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Schizotypal Traits among Patients with Schizophrenia, Their Non-Psychotic First-Degree Relatives, and Normal Controls

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Ali Mohamadzadeh[*1]
Vahid Khosravani[2]
Nadereh Mahdavai Heris[3]

Abstract

Evidence has suggested that risk for schizophrenia is likely to occur in non-psychotic first-degree relatives of patients with schizophrenia. On the other hand, schizotypal personality disorder is genetically related to schizophrenia. The aim of this study was to compare schizotypal traits (i.e., positive schizotypy, negative schizotypy, cognitive disorganization, and impulsive nonconformity) among patients with schizophrenia and their non-psychotic first-degree relatives as well as normal controls. Thirty-four patients with schizophrenia and 50 of their non-psychotic first-degree relatives as well as 34 normal controls were included in this study. The data were collected by the Oxford-Liverpool Inventory of Feelings and Experiences, short version (sO-LIFE). The results showed that the three groups were significantly different from each other regarding mean scores of schizotypal traits. Patients with schizophrenia and their non-psychotic first-degree relatives exceeded normal controls on schizotypal traits. Patients with schizophrenia had higher scores on total schizotypal traits, positive schizotypy, cognitive disorganization, and impulsive nonconformity than their non-psychotic first-degree relatives. The findings revealed that hereditary factors had an important role in the development of schizophrenia spectrum disorders, and it is explainable by the stress-vulnerability model.

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1. *Department of Psychology, Payam-e-Noor University, Tehran, Iran, e-mail: A_mohammadzadeh@pnu.ac.ir
2. Clinical Research Development Center of Loghman Hakim Hospital, Shahid Beheshti University of Medical Sciences, Iran
3. M.A. in General Psychology, Islamic Azad University, Tabriz Branch, Tabriz, Iran. nadere.mah@gmail.com

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Introduction

Schizophrenia as a psychiatric disorder is marked by experiences such as hallucination, thought disorder, delusion, personality changes, disrupted social communications, weird behaviors, and problems in performing daily affairs (Compton & Broussard, 2009). Schizotypy as a latent personality trait is considered as the vulnerability factor for schizophrenia spectrum disorders (SSD) (Lenzenweger, 2011). This trait includes the manifestation of psychosis symptoms and deficits ranging from non-clinical and subclinical levels to complete psychosis (Kwapil & Barrantes-Vidal, 2014).

Schizotypy is characterized as a probable phenotypic determinant for the development of psychosis. Schizotypal traits are thought to be beneficial as a possible phenotypic indicator which may lead to psychosis spectrum disorders (Fonseca Pedrero & Debbané, 2017). For example, research studies conducted on the general population and clinical samples have suggested the association of schizotypal traits with psychiatric conditions, especially SSD (Debbané et al., 2014). The phenotypic manifestation of schizotypy such as schizotypal traits demonstrates the behavioral expression of this covert vulnerability and could be viewed as a clinical risk factor for psychosis (Kwapil & Barrantes-Vidal, 2014) and mental disorders (Fisher et al., 2013).

The concept of continuum between schizotypy and schizophrenia has been hypothesized and confirmed due to high prevalence of transient unusual experiences among the general population (Van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009). Schizotypal traits have been found to be associated with neurobiological dysfunctions such as deficits in cognition, sensorimotor gating, and perception; neurological soft signs; and elevated dopamine function in schizophrenia (Ettinger et al., 2015; Woodward et al., 2011).

Brosey and Woodward (2015) have suggested that psychosis patients including those suffering from schizophrenia and bipolar disorders with psychotic features had higher scores on all schizotypy scales such as the Perceptual Aberration Scale (PAS) (Chapman, Chapman, & Raulin, 1978), the Revised Social Anhedonia Scale

(RSAS) (Chapman, Chapman, & Raulin, 1976), and the Schizotypal Personality Questionnaire (SPQ) (Raine, 1991) than healthy subjects. Torgersen et al. (2002) have concluded that schizotypal patterns are high among relatives of patients with schizophrenia. Nicolson et al. (2003) have revealed that the parents of patients with schizophrenia who had early childhood-onset schizophrenia are more susceptible to SSD than those with adult-onset schizophrenia. Therefore, childhood-onset schizophrenia is significant, because there is a kind of family vulnerability for schizophrenia. Onstad, Skre, Edvardsen, Torgersen, and Kringlen (1991) have found that personality disorders and schizophrenia are prevalent among first-degree relatives of patients with schizophrenia. Also, these authors suggested that the families of patients with schizophrenia had more SSD than those of patients with mood disorder. Solanki, Swami, Singh, and Gupta (2012) have demonstrated that the first-degree relatives of patients with schizophrenia show higher schizotypal scores than normal controls. They also indicated that the first-degree relatives of patients with schizophrenia were more likely to use a variety of signs of vulnerability (nervous irritabilities), which could increase the risk of schizophrenia. Hajnal et al. (2014) have reported that social functioning impairment was high in the first-degree relatives of patients with schizophrenia and individuals at ultra-high risk for psychosis. Laurent et al. (2000) have shown that patients with schizophrenia and their non-psychotic first-degree relatives have higher levels of schizotypy including social and physical anhedonia, perceptual aberration, and magical ideation scales than normal controls. They also revealed that patients with schizophrenia had higher scores on these scales vs. their non-psychotic first-degree relatives. Horan, Reise, Subotnik, Ventura, and Nuechterlein (2008) have represented that schizotypal traits including physical anhedonia, perceptual aberrations, and magical ideation are high in patients with schizophrenia. Other studies have reported that some dimensions of schizotypy are high in patients with psychotic bipolar disorder and their unaffected relatives (Etain et al., 2007; Schürhoff, Laguerre, Szöke, Méary, & Leboyer, 2005; Schürhoff et al., 2003).

The relatively genetic nature of schizophrenia exposes some of non-psychotic family members of patients with schizophrenia to the risk of schizophrenia and pseudo-schizophrenic features. Although there were some studies in relation to assessment of schizotypal traits

among patients with schizophrenia, their first-degree relatives and normal controls, there were some reasons to perform the present research. Since cultural differences are important in the schizotypal traits (Zhao, 2016), performing research to examine schizotypal traits in a continuum of psychosis in Iranian culture is necessary. The previous studies conducted in this regard applied different scales such as the PAS (Chapman et al., 1978), the RSAS (Chapman et al., 1976), and the SPQ (Raine, 1991). Therefore, the present study addresses the schizotypal traits in patients with schizophrenia, their first- degree relatives, and normal controls by using the Oxford-Liverpool Inventory of Feelings and Experiences short version (sO-LIFE; Mason, Linney, & Claridge, 2005). The sO-LIFE is a tool that, in addition to considering the DSM criteria and the heterogeneous schizotypal nature, also covers other syndromes of schizotypy. This questionnaire is one of the most comprehensive tools developed by Mason, Claridge, and Jackson (1991) to evaluate schizotypy that measures a wide range of schizotypal scales. Therefore, the present study aimed to compare schizotypal traits among patients with schizophrenia, their non-psychotic first-degree relatives, and normal controls. According to the above- mentioned studies, we hypothesized that patients with schizophrenia and their first-degree relatives would show higher scores on schizotypal traits than normal controls (hypothesis 1). Also, we expected that patients with schizophrenia would reveal more levels of schizotypal traits compared to their first-degree relatives (hypothesis 2).

Method

Participants: Thirty-four inpatients with principal diagnoses of schizophrenia (17 females: age range = 23-75 years, mean age = 42 years, SD = 12.76; 17 males: age range = 23-75 years, mean age = 44.86 years, SD = 11.7; whole patients with schizophrenia: mean age = 43.03 years, SD = 12.1) were selected from Razi hospital, Tabriz, Iran for this study. The psychiatric diagnoses were determined by a clinical psychologist (Dr. A.M) based on the Structured Clinical Interview for DSM-IV-TR Axis I Disorders-Patient Edition (SCID-I/P)(First, Spitzer, Gibbon, & Williams, 2002). Neurological and medical diseases, substance abuse, psychosis NOS or psychosis due to general medical conditions, the presence of active psychosis at the time of assessment, and any current comorbid Axis I disorders were

exclusion criteria for patients diagnosed with schizophrenia. All patients received antipsychotic medication at the time of participation in the study. Fifty non-psychotic first-degree relatives of patients with schizophrenia (29 females and 21 males: age range = 18-81 years) and 34 community controls (17 females and 17 males: age range = 27-62 years) were selected. They had no previous or current history of psychiatric or neurological diagnoses based on the Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Non-Patient Edition (SCID-I/NP) (First, Spitzer, Gibbon, & Williams, 2002) and the General Health Questionnaire (Goldberg & Williams, 2000)

Measures: The Oxford-Liverpool Inventory of Feelings and Experiences short version (sO-LIFE) (Mason et al., 2005): The sO-LIFE is a 43-item scale (yes/no format) to evaluate schizotypy in clinical and non-clinical samples. It assesses positive schizotypy (12 items for unusual experiences), negative schizotypy (10 items for introvertive anhedonia), cognitive disorganization (11 items), and impulsive nonconformity (10 items). Previous studies have shown that the sO-LIFE is a valid scale (Fonseca-Pedrero et al., 2015; Sierro, Rossier, Mason, & Mohr, 2015). The Cronbach s alphas for positive schizotypy, negative schizotypy, cognitive disorganization, and negative schizotypy were 0.89, 0.82, 0.87, and 0.77 respectively (Mason et al., 2005). In the current study, the Persian version of the sO-LIFE (Yaghoubi & Mohammadzadeh, 2012) was used. The Cronbach s alpha for the Persian version of the sO-LIFE was 0.82. The Cronbach slpha for the scale in the present research was 0.86.

The Positive and Negative Syndrome Scale (PANSS) (Kay, Fiszbein, & Opfer, 1987): The PANSS is a 30-item semi-structured interview which is performed by a specialist researcher. It is most widely used to measure positive and negative symptoms of psychosis. In the study, the Persian version of the PANSS (Ghamari Givi, Moulavi, & Heshmati, 2010) was used. Ghamari Givi et al. (2010) have shown that the Persian version of the PANSS has adequate construct validity in the diagnosis and clinical studies.

General Health Questionnaire (GHQ) (Goldberg & Williams, 2000): The 28-item GHQ is a screening scale to detect individuals with psychiatric disorders. It measures four subscales including somatic symptoms, anxiety/insomnia, social dysfunction, and severe depression. All items are rated on a scale ranging from 0 to 3. This screening questionnaire has cut-off scores of 23 or higher,

representing one s mental health problems (Sterling, 2011). We used the Persian version of the GHQ (Nazifi et al., 2014) in this study for screening normal controls. The Cronbach s alpha for the GHQ was 0.92 (Nazifi et al., 2014). The Cronbach s alpha for the GHQ in the current research was 0.91.

Statistical analyses

The socio-demographic and clinical characteristics of patients with schizophrenia, their first-degree relatives, and normal controls were compared using chi-square and the analysis of variance (ANOVA). To compare schizotypal traits among the three groups, the multivariate analysis of variance (MANOVA) was applied. Additionally, the power analysis was computed using the effect size partial eta square (η^2). A small effect is rated for η^2>0.01, a medium effect for η^2>0.06, and a large effect for η^2>0.14. The results of the Kolmogorov-Smirnov test revealed that the data were normal. There were no missing values. The data confirmed the assumptions of performing ANOVA and MANOVA. The reliability of the applied scales was assessed using the Cronbach's alpha. Data were analyzed using SPSS® version 22.0 for Windows. P<0.05 was considered statistically significant and all tests were two-tailed. The false discovery rate (FDR) correction was used to control type 1 error inflation (Benjamini & Hochberg, 2000).

Results

Demographic and clinical characteristics of participants are presented in Table 1. The results revealed no differences among the three groups regarding gender. Patients with schizophrenia were older than their relatives and normal controls (p<0.05). Patients with schizophrenia and their relatives had significantly lower education and higher levels of being single compared to normal controls (p<0.001). Also, the schizophrenic group showed lower education and higher levels of being single vs. their relatives and normal controls (p<0.001).

Table 1. Demographic and clinical characteristics of patients with schizophrenia, their first-degree relatives, and normal controls (mean ± SD).

Characteristics				Statistics	
	Schizophrenic group (*n*= 34)	First-degree relatives group (*n*=50)	Normal control group (*n*=34)	*F* or χ^2	*p*
Male/Female, *n*	17/17	21/29	17/17	4.30	0.06
Age, year	44.86±11.07	42.86±15.04	42.17±7.48	121.22	<0.05*
Education level				32.80	<0.001**
Primary school	19 (55.9%)	19 (38%)	4 (11.7%)		
High school graduate (diploma)	12 (35.3%)	19 (38%)	9 (26.5%)		
College graduate or higher	3 (8.8%)	12 (24%)	21 (61.8%)		
Marital status				32.20	<0.001**
Single, *n* (%)	22 (64.7%)	17 (34%)	0		
Married, *n* (%)	12 (35.3%)	33 (66%)	34 (100%)		
Positive symptoms	16.76±4.99	-	-	-	-
Negative symptoms	21.39±7.59	-	-	-	-

* $p<0.05$.
** $p<0.001$.

The results of MANOVA indicated significant differences among the three groups (Wilks $\lambda=0.53$; $F = 10.32$; $p<0.001$). Similarly, the findings indicated that patients with schizophrenia and their relatives had higher scores on total schizotypal traits ($F=36.75$, $p<0.001$) than normal controls. Both patients with schizophrenia and their relatives exceeded normal controls on negative schizotypy ($F=15.58$), cognitive disorganization ($F=33.26$), and impulsive nonconformity ($F=26.62$) ($p<0.001$). Patients with schizophrenia gained higher scores on positive schizotypy ($F=9.49$, $p<0.001$) vs. normal controls. Also, the schizophrenic group presented more severe positive schizotypy, cognitive disorganization, and impulsive nonconformity than their relatives ($p<0.001$) (Table 2). In addition, the effect sizes for total schizotypal traits ($\eta^2=0.39$), positive schizotypy ($\eta^2=0.14$), negative schizotypy ($\eta^2=0.21$), cognitive disorganization ($\eta^2=0.37$), and impulsive nonconformity ($\eta^2=0.32$) were large ($\eta^2>0.14$). Therefore, according to the results, hypotheses 1 and 2 were confirmed.

Table 2. Comparison of schizotypal traits among patients with schizophrenia, their non-psychotic first-degree relatives, and normal controls (mean ± SD).

	Patients with schizophrenia (*n*=34)	First-degree relatives (*n*=50)	Normal controls (*n*=34)	*F* [a]	Post-hoc [b]	η^2 [c]
Total schizotypal traits	22.55±6.71	14.08±8.23	7.28±5.55	36.75*	SC> Relatives>Normal	0.39
Positive schizotypy	5.38±3.47	3.22±2.93	2.35±2.44	9.49*	SC>Relatives=Normal	0.14
Negative schizotypy	5.44±2.24	5.42±2.77	4.10±2.66	15.58*	SC=Relatives>Normal	0.21
Cognitive disorganization	7.32±2.55	4.06±3.17	1.97±2.13	33.26*	SC>Relatives>Normal	0.37
Impulsive nonconformity	4.41±1.86	2.38±2.03	1.17±1.52	26.62*	SC> Relatives>Normal	0.32

Note.
SC: schizophrenia group.
[a] Analysis of variance (ANOVA).
[b] With the Tukey HSD correction.
[c] Effect sizes were assessed by partial eta squared (η^2).
* p<0.001.

Discussion & Conclusions

This study aimed to compare schizotypal traits among patients with schizophrenia, their non-psychotic first-degree relatives, and normal controls. Our results showed that patients with schizophrenia and their first-degree relatives had higher schizotypal traits than normal controls. Also, patients with schizophrenia had more severe schizotypal traits and their related dimensions such as positive schizotypy and impulsive nonconformity compared to their first-degree relatives. These findings were in line with previous studies (Brosey & Woodward, 2015; Etain et al., 2007; Hajnal et al., 2014; Horan et al., 2008; Laurent et al., 2000; Nicolson et al., 2003; Onstad et al., 1991; Schürhoff et al., 2005; Schürhoff et al., 2003; Solanki et al., 2012; Torgersen et al., 2002). Our results were also consistent with prior studies (Goulding, 2004; van Kampen, 2006), suggesting that schizotypy is a mild and non-clinical form of schizophrenia. Schizotypy was found to be a vulnerability factor for schizophrenia (Rossi & Daneluzzo, 2002). This vulnerability is characterized by neurological, neuro-biological, psychiatric, and neuro-psychological

damages in first-degree non-psychotic relatives of patients with schizophrenia (Tsuang, Stone, Tarbox, & Faraone, 2002). Schizotypal personality disorder is commonly found in relatives of patients with schizophrenia, and in some individuals, the schizotypal personality disorder is a symptom of the onset of schizophrenia (Moreno-Izco et al., 2015; Wolfradt & Straube, 1998). Moreno-Izco et al. (2015) have demonstrated that patients with schizophrenia report higher scores on the schizotypal personality questionnaire-brief than their siblings (Raine & Benishay, 1995) as a measure of schizotypal personality disorder. As previously mentioned, previous studies have examined schizotypal traits, but our study applied a widely used tool that meets DSM standards. Therefore, measuring a factor using various tools can provide comprehensive information. So far, no research has been performed on the comparison of schizotypal traits among patients with schizophrenia, their non-psychotic first-degree relatives, and normal controls in Iranian population. As previously mentioned, culture has a significant effect on the quality of schizotypal traits. Therefore, the present study evaluated the schizotypal traits in Iranian society using a widely used tool in accordance with DSM criteria.

Schizotypy was found to be high in patients with SSD and their relatives (Brosey & Woodward, 2015; Calkins, Curtis, & Grove, 2004; Moreno-Izco et al., 2015). Furthermore, it was shown that both schizotypy and schizophrenia co-occur in the same families (Mata et al., 2000). It was also suggested that normal parents of patients with schizophrenia who have a family history of SSD revealed higher schizotypal traits vs. those without a family background (Appels, Sitskoorn, Vollema, & Kahn, 2004). Soler et al. (2017) have concluded that schizotypy is a vulnerability factor for SSD that occurs in some family members. Previous studies have found that schizotypy is a genetically influenced disorder, with approximately 50% heritability (Linney et al., 2003).

The findings of the present study are explainable through the stress-vulnerability model. According to this model, psychosocial borderline features (schizotypal and borderline traits) and schizophrenia have been conceptualized as different points on a continuum, suggesting different degrees of risk for psychosis. Although psychotic disorders such as schizophrenia have undoubtedly biologic origins, psychological factors are important in their onset and duration (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001;

Kinderman, 2005). The potential interactions of stress experiences and the course of psychosis are of particular interest. The stress-vulnerability model (Zubin & Spring, 1977) suggests that stress experiences play a pivotal role in the onset of acute psychosis. Based on this model, vulnerability as well as internal and physical preparedness in the interaction with external and internal stressors contributes to the development of psychotic disorders. According to Zubin and Spring (1977), each individual has a degree of vulnerability in him/herself which will manifest periodically as schizophrenia under favorable conditions.

The stress-vulnerability model of schizophrenia explains how the symptoms of schizophrenia develop over time (Rudnick & Lundberg, 2012). It seems that unmanageable stressful experiences leading to distress and anxiety may accelerate the manifestation of psychotic symptoms in individuals who have already been highly vulnerable. Also, this model may provide a possible explanation for some of the other unexplained features of psychotic disorders. Patients with psychosis also refer to the relationship among experiencing stressors, discomfort level, and psychotic symptoms. The stress-vulnerability model suggests the possibility of treating symptoms and preventive interventions, especially through promoting psychological management strategies and adaptation to stress. For example, British researchers have developed a cognitive-psychological model of the role of stressors in the creation and maintenance of psychotic symptoms (Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002; Garety et al., 2001). These researchers proposed that the experience of being victim may lead individuals to believe their vulnerability and weakness, and that the world and others are violent and threatening. Next stressful events seem to trigger psychotic symptoms under such conditions (Freeman et al., 2002; Garety et al., 2001). Recent studies which had implications for treatment supported this model (Dudley & Over, 2003; Freeman et al., 2004). In addition, in relation to genetic factors, Walter et al. (2016) have shown that schizotypal traits are enhanced with elevated genetic proximity to schizophrenia and that some chromosomal regions are related to schizotypy. These authors also found that genes connected to schizophrenia created the initial start point for the study of candidate genes for schizotypal traits.

This research had some limitations. First, conclusions drawn from an inpatient sample may limit generalization to samples in non-acute

phases of illness. For example, the use of antipsychotic drugs reduces the severity of patients symptoms and makes them embarrassed to talk about their previous extraneous and exotic behaviors, and thus they deny their symptoms (as a defensive behavior). Further research is suggested to be conducted by controlling for the effects of medications on the evaluation of schizotypal traits in patients with schizophrenia and their families in order to further clarify the findings of the present study. Second, in the present research, the subtypes of schizophrenia were not investigated because of limited sample size. Further research is needed to be conducted on the differentiation of schizophrenic subtypes concerning schizotypal personality traits in patients with schizophrenia and their families. Third, this study was a cross-sectional design which does not allow for an examination of causation and longitudinal research. Forth, we did not control the effects of depression as a covariate. Fifth, another limitation concerns the use of self-report measures. Finally, this research was limited to a scale for the assessment of schizotypal traits named the sO-LIFE (Mason et al., 2005) and it did not include additional important scales of schizotypy traits such as the PAS (Chapman et al., 1976) and the SPQ (Raine, 1991).

Regardless of these limitations, the present research may have implications for secondary prevention. Since first-degree relatives of patients with schizophrenia are at risk for schizotypal and borderline pseudo-psychosis disorders and possibly a complete psychosis of schizophrenia, providing supportive care for the relatives of these patients and, if possible, more accurate assessment on the likelihood of developing schizophrenia in certain time periods are suggested.

The findings indicated that schizotypal traits are vulnerability factors for developing schizophrenia, and heredity is an important factor in schizophrenia.

Compliance with Ethical Standards

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or non-profit sectors.

Ethical Approval: This article has been extracted form an M.A. thesis in general psychology from Tabriz Azad University. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from all individual participants included in the study.

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