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The relationship of social function to depressive and negative symptoms in individuals at clinical high risk for psychosis

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Abstract

Background—Social dysfunction is a hallmark symptom of schizophrenia which commonly precedes the onset of psychosis. It is unclear if social symptoms in clinical high-risk patients reflect depressive symptoms or are a manifestation of negative symptoms.

Method—We compared social function scores on the Social Adjustment Scale-Self Report between 56 young people (aged 13–27 years) at clinical high risk for psychosis and 22 healthy controls. The cases were also assessed for depressive and 'prodromal' symptoms (subthreshold positive, negative, disorganized and general symptoms).

Results—Poor social function was related to both depressive and negative symptoms, as well as to disorganized and general symptoms. The symptoms were highly intercorrelated but linear regression analysis demonstrated that poor social function was primarily explained by negative symptoms within this cohort, particularly in ethnic minority patients.

Conclusions—Although this study demonstrated a relationship between social dysfunction and depressive symptoms in clinical high-risk cases, this association was primarily explained by the relationship of each of these to negative symptoms. In individuals at heightened risk for psychosis, affective changes may be related to a progressive decrease in social interaction and loss of reinforcement of social behaviors. These findings have relevance for potential treatment strategies for social dysfunction in schizophrenia and its risk states and predict that antidepressant drugs, cognitive behavioral therapy and/or social skills training may be effective.

Keywords

Clinical high risk; negative symptoms; psychosis; social function; ultra high risk

Introduction

For several decades, social dysfunction has been identified as a core feature of schizophrenia (Bellack *et al.* 1990), and is now one of the criteria for diagnosis (APA, 2000). Social dysfunction is often evident early in the course of the disorder (Meares, 1959), first subtly during the pre-morbid period in childhood, expressed as difficulty in establishing

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Declaration of Interest
None.

relationships (Jones *et al.* 1994), as well as social 'over-reactivity' (social anxiety, 'acting out') in boys and as 'under-reactivity' in girls (Done *et al.* 1994). In adolescence, individuals at genetic risk for schizophrenia are more likely to have poor peer engagement and unpopularity with peers (as compared both with healthy controls and with individuals at genetic risk for bipolar disorder) (Dworkin *et al.* 1990; Hans *et al.* 2000), phenomena which cannot be accounted for by co-morbid diagnoses (Hans *et al.* 2000). Retrospective studies of individuals with non-affective psychosis demonstrate that active social withdrawal is a common early behavioral change in the prodromal period, accompanied frequently by dysphoria (Yung & McGorry, 1996; Häfner & Maurer, 2003; Møller & Husby, 2000; Tan & Ang, 2001; Corcoran *et al.* 2007). Some studies also show that social dysfunction during the prodromal period of schizophrenia is predictive of poor social outcome 5 years beyond a first episode of psychosis (Häfner *et al.* 2003). Social dysfunction is also pervasive among young people identified as at heightened clinical risk for psychosis more generally (Cornblatt *et al.* 2007*a*), at levels comparable with that seen in individuals with a first episode (Ballon *et al.* 2007) or even multiple episodes (Addington *et al.* 2008*a*) of psychosis. Social dysfunction in these clinical high-risk (CHR) patients persists over time (Niendam *et al.* 2007), and is one factor among others that predicts the later development of overt psychosis (Cannon *et al.* 2008).

In chronic schizophrenia, negative symptoms have been repeatedly identified as the symptom type most closely associated with social dysfunction (Chaves *et al.* 1993; Blanchard *et al.* 1998; Dickerson *et al.* 1999; Smith *et al.* 1999, 2002; Hofer *et al.* 2006; Wittorf *et al.* 2008) whereas only a few studies have identified positive symptoms (specifically in females) (Chaves *et al.* 1993; MacEwan & Athawes, 1997), disorganized symptoms (Smith *et al.* 2002), general psychopathology (MacEwan & Athawes, 1997), depression (Smith *et al.* 1999; Jin *et al.* 2001) and anxiety (Lysaker & Salyers, 2007) as associated with social function. Likewise, among individuals with a first episode of psychosis, negative symptoms predict social function both in cross-sectional (Voges & Addington, 2005) and in longitudinal studies (Ho *et al.* 1998; Milev *et al.* 2005). However, in a predominantly African-American cohort of patients with a first episode of non-affective psychosis, social impairment was associated with not only negative but also depressive and general symptoms (Goulding *et al.* 2010).

Given its persistence and its impact on morbidity, social dysfunction in schizophrenia and other psychotic disorders is important to understand, especially in its early and incipient phases, when the contribution of chronic illness and medication to social dysfunction is minimal. CHR cohorts are enriched with individuals in early stages of schizophrenia and other psychotic disorders, so their study affords the opportunity to evaluate clinical correlates of social dysfunction, such that effective treatment strategies can be developed. Along these lines, negative symptoms were found to be related to social dysfunction in a heterogeneous sample meeting 'clinical high risk criteria' ; however, some of the subjects were non-help-seeking individuals from the community as old as 54 years (Svirskis *et al.* 2007). More recently, Ruhrmann *et al.* (2008) reported that in a large German cohort of young help-seeking CHR patients, depressive (but not negative) symptoms reliably correlated with impaired quality of life, including subjective satisfaction with social function. Although the measure of social function used by Ruhrmann *et al.* (2008) has well-established psychometric properties and has been used in both schizophrenia and major depressive disorder patient cohorts (Pukrop *et al.* 2000), it does not quantify specific behavioral domains relevant to current social functioning. Moreover, a recent, perhaps underpowered, study failed to find an association of social dysfunction, as assessed with the Social Functioning Scale, with symptoms (positive, negative, disorganized, general, depressive, anxiety) in CHR patients (Shim *et al.* 2008). Thus, at present, there is a need for

further study of how symptoms experienced by patients at clinical high risk for psychosis may be related to social function.

In the present study we aimed to characterize the relationship(s) between social function, as measured with the more behaviorally based Social Adjustment Scale-Self Report (SAS-SR; Weissman & Bothwell, 1976) and negative and/or depressive symptoms in young CHR patients. We hypothesized that social dysfunction would be evident in CHR patients, in whom it would be associated with depressive, but not negative, symptoms, as was found by Ruhrmann *et al.* (2008).

Method

Subjects

CHR patients were help-seeking individuals who met criteria for at least one of three 'prodromal' syndromes, as assessed with the Structured Interview for Prodromal Syndromes (SIPS; Miller *et al.* 2003): (1) attenuated positive symptom syndrome; (2) genetic risk and deterioration syndrome; and/or (3) brief intermittent psychotic syndrome. Attenuated positive symptoms could not have occurred solely in the context of substance abuse or withdrawal (by history). Both patients and healthy controls were between the ages of 12 and 30 years, and English-speaking. Exclusion criteria for all participants included a history of psychosis, serious risk of harm to self or others, major medical or neurological disorders, and mental retardation [intelligence quotient (IQ) <70 with functional impairment]. Specific exclusion criteria for control participants included: (1) a family history of psychosis; (2) a history of adoption; (3) a diagnosis of a DSM-IV cluster A personality disorder ; and (4) any Axis I diagnosis in the preceding 2 years (excluding substance use-related diagnoses).

Patients were ascertained generally through referrals from schools and clinicians in the New York metropolitan area, and through the Internet. Recruitment strategies for patients included presentations, mass mailings of brochures, and the creation of a website. Recruitment and ascertainment of healthy controls were focused on the same source population, and also included flyers and postings on craigslist. The clinical data described in this paper were collected as part of a larger longitudinal cohort study of psychosis risk at the Center of Prevention and Evaluation at New York State Psychiatric Institute at Columbia, which was approved by the Institute's and University's institutional review boards. All participants aged ≥18 years provided written informed consent for participation. All participants aged <18 years provided written assent; for these younger subjects, written informed consent was provided by a parent.

Measures of symptoms, IQ and social function

Prodromal symptom severity was rated using the Scale of Prodromal Symptoms (SOPS) (Miller *et al.* 2003), which probes positive, negative, disorganized and general symptoms (with factors for positive, negative and disorganized symptom types; Hawkins *et al.* 2004). Individual items are rated from 0 (absent) to 6 (suprathreshold), with a prodromal range considered to lie between the scores of 3 and 5. Reliability for the SIPS/SOPS was established with the Recognition and Prevention clinical high-risk research program at Hillside Hospital (intraclass correlations >0.70 for individual scale items and 1.00 for syndrome ratings). This is consistent with excellent to near excellent inter-rater reliability for individual SOPS items (Miller *et al.* 2003). Depressive symptoms were evaluated using the 21-item Hamilton Rating Scale for Depression (HAMD; Hamilton, 1960), which has been employed in both adult and adolescent samples. The HAMD was administered by a postdoctoral psychologist trained at the Lieber Center for Schizophrenia Research, who had achieved κ >0.75 for symptom ratings. Information was available for a subgroup of 40

patients and 16 controls on IQ, measured using the Wechsler Adult Intelligence Scale (Wechsler, 1981) or Wechsler Intelligence Scale for Children, 3rd edition (Wechsler, 1991), depending on age.

Social function was assessed at study entry using the SAS-SR (Weissman & Bothwell, 1976), which probes performance over the last 2 weeks in six domains of functioning: work (as a paid worker, unpaid homemaker or student), social and leisure activities, relationships with extended family, role as a marital partner, parental role, and role within the family unit. The SAS-SR was read aloud to participants by a research coordinator with a master's level of clinical training, who then verbally indicated their responses. Each of the 54 items was rated along a five-point Likert scale, with higher scores indicating greater impairment. Up to seven mean scores can be generated, including one for each of six possible domains, and one overall mean score. Scores are calculated by dividing summary scores (per domain and overall) by the number of relevant items responded to. Domains were not considered for individuals if not appropriate (e.g. 'marital ' if unmarried, 'parental' if no children, 'work' if no work or study endorsed). The SAS-SR (Weissman & Bothwell, 1976) has been used in both community and clinical samples, and yields results similar to those obtained by the interview format of the SAS both in individuals with depression (Weissman *et al.* 1978) and those with schizophrenia (McCreadie & Barron, 1984). (The comparability of the SAS-SR to the interview format, however, has not been evaluated in adolescent and early adult clinical cohorts.) Although no collateral information was collected from family and clinicians specifically for ratings of social adjustment using the SAS-SR, each patient participant had a comprehensive clinical evaluation based on data from all sources, and it has been observed that patients are generally reliable and consistent in their description of their social and work function. Demographic information was also obtained.

Data analysis

Patients were compared with controls in terms of demographics, IQ, social function and symptom severity. It was hypothesized that patients with comparable demographics to controls would show impaired social function and worse symptoms. Then, within the patient cohort, potential associations of both social function and symptoms with demographic variables and IQ were examined, and partial correlations/regression analyses were considered for any potential confounding demographic variables and to evaluate primary associations of demographic variables with social function. Non-parametric Spearman analyses were used as a conservative assessment of all potential associations. Linear regression analyses were done to examine together demographic and symptom correlates of overall social function and ' social/ leisure '.

We set α at 0.05 for associations of overall social adjustment score with depression (hypothesized) and negative symptom severity. Bonferroni correction (0.05/10 tests=adjusted α of 0.005) was used to reduce type I error for other analyses, specifically (1) overall social adjustment with positive, disorganized and general symptoms, and (2) exploratory analyses of specifically ' social/leisure ' with symptoms (negative and other), based on previous findings of an association of the SAS-SR 'social/leisure' scale with negative symptoms in schizophrenia cohorts (Wittorf *et al.* 2008), and (3) correlations of social function with depressive and negative symptoms within the Caucasian and ethnic minority subgroups, given disparate findings in cohorts of different ethnic composition (Ruhrmann *et al.* 2008; Goulding *et al.* 2010). In exploratory analyses, social function and symptoms were also examined in respect to depression diagnoses, family history and later transition to psychosis.

Results

There were 56 CHR patients and 22 healthy controls who were ethnically diverse, and comparable in age (mean about 19–21 years), sex (primarily male), IQ [mean 107 (S.D.=11) *v.* mean 106 (S.D.=19)], employment status (primarily students) and socio-economic status (based on paternal education) (Table 1). Of the patients, 55 met criteria for attenuated positive symptom syndrome (of whom 16 also met genetic risk and deterioration syndrome criteria) and one patient met criteria for brief intermittent psychotic syndrome. As expected, patients had greater symptom severity, with subthreshold psychotic and other 'prodromal' symptoms and mild to moderate depression (Table 1). [Mean symptom scores are comparable with those reported for other CHR cohorts, e.g. total positive 12.0 (S.D.=4.1) and negative 12.1 (S.D.=6.8) symptoms (Cannon *et al.* 2008)]. Patients also had worse social adjustment, specifically in the 'social/leisure' domain, though interestingly had comparable work and school function (determined only for those patients who held a job or attended school). As no participants were married or had children, the marital and parental function domains of the SAS-SR were not applied. Of note, social dysfunction scores obtained were comparable with those obtained in a similar CHR cohort using the same instrument (Ballon *et al.* 2007). Further, the cohort is similar to other CHR cohorts in North America in terms of age (19 years), high prevalence of the attenuated positive symptom syndrome, mean total positive and negative symptom scores, family history of psychosis (28.6%) and transition rates to psychosis (25%) (Cannon *et al.* 2008).

Social adjustment, both overall and specifically in the 'social/leisure' domain, was unrelated to age, sex and IQ, both for the entire sample and in analyses confined to the patient subgroup. All symptom domains were unrelated to age. As for sex, male patients only had significantly more negative symptoms [14.5 (S.D.=6.2) *v.* 7.9 (S.D.=4.6) for women, p=0.001]. By contrast, patients from ethnic minorities had significantly worse overall [2.7 (S.D.=0.6) *v.* 2.3 (S.D.=0.6), t_{54}=3.057, p=0.003] and 'social/leisure' [3.1 (S.D.=0.9) *v.* 2.5 (S.D.=0.8), t_{54}=2.861, p=0.006] social function, and significantly greater negative [15.8 (S.D.=6.6) *v.* 10.5 (S.D.=5.3), t_{51}=3.24, p=0.002] and disorganized [8.4 (S.D.=3.7) *v.* 5.5 (S.D.=3.4), t_{54}=2.754, p=0.008] symptoms. As ethnicity was associated with both overall social function and symptom types of interest, it was included in the final regression model.

Correlational analyses demonstrated that overall social function was, as hypothesized, associated with depressive symptoms, as well as with negative, disorganized and general (but not positive) prodromal symptoms, even correcting for multiple comparisons (i.e. α = 0.005 ; see Table 2). The 'social/leisure' domain was also associated with depressive, negative and disorganized (but not positive or general) symptoms, again correcting for multiple comparisons (Table 2). Of note, these various symptom domains (except for positive symptoms) were also significantly associated with one another (see Table 2).

Linear regression models were constructed for the outcome variables of overall social dysfunction and for specifically the 'social/leisure' domain, including in the models both ethnicity and those symptom types which were individually related to social function (depressive, negative, disorganized and general) (Table 3). These regression models demonstrate the association of negative symptoms with overall social function, as well as with the 'social/leisure' subdomain, while adjusting for ethnicity and other symptoms. Of note, the ethnic difference in overall social function also persisted with adjustment for symptoms.

Exploratory analyses were done to examine correlations within subgroups by race, given ethnic differences in symptoms and social function. Although not surviving Bonferroni correction, for minority patients, social function appeared to be correlated primarily with

negative symptoms (Spearman $\rho = 0.54$, $p=0.006$). By contrast, for Caucasian patients, social function appeared to be correlated with general symptoms (Spearman $\rho = 0.50$, $p=0.008$).

There were data available for 39 patients as to diagnoses obtained by structured Diagnostic Interview with Genetic Studies (DIGS; Nurnberger *et al.* 1994) (with data cleaned, consensus diagnoses achieved, and data entered). Of these 39 patients, 20 met criteria for having had a major depressive episode during their lifetime : these patients had similar social function (2.6 *v.* 2.4, $p=0.15$) and symptom profiles (data not shown). Chart review of diagnosis based on study entry clinical evaluation done to augment the DIGS yielded 27 (of 56) patients with a history of major depressive disorder: these patients also did not differ in social function (2.4 *v.* 2.5, $p=0.52$) and symptom profiles (data not shown). Of the sample, 28.6% had a known first-degree family member with psychosis: they did not differ from the rest of the cohort in terms of social function or symptoms (data not shown).

Of note, this cohort is enriched with individuals in the early stages of schizophrenia and other psychotic disorders, as 11 of 56 patients (19.6%) developed schizophrenia (three others developed affective psychosis and one a substance-induced psychosis). Those patients who made the transition to psychosis did not differ in baseline social function or by depressive or prodromal symptoms (data not shown). (The length of follow-up varied greatly, however, ranging from 1 month to 3 years).

Discussion

Consistent with previous studies, social function was significantly impaired in this CHR cohort, as compared with healthy controls ascertained from the same source population, and with similar demographic characteristics. In fact, social dysfunction scores obtained were comparable with those obtained in a similar CHR cohort using the same instrument – the SAS-SR: overall scores 2.5 (our study) *v.* 2.4 and social/leisure scores 2.8 (our study) *v.* 2.6 (Ballon *et al.* 2007).

Further, in this cohort of CHR patients, social dysfunction was associated with depressive symptoms, a finding consistent with the only other published study identifying symptom correlates of social function in a CHR cohort (Ruhrmann *et al.* 2008). We build on the findings from that study in employing scales specifically designed to evaluate depression (HAMD; Hamilton, 1960) and social function [SAS-SR (Weissman & Bothwell, 1976) *versus* a quality of life scale, known as MSQOL, of which social function is only one of seven domains (Pukrop *et al.* 2000)]. Our findings complement theirs in that whereas they evaluated ‘subjective’ satisfaction with social function (Ruhrmann *et al.* 2008), our methods measured the frequency of specific behaviors related to social function. The CHR state for psychosis was identified in the present study using the SIPS/SOPS (Miller *et al.* 2003) and was operationalized similarly to the late initial prodromal state (‘LIPS’) or ‘Late Prodromal Syndrome’ in the study by Ruhrmann *et al.* (2008), further supporting that this replication illustrates a reliable relationship between depressive symptoms and social function in young people at heightened clinical risk for psychosis.

Unlike the Rurhmann *et al.* (2008) study, however, the present study revealed an association between negative symptoms and social dysfunction, which could not be accounted for by the association of either with depressive symptoms. This was particularly surprising given that the former study arguably offered better statistical power to detect such an association. However, our current finding of an association of negative symptoms with social dysfunction is consistent with multiple reports of such an association in schizophrenia patients, including in some cohorts of first-episode psychosis patients (Blanchard *et al.*

1998; Chaves *et al.* 1993; Dickerson *et al.* 1999; Smith *et al.* 1999, 2002; Hofer *et al.* 2006; Wittorf *et al.* 2008; Goulding *et al.* 2010).

One potential explanation for the discrepancy between the present study and Ruhrmann *et al.* (2008) is differences in the cohorts and their ascertainment. Their German CHR cohort was older (mean age 26 years *v.* mean age 20 years in our cohort), and more likely to have a 'regular occupation' (72% *v.* 12% with 'paid employment' in our cohort) and to be in a 'steady partnership' (32% *v.* 0% in our cohort). Also, it is not clear if the two cohorts are comparable in the proportion of individuals who develop schizophrenia: whereas in the present cohort, the proportion is 19.6%, this statistic was not provided for the Ruhrmann *et al.* (2008) cohort. Ethnic differences may account for the disparate findings, as in both the German cohort (Ruhrmann *et al.* 2008) and in the current study's Caucasian subgroup, social function was unrelated to negative symptoms, whereas that association existed both in the current study's ethnic minority subgroup and in a predominantly African-African first-episode cohort (Goulding *et al.* 2010).

The ability of the current study to detect a significant association between negative symptoms and social dysfunction may, however, be related to differences in the measures used to characterize social function. Subjective satisfaction with social function, as measured using quality-of-life scales, may be related more to affective symptoms, whereas specific social behavior may be also correlated with negative symptoms. This is supported by the existing literature on clinical correlates of social dysfunction in patients with schizophrenia. Those studies which found an association with depressive symptoms in schizophrenia patients used quality-of-life scales to assess social function (Smith *et al.* 1999; Jin *et al.* 2001) whereas those documenting an association with negative symptoms used measures that assay social behavior [e.g. Disability Assessment Scale (Chaves *et al.* 1993); Social Functioning Scale (Dickerson *et al.* 1999; Goulding *et al.* 2010), Social Behavior Scale (Smith *et al.* 1999); Social Adjustment Scale (Blanchard *et al.* 1998; Wittorf *et al.* 2008)].

As for negative symptoms, both CHR studies had low mean levels of negative symptoms with similar variance: PANSS mean negative symptom score=13.1 (S.D.=4.9) in the Ruhrmann cohort (possible range= 7–49) and SOPS mean negative symptom score=12.7 (S.D.=5.7) in the present cohort (possible range= 0–36). Therefore, the discrepancy is unlikely to be related to differences in measures used to assess negative symptoms, or in the prevalence of negative symptoms in the two cohorts, especially as the SIPS/ SOPS was in part derived from the PANSS (Miller *et al.* 2003).

In the current study, social impairment was related to multiple symptom domains – depressed, negative, disorganization, general – that were themselves highly correlated with one another. The notable exception was that positive symptoms correlated neither with social function nor with any of the symptom domains related to social function. These findings are consistent with a principal components analysis of the Scale of Prodromal Symptoms, which demonstrated that 11 of 19 SOPS symptoms loaded heavily together on an initial component, 'including all negative symptoms and a mix of disorganization and general symptoms' (p. 343; Hawkins *et al.* 2004). The robust correlations of depressive symptoms with negative and other prodromal symptoms in the current study suggest that these symptoms may be difficult to distinguish while in attenuated form.

Prevalent social impairment and depression have been described for the prodromal period leading to a first episode of psychosis in retrospective studies (Häfner & Maurer, 2003; Møller & Husby, 2000; Tan & Ang, 2001; Corcoran *et al.* 2007; Myles-Worsley *et al.* 2007), with one study suggesting that depressive symptoms occur before social impairment (Häfner

et al. 1999). Likewise, although ascertained on the basis of subthreshold positive symptoms generally, CHR cohorts have prevalent social impairment, and depressive and negative symptoms (Cornblatt *et al.* 2003; Corcoran *et al.* 2003; Yung *et al.* 2004; Lencz *et al.* 2004), which together have been hypothesized to constitute a relatively stable core feature of risk for schizophrenia (Cornblatt *et al.* 2003). Social impairment was identified in one CHR study as the most common presenting symptom, characterizing 62% of the cohort (Lencz *et al.* 2004). Comparably, the rates of depressive disorder reported for other cohorts are as high as that found in the current study (about 50%): 32% (Lencz *et al.* 2004), 50% (Meyer *et al.* 2005) and 59% (Rosen *et al.* 2006).

Further, both social impairment (Cornblatt *et al.* 2007*a* ; Cannon *et al.* 2008) and depressive symptoms (Yung *et al.* 2004) have been identified variably as predictors of later transition to psychosis in larger CHR cohorts. Given this associated elevated risk for psychosis, and their effect on current morbidity, these clinical features deserve attention as potential targets of treatment. The prevalent social dysfunction in CHR patients has been described as non-responsive to pharmacological treatment (Cornblatt *et al.* 2007*a*) yet improvement has been observed in some patients, suggesting remediation may be possible (Niendam *et al.* 2007). Antidepressants may have efficacy not only in treating depressive symptoms in CHR patients but also in preventing the onset of psychosis (Cornblatt *et al.* 2007*b*). Case reports suggest efficacy for cognitive behavioral therapy (CBT) in treating negative symptoms in both schizophrenia (Perivoliotis & Cather, 2009) and its putative clinical risk states (Kimhy & Corcoran, 2008). A small study suggests that CBT may improve social impairment, as well as general, depressive and prodromal symptoms in CHR patients (Bechdolf *et al.* 2005). The feasibility of CBT has been established for CHR patients, in whom it may reduce risk for transition to psychosis (Morrison *et al.* 2004, 2007). Overall, although there is some evidence to suggest that depressive and negative symptoms may be amenable to treatment in CHR patients, more research is required, including clinical trials.

Finally, a curious finding in the current study was that patients from ethnic minorities had worse social function, and greater symptom severity. A likely explanation for this is ascertainment bias, as members of ethnic/racial minority groups are less likely to access mental health care services (Anglin *et al.* 2008) and have greater concerns about stigma (Cooper-Patrick *et al.* 1997; Wong *et al.* 2009). However, there may be real ethnic differences, as the associations of social impairment with negative, depressive and general symptoms seen in the present cohort have as mentioned also been documented in a predominantly African-American cohort with first-episode nonaffective psychosis (Goulding *et al.* 2010). Ethnic differences in symptoms and function among CHR patients merit further study.

Limitations and future directions

There are several limitations to this study. It is a cross-sectional study of a cohort of only 56 CHR patients and 22 controls. Therefore, causation cannot be established and there is increased risk for type II error. The majority of patients were male, thus limiting the characterization of social dysfunction in girls and women at CHR for psychosis. Social function was assessed using a self-report measure and no collateral information was systematically included. No information was available on pre-morbid adjustment, history of social functioning, age of onset of prodromal symptoms, or duration of prodromal symptoms – all of which may be relevant to social function in CHR patients.

Only IQ was measured in this study, and only in a subgroup of the cohort. It will be important in future studies to examine neuropsychological function in greater detail, as social function has been related in schizophrenia to visual attention and psychomotor function (Kurtz *et al.* 2005) and to verbal learning and working memory (Smith *et al.* 2002).

In a CHR cohort, improved social function was associated prospectively with visual memory and processing speed (Niendam *et al.* 2007).

Another limitation of this study is that there is no measure of social cognition, which is related to social function in schizophrenia (Couture *et al.* 2006) and which is impaired in both schizophrenia (Couture *et al.* 2006) and CHR cohorts (Addington *et al.* 2008*b*; Couture *et al.* 2008). It is plausible that the repeated failure to navigate social situations successfully, especially with peers (and its consequent lack of reward), may lead to both affective symptoms and to social anhedonia and withdrawal. Also, there is no measure of social anxiety, which is prevalent in first-episode psychosis patients, in whom it is related to not only social dysfunction but also depression and negative symptoms (Voges & Addington, 2005). Such social anxiety has been associated in individuals with more chronic presentation of schizophrenia with both poor social function (Blanchard *et al.* 1998) and negative symptoms (Penn *et al.* 1994). We now have studies underway to examine in CHR patients the associations of social dysfunction with social anxiety, social anhedonia and impaired social cognition.

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Table 1

Group differences in demographics, symptoms and social function (n=78)

	Patients (n=56)		Healthy controls (n=22)	
	***n* (%)**	**Mean (S.D.)**	***n* (%)**	**Mean (S.D.)**
Demographics				
Age, years		19.6 (3.6)		21.0 (3.5)
Sex				
Male	43 (77)		13 (59)	
Female	13 (23)		9 (41)	
Race				
Caucasian	30 (54)		13 (59)	
Non-Caucasian	26 (46)		9 (41)	
Occupation				
Paid employment	7 (12)		6 (27)	
Student	34 (61)		13 (59)	
Unemployed	15 (27)		3 (14)	
Paternal education				
High school or less	10 (18)		4 (20)	
BA or some college	20 (36)		13 (65)	
Graduate studies	13 (23)		3 (15)	
Symptoms				
Depression (HAMD) **		10.8 (5.7)		1.1 (1.5)
Prodromal (SOPS)[b]				
Total positive **		13.8 (4.7)		0.6 (0.8)
Total negative **		12.7 (5.7)		1.2 (1.8)
Total disorganized **		6.8 (3.3)		0.6 (1.0)
Total general **		9.1 (4.6)		0.7 (1.0)
Social function and its subscales (SAS-SR)[b]				
Overall **		2.5 (0.6)		1.6 (0.3)
Work		3.5 (1.7)		3.2 (1.9)

	Patients (n=56)		Healthy controls (n=22)	
	n **(%)**	**Mean (S.D.)**	***n*** **(%)**	**Mean (S.D.)**
Study		2.2 (0.8)		1.8 (1.8)
Social/leisure **		2.8 (0.9)		1.7 (0.4)
Extended family *		2.1 (0.8)		1.7 (0.6)

S.D., Standard deviation; BA, Bachelor of Arts ; HAMD, Hamilton Rating Scale for Depression ; SOPS, Scale of Prodromal Symptoms; SAS-SR, Social Adjustment Scale-Self Report.

[a] Paternal education was not obtained for all participants.

[b] Possible ranges of scores are SOPS positive (0–30), SOPS negative (0–36), SOPS disorganized (0–24), SOPS general (0–24), SAS-SR overall and subscales (each 0–5).

* $p \leq 0.05$,

** $p \leq 0.001$.

Table 2

Spearman intercorrelations for social function and symptoms

	SAS-SR overall	SAS-SR leisure	Dep	Pos	Neg	Dis	Gen
Leisure	0.809**	–	–	–	–	–	–
Depressive	0.477**	0.406*	–	–	–	–	–
Positive	0.107	−0.008	0.190	–	–	–	–
Negative	0.498**	0.494**	0.391*	0.076	–	–	–
Disorganized	0.510**	0.571**	0.491**	0.174	0.565**	–	–
General	0.446**	0.315	0.644*	0.143	0.295	0.378	–

SAS-SR, Social Adjustment Scale-Self Report ; Dep, Hamilton Rating Scale for Depression ; SOPS, Scale of Prodromal Symptoms; Pos, SOPS positive symptoms; Neg, SOPS negative symptoms; Dis, SOPS disorganized symptoms; Gen, SOPS general symptoms.

* $0.001<p<0.005$,

** $p\leq 0.001$.

Table 3

Linear regression analyses predicting overall and social/leisure function

	β	S.E.	*t*	*p*
Overall social function (SAS-SR) [R^2=0.569, F(5, 33)=8.699, p<0.001]				
HAMD	0.182	0.014	1.142	0.26
SOPS negative symptoms	0.376	0.013	2.696	0.01
SOPS disorganized symptoms	−0.073	0.023	−0.493	0.63
SOPS general symptoms	0.237	0.018	1.563	0.13
Ethnicity (Caucasian v. non-Caucasian)	−0.343	0.140	−2.660	0.01
Social/leisure function (SAS-SR) [R^2=0.355, F(5, 33)=3.626, p=0.01]				
HAMD	0.060	0.027	00.308	0.76
SOPS negative symptoms	0.391	0.024	2.295	0.03
SOPS disorganized symptoms	0.035	0.044	0.195	0.85
SOPS general symptoms	0.117	0.033	0.633	0.53
Ethnicity (Caucasian v. non-Caucasian)	−0.200	0.265	−1.272	0.21

S.E., Standard error ; SAS-SR, Social Adjustment Scale-Self Report ; HAMD, Hamilton Rating Scale for Depression ; SOPS, Scale of Prodromal Symptoms.