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
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.,
and neuropsychological impairment is one of the most robust predictors of functional outcome in patients with schizophrenia (Green et al., 2000). As yet however, the relationship between neuropsychological deficits and clinical characteristics in individuals with social anhedonia has received limited empirical attention. Thus, the secondary aim of this study was to examine the relationship between neuropsychological functioning, schizophrenia-spectrum symptomatology and general functioning levels in the socially anhedonic participants.

2. Methods

2.1. Participants

The general methodology of the MLSS will be briefly described here. For a fuller description of the MLSS methodology, see Collins et al. (2005) and Blanchard et al. (submitted for publication). A cohort of 18-year old individuals (N = 3498) who lived within a 20-mile radius of the University of Maryland, College Park campus was identified using random-digit-dial methods and mailed a consent form and a screening self-report measure that included items from the Chapman schizotypy scales, including 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. The majority of the prospective participants (n = 2434; 69%) returned their materials. Extreme scorers on the RSAS, identified by one of two methods, were selected as potential candidates for the social anhedonia group. The first method, as used in other high-risk paradigms (Chapman et al., 1994; Gooding et al., 2005b; Kwapil, 1998), involved identifying individuals with RSAS scores 1.9 standard deviations above the group mean, determined separately for each gender and minority vs. non-minority group. The second method involved using the Maximum Covariate Analysis taxometric method (MAXCOV; see Horan et al., 2004 for a review of this methodology). All individuals with Bayesian probabilities (of belonging to a social anhedonia taxon) greater or equal to .50 were included in the social anhedonia group. Using these methods, 86 socially anhedonic individuals were identified who agreed to participate in the present study. The social anhedonia group was not selected based on the PerAb or MagId scores.

Individuals were considered eligible for the control group if their scores on the RSAS, Perceptual Aberrations, and Magical Ideation scales were each below .50 standard deviations from the gender and race derived group means. Additionally, controls needed to have RSAS scores with a Bayesian probability of taxon membership less than .50 using the MAXCOV taxometric procedure. The control participant group was matched to the social anhedonia group on gender and race variables. Using these methods, 89 controls were identified who agreed to participate in the present study.

Three participants, one from the social anhedonia group and two from the non-social anhedonia group, were excluded from the present analyses because they met criteria for a lifetime psychotic disorder. The rationale for this decision was that the present study focused on neuropsychological functioning in individuals at-risk for psychotic disorders. Given that psychosis has shown robust relationships to global neuropsychological deficits (Heinrichs and Zakzanis, 1998), inclusion of these participants may have obscured the findings between social anhedonia and neuropsychological functioning. The demographic 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 mea-

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1 Fourteen cases from the social anhedonia group were identified through taxometric analysis, while 72 were identified using the gender and ethnicity derived norms. Twenty-one cases met both criteria suggesting there was some overlap in these methods. To investigate whether social anhedonia participants identified through these two methods differed in neuropsychological functioning, we compared their performance on each of the neuropsychological measures. None of the differences were statistically significant (all p values > .05), suggesting that there were no demonstrable differences in neuropsychological functioning between these two groups. Accordingly, the two groups were collapsed into a common social anhedonia group for the present project. For further discussion on these methods of identifying social anhedonia participants, the reader is referred to Blanchard et al. (submitted for publication).
Table 1: Demographic and clinical data for social anhedonics (soc. anhedonia; n=85) and controls (n=87) excluding three participants who met the criteria for a lifetime psychotic disorder.

<table>
<thead>
<tr>
<th>Demographic characteristics</th>
<th>Social anhedonia</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>36 (42%)</td>
<td>41 (47%)</td>
</tr>
<tr>
<td>Female</td>
<td>49 (58%)</td>
<td>46 (53%)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>38 (45%)</td>
<td>40 (46%)</td>
</tr>
<tr>
<td>African-American</td>
<td>40 (47%)</td>
<td>36 (41%)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>5 (6%)</td>
<td>7 (8%)</td>
</tr>
<tr>
<td>Asian</td>
<td>1 (1%)</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>Other/refused</td>
<td>1 (1%)</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>Level of education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Did not graduate high school</td>
<td>1 (1%)</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>High school grad., no college</td>
<td>25 (29%)</td>
<td>9 (10%)</td>
</tr>
<tr>
<td>High school grad., part college</td>
<td>59 (70%)</td>
<td>77 (89%)</td>
</tr>
<tr>
<td>Parental socio-economic index scores</td>
<td>41.71 ± 13.31</td>
<td>41.67 ± 12.18</td>
</tr>
</tbody>
</table>

Schizophrenia-spectrum measures

- Schizotypal: 1.22 ± 1.52 .33 ± 0.86
- Schizoid: 1.36 ± 1.84 .28 ± 0.71
- Paranoid: 1.24 ± 1.84 .47 ± 1.02

Overall functioning

Global assessment of functioning scores: 71.54 ± 16.15 81.86 ± 12.38

Lifetime Axis I disorders:

- Mood disorder: 26 (31%) 7 (8%)
- Alcohol abuse disorder: 9 (11%) 14 (16%)
- Substance abuse d/o: 8 (9%) 13 (15%)
- History of any lifetime Axis I disorder: 31 (37%) 21 (24%)

a Increasing scores reflect higher socio-economic status, b increasing scores reflect more severe symptomatology, and c increasing scores reflect better functioning.

The MagId is a 30 item true–false self-report questionnaire designed to measure beliefs about causation that deviate from the norm, and includes items such as “I have sometimes felt that strangers were reading my mind” (keyed true). 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.

The infrequency scale (Chapman and Chapman, 1976) was included to determine the extent to which participants’ responses were valid. The infrequency scale is a 17 true–false questionnaire including such items as “I visited Easter Island last year” (keyed true). As in other studies (e.g., Luh and Gooding, 1999), individuals who endorsed three or more infrequency items were excluded from participation in this study.

2.3. Diagnostic interviews

Participants were administered a series of semi-structured diagnostic interviews during their baseline assessment. The mood, psychosis, and substance abuse modules from the Structured Clinical Interview for the Diagnostic and Statistical Manual for Mental Disorders, Fourth edition (American Psychiatric Association (APA), 1994) were used to determine Axis I diagnoses. Schizophrenia-spectrum symptomatology was determined using the schizoid, schizotypal and paranoid personality disorder modules from the International Personality Disorders Examination (Loranger et al., 1994). The IPDE was selected for use in this study because it yields both categorical and dimensional ratings of Axis II disorders, and has been used in prior studies of psychosis-proneness (Chapman et al., 1994). Diagnostic interviews were administered by doctoral level graduate students who had received rigorous training in both SCID and IPDE administration. Supervision was provided by the principle investigator (J. Blanchard) who has extensive experience with the SCID and IPDE administration.

Each interview was video-taped and reviewed by a pair of advanced level graduate students. Cases were then discussed during monthly case conferences until consensus diagnoses had been reached between the case conference group members, which included the principle investigator (J. Blanchard) and at least two doctoral level graduate students who reviewed the
videotaped interviews. The participants, interviewers and consensus judges were each blind to the participants’ group classification. As noted previously, one participant from the social anhedonia and two from the non-anhedonia group met the criteria for psychotic disorders and were excluded from this study. The descriptive data for lifetime diagnoses are presented in Table 1.

2.4. Neuropsychological measures

Informed by prior investigations of neuropsychological functioning in schizophrenia and individuals at-risk for schizophrenia more generally (e.g., Heinrichs and Zakzanis, 1998; Lyons et al., 1995; Park et al., 1995), a battery of tests was selected which tapped into verbal and nonverbal memory (immediate and delayed), working memory, sustained attention, and general cognitive abilities. Memory tests included: the total recall scores from the Logical Memory I and II tests (Wechsler, 1997b) as measures of immediate and delayed verbal memory respectively; and the total correct scores from the Visual Reproduction I and II tests (Wechsler, 1997a) as measures of immediate and delayed visual–spatial memory respectively. Working memory was assessed with the following tests: the total score from the Digit Span test (Wechsler, 1981) as a measure of immediate memory ability, the total correct score from the Spatial Span test (Wechsler, 1997a) as a measure of visual–spatial attention/working memory, and the total correct from the Letter Number sequencing test (Wechsler, 1997a) as a measure of verbal working memory. The Degraded Stimuli-Continuous Performance Test (DSCPT; Nuechterlein and Asarnow, 1992), a measure of sustained attention that is thought to reflect a vulnerability factor for psychotic symptoms, was also used. Finally, the total correct score from the Vocabulary and the Block Design from the WAIS-III (Wechsler, 1997a) tests were used as measures of verbal and visual–spatial performance ability and general cognitive ability. Validity and reliability data for each of these tests is provided in their respective administration manuals. Raw scores, as opposed to age corrected scores, were used for each of the Wechsler variables because all of the participants in this study were in the same age range, and because raw scores provided a greater range for the statistical analyses. Increasing scores for each of these variables reflects better performance. DSCPT data were missing for two of the social anhedonia participants due to equipment problems, and Delayed Visual Reproduction data was missing for one control participant.

2.5. General functioning

The participants’ general level of functioning was measured using the Global Assessment of Functioning (GAF; APA, 1994) scores. GAF scores, based on a scale from 1 to 100, reflect a measure of an individual’s psychological, social and occupational functioning for the prior month. Increasing scores reflect better functioning. The team consensus approach, outlined above, was used to assign GAF scores for each case.

2.6. Socio-economic status

Socio-economic status for the participants’ father and mother was separately determined using the Hollingshead scale (Hollingshead, 1975). When both scores were available, the two scores were averaged together.

2.7. Statistical analyses

Analyses were conducted in three steps. First, we compared the social anhedonia and control participants in descriptive characteristics. Second, in order to determine whether the social anhedonia and control participant groups statistically differed in neuropsychological performance, a series of independent-sample t-tests were computed. Effect size values were also computed to help evaluate the extent to which these groups differed. Finally, in order to determine the extent to which neuropsychological performance was associated with clinical and functioning variables within the social anhedonia participants, bivariate correlations were computed between the neuropsychological test scores and the paranoid, schizoid and schizotypal dimensional scores from the IPDE and the GAF scores. The paranoid, schizoid and schizotypal dimensional scores from the IPDE were square-root transformed to compensate for excessive positive skew (skew > 1.5).
3. Results

3.1. Sample characteristics

The sample characteristics are described and analyzed more fully in Collins et al. (2005) and Blanchard et al. (submitted for publication), but are briefly discussed here. The two groups were not significantly different in gender or ethnic composition or parental socio-economic status scores (all p values > .05). Significantly fewer anhedonia participants had enrolled in college ($\chi^2 [2, 172] = 9.89, p < .01$). The two groups were not statistically different with respect to the total number of alcohol and substance abuse disorders, but more participants from the social anhedonia group had lifetime mood disorders ($\chi^2 = 15.25, p < .00$) than the control group. Anhedonia participants had more severe schizotypal ($t[170]= 4.76, p < .00$), schizoid ($t[170]= 5.13, p < .00$) and paranoid ($t[170]= 3.38, p < .00$) spectrum-symptomatology, and poorer general functioning ($t[170]= 4.71, p < .00$) than controls.

3.2. Social anhedonia vs. control participants: neuropsychological functioning

Table 2 contains the means, standard deviations, $t$-values, and effect sizes for the between-group comparisons of neuropsychological functioning. Social anhedonia participants performed significantly poorer on the Visual Reproduction immediate and delayed recall tests, and the Block Design test. It is also noteworthy that, although not statistically different, group differences for the Logical Memory immediate recall, Letter Number sequencing and Digit Span tests each showed “small effect” size values, defined using Cohen’s (1987) guidelines for evaluating effect sizes. In sum, performances on three of the four tests with a prominent visual–spatial component were significantly different between the two groups. The two groups showed small, but nonsignificant differences on tests with working memory and verbal memory components. Group differences were negligible for one of the visual–spatial attention/working memory tests and for the delayed verbal memory, general verbal ability, and sustained attention components.

3.3. Group differences in neuropsychological functioning excluding participants with a history of Axis I pathology

The social anhedonia group had higher rates of Axis I disorders than the control group, so it is possible that group differences in neuropsychological functioning reflected differences in psychopathology. In order to address this issue, group comparisons were recomputed excluding all participants with lifetime Axis I disorders. We adopted this conservative strategy because participants with lifetime Axis I pathology have largely been excluded from prior studies that have examined neuropsychological correlates of social anhedonia (e.g., Gooding and Tallent, 2003).

Table 2

<table>
<thead>
<tr>
<th>Neuropsychological test</th>
<th>Soc. anhedonia</th>
<th>Controls</th>
<th>$t$-value</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual Reproduction I (recall)</td>
<td>90.72 ± 11.80</td>
<td>93.75 ± 7.74</td>
<td>2.00*</td>
<td>0.31</td>
</tr>
<tr>
<td>Visual Reproduction II (recall)</td>
<td>78.98 ± 16.92</td>
<td>85.05 ± 13.88</td>
<td>2.58*</td>
<td>0.39</td>
</tr>
<tr>
<td>Block Design</td>
<td>41.62 ± 12.18</td>
<td>45.48 ± 11.18</td>
<td>2.17*</td>
<td>0.33</td>
</tr>
<tr>
<td>Spatial Span total</td>
<td>17.27 ± 3.31</td>
<td>17.37 ± 3.36</td>
<td>.19</td>
<td>0.03</td>
</tr>
<tr>
<td>Logical Memory I (recall)</td>
<td>42.11 ± 10.47</td>
<td>44.31 ± 9.97</td>
<td>1.42</td>
<td>0.22</td>
</tr>
<tr>
<td>Logical Memory II (recall)</td>
<td>27.05 ± 7.32</td>
<td>28.03 ± 7.65</td>
<td>.87</td>
<td>0.13</td>
</tr>
<tr>
<td>Letter Number sequencing</td>
<td>11.00 ± 2.55</td>
<td>11.61 ± 2.63</td>
<td>1.54</td>
<td>0.24</td>
</tr>
<tr>
<td>Digit Span total</td>
<td>17.38 ± 4.00</td>
<td>18.33 ± 4.17</td>
<td>1.53</td>
<td>0.23</td>
</tr>
<tr>
<td>Vocabulary</td>
<td>44.53 ± 11.31</td>
<td>44.79 ± 10.46</td>
<td>.16</td>
<td>0.02</td>
</tr>
<tr>
<td>Degraded CPT (d prime)</td>
<td>2.09 ± 1.07</td>
<td>2.24 ± 0.94</td>
<td>.97</td>
<td>0.15</td>
</tr>
</tbody>
</table>

*p < .05. * Increasing neuropsychological test scores reflect better performance, b missing data for 1 control participant, and c missing data for 2 social anhedonia participants.
2003; Gooding et al., 1999). Nineteen females and 12 males from the social anhedonia group, and eight females and 13 males from the control group met criteria for a lifetime mood, alcohol abuse or substance abuse disorder. A Chi-square analysis indicated that the reduced social anhedonia and control groups without a history of Axis I disorders remained comparable in gender ($\chi^2=.05, \text{ns.}$) and ethnic ($\chi^2=1.84, \text{ns.}$) composition. Comparisons among the social anhedonia participants indicated that those with vs. without Axis I disorders did not significantly differ for gender ($\chi^2=.27, \text{ns.}$), ethnicity ($\chi^2=6.10, \text{ns.}$), or any of the neuropsychological variables (all $t$-values $<1.58$).

Table 3 contains the means, standard deviations, $t$-values, and effect sizes for the between-group comparisons using this subset of participants on neuropsychological functioning. As with the full-sample analyses, social anhedonia participants without Axis I disorders performed significantly poorer on the Visual Reproduction immediate and delayed recall tests, but the Block Design test $t$-value was no longer significant. “Small” effect size values were seen for the Visual Reproduction immediate and delayed recall, Block Design, Logical Memory immediate and delayed recall, Letter Number sequencing, and Digit Span tests. The effect size magnitude of the group differences from the DSCPT test was upgraded from “negligible” to “small”. Overall, there was little change in group comparisons of cognitive functioning when participants with lifetime histories of Axis I disorders were excluded from the analyses.

### Table 3
Neuropsychological test performance in social anhedonics (soc. anhedonia; $n=54$) and controls ($n=66$), excluding all participants who met the criteria for lifetime mood, psychotic or substance abuse disorder

<table>
<thead>
<tr>
<th>Neuropsychological tests$^a$</th>
<th>Soc. anhedonia</th>
<th>Controls</th>
<th>$t$-value</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual Reproduction I (recall)</td>
<td>89.41 ± 13.38</td>
<td>94.00 ± 7.22</td>
<td>2.40*</td>
<td>0.44</td>
</tr>
<tr>
<td>Visual Reproduction II (recall)$^b$</td>
<td>78.09 ± 17.84</td>
<td>84.58 ± 13.59</td>
<td>2.30*</td>
<td>0.43</td>
</tr>
<tr>
<td>Block Design</td>
<td>41.54 ± 12.19</td>
<td>44.44 ± 10.80</td>
<td>1.38</td>
<td>0.26</td>
</tr>
<tr>
<td>Spatial Span total</td>
<td>17.39 ± 3.03</td>
<td>17.55 ± 3.25</td>
<td>0.27</td>
<td>0.05</td>
</tr>
<tr>
<td>Logical Memory I (recall)</td>
<td>40.85 ± 9.38</td>
<td>44.14 ± 10.12</td>
<td>1.83</td>
<td>0.34</td>
</tr>
<tr>
<td>Logical Memory II (recall)</td>
<td>26.30 ± 6.79</td>
<td>27.95 ± 7.49</td>
<td>1.26</td>
<td>0.23</td>
</tr>
<tr>
<td>Letter Number sequencing</td>
<td>10.98 ± 2.52</td>
<td>11.64 ± 2.46</td>
<td>1.43</td>
<td>0.30</td>
</tr>
<tr>
<td>Digit Span total</td>
<td>17.17 ± 3.94</td>
<td>18.18 ± 3.72</td>
<td>1.45</td>
<td>0.26</td>
</tr>
<tr>
<td>Vocabulary</td>
<td>43.43 ± 11.11</td>
<td>44.88 ± 9.92</td>
<td>.76</td>
<td>0.14</td>
</tr>
<tr>
<td>Degraded CPT (d prime)$^c$</td>
<td>1.96 ± .93</td>
<td>2.31 ± .99</td>
<td>1.98</td>
<td>0.37</td>
</tr>
</tbody>
</table>

$^a p < .05, \quad ^b$ missing data for 1 control participant, and $^c$ missing data for 2 social anhedonia participants.

3.4. Within the social anhedonia group: relationship between neuropsychological functioning and clinical and functioning measures

For space considerations, these results are presented in text as opposed to tabular format. Severity of paranoid-spectrum scores was associated with poorer visual–spatial working memory ability ($r=.29, \text{p}<.05$), but none of the other clinical or general functioning variables were significantly associated with neuropsychological performance (all $p$ values $>.05$). These findings suggest that, at baseline, neuropsychological liabilities had not manifested in a poorer clinical presentation within the anhedonia group.

4. Discussion

The present study is the first to examine the neuropsychological correlates of social anhedonia in a representative community sample of young adults. In contrast to prior studies of this kind, there was considerable diversity within our sample in terms of ethnicity, level of academic achievement and psychological health. We sought to examine the hypothesis that, compared to controls, participants with social anhedonia would evidence poorer neuropsychological performance in domains implicated in prior studies of schizophrenia and schizotypy. Additionally, we hypothesized that neuropsychological performance would be associated with greater clinical symptoms...
and worse overall functioning within the anhedonia group.

Results indicated that, consistent with other studies (e.g., Gooding and Braun, 2004; Luh and Gooding, 1999), participants with social anhedonia performed more poorly than community controls on tasks of immediate and delayed visual–spatial memory and visual–spatial construction. No other significant group differences in neuropsychological performance were observed, thus raising the possibility that impairment in visual–spatial abilities, which involve frontal and right hemisphere parietal lobe functions, reflects a differential deficit in certain individuals who are at-risk for schizophrenia-spectrum disorders. Although we had no a priori hypotheses regarding this differential deficit, the link between social anhedonia and these neuropsychological liabilities is consistent with other research on affectivity and social behavior. Evidence from the affective neuroscience literature suggests that the right inferior parietal and frontal regions are crucial for processing social-based emotions (e.g., Ruby and Decety, 2004), and that reduced right parietal lobe activation is associated with decreased “approach” social behaviors (e.g., Heilman, 1997; Heller, 1993). Accordingly, one might speculate that abnormalities in the frontal and right hemisphere parietal neurocircuitry are central in understanding social dysfunction in the schizophrenia prodrome.

With respect to cognitive correlates within social anhedonic participants, neuropsychological functioning was largely unrelated to schizophrenia-spectrum symptoms or general functioning. It is important to note that while schizophrenia-spectrum symptomatology was elevated in the social anhedonia group, these symptoms tended to be relatively benign in severity. Participants were recruited at an age that is below the expected onset of manifest illness so it is possible that neuropsychological deficits will become more of a liability as schizophrenia-spectrum symptomatology begins to emerge. It could also be the case that neuropsychological deficits will become more severe as clinical state worsens. While there is little empirical evidence that patients as a group show “neurodegeneration” as they progress through the early phases of the illness (see Kurtz, 2005 for a review), changes in the severity of negative symptoms in patients have been associated with a worsening of cognitive functioning in longitudinal studies of schizophrenia patients (Gold et al., 1999; Stirling et al., 2003).

Limitations regarding the neuropsychological tests warrant mention. First, it is possible that some of our neuropsychological tests were not ideal for detecting group differences. Visual–spatial working memory (as measured by the Spatial Span task) was not more impaired in the social anhedonia group compared to controls. In light of findings that individuals with social anhedonia show worse performance on computerized delayed match-to-sample visual–spatial tasks (Tallent and Gooding, 1999; Gooding and Tallent, 2003), our null finding may have been because the Spatial Span task lacks sufficient sensitivity. However, in the present study, visual–spatial working memory was the only task to be correlated with schizophrenia-spectrum symptomatology (i.e., paranoid dimensional scores), suggesting that visual–spatial working memory deficits reflect an important characteristic of the schizophrenia prodrome in at least some regards. Another unexpected finding was that individuals with social anhedonia showed relatively normal attentional vigilance ability. We used a degraded CPT which has shown promise in schizophrenia research (e.g., Nuechterlein et al., 1994), but has not been studied rigorously in at-risk populations. Other CPT tasks which have a working memory component, such as the CPT-IP (Cornblatt and Keilp, 1994), have shown discrimination between social anhedonics and controls (Gooding et al., 2006). It may be the case that working memory is an important aspect of functioning in psychometrically identified schizotypes.

A second limitation is that, although the present study employed a broad battery to measure neuropsychological functioning, several key neuropsychological abilities were not assessed. Executive functions, such as those measured by the Wisconsin Card Sorting Task, are particularly relevant because they are impaired in many patients with schizophrenia (Heinrichs and Zakzanis, 1998) and individuals thought to be at genetic risk for the disorder (Pardo et al., 2000; Sitskoorn et al., 2004). Another promising neuropsychological liability that may characterize individuals with social anhedonia involves olfactory discrimination ability. Olfactory discrimination deficits have been related to reduced affiliative behavior in non-human mammals (Baum, 2004), and have been
found to be impaired in schizophrenia patients with the deficit syndrome (Malaspina and Coleman, 2003). Longitudinal assessment of a broader range of neuropsychological abilities including executive functions and olfactory discrimination ability is currently underway for the MLSS participants, and will hopefully shed some light on these issues.

In summary, results from the baseline analyses of neuropsychological functioning from participants in this community sample provide further evidence of neuropsychological deficits in individuals with social anhedonia. Our findings also raise questions about whether social anhedonia is characterized by a differential deficit in visual–spatial abilities. Follow-up data from the longitudinal assessment of these participants as they enter the “window of risk” will hopefully provide further insights into the neuropsychology of social anhedonia, and improve efforts to identify individuals at-risk for developing schizophrenia-spectrum disorders.

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