



Neuropsychological functioning and social anhedonia: Results from a community high-risk study

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Abstract

Social anhedonia has shown promise as a vulnerability marker for schizophrenia-spectrum pathology. Validity data have come, in part, from findings indicating that cognitive deficits occurring in schizophrenia are also evident in individuals with elevated levels of social anhedonia. However, prior research on this topic has been limited because it has been based almost exclusively on the study of selective samples of college students. The present article reports baseline findings of neuropsychological functioning in social anhedonics and controls from a representative community sample. Data on a wide array of neuropsychological abilities from 18–19 year-old participants with ($n=85$) vs. without ($n=87$) elevated levels of social anhedonia were analyzed. We hypothesized that, compared to controls, social anhedonics would show impairments in memory and sustained attention. Additionally, we sought to determine if more severe cognitive impairment in anhedonics was associated with greater schizophrenia-spectrum pathology and poorer overall functioning. Compared to controls, socially anhedonic participants performed more poorly on two visual-spatial memory tasks and a test of visual-spatial construction. The groups did not statistically differ on any of the other neuropsychological measures including general cognitive ability and sustained attention. Group differences were not the result of depression, bipolar or substance abuse disorders. Neuropsychological functioning showed little relationship to current clinical symptoms and functioning. Longitudinal assessment of these participants as they move through the risk period should provide important insights into the neuropsychological correlates of the schizophrenia prodrome.

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1. Introduction

In support of Meehl's (1962) conjecture that a deficit in the experience of social pleasure reflects a core feature of schizotypy, there is growing evidence for the validity of social anhedonia as a risk marker for schizophrenia-spectrum disorders. In patients with

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schizophrenia, social anhedonia is elevated, has been associated with poorer social functioning (Blanchard et al., 1998; Cohen et al., 2005) and seems to be particularly pronounced in patients with the deficit syndrome (Kirkpatrick and Buchanan, 1990). Elevated levels of social anhedonia have also been found in first-degree relatives of patients with schizophrenia (Kendler et al., 1996), supporting its potential role as an indicator of genetic risk. Moreover, baseline elevations in social anhedonia have been associated with the emergence of schizophrenia-spectrum disorders in longitudinal psychometric “high-risk” paradigms using college student participants (e.g., Gooding et al., 2005b; Kwapil, 1998). Finally, cross-sectional studies have found that socially anhedonic participants tend to evidence a number of neuropsychological and physiological abnormalities that are consistent with those seen in schizophrenia, albeit in attenuated form (Gooding et al., 2005a; Gooding and Tallent, 2003).

Investigations into the neuropsychological correlates of social anhedonia have yielded compelling support for the construct validity of social anhedonia as a putative risk marker for schizophrenia. Neuropsychological deficits are prominent in patients with schizophrenia (Heinrichs and Zakzanis, 1998), discordant dizygotic twins of patients (Pardo et al., 2000), and relatives of patients (Sitskoorn et al., 2004), and are considered to be endophenotypic indicators (Gottesman and Gould, 2003) of schizophrenia vulnerability (e.g., Cornblatt and Keilp, 1994; Nuechterlein et al., 1994). Similarly, individuals with elevated levels of social anhedonia have demonstrated a number of neuropsychological deficits, including impairments in visual–spatial working memory (Gooding and Tallent, 2003; Tallent and Gooding, 1999), visual–spatial construction (Gooding and Braun, 2004), visual–spatial delayed memory (Gooding and Braun, 2004), sustained visual attention (Gooding et al., 2006) and executive functioning (Gooding et al., 1999; Tallent and Gooding, 1999 but see Barrantes-Vidal et al., 2003) compared to non-anhedonic controls. Moreover, individuals with social anhedonia have shown an attenuated left-field perceptual bias during a chimeric emotion perception test (Luh and Gooding, 1999), providing evidence of attenuated right hemisphere activity in response to visual stimuli. However, other studies of social

anhedonia have shown normative performance in overall cognitive functioning (Gooding et al., 1999, 2001), motor functioning (Tallent and Gooding, 1999), verbal memory (LaPorte et al., 1994) and brief concentration/attention (Barrantes-Vidal et al., 2003; Tallent and Gooding, 1999), suggesting that the neuropsychological liabilities associated with social anhedonia may reflect abnormalities in relatively specific neuropsychological processes.

Although highly informative, prior research regarding neuropsychological functioning in social anhedonia has been limited by the study of non-representative college samples. Consider that these samples have typically been composed of Caucasian (e.g., Gooding and Tallent, 2003) college student participants with above average intelligence (e.g., Gooding and Braun, 2004) from select universities. Moreover, many prior studies investigating the neuropsychological correlates of social anhedonia have excluded all participants with a history of Axis I mood or substance use disorders (e.g., Gooding and Tallent, 2003; Gooding et al., 1999). Given that low IQ scores have been identified as a risk factor for schizophrenia in some studies (David et al., 1997), and that mood and substance use disorders are often present in the schizophrenia prodrome (Erlenmeyer-Kimling et al., 1997), concerns are raised regarding the generalizability of findings to the general population. The Maryland Longitudinal Study of Schizotypy (MLSS; Blanchard et al., submitted for publication; Collins et al., 2005) was designed to address these concerns by applying the psychometric high-risk paradigm to a representative community sample. The present study reports findings on the neuropsychological functioning of the MLSS participants.

It is also important to note that there is considerable variability in clinical outcomes across individuals who are considered “vulnerable” for schizophrenia-spectrum disorders as identified by social anhedonia (Gooding and Tallent, 2003; Gooding et al., 1999). It stands to reason that neuropsychological functioning may be important in understanding this variability, possibly by playing a role in potentiating the expression of the illness. In support of these explanations, neuropsychological deficits have been associated with more severe psychotic symptomatology in “at-risk” individuals more generally (Cornblatt et al.,

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