Schizotypal, schizoid and paranoid characteristics in the biological parents of social anhedonics

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ABSTRACT

Mounting evidence suggests that social anhedonia may be a marker of genetic liability for schizophrenia-spectrum pathology. To examine this hypothesis, we conducted a study of severity of schizotypal, schizoid and paranoid pathology (i.e., Cluster A personality disorders) in the biological parents of individuals with high levels of social anhedonia and healthy controls. Eighty-six individuals with social anhedonia, 89 healthy controls and their biological parents were recruited from a large community. Structured clinical interviews were conducted to obtain Cluster A diagnoses and symptom ratings for parents. The biological parents of socially anhedonic probands had elevated rates of Cluster A disorders (24%) compared with the parents of control probands (12%). Post hoc analyses revealed that these group differences were the result of elevated rates of diagnoses in the fathers of social anhedonic probands, but not the mothers. This finding was replicated when Cluster A symptoms were examined dimensionally. These findings are consistent with the hypothesis that social anhedonia is a promising indicator of the genetic vulnerability to schizophrenia-spectrum pathology. The unexpected findings of elevated pathology in fathers, but not mothers of socially anhedonic probands, require further exploration.

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1. Introduction

Social anhedonia is a promising indicator of schizotypy, the personality organisation conjectured to characterize individuals with genetic liability for schizophrenia (Meehl, 1962; Blanchard et al., 2000 but see also Meehl, 1990, 2001 and Linscott, 2007). In support of this notion, elevated levels of social anhedonia have been seen in patients with schizophrenia (e.g., Chapman et al., 1976; Blanchard et al., 2001, 1998; Cohen et al., 2005). Moreover, non-psychotic individuals with high levels of social anhedonia have shown a wide variety of characteristics similar to those seen in patients with schizophrenia such as neurocognitive deficits (e.g., Cohen et al., 2006; Gooding et al., 1999), eye tracking deficits (Gooding, 1999; Gooding et al., 2000), minor physical anomalies (Chok and Kwapil, 2005; Chok et al., 2005) and the occurrence of subclinical psychotic symptoms (Gooding et al., 2001; Mishlove and Chapman, 1985). Longitudinal examination of these socially anhedonic individuals has revealed a relatively high risk for the development of schizophrenia-spectrum disorders (Gooding et al., 2005; Kwapil, 1998). To test the hypothesis that social anhedonia identifies a genetic risk for

schizophrenia-spectrum disorders, studies have also examined the familiality of hedonic capacity. Findings from this literature suggest that biological relatives of patients with schizophrenia report experiencing significantly higher levels of social anhedonia than relatives of non-patients (Katsanis et al., 1990; Kendler et al., 1996; Vollema et al., 2002 but see also Appels et al., 2004; Yaralian et al., 2000) and that social anhedonia is correlated with severity of schizoid and paranoid personality disorder characteristics in relatives of individuals with schizophrenia (Lyons et al. 1995). In non-patient samples, probands have tended to resemble their biological family members in severity of social anhedonia (Berenbaum and McGrew, 1993; Meyer and Hautzinger, 2001). Finally, a series of non-patient twin studies has suggested that social anhedonia shows moderate levels of heritability, on the order of .32 to .67 (Kendler and Hewitt, 1992; Linney et al., 2003; MacDonald et al., 2001).

Although the above studies are consistent with the view that social anhedonia is familial and is elevated in relatives of probands with schizophrenia, these results do not directly address whether social anhedonia assessed in non-clinical samples identifies individuals with increased familial risk for schizophrenia. That is, if social anhedonics are at genetic risk for schizophrenia and related disorders, this risk would have been conferred through their biological parents. To date, we are not aware of any published study that has directly evaluated the biological relatives of individuals who are psychometrically identified as schizotypes using social anhedonia as a criterion. In

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examining other putative schizotypy questionnaires (e.g., magical ideation, perceptual aberration), Chapman et al. (1994) found that proband reports of psychosis (not differentiating the type of psychosis nor including schizophrenia-spectrum personality disorders) in their first- and second-degree relatives of schizotypes were twice as frequent compared with controls (15% vs. 7%). Lenzenweger and Loranger (1989) reported similar findings when a more detailed approach was employed to understanding the familial rates of Axis I schizophrenia and affective disorders in individuals with relatively high levels of perceptual aberration. However, none of these studies have reported on the clinical status of the family members of individuals scoring high in social anhedonia.

The present study examined schizotypal, schizoid and paranoid characteristics (i.e., Cluster A personality disorders) in the biological relatives of psychometrically identified schizotypes. Specifically, this study used data from the Maryland Longitudinal Schizotypy Study (MLSS; Blanchard et al., submitted for publication; Cohen et al., 2006; Collins et al., 2005) to examine the hypothesis that biological parents of social anhedonic probands would evidence elevated Cluster A characteristics compared with biological parents of controls. An advantage of this study is the use of a community sample. This addresses a significant limitation of most prior research on social anhedonia, namely the reliance on high functioning college-student samples who are predominantly Caucasian (Kwapil et al., 2002; see also Blanchard et al., submitted for publication for further discussion of this point).

2. Methods

2.1. Subjects

After offering informed consent, probands and parents were administered a series of diagnostic interviews, symptom severity measures and family inventories. Following completion of the study tasks, subjects were fully debriefed as to the nature of the study and provided with diagnostic feedback and clinical referrals if warranted. Subjects received $100 for their participation.

2.1.1. Probands

The present study recruited subjects from the community and surrounding areas of the University of Maryland at College Park (UMCP) as part of the Maryland Longitudinal Study of Schizotypy (see MLSS, Blanchard et al., submitted for publication for an in-depth explication on the recruiting procedures). The MLSS subjects included a subset of 2434 18- to 19-year-olds recruited by the UMCP Survey Research Center using random digit dial methods. Subjects were mailed a consent form (N = 3498; response rate = 70%) and a screening questionnaire that included the Revised Social Anhedonia Scale (SocAnh Scale; Eckblad et al., 1982), Perceptual Aberrations Scale (PerAb; Chapman et al., 1980) and Magical Ideation Scale (MagicID; Eckblad and Chapman, 1983). A validity scale (The Infrequency Scale; Chapman et al., 1976) was embedded within the screening questionnaire, and individuals who endorsed three or more items in the unexpected direction were excluded from the study (as in Chapman and Chapman, 1976). Subsequent selection and recruitment was based solely on responses to the Social Anhedonia questionnaire.

Eighty-six individuals identified by extreme social anhedonia scores participated in this study. Two methods were used to identify these subjects. The first method involved identifying individuals at least 1.9 standard deviations above SocAnh Scale mean (n = 71). This cut-off score was adopted after consulting prior published social anhedonia studies (notably Gooding et al., 2005 and Kwapil, 1998). Cut-off scores were determined separately for gender and ethnicity groups in light of research showing group differences on these variables for the SocAnh Scale (Kwapil, Crump and Pickup, 2002). The second selection method involved using the taxometric method of maximum covariance analysis (MAXCOV-HITMAX; Waller and Meehl, 1998) using critical SocAnh items identified in prior taxometric studies of the Chapman Schizotypy scales (see Blanchard et al., 2000; Horan et al., 2004 for a review of this methodology and a list of critical items). Individuals with Bayesian probabilities ≥ to 0.50 were assigned to the social anhedonia taxon group. This method identified 34 social anhedonia subjects, 15 of whom were undetected by the cut-off score method. There were no statistically significant differences between subjects identified through these two procedures in mean SocAnh scale score or for any of the dependent variables examined in this study (all Ps>0.10, range of Cohen's d=−0.20–0.20).

The control group consisted of 89 individuals without elevated scores on the SocAnh Scale, operationalised as a SocAnh score 0.50 standard deviations below the gender and race group derived mean and a Bayesian probability of taxon membership below 0.50. An additional inclusion criterion specified that control subjects not score higher than 0.50 standard deviations above the mean on the PerAb or MagicID scales of psychosis proneness. Control subjects were matched to the social anhedonia group on gender and ethnicity variables.

2.1.2. Biological parents

The biological mothers and fathers, identified by the proband, were each invited to participate in the study. Mother participation (58% [n = 50] and 55% [n = 47]) from the social anhedonia and control groups respectively) in the study was somewhat higher than father participation overall (32% [n = 28] and 28% [n = 24]) from the social anhedonia and control groups respectively). The rates of participation did not significantly differ between the participant groups (all Ps>0.10, range of Cohen's d=−0.20–0.20).

2.2. Measures

2.2.1. Social anhedonia

The SocAnh scale was administered as part of the initial screening questionnaire. The SocAnh Scale is a 40-item true–false self-report questionnaire designed to assess decreased pleasure from interpersonal sources. Examples of items include "If given the choice, I would much rather be with others than be alone" (keyed false) and "Just being with friends can make me feel really good" (keyed false). The SocAnh Scale has demonstrated good internal consistency reliability (Blanchard et al., 1998; Mishlove and Chapman, 1985) and test–retest reliability (Blanchard et al., 2001, 1998).

2.2.2. Cluster A symptoms

Cluster A symptoms (defined dimensionally) and disorders (defined categorically) of the biological parents were measured using two different approaches. The first employed a face-to-face interview with the biological relative using the schizoid, schizotypal, and paranoid modules from the International Personality Disorders Examination (IPDE; Loranger et al., 1994). The IPDE is compatible with the (Diagnostic and Statistical Manual of Mental Disorders DSM-III-R) (American Psychiatric Association, 1987) and has been used to assess Cluster A disorders in other at-risk studies (e.g., Chapman et al., 1994). The IPDE uses a three-point scoring system where '0' reflects an absence of the symptom, a '1' reflects a subclinical symptom presence, and a '2' represents a clinically significant symptom. Advanced doctoral students conducted these interviews. Interviews were video-taped and reviewed by a pair of doctoral level graduate students. Cases were then discussed during monthly case conferences until consensus diagnoses had been reached between the case conference group members. The subjects, interviewers and consensus judges were each blind to the subjects' group. Categorical diagnoses were made using DSM-IV criteria (i.e., five or more 'clinically significant' symptoms to meet criteria for schizotypal personality disorder, four or more 'clinically significant' symptoms to meet criteria for schizoid or paranoid personality disorders).

The second approach, using the Family Interview for Genetic Studies (FIGS; NIMH, 1992), was used when the biological relatives were not available for an in-person interview. The FIGS is a semi-structured interview designed specifically for use in family genetic studies for the purpose of gathering information. The FIGS has been used in a number of prior studies (e.g., Edmonds et al., 1998). In the present study, the FIGS was administered to probands (asking about their biological mother and father), to mothers (asking about the proband and the proband's father), and to fathers (asking about the proband and the proband's mother). FIGS ratings are dichotomous (i.e., present or absent). Because of time limitations in conducting clinical interviews (involving SCID assessments and FIGS interviews on family members), the FIGS interview focussed on Cluster A disorders (schizotypal, schizoid and paranoid personality disorders). We expected these characteristics to be those most promising for differentiating between social anhedonia and control relatives. Unfortunately, this does not provide information on the occurrence of Axis I disorders or other personality disorders in those individuals for whom we have only FIGS assessments (i.e., those parents not available for in-person diagnostic interviews).

Mother participation (58% [n = 50] and 55% [n = 47] from the social anhedonia and control groups respectively) in the study was somewhat higher than father participation overall (32% [n = 28] and 28% [n = 24] from the social anhedonia and control groups respectively). We adopted a hierarchical data replacement strategy where symptom ratings from the FIGS were used when in-person ratings from the IPDE were unavailable for a parent. In those cases where a parent did not complete an in-person diagnostic interview we gave priority to replacing these data with FIGS ratings provided by the spouse as it was assumed that the spouse of the missing parent would have the greatest familiarity with the individual. If the spouse was also not available to provide FIGS ratings on the other parent, we then relied on FIGS data provided by the proband. IPDE data were available for 58% (n = 50) of the social anhedonia and 55% (n = 47) of the control probands mothers and 32% (n = 28) of the social anhedonia and 28% (n = 24) of the control proband fathers. FIGS data were used for 42% (n = 36) of the social anhedonia and 45% (n = 42) of the control probands mothers and 68% (n = 58) of the social anhedonia and 72% (n = 65) of the control proband fathers. Between the social anhedonia and control groups, mother's FIGS ratings of fathers (n = 27 and n = 26 respectively), father's ratings of mothers (n = 5 and n = 3 respectively) and proband's FIGS ratings of their parents (n = 62 and n = 78 respectively) were similar. Given that diagnostic information produced by informants has been shown to underestimate the overall level of pathology (Kendler et al., 1996), we believe that our data replacement strategy is a relatively conservative approach. In order to make the two instruments compatible, we were required to recode IPDE in a dichotomous manner (e.g., '1' scores as '0's).

2.3. Statistical analyses

The analyses were conducted in two steps. First, we evaluated the hypothesis that biological parents of individuals with social anhedonia would show more frequent rates of cluster A disorder than biological parents of controls. Second, Cluster A pathology was

examined in a dimensional manner between the biological parents of social anhedonics and those of controls. We employed non-parametric statistical tests because the data examined here were ordinal, and thus, inappropriate for parametric statistics. Moreover, the distributions of the Cluster A diagnoses and dimensional symptom ratings were highly skewed.

3. Results

3.1. Descriptive and clinical characteristics of the probands

The demographic and clinical characteristics of the probands are presented in depth elsewhere (Blanchard et al., submitted for publication; Cohen et al., 2006; Collins et al., 2005), but will be presented briefly here. The social anhedonia and control groups did not differ on gender (57% and 54% female respectively; χ^2 [1] = 0.22, ns; Cohen's $d < 0.07$) or ethnicity (44%/47% and 45%/42% Caucasian/African-American respectively; χ^2 [3] = 1.13, ns; Cohen's $d = 0.37$). Social anhedonic subjects were significantly less educated (χ^2 [3] = 9.44, $P < 0.01$; Cohen's $d = 1.24$) with 30% not having attended any college, versus 11% of controls. With respect to clinical disorders, social anhedonia subjects had significantly more frequent mood disorders than controls (lifetime rates of 30% and 9% respectively; χ^2 [1] = 12.61, $P < 0.01$; Cohen's $d = 1.54$), but did not differ in psychotic (lifetime rates of 1% and 2% respectively; χ^2 [1] = 0.31, ns; Cohen's $d < 0.08$) or substance abuse (lifetime rates of 15% and 19% respectively; χ^2 [1] = 0.49, ns; Cohen's $d = 0.24$) disorders. Subjects with social anhedonia versus controls had significantly higher social anhedonia (24.26 ± 5.58 and 8.12 ± 3.5 respectively; $t[173] = 23.03$, $P < 0.001$; Cohen's $d = 3.50$), magical ideation (9.93 ± 5.68 and 6.53 ± 2.74 respectively; $t[173] = 5.07$, $P < 0.001$; Cohen's $d = 0.77$) and perceptual aberration (6.85 ± 5.44 and 2.66 ± 2.06 respectively; $t[173] = 6.78$, $P < 0.001$; Cohen's $d = 1.03$) scores and poorer general functioning (71.12 ± 16.53 and 81.15 ± 13.13 respectively; $t[173] = 4.45$, $P < 0.001$; Cohen's $d = 0.68$), assessed using the Global Assessment of Functioning Scale (GAF; American Psychiatric Association, 1994).

3.2. Cluster A disorders in the biological parents of social anhedonics versus controls

Rates of schizotypal (χ^2 [1] = 1.01, ns; Cohen's $d = 0.21$), schizoid (χ^2 [1] = 3.67, ns; Cohen's $d = 0.41$) or paranoid (χ^2 [1] = 1.32, ns; Cohen's $d = 0.25$) personality disorder diagnosis between the parents of the social anhedonia vs. control groups did not significantly differ between (see Table 1) However, probands with social anhedonia versus controls were significantly more likely to have a parent with a Cluster A disorder (χ^2 [1] = 4.91, $P < 0.05$; Cohen's $d = 0.48$).

As part of a *post hoc* set of analyses, we attempted to ascertain whether there were differences between biological mothers and fathers in their rates of cluster A disorders. In comparing these groups, mothers of social anhedonics and those of controls did not significantly differ in rates of diagnoses (χ^2 [1] = 0.55, ns; Cohen's $d < 0.11$). On the other hand, Cluster A diagnoses occurred significantly more in the fathers of social anhedonic probands than the control group (χ^2 [1] = 3.96, $P < 0.05$ Cohen's $d = 0.43$). In sum, Cluster A diagnoses were higher in the parents of social anhedonics compared

Table 1
Biological parents of social anhedonic ($n = 86$) vs. those of control subjects ($n = 89$): frequency of Cluster A diagnoses.

	Parents of controls % (n)	Parents of social anhedonics % (n)
Schizotypal	0% (0)	1% (1)
Schizoid	5% (4)	12% (11)
Paranoid	10% (9)	16% (14)
Any Cluster A disorder*	12% (10)	24% (21)

* $P < 0.05$.

Table 2
Biological parents of social anhedonic vs. those of control subjects: dimensional Cluster A scores.

	Parents of controls		Parents of social anhedonics		
	Mean	S.D.	Mean	S.D.	Mann Whitney U test (Z-score)
Mothers					
Schizotypal	0.28	0.71	0.21	0.72	1.03
Schizoid	0.32	0.64	0.47	1.01	.26
Paranoid	0.59	1.30	0.57	1.32	.29
Total	1.18	2.03	1.24	2.46	.56
Fathers					
Schizotypal	0.22	0.61	0.43	0.82	2.12*
Schizoid	0.77	1.30	1.20	1.59	1.97*
Paranoid	0.74	1.34	1.15	1.81	1.34*
Total	1.73	2.71	2.78	3.28	2.42*

* $P < 0.05$.

with controls. However, follow-up analyses indicated that this parental difference was limited to the fathers of social anhedonics as rates of diagnoses did not differ in the mothers of the two groups.

3.3. Cluster A symptoms in the biological parents of social anhedonics versus controls

The above comparisons focussed on categorical diagnoses. Given that elevations in Cluster A characteristics might occur in the absence of meeting full DSM diagnostic threshold, we conducted another set of group comparisons using dimensional scores for Cluster A characteristics. This allowed us to explore whether, despite not evidencing increased rates of Cluster A disorders, mothers of social anhedonics might display elevations in these characteristics rated dimensionally. The dimensional symptom scores for Cluster A characteristics were computed and compared using a series of Mann–Whitney U tests, conducted separately for mothers and fathers. These findings are presented in Table 2. The fathers of social anhedonia subjects had significantly more severe symptoms than control fathers on each of the measures and the total scores (Ps < 0.05; range of Cohen's $d = 0.29$–0.54). The mothers of social anhedonia and control subjects did not significantly differ on any of these variables (all Ps > 0.05; range of Cohen's $d = -0.20$–0.20). Wilcoxon signed rank tests revealed that the mothers and fathers of social anhedonic subjects significantly differed for the schizotypal ($Z = 2.18$, $P < 0.05$; Cohen's $d = 0.48$), schizoid ($Z = 3.55$, $P < .001$; Cohen's $d = 0.83$), paranoid ($Z = 2.36$, $P < 0.05$; Cohen's $d = 0.53$) and total IPDE ($Z = 3.89$, $P < 0.001$; Cohen's $d = 0.92$) scores, These results indicate that the fathers, but not mothers, of probands with social anhedonia had more severe Cluster A symptomatology[1].

Because parental ratings included both in-person (IPDE) and informant-based (FIGS) assessments, we examined whether the above results would hold-up using just the in-person diagnostic assessments (i.e., IPDE ratings). In this smaller sample, the parents of social anhedonics and controls did not significantly differ in frequency of categorical Cluster A diagnoses (all Ps > 0.05; range of Cohen's $d = 0$–0.21). Consistent with what was seen in the full sample however, the fathers of social anhedonics showed significantly more severe schizoid symptoms ($t[50] = 2.06$, $P < 0.05$; Cohen's $d = 0.58$) and a trend for more severe overall Cluster A symptoms ($t[50] = 1.88$, $P < 0.10$; Cohen's $d = 0.53$). Although these analyses have significantly less power than the combined IPDE and FIGS data, the results provide

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[1] To address the concern that that the SocAn scales can yield 'false' positives due to mood disorders, all analyses in this project were recomputed excluding cases where the proband met criteria for a current Axis I mood disorder ($n = 2$ and $n = 5$ from the control and social anhedonia groups respectively), as these individuals may reflect "false" schizotypes. None of the results of this project changed.

assurance that the overall pattern of findings was relatively consistent whether or not parents directly participated in the study.

4. Discussion

The present study found that almost a quarter of socially anhedonic probands had a biological parent with a diagnosable Cluster A disorder, a rate that was twice that seen in the control group. This finding is in line with prior studies on the familiality of social anhedonia (Katsanis et al., 1990, Kendler et al., 1996; Vollema et al., 2002; Lyons et al., 1995; Kendler and Hewitt, 1992; Linney et al., 2003; MacDonald et al., 2001), thus bolstering the notion that social anhedonia reflects a vulnerability marker for schizophrenia diathesis. The present findings are also important in that they contribute to the growing construct validity for the use of the Chapman Social Anhedonia Scale as a tool for the psychometric identification of at-risk individuals (e.g., Kwapil, 1998; Gooding et al., 2005).

An interesting, and entirely unexpected result that emerged from this study was that elevated rates of Cluster A diagnoses and characteristics were limited to the fathers of social anhedonics (mothers in the two groups did not differ in clinical assessments). This finding might be understood in the context of increasing evidence that the familial transmission of schizophrenia may be sex-linked in some fashion. For example, the first-degree relatives of female patients with schizophrenia have shown a higher incidence of schizophrenia and psychotic disorders compared to male patients, who tend to have first degree relatives with a higher incidence of Cluster A personality disorders and more severe flat affect (Goldstein et al., 1990, 1995). While the present study is unable to examine the differential parental contribution to proband psychopathology in a systematic way, the finding that fathers of individuals high in social anhedonia had high rates of Cluster A disorders is an intriguing result that requires replication.

Limitations of the present project warrant mention. First, to minimise the impact of missing parental data we employed a data replacement strategy that often used family member ratings of parental pathology rather than data from semi-structured interviews. It is unclear to what degree this may have affected the results. Prior research has suggested that diagnostic information produced by informants tends to underestimate the overall level of pathology; therefore, the parental pathology levels may be artificially low (Kendler et al., 1996). Due to missing parental data, we were unable to examine the potential impact of important variables on the present results. For example, the parents who participated could have been more similar to their offspring than the parents who did not for a variety of non-familial reasons, including familiarity, increased contact or shared environment. Second, we lacked parental data for Axis I disorders, so it was unclear whether any of the missing biological parents may have met criteria for schizophrenia (although none of those completing in-person assessments met criteria for this disorder). Third, the present study was unable to tease apart genetic and environmental factors when examining familial contribution, so we were unable to examine familial factors in any meaningful manner. Fourth, we were unable to confirm that the probands parents were biological, introducing potential noise in our data. Nonetheless, a familial link in symptoms was established.

The present findings are consistent with the hypothesis that social anhedonia is a promising indicator for schizophrenia-spectrum liability. Results indicated that Cluster A characteristics extended beyond probands identified by elevations in social anhedonia and were evident in a biological relatives, specifically fathers. The unexpected findings of elevated pathology limited to fathers of socially anhedonic probands requires further exploration.

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