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

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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) vs. 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.

Keywords

Social anhedonia; schizophrenia; schizotypy; prodrome; risk; neurocognitive; neuropsychological

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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, Emmerson, Mann, Forbes, & Blanchard, 2010; Kendler, Thacker, & Walsh, 1996), is associated with reduced functioning (Blanchard, Collins, Aghevli, Leung, & Cohen, 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, Tallent, & Matts, 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 & Tallent, 2004; Gooding & Tallent, 2003; Tallent & Gooding, 1999), visual spatial construction (Gooding & Braun, 2004), visual spatial delayed memory (Gooding & Tallent, 2004), sustained visual attention (Gooding, Matts, & Rollmann, 2006) and executive functioning (Tallent & Gooding, 1999) compared to non-anhedonic controls. Given the importance of neurocognitive abnormalities for understanding schizophrenia (Green, 1996) and schizotypy (e.g., Gooding & Tallent, 2004; Gooding & Tallent, 2003; Tallent & 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 & Tallent, 2003). The study of samples that are college-educated may limit our 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, Leung, Saperstein, & Blanchard, 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, Kwapil, & Tallent, 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, Hahn, Borgmann-Winter, & Moberg, 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, Chapman, Chapman, & Mishlove, 1982), Perceptual Aberrations (PerAb; Chapman & Chapman, 1978), Magical Ideation (MagId; Eckblad & Chapman, 1983) and Infrequency (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, Chapman, Kwapil, Eckblad, & Zinser, 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, Blanchard, Gangestad, & Kwapil, 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 et al., 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.

The Infrequency Scale (Chapman, 1976) was included to determine the extent to which participants' responses were valid. The infrequency scale is a 17 item true-false questionnaire. As in other studies (e.g., Luh & 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 and follow-up assessments. 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, 1994) were administered to evaluate 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). Dimensional scores, computed as a sum of the respective ratings from the schizoid, schizotypal and paranoid modules, were computed and examined in this study. Diagnostic interviews were administered by doctoral level graduate students who had received rigorous training in both SCID and IPDE administration. Supervision was provided by two doctoral psychologists (J. Blanchard & A. Cohen) with extensive experience in SCID and IPDE administration. Final diagnoses and ratings were 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 in our baseline study (Cohen et al., 2006), one participant from the social anhedonia and two from the control groups met criteria for psychotic disorders and were excluded from the studies. Individuals who developed psychotic symptoms after baseline were included in the present study. The rationale for this was that we were primarily interested in individuals at elevated risk for schizophrenia as opposed to those already meeting criteria for the disorder. The descriptive data for lifetime diagnoses are presented in Table 1.

2.4 Neuropsychological Measures

Measures from the baseline assessment, selected based on prior investigations of neuropsychological functioning in schizophrenia and individuals at-risk for schizophrenia more generally (Heinrichs & Zakzanis, 1998), were readministered during the follow-up session. Memory tests included: the total recall scores from the Logical Memory I and II tests (Wechsler, 1987; Wechsler, 1997) as measures of immediate and delayed verbal memory respectively and the total correct scores from the Visual Reproduction I and II tests (Wechsler, 1987) 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, 1987) as a measure of immediate memory ability, the total correct score from the Spatial Span test (Wechsler, 1987) as a measure of visual-spatial attention/working memory, and the total correct from the Letter Number Sequencing test (Wechsler, 1987) as a measure of verbal working memory. The Degraded Stimuli – Continuous Performance Test (DSCPT; Nuechterlein, 1992), a measure of sustained attention was also used. 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 provide greater variability. Increasing scores for each of these variables reflects better performance. The block design and vocabulary tests (Wechsler, 1997), which were administered as proxies for general cognitive ability at baseline, were not re-administered at follow-up.

We introduced three additional measures for the follow-up assessment. First, to expand the measures of executive functions, we included the total correct and number of perseverative

errors variables from the Wisconsin Card Sorting Task (WCST; Heaton & PAR Staff, 1999) and the total seconds to completion from the Trails B test (a.k.a. "complex" processing speed; Boll, 1981). As a measure of simple processing speed, we included total seconds to completion from the Trails A (Boll, 1981). Finally, we administered a measure of olfaction recognition (UPSIT; Doty, 1984). Missing data included: one control and one social anhedonic subject for the WCST, one control and two social anhedonics for the UPSIT, and one control and four social anehdonics for the DSCPT.

2.5 General Functioning

Participants' general level of functioning was measured using the Global Assessment of Functioning (GAF; American Psychiatric Association, 1994) scale. GAF scores, based on a scale from one 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 five steps. First, we compared the social anhedonia and control groups in descriptive characteristics to ensure there were no confounding variables worthy of consideration in the subsequent analyses. Second, we employed a series of time (i.e., baseline and follow-up) by group (i.e., social anhedonia versus controls) repeated-measure ANOVAs to evaluate neurocognitive performance over time across the two groups. Third, we compared the social anhedonia and control groups on performance for the measures that were only administered at follow-up, specifically those tapping executive, processing speed and olfaction abilities, using a series of t-tests. Fourth, in order to determine the extent to which neurocognitive performance was associated with symptom and functioning variables within the social anhedonia group, bivariate correlations were computed between the neurocognitive test scores and the paranoid, schizoid and schizotypal and GAF scores for the social anhedonia group only. Finally, we examined the relationship between baseline neurocognitive measures and follow-up schizotypal symptom and functioning scores. This analysis helped evaluate the predictive power of neurocognitive performance in terms of symptom severity and functioning. The paranoid, schizoid and schizotypal dimensional scores from the IPDE were square-root transformed to compensate for excessive positive skew (skew > 1.5). Unless otherwise noted, all variables are normally distributed and all statistical tests are two-tailed.

3. Results

3.1 Descriptive and clinical characteristics

Descriptive statistics were computed and compared between the social anhedonia and control groups for follow-up data (see Table 1). The groups were not statistically different in sex, ethnicity, age, time between baseline and follow-up assessments, or history of substance abuse or schizophrenia diagnoses (p's > .10). The social anhedonia group had significantly greater schizotypal ($t[153] = 3.54$, $p = .001$), schizoid ($t[153] = 4.51$, $p < .001$) and paranoid ($t[153] = 4.02$, $p < .001$) ratings, poorer functioning ($t[153] = 4.84$, $p < .001$), higher frequency of inpatient (χ^2 [1] = 4.66, $p = .03$) and outpatient (χ^2 [1] = 5.21, $p = .02$)

treatment history and history of major depressive disorder (χ^2 [1] = 9.23, p = .002). For more information regarding these variables, the author is referred to Blanchard et al. (2011).

3.2 Group differences in neurocognitive performance: baseline to follow-up

Table 2 contains the means, standard deviations, and test statistics for the social anhedonia and control groups for neurocognitive performance across baseline and follow-up sessions. Significant time effects indicated that, across groups, performance significantly improved across the three-year epoch in immediate and delayed verbal and visual memory, and for verbal working memory. Significant main effects for group, indicated that, compared to controls, the social anhedonia group was significantly poorer in delayed visual memory and attentional vigilance performance, but the groups did not differ on any of the other tasks. There were no significant group by time interactions. Our expectation that individuals with social anhedonia would show poorer neurocognitive functioning was generally not supported.

It is noteworthy that we reported baseline group differences in visual memory and attentional vigilance performance in our prior publication of baseline differences (Cohen et al., 2006), yet no baseline differences were observed in the present analyses of baseline performance (which only contains those individuals who completed the follow-up). This disparity is because the five individuals with social anhedonia lost to the follow-up had significantly poorer neurocognitive functioning on these tasks than those retained in the study (t's[83] = 2.44 and 2.96, p's = .02 and .004, respectively).

3.3 Group differences in neurocognitive task performance assessed at follow-up

Table 3 contains means, standard deviations and test statistics for those tests administered only at follow-up for the social anhedonia and control groups. The social anhedonia group was significantly slower on the processing speed task and significantly more accurate on the olfaction recognition test. In general, expected group differences were not seen with the additional neurocognitive tasks assessed at follow-up.

3.4 The relationship between global functioning, symptoms and neurocognitive performance (within the social anhedonia group) at follow-up

Correlations computed between the functioning, schizotypal, schizoid and paranoid symptom and neurocognitive performance scores are in Table 4. Increasing schizoid symptoms corresponded to poorer immediate and delayed verbal and visual memory, poorer olfaction recognition and slower processing speed for both simple (i.e., Trails A) and complex (i.e., Trails B) processing speed tasks. Severity of schizotypal symptoms was associated with poorer immediate verbal memory and olfaction recognition. Neither functioning nor paranoid symptoms were significantly associated with any neurocognitive performance variables. In sum, schizoid and, to a lesser extent, schizotypal, but not paranoid symptoms were associated with a range of neurocognitive impairments. Neurocognitive variables were not significantly related to functioning as hypothesized.

3.5 The relationship between baseline neurocognition and follow-up symptom and functioning measures

Correlations computed between the baseline neurocognitive measures and the follow-up schizotypal symptoms and GAF scores were then computed. Poorer visual-spatial working memory performance was associated with more severe follow-up paranoia symptoms (r[78] = −.28, p = .01). Beyond this, baseline neurocognition performance was not significantly associated with any of the schizotypal, paranoid or schizoid symptom scores (p's > .05). With respect to functioning, none of the baseline neurocognition scores were significantly

associated with any of the follow-up functioning scores in the expected direction. In sum, baseline neurocognition held little power in predicting follow-up schizotypal symptoms or functioning.

4. Discussion

This study examined neurocognitive functioning in individuals with psychometrically-defined social anhedonia and controls. There are four relatively unique features of this study. First, in contrast to most studies of this kind that recruit from college student populations, participants were recruited from a large metropolitan region. Second, due to the longitudinal design of this study and the excellent retention for study participants, we were able to examine potential changes in neurocognitive performance across a three-year epoch. Third, we employed a relatively large battery of standard neurocognitive instruments tapping a broad range of abilities. Finally, we employed measures of functioning and schizophrenia-spectrum pathology, thus offering insight into the correlates of neurocognitive impairments in individuals with social anhedonia.

In contrast to our expectations, there were very few neurocognitive impairments in the social anhedonia group at follow-up. Only two measures – one tapping attentional vigilance and the other simple processing speed, were significantly different between the groups at follow-up. In contrast, immediate and delayed memory across both verbal and visual domains, working memory, olfaction recognition and a range of executive functions were preserved in the social anhedonia group. These findings are inconsistent with what was observed at baseline, where the social anhedonia group demonstrated a broader range of visual memory impairments. The reasons for this seeming discrepancy reflect that the five subjects lost to follow-up tended to have poorer visual memory, and that the group differences (which were at a small effect size at baseline) narrowed over time. Our findings are inconsistent with many published studies on neurocognition in college students with social anhedonia (e.g., Gooding & Tallent, 2004), and the reasons for this disparity are unclear at the present time. It is worth noting that there are a number of null findings with respect to neurocognition in psychometrically-defined schizotypy more generally (e.g., Lenzenweger & Gold, 2000, Cohen, Iglesias & Minor, 2009) – so the present findings are not wholly inconsistent with the extant literature. For what it's worth, it is clear that the samples employed in the present study were much more representative of the general US population in terms of ethnicity, education, substance use/abuse and global cognitive ability (see Blanchard et al., 2011 and Cohen et al., 2006), though it is unclear if sample differences between this and prior social anhedonia studies may have contributed to the null findings. Regardless, care is recommended in the assumption of generalizability from college samples to more representative community populations.

The present data did not support the notion that neurocognitive performance was, for the most part, predictive of functioning. Interestingly, this finding is consistent with that reported by Gooding & Tallent (2005) in that baseline laboratory metrics were not predictive of symptom severity after a five year epoch. In that study, plexus visibility, smooth pursuit eye tracking and antisaccade task errors were the metrics of interest. However, our findings are inconsistent with what is seen in schizophrenia, as neurocognitive dysfunction has been shown to be predictive of functioning and illness severity over time (e.g., Milev, Ho, Arndt, & Andreasen, 2005). Clarifying why neurocognition and illness course are related in patients with schizophrenia, but not in individuals with social anhedonia is a potentially interesting future research line.

It is noteworthy that neurocognitive performance was relatively poor in some socially anhedonic individuals. At follow-up, within the social anhedonia group, severity of schizoid

symptoms was associated with poorer neurocognitive performance in a range of domains, including immediate and delayed verbal and visual memory, olfaction recognition and processing speed. Moreover, severity of schizotypal symptoms was associated with poorer performance on immediate verbal memory and olfaction recognition tests. This suggests that a subset of individuals with social anhedonia – those that are manifesting schizoid and schizotypal symptoms, show neurocognitive dysfunctions. It is curious that schizoid symptoms, as opposed to paranoid symptoms were associated with poorer neurocognitive performance. While the meaning of this finding is unclear at the present time, it echoes findings from the schizophrenia literature that patients with prominent negative symptoms have poorer neurocognitive functioning than patients with positive or disorganization symptoms (Cohen et al., 2007). Thus, schizoid symptoms may reflect a marker of neurocognitive pathology in individuals with social anhedonia. The nature of the relationship between schizotypal symptoms and neurocognitive deficits is unclear at the present time. Further examining the relationship between these variables, and whether they share pathophysiological substrata, reflects an important line for future research.

Methodological issues may have contributed to two of the major null findings in this study. First, in contrast to what is seen in the literature on schizophrenia (Green, 1996), global functioning was not significantly related to neurocognitive performance. This is an important finding because it suggests that, in individuals with social anhedonia, global functioning impairments do not manifest as a function of neurocognitive impairments. However, this null finding might reflect the measure of functioning employed not being particularly sensitive and reflecting an overly broad range of symptom, occupational and social domains. For this reason, examining the functional sequelae of social anhedonia as they independently manifest across a broad range of social, academic and occupational domains is potentially important for future research. Second, although the neurocognitive tests employed in this study are commonly used in clinical practice and schizophrenia research more generally, they may be insufficiently sensitive for detecting differences in at-risk individuals. It seems unlikely that insensitivity wholly accounts for the lack of group differences in neurocognitive functioning, as there was sufficient variability in performance across individuals with social anhedonia to be significantly correlated to schizoid and schizotypal symptoms. Nonetheless, further research, perhaps employing methods derived from cognitive sciences (MacDonald & Carter, 2002), would address this issue. Finally, while this study had excellent subject retention from the baseline to follow-up assessments, a handful of subjects from the social anhedonia group were lost due to attrition. It appeared to be the case that, at least in some respects, these individuals had relatively poor neurocognitive functioning. Moreover, it stands to reason that they had a higher risk of manifesting schizophrenia.

In summary, results from the three-year follow-up assessment of neurocognitive functioning from subjects in the MLSS provide little evidence of impairments in individuals with social anhedonia. It is noteworthy that subjects in this study were young adults and still progressing through the “window of risk”. Further longitudinal assessment of these individuals will hopefully provide further insights into the neurocognition of social anhedonia.

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Table 1

Follow-up demographic and clinical data for Social Anhedonics (n = 78) and Controls (n = 77)[1]

	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[2]		
Schizotypal	.53 ± 1.00	.08 ± .48
Schizoid	.77 ± 1.18	.10 ± .53
Paranoid	.65 ± 1.04	.12 ± .54
Overall Functioning		
Revised Social Anhedonia Scale scores[2]	15.13 ± 6.99	6.58 ± 3.93
Global Assessment of Functioning scores[2]	73.76 ± 13.62	83.30 ± 10.73
Treatment History[2]		
Mood disorder diagnosis	34 (44%)	16 (21%)
Substance abuse diagnosis	18 (23%)	21 (27%)
Psychosis diagnosis	1 (1%)	1 (1%)
Inpatient Treatment[2]	7 (9%)	1 (1.3%)
Outpatient Treatment[2]	28 (36%)	15 (20%)

[1]Three participants who met criteria for a lifetime psychotic disorder at baseline were excluded.

[2]Data from the follow-up assessment.

Table 2

Neurocognitive performance between baseline and three-year follow-up administrations for controls versus social anhedonia (S. A.) groups.

	Time	Control M±SD	Social Anhedonia M±SD	Time	F Between	Interaction	Time	d Between: Control	Between: S. A.
Immediate Verbal Memory	Baseline	44.31 ± 9.89	42.63 ± 10.29	21.51***	1.06	0.04	0.17	−0.35	−0.35
	Follow-up	47.84 ± 10.17	46.46 ± 11.60				0.13		
Delayed Verbal Memory	Baseline	27.88 ± 7.59	27.14 ± 7.24	51.81***	1.29	1.08	0.10	−0.53	−0.40
	Follow-up	31.84 ± 7.41	30.10 ± 7.49				0.23		
Immediate Visual Memory	Baseline	93.68 ± 7.95	91.56 ± 10.90	27.35***	1.89	0.55	0.22	−0.43	−0.49
	Follow-up	96.97 ± 7.38	95.95 ± 6.96				0.14		
Delayed Visual Memory	Baseline	84.70 ± 13.90	80.54 ± 16.06	20.67***	4.63*	0.01	0.28	−0.41	−0.36
	Follow-up	90.29 ± 13.35	85.94 ± 14.24				0.32		
Verbal Working Memory (LN Seq)	Baseline	11.66 ± 2.74	11.05 ± 2.56	3.24	1.27	1.02	0.23	−0.05	−0.18
	Follow-up	11.79 ± 2.73	11.51 ± 2.63				0.10		
Visual Working Memory (Spatial Span)	Baseline	17.52 ± 3.30	17.31 ± 3.12	0.27	1.38	2.94	0.07	−0.08	0.14
	Follow-up	17.77 ± 3.31	16.85 ± 3.33				0.28		
Verbal Working Memory (Digit Span)	Baseline	18.30 ± 4.24	17.45 ± 3.88	12.54***	2.22	0.07	0.21	−0.21	−0.19
	Follow-up	19.19 ± 4.16	18.22 ± 4.06				0.24		
Attentional Vigilance: d prime	Baseline	2.30 ± 0.91	2.03 ± 0.95	0.37	4.77*	0.91	0.26	−0.02	0.13
	Follow-up	2.32 ± 0.95	1.93 ± 1.09				0.38		

* = p < .05,

*** = p < .001

Table 3

Neurocognitive measures only available at follow-up

	Control		Social Anhedonia			
	M	**SD**	**M**	**SD**	**t**	**d**
WCST (Total Correct)	68.92	9.32	69.51	8.57	.41	.07
WCST (Perseverative Errors)	9.22	10.73	10.50	8.65	.82	.13
Olfaction Functioning	33.26	4.05	34.66	3.37	2.35*	.38
Simple Processing Speed (Trails A)	25.14	9.63	28.85	12.14	2.13*	.34
Complex Processing Speed (Trails B)	56.52	19.14	62.96	30.07	1.61	.26

* = $p < .05$

Table 4

Bivariate correlations between clinical and neurocognitive functioning variables at follow-up, within the social anhedonia group.

	Functioning	IPDE Schizophrenia−Spectrum Traits		
	GAF	Schizotypy	Schizoid	Paranoia
Immediate Verbal Memory	.22	−.24*	−.33**	−.14
Delayed Verbal Memory	.12	−.18	−.29*	−.10
Immediate Visual Memory	−.04	.01	−.25*	−.01
Delayed Visual Memory	.02	−.02	−.22*	.00
Verbal Working Memory (LN Seq)	−.17	−.12	.00	.11
Visual Working Memory (Spatial Span)	.16	−.19	−.19	−.21
Verbal Working Memory (Digit Span)	−.01	−.11	−.11	.04
WCST (Total Correct)	−.08	−.02	.03	−.03
WCST (Perseverative Errors)	.04	.00	−.09	.02
Olfaction Functioning	.11	−.24*	−.25*	.08
Simple Processing Speed (Trails A)	−.08	.18	.25*	.08
Complex Processing Speed (Trails B)	−.11	.18	.24*	−.02
Attentional Vigilance: d prime	−.04	−.02	−.18	−.04

Note – Schizophrenia-spectrum scores square root transformed to compensate for excessive skew

* = p< .05,

** = p< .01