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Family history of psychosis as a predictor or protective factor of social maladjustment in a population at clinical high risk for psychosis

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Abstract

Literature suggests that social maladjustment is predictive of psychosis. We assessed 70 clinical high risk (CHR) patients for social maladjustment. There were no significant differences between patients with a positive or negative family history, suggesting that the relationship between social maladjustment and psychosis found in the recent literature may not translate to a relationship between social maladjustment and family history of psychosis in a CHR population.

Keywords

Prodromal; Attenuated positive symptom; psychosis-risk syndrome; Social adjustment scale

1. Introduction

The endeavor to better understand and identify the psychosis risk state, even in the clinical high risk (CHR) state or ‘prodromal’ period before onset of frank psychosis, is critical for a number of reasons, with a primary goal to develop treatments to prevent psychosis (Fusar-Poli et al., 2013).

To address the issue of recognizing elevated risk factors for conversion to psychosis in these high-risk groups, researchers have increasingly turned to measures of functioning. Social adjustment, defined as the interaction between individuals and their social environment (Weissman, 1975), has been well studied in schizophrenia (Cannon et al., 1997, Malmberg et al., 1998, Arajärvi et al., 2006). Several studies have recently shown that poor social functioning, or social maladjustment, is present in the CHR state as well (Ballon et al., 2007; Carrion et al., 2011). Clinical correlations with neurocognition, in particular processing speed, and negative symptoms have been linked to social functioning in high-risk cohorts (Carrion et al., 2011; Corcoran et al., 2011). In addition, Cannon et al. identified social dysfunction and genetic risk with functional decline as important predictors of psychosis risk (Cannon et al., 2008), underscoring the importance of this measure for characterization, prognosis, and, therefore, potentially treatment. Therefore, our prime interest in this

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investigation is the degree to which having a positive family history of psychosis is predictive of maladjustment.

We hypothesize that patients with a first-degree relative with a diagnosis of psychosis (FX+) have greater odds of maladjustment as patients without this family history. If our results support our hypothesis, it may suggest that having the genetic factor independently contributes to a patient's risk for functional impairment within the context of emergent psychosis.

2. Methods

2.1. Participants

The Center of Prevention and Evaluation (COPE) research clinic at the New York State Psychiatric Institute (NYSPI) and Columbia University Medical Center recruits patients aged 14–30 who meet CHR criteria on the Structured Interview for Prodromal Syndromes. Eligible patients meet for either 1) Attenuated Positive Symptom Psychosis Risk Syndrome (APSS); 2) Genetic Risk and Deterioration Psychosis-Risk Syndrome (GRD); and/or 3) Brief Intermittent Psychotic Symptom Psychosis-Risk Syndrome (BIPS) as assessed by the SIPS (Miller et al., 2003). Exclusion criteria include a history of psychosis beyond BIPS criteria, an IQ<70, APSS occurring solely in the context of substance use, medical or neurological disorders that can have a significant effect on the brain, and a serious risk of harm to self or others. COPE patients are mainly ascertained through referrals from schools or clinicians in the New York City area. We also included 49 healthy control subjects who were medically healthy, had no history of axis I or axis II psychiatric conditions including substance use disorders, had normal intelligence, and had no family history of psychotic disorders. Control subjects were recruited from the surrounding community via Internet ads and compensated for their participation in this study. The Institutional Review Board at NYSPI approved this study. Informed consent (or informed assent and parental consent if the patient was less than 18 years of age) was obtained before any research procedures were performed.

We had data available on social adjustment, family history and symptoms for 70 CHR patients. Incomplete data included baseline social adjustment (20) and demographic information (16). Patient information, including demographics, various symptomologies, and ranges of functioning, was collected at baseline. All information was obtained by self-report from participants.

2.2. Assessments

Family history of psychosis was ascertained using a demographic informational questionnaire, specifically asking for the patient to list “any known relative with a history of psychosis.” Social adjustment was assessed using the Spare Time section of the Social Adjustment Scale. The Social Adjustment Scale (Weissman & Bothwell, 1976) serves as a quantitative measure of social maladjustment for which adjustment categories can be looked at based on the population's score distribution. The Spare Time section is used to assess a patient's degree of social satisfaction in terms of how they use their spare time. This section

was chosen for this study as it was the only section common to all patients (e.g., the “for students” section would not have been filled out by patients working full time). Scores on the Social Adjustment Scale range from 9 (most socially-adjusted) to 57 (most socially-maladjusted).

Maladjustment was defined using the distribution of scores from the 70 patients. Patients with scores in the lowest, first quartile represented the patients with the healthiest responses on the Social Adjustment Scale (spare) and were categorized into the dichotomous well-adjusted category. The middle two quartiles of the distribution represent patients that did not stand out as being either well-adjusted or maladjusted and are designated normally-adjusted. This group served as the reference in the multinomial regression analysis. Patients with Social Adjustment Scale (spare) scores in the highest quartile gave the least healthy responses and were categorized in the binary maladjustment category.

2.3. Statistical analysis

Statistical analysis was performed using SPSS version 20. Wald chi-square tests for differences in proportions among demographic groups were calculated and Student's *t*-test was used for the continuous variable (age). An independent *t* test to compare means of controls' and patients' social adjustment scores was performed. The multinomial logistic regression assessed the odds of being FX+ in the three independent-variable categories: maladjusted, normally adjusted, and well adjusted. An adjusted version of this analysis was run for both age and gender covariates because of their hypothesized effects on respondents' access to social resources. A binomial logistic regression assessed the odds that FX+ patients would be maladjusted with a composite reference group including both normally and well-adjusted patients. Last, a linear regression model assessed the relationship between FX+ on continuous-variable adjustment.

3. Results

FX+ patients ($n=24$, 34%) and FX- patients ($n=46$, 66%) did not significantly differ from each other on demographic variables (Table 1). Results from an independent sample *t* test show significant differences between patients and controls on the adjustment measure. In addition, an independent sample *t* test comparing mean age, and chi-squared tests comparing gender, race and education, all show non-significant differences between the patient and control groups. The mean difference on the Social Adjustment Scale (spare) score between patients and controls is 9.34 ($P= <0.001$, CI=6.90–11.8).

Normally adjusted patients ($n=36$) served as the reference in the multinomial regression for maladjusted ($n=18$) and well adjusted ($n=16$) groups. Results from the multinomial logistic regression show FX+ patients to have approximately half the odds of maladjustment compared to FX- patients at a trend level (OR=0.45, $P=0.23$) and the same odds of being well adjusted at an insignificant *P* value (OR=0.94, $P=0.92$) (Table 1). Results were similar when adjusted for gender and age (Table 1). The linear regression analysis shows FX+ scoring 3.63 points better on the adjustment scale ($P=0.076$).

Results from the unadjusted binomial logistic regression model indicate at trend level that FX+ patients have half the odds of being maladjusted as compared to FX- patients (OR=0.46, $P=0.22$) (Table 1). Results were similar when adjusted for gender and age. Neither of these measures of association were statistically significant at the alpha 0.05 level (Table 1).

4. Discussion

We set out to answer the question of whether FX+ is a predictor for maladjustment in a CHR patient population. Other studies, which have examined social function in genetic high risk adolescents, who are by definition FX+ but not necessarily symptomatic, found FX+ to associate with poorer social outcomes (Freedman et al., 1998). Freedman et al. (1998) compared an FX+ high risk group, a group of subjects with parents with major affective illness, and a control group comprised of subjects with parents who have no psychiatric illness, on three measures of social outcome in adulthood ('Suspicious Solitude', 'Social Insecurity', and 'Lack of Empathy'), derived from the Personality Disorders Examination. Participants in the FX+ high risk group showed relationships to increased Suspicious Solitude, Social Insecurity and Lack of Empathy in adulthood, all of which were mediated through their scores on the Attention Deviance Index and the Physical Anhedonia Scale.

In other populations, FX+ has failed to predict social outcome measures. Cannon et al. (1997) retrospectively assessed the pre-morbid sociability scores of individuals with schizophrenia and bipolar disorder as compared to healthy controls and found bipolar patients to have had significantly lower social adjustment in childhood than controls and higher scores than schizophrenic patients obtained in their premorbid stage (Cannon et al., 1997). However, there was no relationship between FX+ and social adjustment. FX+ information was obtained by asking one informant about the psychiatric history of all his or her relatives; a similar approach of identification was utilized in our study protocol. The authors proposed that their method of FX+ identification is less effective than interviewing all family members and therefore may have contributed to insignificant power for detection of an association. Likewise, this is a limitation in our study technique.

Our data suggest that the answer to our question is no, FX+ does not appear to be a predictor of maladjustment in a CHR population. Rather, our trend-level results suggest that FX+ patients are underrepresented in the social maladjustment category compared to FX- patients in a population at risk for maladjustment. It is unclear what the mechanism involved in lowering the odds for a FX+ patient for maladjustment could be. FX+ patients may ask and receive help differently than FX- patients. This is relevant when considering COPE patients, all of whom are help-seeking. FX+ patients may be more comfortable navigating themselves within the mental health system. Psychiatric professionals may also see FX+ patients earlier in their disease course because of their additional risk factor. In addition, the family of FX+ patients may, being familiar with the disease, bring the FX+ patient into the clinic at an earlier stage of the risk state. This may be in contrast to the family of FX- patients who presumably rely more on the attenuated symptoms of psychosis and social maladjustment as primary cues to seek help for their family member. The ability of FX+ patients to seek help compared to FX- patients, who assumedly have higher thresholds of

concern, may ultimately be the protective consideration for maladjustment. Corcoran et al. (2003), in a qualitative account of CHR patients' symptom evolution, quote family members of FX+ prodromal patients as they recognize signs and symptoms similar to psychotic relatives. For instance, "One father reported that many people have noticed that his ill wife and now prodromal daughter are very similar, in terms of being very compliant, saying off the wall things, and in being inattentive to clutter" is an example that could be categorized as the father's attempt at determining the causes of the problem he perceives. In this context, we could therefore expect that such an observation could make the difference between bringing a CHR child in earlier in the disease course and not. Many additional papers have discussed the importance of social networks in the development of children of psychotic parents (Wang & Goldschmidt, 1996; Miller, 1997; Nicholson, et al., 1998; Hearle et al., 1999). However, this explanation remains speculative. This consideration may limit the generalizability of this study.

Our study was also limited by a small sample size. The attributable risk to maladjustment from family history is small enough that such an investigation would benefit from a larger sample size. Indeed, although our hypotheses were made *a priori*, the subjects in this sample were not recruited specifically for an assessment between social maladjustment and family history, and therefore this sample was not powered to observe such an effect. Other potential study limitations include recall and response bias. Younger participants may not know the psychiatric history of relatives, particularly more distant ones, and they may recall certain histories involving relatives incorrectly. Furthermore, due to the stigma of mental illness, some participants may feel the need to hide their family history of mental illness, especially if they have had previous negative experiences with the mental health system. Finally, because SAS is a self-report measure, as opposed to a clinician's assessment of the individual patient's functioning, we gain standardization in one respect, but we are limited once again by response bias.

Further research might enable more detailed answers as to whether FX+ patients are better adjusted and, if so, why this is the case. It is important, given the evidence of the benefits of early intervention, that it be better understood what brings young CHR patients to the clinic at early stages of their disease. This is especially true if we consider preventable impediments to arrival such as not being familiar with the symptoms, the severity of the disease or what kind of provider can help. This might include an assessment of types of recruitment as possible interaction terms to explore whether how patients are recruited into the study relates to their underlying adjustment level.

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Table 1

Regression analyses and tests for confounding.

	FX–	FX+	Chi-square	P-value
Gender			1.63	^a 0.25
<i>Female</i>	9 (12.9%)	8 (11.4%)		
<i>Male</i>	37 (52.9%)	16 (22.9%)		
Race			1.429	0.699
<i>White</i>	18 (25.7%)	11 (15.7%)		
<i>Black</i>	11 (15.7%)	6 (8.6%)		
<i>Asian</i>	6 (8.6%)	1 (1.4%)		
<i>Other</i>	11 (15.7%)	6 (8.6%)		
Education			4.384	0.357
<i>Less than high school</i>	9 (12.9%)	9 (12.9%)		
<i>High school and some college</i>	30 (42.9%)	13 (18.6%)		
<i>College degree or more</i>	7 (10%)	2 (2.9%)		
			Mean difference	
Age			^b 1.041	0.26
<i>Mean value</i>	20.9	19.8		
Multinomial regression				
Maladjusted vs. normally adjusted	Odds ratio	P-value	95% CI (lower)	95% CI (upper)
<i>Unadjusted (FX+)</i>	0.45	0.23	0.12	1.64
<i>FX+ adjusted for gender (female)</i>	0.37	0.15	0.10	1.45
<i>FX+ adjusted for age</i>	0.47	0.26	0.13	1.72
Well-adjusted vs. normally adjusted				
<i>Unadjusted (FX+)</i>	0.94	0.92	0.28	3.13
<i>FX+ adjusted for gender (female)</i>	0.82	0.76	0.23	2.86
<i>FX+ adjusted for age</i>	0.97	0.96	0.28	3.33
Binomial regression				
Maladjusted vs normally-+well-adjusted				
<i>Unadjusted (FX+)</i>	0.46	0.22	0.13	1.59
<i>FX+ adjusted for gender (female)</i>	0.40	0.16	0.11	1.44
<i>FX+ adjusted for age</i>	0.47	0.24	0.14	1.65

^aResult of Fisher's exact test.^bResult of Student's *t* test (equal variances assumed).