulness, impulsivity and even self-directed aggression (American Psychiatric Association, 2000; Kendler et al., 2008). In fact, some data suggest that genetic risk for personality traits do not necessarily overlap with DSM-IV categories, thus suggesting that genetic risk for schizophrenia may in turn result in diverse personality traits (Kendler et al., 2008). In the last decade, several groups have attempted to define prodromal traits predictive of future conversion to schizophrenia in at-risk subjects, especially after demonstration that early intervention shortening the duration of active symptoms improves the ominous prognosis of schizophrenia (Klosterkotter et al., 2011). Most efforts in this direction have involved either the definition of early or subsyndromal positive manifestations of psychosis, or neuropsychological deficits (Klosterkotter et al., 2011; Stanford et al., 2011). However, the predictive ability of these manifestations has been relatively modest even in large samples, ranging between 13% and 50% for transition to a psychotic episode, and with substantial variance even in the same center. Ideally, putative clinical and neuropsychological predictors could be complemented with neurobiological predictors.
as exemplified by the model predicting conversion from amnestic mild cognitive impairment to Alzheimer's dementia (Westman et al., 2012).

We recently described a specific failure in activation of right-hemisphere structures concerned with social cognition in patients with schizophrenia and their nonpsychotic siblings (de Achaval et al., 2012). In the present study we sought to establish if specific personality traits in that sample are associated with brain activation abnormalities characteristic of the full-blown syndrome. Available studies describe cluster A personality disorders and traits as clinically and biologically similar to schizophrenia (e.g., Tarbox and Pogue-Geile, 2011). In addition, recent work suggests that cluster A schizophrenia traits in patients at risk for psychosis are significant predictors of conversion, thus underscoring the relationship this symptom dimension exhibits with full-blown psychosis (Schultze-Lutter et al., 2012). This personality trait dimension involves persistent deficits in social functioning akin to those seen in schizophrenia. This led us to the hypothesis that cluster A traits account for shared neurobiological alterations underlying social deficits, between siblings discordant for schizophrenia, as previously demonstrated (de Achaval et al., 2012). In the present study, we sought to establish if specific personality traits in the nonpsychotic siblings of schizophrenia patients in that sample are associated with brain activation abnormalities characteristic of the full-blown syndrome. We predicted that cluster A traits would be associated with both a deficit in social functioning and a failure to recruit right-hemisphere structures concerned with social cognition (i.e., inferior frontal gyrus, and superior temporal sulcus/temporoparietal junction).

Based upon previous data, we explored the relationship between brain activation during social cognitive tasks and personality traits in three areas, namely the temporoparietal junction (TPJ), and the inferior (IFG) and middle (MFC) frontal gyri. Among these, different studies have assigned a critical role to the TPJ, especially on the right hemisphere, for the processing of social cognitive information, both verbal and nonverbal (Völlm et al., 2006; Decety and Lamm, 2007; Morishima et al., 2012; Santiesteban et al., 2012). Our group and others have recently described deficits in activation of the rTPJ in patients with schizophrenia, suggesting this is a finding characteristic of the disease (Das et al., 2012; de Achaval et al., 2012), and thus probably related to their deficits in social function. IFG and MFC have also been implicated in different aspects of emotion processing and empathy and theory of mind (Shamay and Tsoor, 2011; Bereczkei et al., 2013), along with structural alterations in schizophrenia (Kikinis et al., 2010; Yang et al., 2010), and displayed deficits of activation in the right hemisphere in a previous study (de Achaval et al., 2012).

On the basis of these observations, we predicted that cluster A traits would be associated with a failure to recruit right-hemisphere structures concerned with social cognition (i.e., inferior frontal gyrus, superior temporal sulcus/temporoparietal junction). Moreover, we expected that such neural activity abnormalities would be related to actual social competence deficits in persons who are at heightened genetic risk for schizophrenia. To test these hypotheses, we employed a functional magnetic resonance imaging (fMRI) paradigm of identification of basic emotions in faces, and a recently developed test of social competence in schizophrenia, the test of adaptive behavior in schizophrenia (TABS) (Velligan et al., 2007).
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