Schizotypal Symptoms in the Relatives of Schizophrenia Patients: An Empirical Analysis of the Factor Structure

by Andrea J. Bergman, Jeremy M. Silverman, Philip D. Harvey, Christopher J. Smith, and Larry J. Siever

Abstract

This study examined the nature of schizotypal symptoms in the relatives of schizophrenia patients and investigated phenomenological differences in symptomatology manifested by a familial sample and a clinical sample of personality disorder patients. Confirmatory factor analyses were used to test models of DSM-III-R schizotypal symptoms in the first degree relatives (n = 172) of schizophrenia patients. A multisample analysis was conducted to determine whether the same model adequately described the schizotypal symptoms rated in the relatives of schizophrenia patients and in clinically selected personality disorder patients. The results indicated that a three-factor model consisting of cognitive/perceptual, interpersonal, and disorganization factors yielded the best fit to the data from the relatives of schizophrenia patients, but that this model did not adequately describe both the relatives of schizophrenia patients and personality disorder patients. These findings indicate that the structure of schizotypal symptoms in the relatives of schizophrenia patients is similar to the three-factor model of schizophrenia symptoms often reported, but not the same as the structure of schizotypal symptoms in clinically selected personality disorder patients.

Keywords: Schizotypal, confirmatory factor analysis.


The concept of a schizophrenia spectrum dates back to observations that there may be gradations of schizophrenia-related disorders, with the relatives of schizophrenia patients displaying mild psychotic-like symptoms and asociality that are similar to those observed in patients with chronic schizophrenia (Kraepelin 1919/1971; Bleuler 1950). Formulations by Rado (1953) and Meehl (1962, 1990) suggested that individuals with a genetic predisposition for schizophrenia may not manifest all of the signs of the illness but may display some evidence of deviant psychological functioning, which is called “schizotypy.” The diagnosis of schizotypal personality disorder (SPD) was introduced in DSM-III in 1980 and was based on the clinical profiles of patients and relatives with “borderline schizophrenia” in the adoption studies of Kety, Rosenthal, and Wender (e.g., Kety et al. 1975).

Two major traditions—familial and clinical—have influenced the conceptualization of schizotypal personality disorder (Kendler 1985). The familial approach focuses on the traits found in the behaviorally deviant but nonpsychotic relatives of schizophrenia patients. The clinical approach features individuals who exhibit the fundamental traits of schizophrenia without chronic psychosis or severe deterioration. A key question is whether these models are describing different syndromes. One issue that has been raised relates to the structure of schizotypal symptoms, which has been explored in a variety of populations with various assessment instruments. While the vast majority of studies in this area have indicated that schizotypy, like schizophrenia, is a multidimensional construct, many questions remain about how different schizotypal dimensions relate to the familial liability to schizophrenia (Kendler et al. 1995). As Raine and Lencz (1995) point out, an important question that needs to be addressed is whether there are phenomenological differences in the presenting symptoms between SPD individuals with and without a family history of schizophrenia.

It has been proposed that schizotypal relatives of patients with schizophrenia may be different from clinically selected schizotypal patients (Kendler 1985). Some
independent patterns of neurobiological correlates associated with distinct symptom clusters in different SPD populations (Siever 1991; Amin et al. 1993). One hypothesis is that schizotypal first degree relatives of schizophrenia patients may have reduced dopaminergic activity in cortical areas, associated with deficit-like symptoms. While in clinically selected schizotypal patients, increases in dopaminergic functioning may be associated with psychotic-like symptoms (Siever 1991; Siever et al. 1993, 1995).

Both theory and previous empirical research provide a variety of possible models of schizotypal symptoms. Historically, SPD has been viewed within a two-factor model, with a "positive" factor consisting of psychotic-like cognitive and perceptual experiences, and a factor comprised of "negative" characteristics or deficits in interpersonal functioning (Siever and Gunderson 1983; Widger et al. 1986). Much empirical evidence has supported these two factors (Muntaner et al. 1988; Bentall et al. 1989; Hewitt and Claridge 1989; Raine and Allbutt 1989; Rosenberger and Miller 1989; Venables 1990; Kendler and Hewitt 1992; Raine et al. 1994; Battaglia et al. 1997). However, many of these studies of schizotypal personality have suggested additional dimensions, such as cognitive disorganization (Bentall et al. 1989; Raine et al. 1994; Battaglia et al. 1997), paranoid ideation (Rosenberger and Miller 1989; Bergman et al. 1996), and nonconformity (Muntaner et al. 1988; Bentall et al. 1989; Raine and Allbutt 1989; Kendler and Hewitt 1992). Theoretically, if schizotypal traits are on the milder end of a continuum with schizophrenia symptoms, a model of schizotypal traits might be predicted that is parallel to the three-factor models proposed for schizophrenia symptomatology (e.g., Bilder et al. 1985; Liddle 1987; Liddle and Barnes 1990; Arndt et al. 1991). These models typically include dimensions of positive symptoms, negative symptoms, and disorganization symptoms.

It is noteworthy that few studies have examined the structure of schizotypal symptoms in the relatives of schizophrenia patients. Relatives may be most likely to resemble schizophrenia patients because they may share a genetic predisposition to schizophrenia. In an exploratory factor analysis of 25 schizotypal items assessed in the first degree relatives of five proband groups (schizophrenia, other nonaffective psychoses, psychotic affective illness, nonpsychotic affective illness, and matched controls), Kendler et al. (1995) identified seven factors: negative schizotypy, positive schizotypy, borderline symptoms, social dysfunction, avoidant symptoms, odd speech, and suspicious behavior. No studies to date have used confirmatory factor analytic techniques to compare different models of the factor structure of schizotypal symptoms in the first degree relatives of patients with schizophrenia.

One purpose of this study was to examine the factor structure of clinically rated schizotypal symptoms in the first degree relatives of schizophrenia patients. Specifically, the goal was to empirically assess five competing models of schizotypal symptoms (a null model positing no underlying structure, a unidimensional severity model, a two-factor model with positive and negative factors, and two three-factor models based on previous empirical studies). Confirmatory factor analysis was used to test competing models because such analysis provides statistics to assess the goodness-of-fit of each model to the actual data. This type of analysis, as opposed to exploratory factor analysis, is a rigorous approach to the investigation of the underlying factor structure, in that it requires the specification of a priori models and allows for direct comparisons of the relative fit of alternative models (Lenzenweger et al. 1989; Harvey et al. 1992; Raine et al. 1994; Lenzenweger and Dworkin 1996).

Previous studies examining the structure of schizotypal symptoms have involved both nonclinical populations (e.g., college students) and clinical populations (e.g., personality disorder patients) using both self-report questionnaires and clinical ratings. Thus, the variation in results across studies may reflect the different instruments used (because the results of a factor analysis are dependent on the items included in the analysis) and/or reflect actual differences related to the different populations used. Comparisons between populations are difficult if different symptoms are evaluated using different methodologies. Another goal of this study was to directly compare the factor structure of clinically rated DSM-III-R schizotypal symptoms in two populations: the first degree relatives of schizophrenia patients and a sample of clinically identified personality disorder patients, reported on in a previous study (Bergman et al. 1996).

Methods

Participants. The sample consisted of 172 (67 males and 105 females) nonpsychotic first degree relatives of schizophrenia patients who participated in a family/genetic study of schizophrenia conducted at the Mount Sinai School of Medicine and associated facilities. The mean age of the relatives was 45.25 years (standard deviation [SD] = 16.74 years); the age range was 18 to 82 years. Mean education was 13.05 years (SD = 3.08). Relatives were excluded if they met criteria for any Axis I organic disorder, such as dementia, based on the lengthy diagnostic interview as described below.
Ascertainment and diagnosis of schizophrenia patients. Patients diagnosed with schizophrenia who were entered into the research programs of the Mount Sinai School of Medicine and associated facilities were referred to the Family Studies Program for family history evaluation. All schizophrenia patients \((n = 55)\) were interviewed with the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott and Spitzer 1978) and met both Research Diagnostic Criteria (RDC; Spitzer et al. 1972) and DSM-III-R (American Psychiatric Association 1987) criteria for schizophrenia without a current RDC or DSM-III-R substance abuse/dependence disorder. See Silverman et al. (1993) for a more complete description of the procedures for proband diagnoses.

Assessment of relatives. Adult, first degree relatives of each proband were recruited for study, and demographic data were collected. Face-to-face interviews with the relatives, blind to proband diagnosis, were conducted to assess for the presence or absence of Axis I and Axis II disorders (see Silverman et al. 1996 for additional description of assessment procedures). For Axis I disorders, the SADS-Lifetime (SADS-L) version of the Comprehensive Assessment of Symptoms and History (Andreasen 1985), or most recently a modified version supplemented by sections of the SADS-L, was used. To obtain a detailed picture of each relative’s personality characteristics, behavior, and functioning, relatives were also interviewed using the Structured Interview for Diagnosing DSM-III-R Personality Disorders (SIDP-R; Stangl et al. 1985). Integrated into the SIDP-R was the Supplemental Questions for Assessing Schizotypal Symptoms (Silverman et al. 1998), which includes questions from the Structured Interview for Schizotypy (Kendler et al. 1989). The SIDP-R items were scored according to the standard procedures used with this instrument. In addition to the face-to-face interview, informant interviews were employed on every relative, using the same interview structure. Two different diagnosticians completed the direct and informant interviews. They then met to arrive on a preliminary consensus diagnosis. A final consensus diagnosis was established for each relative at a consensus meeting led by L.J.S. Based on the responses to the interview items, the DSM-III-R criteria were rated on a 4-point scale \((0 = \text{absent}, 0.5 = \text{possible/uncertain}, 1.0 = \text{definitely present}, 2.0 = \text{severe}, \text{pervasive}, \text{prototypic})\). The diagnoses for the relatives are presented in table 1. As part of field trials for proposed DSM-IV SPD, we assessed interrater reliability of total and individual items (Silverman et al. 1998). Relatives \((n = 21)\) were diagnosed by two independent raters. Agreement between raters for total items was good (intraclass correlation = 0.84). Kappa statistics for individual SPD items ranged from 0.64 to 0.83.

Table 1. Subjects meeting lifetime criteria for DSM-III-R diagnoses

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manic episode</td>
<td>0</td>
</tr>
<tr>
<td>Hypomanic episode</td>
<td>12</td>
</tr>
<tr>
<td>Major depressive episode</td>
<td>53</td>
</tr>
<tr>
<td>No affective lifetime diagnosis</td>
<td>114</td>
</tr>
<tr>
<td>Alcohol dependence</td>
<td>29</td>
</tr>
<tr>
<td>Substance dependence</td>
<td>23</td>
</tr>
<tr>
<td>No substance dependence lifetime diagnosis</td>
<td>135</td>
</tr>
<tr>
<td>Paranoid personality disorder</td>
<td>27</td>
</tr>
<tr>
<td>Schizoid personality disorder</td>
<td>9</td>
</tr>
<tr>
<td>Schizotypal personality disorder</td>
<td>20</td>
</tr>
<tr>
<td>Compulsive personality disorder</td>
<td>18</td>
</tr>
<tr>
<td>Histrionic personality disorder</td>
<td>15</td>
</tr>
<tr>
<td>Dependent personality disorder</td>
<td>3</td>
</tr>
<tr>
<td>Antisocial personality disorder</td>
<td>7</td>
</tr>
<tr>
<td>Narcissistic personality disorder</td>
<td>10</td>
</tr>
<tr>
<td>Avoidant personality disorder</td>
<td>12</td>
</tr>
<tr>
<td>Borderline personality disorder</td>
<td>8</td>
</tr>
<tr>
<td>Passive-aggressive personality disorder</td>
<td>7</td>
</tr>
<tr>
<td>Mixed personality disorder</td>
<td>5</td>
</tr>
<tr>
<td>No personality disorder diagnosis</td>
<td>93</td>
</tr>
</tbody>
</table>

Data Analysis. Five models of the structure of DSM-III-R schizotypal symptoms were tested against each other, with ratings for the nine DSM-III-R schizotypal symptoms entered into the factor analyses. All nine of the symptoms were present at a level of 1.0 (definitely present) or higher in at least 5 percent of the cases, and the average severity rating of each symptom was significantly different from zero based on t tests \((t > 4.69, \text{all } p < 0.001)\). Information on the SIDP-R ratings is presented in table 2.

The first model examined was a null model, which assumed the complete absence of latent structure and therefore hypothesized zero correlations between all of the schizotypal symptoms. The unifactorial severity model assumed that all nine traits were best described through a single underlying dimension. The next model, a two-factor model (Siever and Gunderson 1983; Widiger et al. 1986), divided the schizotypal symptoms into two factors: "cognitive/perceptual" (ideas of reference, magical thinking, unusual perceptual experiences, suspiciousness, odd behavior, odd speech) and "deficit-related" (social anxiety, no close friends, constricted affect). Two three-factor models were examined based on the results of previous research in both schizotypy and schizophrenia. One model, the "disorganization" model, is based on the
results of a confirmatory factor analytic study conducted by Raine et al. (1994) using self-report items of schizotypal symptoms with a nonclinical population, and is similar to the three-factor model proposed by Battaglia et al. (1997). This model is also theoretically consistent with the three-factor models of schizophrenia symptoms. The second three-factor model, the “paranoid model,” is based on the results of a confirmatory factor analytic study (Bergman et al. 1996) using clinical ratings of SPD diagnostic criteria with a clinical sample of patients with personality disorders. Both of the three-factor models are depicted in table 3.

To identify the best fitting factor structure of the *DSM-III-R* symptoms of schizotypal personality disorder, a confirmatory factor analysis (CFA) was calculated with the EQS (Version 4.02; Bentler 1993) program. In CFA, a measurement model (i.e., a hypothetical factor structure) is constructed by the investigator, delineating how covariances in a group of variables should have been caused by latent variables (i.e., underlying factors). Model parameters are estimated based on the measurement model, and then the estimated covariance matrix is compared to the actual input covariance matrix using procedures based on maximum likelihood. A good fit with the data is indicated when a measurement model produces a solution closely matching the input covariance matrix.

The chi-square test is a way to statistically evaluate the quality of the fit between estimated and solution models (Long 1983; Joreskog and Sorbom 1984). In this case, the null hypothesis is that all of the population covariance has been extracted from the covariance matrix by the measurement model, such that a statistically significant chi-square may be generated even though a model may provide a good fit to observed data (Bentler and Bonett 1980; Marsh et al. 1988). In such instances chi-square contrasts and incremental fit indexes are often used to assess the relative fits of competing models (Bentler and Bonett 1980; Marsh et al. 1988). However, as Bollen and Long (1993) point out, the controversy about measures of overall fit makes it advisable to judge model fit by using several measures of overall fit rather than one fit index. Therefore, the following six indices were used to assess fit: goodness-of-fit index (Joreskog and Sorbom 1989), normed fit index (Bentler and Bonett 1980), incremental fit index (Bollen 1989), comparative fit index (CFI; Bentler 1990), Satorra-Bentler corrected CFI (Satorra and Bentler 1988), and root mean square error of approximation (RMSEA, obtained from LISREL-8; Joreskog and Sorbom 1993).

As pointed out by Lenzenweger and Dworkin (1996), symptom ratings in psychopathology research can often
be skewed, potentially violating assumptions of multivariate normality that underlie maximum likelihood computations. As recommended by these authors, we have used a statistic corrected for nonnormality when using an estimation method based on the assumption of underlying normality (i.e., maximum likelihood). The Satorra-Bentler scaled statistic (Satorra and Bentler 1988) was used to calculate a corrected CFI because it has been shown to be a highly reliable test statistic for evaluating covariance structure models under various distributions and sample sizes (Hu et al. 1992; Byrne 1994). In addition, a parallel set of analyses was obtained with LISREL-8 (Joreskog and Sorbom 1993) to verify the EQS results and to provide an important test statistic not available in EQS (i.e., RMSEA).

The input data for these analyses were raw data that were transformed into a covariance matrix by the EQS program (and the LISREL-8 program). To quantify the underlying latent traits, their variance was fixed to 1.0, while the factor loadings between the schizotypal symptoms and the latent traits were all free to vary, as were the error terms of the measures and the factor correlations.

**Results**

Results of the EQS analyses evaluating goodness-of-fit for each model are contained in the top of table 4. The results for the parallel set of CFAs derived from LISREL were very similar to those reported here using EQS (these data are available upon request). The evaluation of incremental fit indices is a matter of debate, although values greater than 0.90 (Cole 1987) or 0.95, depending on the standards set by prior work (Bollen 1989), have been proposed as indicative of a good fit. The paranoid model yielded a smaller chi-square value than all previous models, with all five fit indices above 0.90. The disorganization model yielded a nonsignificant chi-square, four of five fit indices at 0.95 or higher, and an RMSEA less than 0.05, suggesting that this model was the best fit to the measured covariance among the variables.

To determine if the less restricted models provided a statistically significant improvement in fit to the data relative to the null model, each of the nested models was compared by contrasting their respective chi-square values. Chi-square differences were calculated for the null

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$p &lt;$</th>
<th>GFI</th>
<th>NFI</th>
<th>IFI</th>
<th>CFI</th>
<th>*CFI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>0. Null</td>
<td>401.55</td>
<td>36</td>
<td>0.001</td>
<td>0.54</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.290</td>
</tr>
<tr>
<td>1. Unidimension</td>
<td>82.203</td>
<td>27</td>
<td>0.001</td>
<td>0.89</td>
<td>0.80</td>
<td>0.85</td>
<td>0.85</td>
<td>0.84</td>
<td>0.110</td>
</tr>
<tr>
<td>2. Two-factor</td>
<td>57.11</td>
<td>26</td>
<td>0.001</td>
<td>0.93</td>
<td>0.86</td>
<td>0.92</td>
<td>0.92</td>
<td>0.92</td>
<td>0.084</td>
</tr>
<tr>
<td>3. Paranoid</td>
<td>47.07</td>
<td>24</td>
<td>0.004</td>
<td>0.94</td>
<td>0.88</td>
<td>0.94</td>
<td>0.94</td>
<td>0.95</td>
<td>0.075</td>
</tr>
<tr>
<td>4. Disorganization</td>
<td>32.19</td>
<td>23</td>
<td>0.10</td>
<td>0.96</td>
<td>0.92</td>
<td>0.98</td>
<td>0.98</td>
<td>1.0</td>
<td>0.048</td>
</tr>
</tbody>
</table>

**Note.**—CFI = comparative fit index; *CFI = corrected comparative fit index computed with Satorra-Bentler scaled statistic; GFI = goodness-of-fit index; IFI = incremental fit index; NFI = normed fit index; RMSEA = root mean square error of approximation (a value of 0.05 or less is believed to indicate a close fit of the model in relation to the degrees of freedom; Browne and Cudeck 1993).
model compared with the unifactorial model, the unifactorial model was compared with the two-factor and three-factor models, and the two-factor and disorganization models were compared. The three-factor models could not be compared with each other because they are not nested. The differences between the chi-square values for each model were calculated and evaluated for statistical significance (Bentler and Bonett 1980); these values are contained in the bottom of table 4. The data indicate that all of the multifactorial models improve significantly on the fit of a single-dimension severity model. Because the disorganization model fit the data significantly better than both the one-factor and two-factor models and also had four of five fit indices equal to or greater than 0.95, the more stringent cutoff for a good fit, the disorganization model appears to be the best of the substantive models tested at describing the data. The factor loadings for the disorganization model are presented in figure 1.

Because the present sample consisted of more than one relative per family, it could be argued that the degree of correlation among the relatives may have affected the indexes and p values obtained. Therefore, a parallel set of analyses was conducted with only one relative per family (n = 65), and the results were consistent in that the disorganization model provided the best fit to the data ($\chi^2(23) = 33.608, p > 0.05; CFI = 0.92$), despite the small sample size.

A multiple sample analysis was conducted to determine whether the same three-factor model could adequately describe two different samples (Byrne 1994): the sample of first degree relatives currently reported, and a sample of 143 clinically selected patients diagnosed with at least one personality disorder. The specific characteristics of the clinical sample can be found in Bergman et al. (1996); the clinical sample was evaluated in much the same way as the currently reported sample, with the same ratings available and used for the factor analysis. A multiple sample analysis is done by fitting an EQS model simultaneously in both groups. There is a single goodness-of-fit chi-square test to evaluate the joint hypothesis specified by the researcher. If there is a model with identical parameters in both groups, the resulting model covariance matrices are identical, and the samples can be interpreted as arising from the same population. This is reflected by a $\chi^2$ statistic of zero. If, on the other hand, models of the various groups have parameters that are different, the resulting model covariance matrices will be different, the various samples must arise from different populations, and the $\chi^2$ will be greater than zero. In this case, the hypotheses tested were that the observed variables are measuring the same factors in each of the groups, although the factor loadings were free to vary. Both the disorganization model and the paranoid model were evaluated in separate multisample analyses. The disorganization and paranoid models both yielded CFI's of 0.87 ($\chi^2 = 139, p < 0.001$ and $\chi^2 = 142, p < 0.001$, respectively), indicating that neither model was a good fit for both samples.

Discussion

The results of the confirmatory factor analysis indicate that a three-factor model consisting of cognitive/perceptual, interpersonal, and disorganization factors is the best description of the interrelations of clinically rated symptoms of SPD in a sample of first degree relatives of schizophrenic patients. This appears to be consistent with a previous study of self-report symptoms in a nonclinical sample (Raine et al. 1994) and with the results of many studies of patients with schizophrenia that found three-factor solutions (e.g., Bilder et al. 1985; Liddle 1987; Liddle and Barnes 1990; Arndt et al. 1991). Furthermore, these results support the validity of the DSM criteria of SPD for the relatives of schizophrenic patients and strengthen the phenomenological association between schizophrenia and SPD.

The findings of this study further suggest that the structure of schizotypal traits in the relatives of schizophrenia patients is not identical to that of clinically
selected personality disorder patients. Multisample analyses conducted for both the paranoid model and the disorganization model indicated that neither model produced a similar fit for both samples. These results suggest that personality disorder patients and the first degree relatives of schizophrenia patients are not sampled from the same population with respect to the structure of schizotypal symptoms. This may be reflective of a difference in the expression of schizotypal traits in what Torgersen et al. (1993) refer to as “true” schizophrenia-related schizotypal disorder and “false” schizotypal disorders (i.e., not genetically related to schizophrenia). It is also possible, however, that sample differences such as gender distribution, age, and diagnoses may have influenced the results of the multiple sample analysis.

The results of this study are somewhat different from those of a recent study that explored the factor structure of schizotypal symptoms in relatives of psychiatric patients and found seven factors (Kendler et al. 1995). There are several possible explanations for these seemingly disparate findings. First, the items used were different; the Kendler et al. (1995) study included 25 schizotypal signs and symptoms versus the 9 DSM-III-R criteria used in the current investigation. In addition, the samples were somewhat different in that we studied only relatives of patients with schizophrenia, rather than relatives of patients with a variety of psychiatric disorders. Finally, it is difficult to compare the results of the two studies directly because different factor analytic procedures were used. Because of the nature of the exploratory factor analysis used by Kendler et al., it is not clear whether our three-factor model would fit the data better than the seven-factor model.

These results should be considered in the context of the limitations of this study. First, one factor in the disorganization model (i.e., the disorganization factor) and one factor in the paranoid model (the cognitive/perceptual factor) were identified by fewer than three symptoms, so these factors could be considered underidentified (Bollen 1989). This was unavoidable because of the limited number of symptoms for SPD in the DSM-III-R, which was the basis for the ratings in this study. Furthermore, the specification of every model was based on previous empirical research. It should also be noted that one of the items, suspiciousness, has a relatively weak loading (0.27) on the interpersonal factor, although it has a stronger loading (0.52) on the cognitive/perceptual factor. This may indicate that suspiciousness is primarily a measure of cognitive/perceptual distortion in this sample. Finally, the sample of relatives examined in this study may be more heterogeneous with respect to age than the samples in other studies investigating SPD symptoms. In particular, there were a number of relatives (n = 31) over the age of 65. Future studies should address possible developmental differences in the expression of SPD symptoms over the lifespan.

In conclusion, the results of the current investigation suggest that a three-factor model may be the best description of clinically rated SPD symptoms in the first degree relatives of schizophrenia patients. This model included a cognitive/perceptual factor, an interpersonal factor, and a disorganization factor, which seem to correspond to many of the three-factor models that have been proposed for schizophrenia symptoms. Furthermore, the contrast between the best fitting model for the relatives (disorganization model) and the best fitting model for a clinical sample (paranoid model; Bergman et al. 1996) supports the idea that the first degree relatives of schizophrenia patients represent a different population, with respect to schizotypal symptoms, from clinically selected samples. It has been proposed that schizotypal symptoms may have different underlying pathophysiological mechanisms in different populations (Siever et al. 1995). While this study does not address pathophysiological differences among the samples, it does indicate that a familial liability for schizophrenia may have implications for the nature of schizotypal symptoms. Future research is needed to validate these models and uncover the meaning of these dimensions.

References


Schizotypal Symptoms in Relatives


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