SCHIZOTYPAL TRAITS IN UNAFFECTED RELATIVES OF PATIENTS IN THE SCHIZOPHRENIA SPECTRUM

Chrysoula Zouraraki, Leda Karagiannopoulou, Penny Karamaouna, & Stella G. Giakoumaki
University of Crete, Greece

Abstract: Schizotypy is defined as liability to schizophrenia, paralleling schizophrenic symptoms. Individuals with a family history of schizophrenia are at increased risk for illness development and their evaluation for schizotypy offers the advantage of examining the background of schizophrenia, while avoiding confounding variables. In this review, research findings regarding the expression of different schizotypal dimensions in unaffected relatives of patients with a diagnosis in the schizophrenia spectrum are summarized. The main findings indicate that first degree relatives express elevated negative schizotypy but results regarding positive and disorganized schizotypal dimensions are less established. Methodological considerations and future directions are discussed.

Keywords: schizotypy, schizotypal traits, relatives of patients in schizophrenia spectrum

INTRODUCTION

Schizophrenia is a complex psychiatric disorder, characterized by social (Dickerson, Boronow, Ringel, & Parente, 1999), cognitive (Heinrichs & Zakzanis, 1998) and generalized functional impairment (Patterson, Goldman, McKibbin, Hughes, & Jeste, 2001). It manifests itself with a significant heterogeneity of clinical symptoms, disease course and outcome (Heckers, 2009) and has a multifactorial etiology, with multiple susceptibility genes interacting with environmental influences (Siever & Davis, 2004). One way to approach the liability to schizophrenia, avoiding the effects of confounding variables such as medication, institutionalization and illness chronicity, is...
via the study of schizotypal traits in genetic high-risk populations, such as the unaffected first degree relatives of patients.

Schizotypy is a multidimensional concept related to the symptom clusters in schizophrenia (Bentall, Claridge, & Slade, 1989; Vollema & Van den Bosch, 1995) as the three schizotypal (i.e., positive, negative, disorganization) factors are considered to be analogous to the schizophrenic symptoms (reality distortion, psychomotor poverty, disorganization) introduced by Liddle (1987). In detail, positive schizotypal traits are similar to the positive symptoms of schizophrenia and include oddities in perception, magical thinking, ideas of reference and suspiciousness/paranoia; negative schizotypal traits are analogous to the negative symptoms of schizophrenia and refer to impoverished interpersonal relationships, constricted affect, excessive social anxiety; disorganized schizotypal traits include unusual speech and eccentric behavior and resemble the bizarre behavior and formal thought disorder observed in schizophrenia. Schizotypy is classically studied through two different approaches, namely the fully dimensional and the quasi-dimensional models. The fully dimensional model was formulated by Claridge (1997) and is based on the idea that schizotypy is a deviant but non-pathological personality trait, which indicates predisposition to psychosis, when exceeding a critical threshold. According to the quasi-dimensional model, schizotypy indicates proneness to psychosis and is part of the schizophrenia-spectrum (Meehl, 1962). This psychiatric viewpoint is based on the clinical observations of Rado (1953), who coined the term “schizotypy”; later on, Meehl (1962) proposed a model for the pathogenesis of schizophrenia, suggesting that both schizotypal and schizophrenic individuals share a common neurodevelopmental vulnerability path.

Schizotypal traits are assessed either with self-report questionnaires or through clinical and structured interviews. According to Vollema and Van den Bosch (1995) and Bentall et al. (1989) the psychometric scales for assessing schizotypy are heterogeneous and can be differentiated into (a) symptom-oriented, such as the Perceptual Aberrations scale, Social and Physical Anhedonia scales (Chapman, Chapman, & Raulin, 1976; Chapman, Chapman, & Raulin, 1978), Magical Ideation (Eckblad & Chapman, 1983) and Impulsive Nonconformity scales (Chapman et al., 1984); (b) syndrome-oriented, such as the Schizotypal Personality Questionnaire (SPQ; Raine, 1991) and the Schizotypal Personality Scale (STA; Claridge & Broks, 1984) and (c) personality-oriented, such as the Eysenck Personality Questionnaire (EPQ; Eysenck, Eysenck, & Barrett, 1985) and the Minnesota Multiphasic Personality Inventory (MMPI) profile for schizotypal traits (Lachar, 1974). Clinical interviews, though, are considered more sensitive for the evaluation of schizotypal traits than self-report questionnaires (Catts, Fox, Ward, & McConaghy, 2000). Some widely used interviews for the evaluation of schizotypal symptoms are the Structured
Clinical Interview for DSM Axis II Disorders - module for schizotypal personality disorder (SCID; Spitzer, Williams, Gibbon, & First, 1990), the Interview for Prodromal Schizophrenia Syndromes (SIPS; Miller et al., 2002), and the Structured Interview for Schizotypy (SIS; Kendler, Lieberman, & Walsh, 1989).

As regards the etiology of schizotypy, although it is not fully clarified in the literature, a significant percentage (approximating 50%) is explained by genetic (Linney et al., 2003) and neuroanatomical factors (Diwadkar, Montrose, Dworakowski, Sweeney, & Keshavan, 2006; Rosso et al., 2010) related to schizophrenia (Ettinger, Meyhöfer, Steffens, Wagner, & Koutsouleris, 2014; Kendler et al., 1993). It is not surprising, therefore, that schizotypal traits are increased in both clinical (Rosell, Futterman, McMaster, & Siever, 2014) and genetic high-risk groups, such as the unaffected relatives of patients (Solanki, Swami, Singh, & Gupta, 2012). Interestingly, it has also been reported that the prevalence of positive or negative symptoms in schizophrenic probands predicts the type of schizotypal features in their unaffected relatives (Fanous, Gardner, Walsh, & Kendler, 2001). However, important environmental parameters, either “biological”, such as pre- and perinatal complications, winter/autumn birth (Lahti et al., 2009), low birth weight, obstetric complications (Foerster, Lewis, Owen, & Murray, 1991), or “psychosocial”, such as childhood trauma (Schürhoff et al., 2009), physical or sexual abuse (Steel, Marzillier, Fearon, & Ruddle, 2009), parental communication deviance (de Sousa, Varese, Sellwood, & Bentall, 2014), and sub-optimal parenting (Giakoumaki et al., 2013) have also been reported to increase the risk for the development of schizotypal traits. The interplay between genetics and environment is well documented in a recent study by Walder, Faraone, Glatt, Tsuang, and Seidman (2014) who proposed a ‘polygenic neurodevelopmental diathesis-stress model’. According to this model, the susceptibility for psychosis “involves the independent and synergistic confluence of (temporally-sensitive) biological and environmental factors across development” (p. 142).

Evidence about the familial aggregation of schizophrenia-like features was consistently provided by the Danish Adoption studies of Kety, Rosenthal, Wender, and Schulsinger (1968). Based on these studies Spitzer, Endicott, and Gibbon (1979) proposed the criteria (i.e., magical thinking, ideas of reference, social isolation, recurrent illusions, odd speech, inadequate rapport, suspiciousness and undue social anxiety) for a new diagnostic entity termed Schizotypal Personality Disorder (SPD), which was included in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980). These criteria were retained in the revisions of DSM (DSM IV, American Psychiatric Association, 1994 and DSM IV-TR, American Psychiatric Association, 2000) that followed. However, Section III of DSM 5th Edition (DSM-5, American Psychiatric Association,
2013) recently introduced significant changes in the diagnosis of SPD. More specifically, a new hybrid model introducing six personality disorder types, including SPD, was proposed. According to this model, personality traits are found in different degrees in every individual and personality disorders represent broad personality domains, which consist of specific maladaptive personality facets. The domains of Psychoticism (i.e., extensive range of odd behaviors/cognitions) and Detachment (i.e., avoidance of social/emotional experiences) are characteristic of the schizotypal personality and are adequately examined by all the scales assessing schizotypy: Positive and Disorganized Schizotypal traits are closely connected with Psychoticism and Negative Schizotypy maps well onto Detachment. Another significant addition in DSM 5 is the inclusion of Attenuated Psychosis Syndrome, which refers to the existence of “state-induced” sub-threshold psychosis-like experiences, which resemble closely schizotypal traits, accompanied by functional impairment. Having a family history of psychosis increases the risk of individuals with attenuated psychosis syndrome for developing psychotic disorders (American Psychiatric Association, 2013). Based on these innovative changes introduced in DSM 5, it is evident that the study of schizotypy in first-degree relatives of schizophrenia patients can further aid clinical practice in two ways: (a) more precise diagnostic criteria aiming to the early identification of individuals at risk for developing psychotic disorders can be developed and (b) both protective and compensating factors that could be incorporated into therapeutic approaches can be recognized.

To this end, although it is has been repeatedly reported (Bora & Veznedaroglu, 2007; Calkins, Curtis, Grove, & Iacono, 2004; Docherty, Sponheim, & Kerns, 2015) that unaffected relatives of schizophrenia patients present with elevated schizotypal traits overall, studies examining which schizotypal dimension (i.e., Positive, Negative or Disorganized) prevails as well as studies examining the severity of schizotypal traits in this group of subjects have yielded discrepant findings. The aim of this review is to summarize these findings in order to further elucidate the topic. For this reason, we performed a PubMed and Scopus search with combinations of the keywords “schizotypal traits”, “schizotypy”, “schizotypal personality”, “relatives”, “parents”, “siblings”, “offspring”, “familial risk for schizophrenia”, “positive schizotypy”, “negative schizotypy”, “disorganized schizotypy”. The search covered publications from 1990 to August 2015. Studies were selected if they met the following criteria: written in English, participants were unaffected first degree relatives of patients within the schizophrenia spectrum, schizotypal traits were assessed with either psychometric self-report questionnaires or clinical interviews, there was a control group and/or a group of patients to be compared with the group of relatives. Based on these criteria, we identified twenty three studies, summarized in Table 1.
### Table 1. Summary of reviewed studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample</th>
<th>Schizotypy Assessment</th>
<th>Group Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appels et al. (2004)</td>
<td>Rel: N = 72; Con: N = 52</td>
<td>SPQ</td>
<td>CPS, Delusional atmosphere, Magical ideation: Rel &lt; Con POS and NEG: Rel with FH &gt; Rel without FH All p values &lt; .05</td>
</tr>
<tr>
<td>Bollini et al. (2007)</td>
<td>Rel: N = 26; Con: N = 38</td>
<td>SCID Interview; SPQ</td>
<td>DIS: Con &gt; Rel; p &lt; .05</td>
</tr>
<tr>
<td>Bora et al. (2007)</td>
<td>Rel: N = 94; Con: N = 75</td>
<td>SPQ-B</td>
<td>SPQ Total and INT: Rel &gt; Con All p values &lt; .005</td>
</tr>
<tr>
<td>Calkins et al. (2004)</td>
<td>Rel: N = 135; Con: N = 112</td>
<td>SPQ; MMPI-2: Lie/defensiveness; PRF: Infrequency</td>
<td>SPQ Total, INT, Social Anxiety, Constricted Affect, No Close Friends, Unusual Perceptual Experiences: Rel &gt; Con All p values &lt; .05</td>
</tr>
<tr>
<td>Catts et al. (2000)</td>
<td>Rel: N = 46; Con1: Healthy N = 38/Con2: Parents of patients with axis I diagnosis N = 40</td>
<td>PAS; PhysAnh; EPQ Psychoticism Scale</td>
<td>Psychoticism: Rel &gt; Con1 Neuroticism: Con2 &gt; Rel PAS: Rel &lt; Con1 All p values &lt; .05</td>
</tr>
<tr>
<td>Clementz et al. (1991)</td>
<td>Rel: N = 148; Con: N = 178</td>
<td>PAS; PhysAnh</td>
<td>PAS: Con &gt; Rel PhysAnh: Rel &gt; Con All p values &lt; .001</td>
</tr>
<tr>
<td>Compton et al. (2007)</td>
<td>Rel: N = 61; Con: N = 57</td>
<td>SPQ-B</td>
<td>CPS, INT and DIS: Rel = Con</td>
</tr>
<tr>
<td>Craver et al. (1999)</td>
<td>Rel: N = 39; Con1: Control Siblings; N = 38; Con2: Control Proband: N = 38</td>
<td>SANS; Revised SocAnh</td>
<td>Negative SANS Symptoms and SocAnh: Rel = Con1 and Rel = Con2</td>
</tr>
<tr>
<td>De la Serna et al. (2011)</td>
<td>Rel: N = 56; Con: N = 33</td>
<td>SIPS</td>
<td>Positive, Disorganized, General and Total Symptoms: Rel &gt; Con All p values &lt; .05</td>
</tr>
<tr>
<td>Docherty et al. (2015)</td>
<td>Rel: N = 33; Con: N = 25</td>
<td>SAE</td>
<td>SocAnh and PAS: Rel &gt; Con All p values &lt; .05</td>
</tr>
<tr>
<td>Franke et al. (1993)</td>
<td>Rel:N=26; Con: N=35</td>
<td>PhysAnh; PAS</td>
<td>PhysAnh: Rel &gt; Con; p &lt; .05</td>
</tr>
<tr>
<td>Glatt et al. (2006)</td>
<td>Rel: N = 35; Con: N = 55</td>
<td>MIS; PAS; PhysAnh</td>
<td>PhysAnh: Rel &gt; Con; p &lt; .005</td>
</tr>
<tr>
<td>Groove et al. (1991)</td>
<td>Rel: N = 61; Con: N = 18</td>
<td>SSP; PhysAnh; PAS; MMPI</td>
<td>SocInt and PhysAnh: Rel &gt; Con All p values &lt; .05</td>
</tr>
<tr>
<td>Katsanis et al. (1990)</td>
<td>Rel: N = 125; Con: N = 117</td>
<td>PhysAnh; SocAnh; PAS</td>
<td>PhysAnh and SocAnh: Rel &gt; Con PAS: Rel &lt; Con All p values &lt; .05</td>
</tr>
<tr>
<td>Kendler et al. (1995)</td>
<td>Rel: N = 314; Con: N = 575</td>
<td>SIS</td>
<td>Negative/Positive schizotypy, Social Dysfunction, Avoidant Symptoms, Odd Speech, Suspicious Behavior: Rel &gt; Con All p values &lt; .005</td>
</tr>
</tbody>
</table>

Continued
Table 1. (Continued)

<table>
<thead>
<tr>
<th>Study</th>
<th>Rel: N =</th>
<th>Con: N =</th>
<th>Measure</th>
<th>Schizotypy Levels:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kimble et al. (2000)</td>
<td>15</td>
<td>15</td>
<td>SIS</td>
<td>Rel = Con</td>
</tr>
<tr>
<td>Kremen et al. (1998)</td>
<td>40</td>
<td>44</td>
<td>SPQ</td>
<td>CPS: Rel &gt; Con; p &lt; .01</td>
</tr>
<tr>
<td>Laurent et al. (2000)</td>
<td>47</td>
<td>34</td>
<td>PhysAnh; SocAnh; PAS; MIS</td>
<td>PAS, PhysAnh, SocAnh, MIS: Rel = Con</td>
</tr>
</tbody>
</table>
| Mata et al. (2000)     | 121      | 90       | KSQ; SAE; Modified IPDE for Cluster A Personality Disorders | Negative Schizotypy, Anhedonia, Having no close friends, Constricted Affect: M Rel > F Rel Schizotypal traits in SCZ patients correlated with KSQ Score in Rel and SAE All p values < .05.
| Mata et al. (2003)     | 263      | 172      | KSQ; SAE; Modified IPDE for Cluster A Personality Disorders | Anhedonia, Inappropriate Affect, Paranoid traits, No close friends: M Rel > F Rel IPDE Schizotypal traits: F Rel > M Rel Delusions-Hallucinations Syndrome in patients correlated positively with the three measures for schizotypy in Relatives All p values < .05 |
| Solanki et al. (2012)  | 50       | 30       | SPQ-B   | SPQ-B Total Score, CPS, INT: Rel > Con All p values < .001 |
| Yaralian et al. (2000) | 13/38    | 13/38    | SPQ     | CPS: Rel > Con1 and Rel > Con2 Ideas of Reference and Unusual Perceptual Experiences: Rel > Con1 All p values < .05 |
| Vollema et al. (2002)  | 117      | 51       | SPQ     | CPS: Siblings and Offsprings > Parents All p values < .05 |

Con = Controls; Rel = Relatives; M = Males; F = Females; SCZ = Schizophrenia, SCT = Schizotypy; FH = Family History; SPQ = Schizotypal Personality Questionnaire; SPQ-B = Schizotypal Personality Questionnaire Brief Version; CPS = Cognitive-Perceptual Schizotypy Factor; INT = Interpersonal Schizotypy Factor; DIS = Disorganized Schizotypy Factor; POS = Positive Schizotypy; NEG = Negative Schizotypy; SCID Interview = Structured Clinical Interview for DSM-Module for schizotypal personality disorder; PRF = Personality Research Form; MMPI-2 = Minnesota Multiphasic Personality Inventory-2; EPQ = Eysenck Psychoticism Scale; SANS = Scale for the Assessment of Negative Symptoms; SIPS = Semi-structured Interview for Prodromal Schizophrenia Syndromes; SocInt = Social Interpersonal Factor; SSP = Schedule for Schizotypal Personalities; SIS = Structured Interview for Schizotypy; MIS = Magical Ideation Scale; PAS = Perceptual Aberration Scale; SocAnh = Social Anhedonia Scale; PhysAnh = Physical Anhedonia Scale; IPDE = Interview for Cluster A' Personality Disorders Examination; SAE = Survey of Attitudes and Experiences Scale; KSQ = Kings Schizotypy Questionnaire.
Positive schizotypal traits

Positive schizotypal traits include symptoms such as magical ideation, ideas of reference, and unusual perceptual experiences (Raine, 1991). De la Serna et al. (2011) examined both offsprings and siblings of schizophrenic patients for positive, negative and disorganized symptoms, using the SIPS. Offsprings manifested higher positive symptoms in comparison with control subjects, while siblings manifested an increase only in disorganized symptoms. Studies employing the SPQ (Raine, 1991) have also revealed that relatives score significantly higher on the Positive Schizotypy factor compared with healthy controls (Kremen, Faraone, Toomey, Seidman, & Tsuang, 1998; Solanki et al., 2012; Yaralian et al., 2000). Vollema, Sitskoorn, Appels, and Kahn (2002) compared parents, siblings and offsprings of schizophrenic patients with the SPQ and found that both siblings and offsprings scored higher on positive schizotypy than parents. Laurent et al. (2000) and Docherty et al. (2015) administered the Chapman’s scales (Chapman, Chapman & Raulin, 1978) and found a trend towards increased perceptual aberrations. Finally, in a study with the SIS Kendler, McGuire, Gruenberg, and Walsh (1995) reported increased positive schizotypy in the relatives’ group.

Despite the aforementioned results, many studies support that either there is no difference in positive schizotypal traits or that relatives express lower positive schizotypal levels than controls. Thus, Appels, Sitskoorn, Vollema, and Kahn (2004) assessed parents of patients with schizophrenia and control parents of healthy individuals using the SPQ. They found that parents of patients scored lower on cognitive-perceptual schizotypy (an aspect of positive schizotypy), especially on the magical ideation subscale. Bollini et al. (2007) administered the SPQ and the SCID-II and found that relatives of schizophrenic patients and control individuals did not differ in either the number of cognitive-perceptual features or on the total score on the cognitive-perceptual dimension of the SPQ. Non-significant differences between relatives and controls in the expression of positive schizotypal traits have also been reported with the SPQ-Brief (Compton, Chien, & Bollini, 2007), the Perceptual Aberration Scale (Catts et al., 2000; Clementz, Grove, Katsanis, & Iacono, 1991; Katsanis, Iacono, & Beiser, 1990) and the SIS (Kimble et al., 2000).

Negative Schizotypal Traits

Negative schizotypy refers to sub-threshold symptoms such as constricted affect, excessive social anxiety and no close friends (Raine, 1991). There are many studies assessing the expression of negative schizotypy in unaffected relatives of
schizophrenic patients, using the Physical Anhedonia scale (Clementz et al., 1991; Franke, Maier, Hardt, Hain, & Cornblatt, 1993; Glatt, Stone, Faraone, Seidman, & Tsuang, 2006; Grove et al., 1991). All these studies concluded that this group has significantly elevated Physical Anhedonia compared with healthy controls. Similar findings have also been reported for Social Anhedonia (Docherty et al., 2015; Katsanis et al., 1990). Two studies (Mata et al., 2000; Mata et al., 2003) employed the Kings Schizotypy Questionnaire (KSQ; Williams, 1993) and the Survey of Attitudes and Experiences Scale (SAE; Wilkins, 1988) for the assessment of schizotypy in parents and siblings of patients. They both found elevated scores on KSQ Negative Schizotypy and on SAE Anhedonia with the male parents’ and siblings’ groups. Interestingly, high rates of premorbid schizoid-schizotypal traits in the schizophrenic probands were correlated with high total scores on the KSQ and the SAE in their relatives (Mata et al., 2000). Similar findings have been obtained with the SPQ, indicating that unaffected relatives have higher negative schizotypy compared with controls (Bora & Veznedaroglu 2007; Calkins et al., 2004; Solanki et al., 2012). Finally, in one study with the SIS, Kendler et al. (1995) also reported increased negative schizotypy in the relatives’ group.

As with positive schizotypal traits, there are also studies which report non-significant differences between unaffected relatives and controls in negative schizotypy as assessed with the Scale for the Assessment of Negative Symptoms (SANS) and the Social Anhedonia Scale (Craver & Pogue'Qeile, 1999), SIS (Kimble et al., 2000), SPQ and SCID-II (Bollini et al., 2007) as well as SPQ-Brief (Compton et al., 2007).

**Disorganized schizotypal traits**

Disorganized Schizotypy includes odd speech and odd/eccentric behavior (Raine, 1991). De la Serna et al. (2011) assessed offsprings and siblings of schizophrenic patients with the SIPS and found significantly increased disorganized symptoms. Moreover, Kendler et al. (1995) supported that relatives of schizophrenic probands can be distinguished from control subjects by their scores in odd speech, which is a trait highly associated with disorganization (Raine, 1991). In a study by Bollini et al. (2007), however, the control group had higher disorganized traits than the unaffected relatives, possibly due to the inclusion of a biased group of relatives, expressing fewer schizotypal traits. Finally, Compton et al. (2007) administered the SPQ-Brief and reported no differences between the group of relatives and controls.
DISCUSSION

The majority of the studies reviewed suggests that unaffected first-degree relatives of schizophrenia patients present with higher schizotypal traits compared to control individuals without a family history of psychosis. However, there is a critical number of studies that provide negative findings. More specifically, eight studies examining positive schizotypy report that the unaffected relatives present with higher schizotypal traits while another seven studies did not find any significant differences between this and the control group. In fact, Appels et al. (2004), Clementz et al. (1991) and Catts et al. (2000) support that the relatives experience even lower positive schizotypal traits. Possible explanations to this could be (a) the inclusion of biased groups of relatives (e.g., relatives who differed in response style); (b) that the questionnaires used are not sensitive enough in the detection of positive schizotypal traits and (c) that cognitive-perceptual schizotypy, which is a central aspect of positive schizotypy, is also part of what is described in the literature as “healthy” or “pseudo-schizotypy” (Mohr & Claridge, 2015; Raine, 2006) and may not be related to the genetic risk for schizophrenia (Torgersen, Onstad, Skre, Edvardsen, & Kringlen, 1993). Findings on disorganized schizotypy are inconclusive, as well. However, the lack of sensitivity of the psychometric instruments in the detection of disorganized traits has already been highlighted (Tarbox & Pogue-Geile, 2011) and studies examining this aspect of schizotypy in unaffected relatives are scarce.

Studies on negative schizotypy support that unaffected relatives experience higher negative schizotypal traits compared with control individuals. However, four studies failed to obtain statistically significant differences: Bollini et al. (2007) and Compton et al. (2007) argued that this is possibly due to the relatives responding in a defensive way, because of their heightened awareness of schizophrenia and the related sociocultural stigma; Craver and Pogue-Geile (1999) proposed that the lack of significant differences is due the inadequacy of the scale used in their study to identify mild deficits in the relatives as a result of floor effects, and the study of Kimble et al. (2000) evaluated a sample with small size. Based on the above, we could conclude that negative schizotypal traits are indeed increased in the unaffected relatives of schizophrenia spectrum patients, indicating a stronger familial association with schizophrenia (Tarbox & Pogue-Geile, 2011).

Overall, there are some additional methodological problems in the existing studies that need to be acknowledged. One limitation is the small sample size employed in several studies. According to Button et al. (2013) small sample sizes reduce the statistical power and the chance of detecting a true effect. Furthermore, inclusion and exclusion criteria for participation are diverse. For example, the lack of
prior psychiatric examination of the relatives, the absence of an assessment for intellectual functioning, substance abuse and pharmacological treatment could have a confounding effect in the findings. The absence of control groups matched for gender, age and education with the relatives is another methodological limitation. Notably, all these three factors have been associated with schizotypy (Bora & Arabaci, 2009; Miettunen et al., 2010; Raine, 1992).

Another important issue is that several studies evaluate schizotypy with the SPQ. One major criticism about the SPQ is its dichotomous forced choice format (true-false). Cohen, Matthews, Najolia, and Brown (2010) and Wuthrich and Bates (2005) used Likert versions of the SPQ and found greater sensitivity, better internal reliability and identified more high scorers for schizotypy, compared with the dichotomous version. Also, a four-factor model of the SPQ, where positive schizotypy is divided into paranoid and cognitive perceptual factors (Tsousis, Zouraraki, Karamaouna, Karagiannopoulou, & Giakoumaki, 2015; Stefanis et al., 2004), better reflects the multidimensionality of schizotypal traits. Interestingly, this model has not thus far been used in studies as those described in the present review. Finally, regardless of the psychometric measure selected, assessments with self-report questionnaires always have the possibility of a defensive or socially desirable response style by the participant (Catts et al., 2000; Peltier & Walsh, 1990), further explaining the contradictory results reported.

To conclude, there is still need for further and more thorough assessments of the different schizotypal dimensions in unaffected relatives of patients in the schizophrenia spectrum. Thus, future studies should include larger samples, should employ stricter inclusion/exclusion criteria and well-defined demographic characteristics for the groups compared. Furthermore, the evaluation of environmental factors along with schizotypy could be informative about the expression of different schizotypal traits in high risk individuals. Future studies should also address the use of self-report questionnaires, in parallel with interview-based assessments for schizotypy.

REFERENCES


subjects at risk for schizophrenia. *Schizophrenia Research, 10*, 77-84. doi: 10.1016/0920-9964(93)90079-X.


