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Neuropsychological functioning and social anhedonia: Three-year follow-up data from a longitudinal community high risk study

Alex S. Cohen [a], Shannon M. Couture [b], Jack J. Blanchard [c,*]

[a] Louisiana State University, Department of Psychology, United States
[b] University of Southern California, Department of Psychology, United States
[c] University of Maryland, College Park, Department of Psychology, MD 20742 4411, United States

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ABSTRACT

Social anhedonia is a promising vulnerability marker for schizophrenia-spectrum pathology. Prior research has demonstrated that individuals with psychometrically-defined social anhedonia show a range of "schizophrenia-like" neurocognitive abnormalities. However, this research is limited in that it is based largely on the study of college students. The present article reports findings from a longitudinal study of social anhedonia recruited from a community sample. As part of this study, a neurocognitive battery was administered at baseline and at three-year follow-up sessions to participants with ($n = 78$) versus without ($n = 77$) social anhedonia. Additional measures of global functioning and schizotypal, schizoid and paranoid schizophrenia-spectrum symptoms were also administered. Across groups, subjects showed significant improvement in neurocognitive functioning over time. Compared to controls, at follow-up, individuals with social anhedonia showed significantly poorer attentional vigilance and simple processing speed, but failed to evidence impairments in immediate or delayed verbal memory, immediate or delayed visual memory, visual or verbal working memory, olfaction or executive abilities. At follow-up, within the social anhedonia group, schizoid (and to a lesser extent, schizotypal) symptom severity was associated with a range of neurocognitive impairments. Neurocognitive impairments were generally not associated with paranoid symptoms or global functioning. Baseline neurocognitive performance was not significantly predictive of follow-up symptom severity or functioning. Collectively, these findings suggest that neurocognitive dysfunctions only characterize a subset of individuals with social anhedonia.

1. Introduction

Social anhedonia, defined in terms of an inability to experience pleasure from social interactions, may be a risk marker for schizophrenia-spectrum disorders. Social anhedonia appears to be modestly heritable (Cohen et al., 2010; Kendler et al., 1996), is associated with reduced functioning (Blanchard et al., 2011), and has been associated with the emergence of schizophrenia-spectrum disorders in longitudinal questionnaire-based psychometric "high risk" paradigms using college student participants (e.g., Gooding et al., 2005; Kwapil, 1998). Cross-sectional studies have also found that socially anhedonic individuals evidence a number of "schizophrenia-like" neurocognitive abnormalities, albeit in attenuated form. For example, individuals with elevated levels of social anhedonia have demonstrated impairments in visual-spatial working memory (Gooding and Tallent, 2003, 2004; Tallent and Gooding, 1999), visual-spatial construction (Gooding and Braun, 2004), visual-spatial delayed memory (Gooding and Tallent, 2004), sustained visual attention (Gooding et al., 2006) and executive functioning (Tallent and Gooding, 1999) compared to non-anhedonic controls. Given the importance of neurocognitive abnormalities for understanding schizophrenia (Green, 1996) and schizotypy (e.g., Gooding and Tallent, 2003, 2004; Tallent and Gooding, 1999) more generally, and the potential use of neurocognitive measures as an indicator of schizophrenia vulnerability (Gur et al., 2007), there is considerable merit to clarifying the relationship between neurocognitive impairments and social anhedonia.

A significant limitation of much prior research on neurocognitive functioning in individuals with social anhedonia is the reliance on non-representative college samples. Consider that these samples are typically composed of Caucasian college students from universities with above average intelligence (e.g., Gooding and Tallent, 2003). The study of samples that are college-educated may limit our

* Corresponding author. Tel.: +1 301 405 8438; fax: +1 301 405 0367.
E-mail addresses: jblancha@umd.edu, acohen@lsu.edu (J.J. Blanchard).

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understanding of cognitive deficits in social anhedonia. The Maryland Longitudinal Study of Schizotypy (MLSS; Blanchard et al., 2011) was designed to address these concerns by applying the psychometric high risk paradigm to a representative community sample. In a prior article, we examined baseline differences in neurocognitive functioning across a wide array of attentional, memory, visual-spatial and language domains between individuals with social anhedonia and controls (Cohen et al., 2006). Our findings suggested that individuals with social anhedonia performed significantly worse than controls on tests of visual-spatial working memory, visual delayed memory and visual-constructional processing, but not on tests of verbal working memory, immediate or delayed memory, attentional vigilance or vocabulary ability. Moreover, neurocognitive performance was not significantly associated with functioning or severity of schizoid, schizotypal or paranoid symptoms, suggesting that neurocognitive abnormalities had not manifested in poorer clinical presentation at baseline. Given that these subjects were each 18-years old when they were recruited, and thus just entering the window of risk for onset of schizophrenia-spectrum disorders, it is important to understand how these neurocognitive impairments potentially changed over time.

The present study reports data from a three-year follow-up assessment from the MLSS. At the follow-up assessment, we repeated administration of the neurocognitive measures from the baseline assessment. This allowed us to determine the extent to which performance changed as individuals progressed through the risk period for schizophrenia-spectrum pathology. The longitudinal design also allowed us to examine whether baseline individual differences in neurocognitive performance were predictive of clinical outcomes at follow-up. We also expanded the scope of neurocognitive measures to include those tapping processing speed, executive functions and olfaction identification abilities – abilities not assessed at baseline but thought to be associated with schizophrenia vulnerability (e.g., Gooding et al., 1999). Olfaction identification is a particularly important neurocognitive ability to examine given recent claims that it reflects a valid vulnerability marker of schizophrenia-spectrum pathology (Turetsky et al., 2009). Finally, we administered measures of functioning and schizophrenia-spectrum symptoms at the follow-up assessment, which allowed us to evaluate whether neurocognitive impairment was related to functioning and schizophrenia-spectrum symptoms at the three-year follow-up assessment. For more detailed information on diagnosis, symptom severity and general functioning at follow-up in the social anhedonia group, the reader is referred to a companion article (Blanchard et al., 2011).

2. Methods

2.1. Participants

During the baseline screening, a cohort of 18-year old individuals ($N = 3508$) who lived within a 20-mile radius of the University of Maryland, College Park campus was identified using random-digit-dial methods. These individuals were each mailed a consent form and a screening self-report measure that included items from the Chapman schizotypy scales: the Revised-Social Anhedonia (RSAS; Eckblad et al., 1982), Perceptual Aberrations (PerAb; Chapman et al., 1978), Magical Ideation (MagId; Eckblad and Chapman, 1983) and Infrequency (Chapman and Chapman, 1976) scales. Response rate was high ($n = 2434$; 69%). Extreme scorers on the RSAS, selected as potential candidates for the social anhedonia group, were defined as either having a) an RSAS score 1.9 standard deviations above their respective gender and ethnicity-centered means (Chapman et al., 1994; Gooding et al., 2005; Kwapil, 1998), and/or b) having a Bayesian probabilities of belonging to the social anhedonia taxon greater or equal to .50 using Maximum Covariate Analysis taxometric method (MAXCOV; See Horan et al., 2004 for a review of this methodology). A cutoff score of 1.9 identifies less than 3% of the population, which is conservative based on the purported 10% prevalence of social anhedonia (e.g., Horan et al., 2004). Using these methods, 86 socially anhedonic individuals were identified who agreed to participate at baseline. Additionally, 89 controls were identified based on their scores on the RSAS, Perceptual Aberrations, and Magical Ideation scales being below .50 standard deviations from the gender and ethnicity derived group means, and Bayesian probabilities of taxon membership less than .50 using the MAXCOV taxometric procedure. There were no statistically significant differences in neurocognitive functioning between subjects identified using extreme scores versus taxon membership. The control participant group was matched to the social anhedonia group on gender and race variables. The follow-up assessment was conducted approximately three years after the completion of the baseline assessment. Retention from the baseline study was similarly excellent between the social anhedonia (91%) and control (87%) groups. For this study, neurocognitive data were available for 78 social anhedonic participants and 77 controls. The descriptive data for these groups are presented in Table 1.

2.2. Psychometric schizotypy measures

The RSAS (Eckblad et al., 1982), was used to measure social anhedonia. The RSAS is a 40 item true–false self-report questionnaire designed to measure deficits in social pleasure. The PerAb (Chapman et al., 1978) and the MagId (Eckblad and Chapman, 1983) scales were used to measure psychosis proneness to screen controls. The PerAb is a 35 item true–false self-report questionnaire designed to measure distortions in the perception of one's own body and environment. The MagId is a 30 item true–false self-report questionnaire designed to measure beliefs about causation that deviate from the norm. The RSAS, PerAb and MagId each have documented validity and reliability, and the reader is referred to their source documents, referenced above, for their psychometric properties.

Table 1
Follow-up demographic and clinical data for social anhedonics ($n = 78$) and controls ($n = 77$).[a]

	Social anhedonia	Controls
Gender		
% Female	45 (58%)	40 (52%)
Ethnicity		
Caucasian	35 (45%)	37 (48%)
African-American	37 (47%)	30 (39%)
Hispanic	4 (5%)	7 (9%)
Asian	1 (1%)	2 (3%)
Other/refused	1 (1%)	1 (1%)
Age	21.45 ± .50	21.52 ± .53
Epoch from baseline to follow-up (in days)	1021.37 ± 74.84	1082.78 ± 46.41
IPDE schizophrenia-spectrum scores[b]		
Schizotypal	.53 ± 1.00	.08 ± .48
Schizoid	.77 ± 1.18	.10 ± .53
Paranoid	.65 ± 1.04	.12 ± .54
Overall functioning		
Revised Social Anhedonia Scale[b]	15.13 ± 6.99	6.58 ± 3.93
Global Assessment of Functioning[b]	73.76 ± 13.62	83.30 ± 10.73
Treatment history:[b]		
Mood disorder diagnosis	34 (44%)	16 (21%)
Substance abuse diagnosis	18 (23%)	21 (27%)
Psychosis diagnosis	1 (1%)	1 (1%)
Inpatient treatment[b]	7 (9%)	1 (1.3%)
Outpatient treatment[b]	28 (36%)	15 (20%)

[a] Three participants who met criteria for a lifetime psychotic disorder at baseline were excluded.
[b] Data from the follow-up assessment.