Schizotypal personality disorder in individuals with and without schizophrenic relatives: similarities and contrasts in neurocognitive and clinical functioning

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Schizophrenia-spectrum disorders may reflect the genotype for schizophrenia. One such disorder, Schizotypal Personality Disorder (SPD), was examined as a function of family history of schizophrenia. Clinical profiles and neurocognitive functioning were evaluated in 25 schizotypal subjects (10 SPD with schizophrenic relatives and 15 SPD without schizophrenic relatives), and in 24 normal controls. The primary finding is that vigilance performance was similarly impaired in both SPD groups. An additional neurocognitive impairment, comprehension of grammatical constructions, was observed only in the SPD group with schizophrenic relatives. Of interest, the clinical profiles of the two SPD groups did not differ significantly. These results suggest that schizotypal personality disorder is associated with a continuum of neurocognitive vulnerability that increases as a function of family history of schizophrenia.

Key words: Vigilance; Grammatical construction; P300; Vulnerability; Schizophrenia-spectrum

INTRODUCTION

It has been suggested that the genotype for schizophrenia is expressed in the schizophrenia-spectrum disorders, with diagnosed schizophrenia being a severe complication that is produced by environmental trauma and associated with neurological sequelae (e.g., Mech, 1962, 1989; Schulsinger et al., in press). The spectrum disorder most closely related to schizophrenia is Schizotypal Personality Disorder (SPD). As defined by DSM-III and DSM-IIIR, SPD actually represents an admixture of clinical symptoms derived from two different perspectives: the family and the clinical traditions (Kendler, 1985; Spitzer et al., 1979). These perspectives differ in terms of the context in which SPD has been identified (i.e., in families of schizophrenics versus in psychiatric settings), and in the clinical features most frequently observed (Kendler, 1985). Negative symptoms, such as social isolation and impairment, predominate in the observations of family members of patients (Gunderson et al., 1983; Kendler, 1985; Torgersen, 1985), while positive symptoms, such as magical thought and illusions, are more commonly noted in the SPD identified in psychiatric settings (Jacobsberg et al., 1986; Kendler, 1985). As a result, some have suggested that SPD be redefined to emphasize those negative or social symptoms that are more closely associated with a family history of schizophrenia (Gunderson et al., 1983; Gunderson and Siever, 1985).

A fundamental question concerns whether common neurocognitive mechanisms characterize individuals who have been diagnosed with SPD, but who differ on the basis of family history of schizophrenia. The presence of similar neurocogni-
tive deficits would suggest that the two groups are related despite their difference in family history of schizophrenia. We report a comparison of the clinical features and neurocognitive functioning of subjects who were diagnosed with DSM III SPD, and who were selected to represent the two populations of SPD with and without schizophrenic relatives: siblings of schizophrenic probands; and subjects without schizophrenic relatives who were recruited from the general population via media advertisement. The neurocognitive measures include tests that have reliably detected deficits in schizophrenic patients. To our knowledge, there are no published studies that compare directly these two types of schizotypy on the combination of neurocognitive measures reported in the present study. With these issues identified, we hypothesized the following:

(1) Schizotypal subjects with a family history of schizophrenia will exhibit more impaired social functioning than that of schizotypal subjects without a family history of schizophrenia;

(2) Schizotypal subjects without a family history of schizophrenia will exhibit more positive symptoms (e.g., magical thought and illusions) than will the schizotypal subjects with schizophrenic relatives; and

(3) Schizotypal subjects will exhibit impaired neurocognitive performance compared to normal controls, regardless of their family history of schizophrenia.

METHODS

Subjects
Subjects included 10 SPD siblings of schizophrenic probands, 15 SPD individuals without a family history of schizophrenia who were recruited from the general population, and 24 normal controls (No Lifetime Diagnosis of Psychiatric Disorder). SPD siblings of schizophrenic probands were identified from among the male siblings (n = 58; Mean age = 37.2 years/S.D. = 8.6) of male schizophrenic probands (n = 50; Mean age = 36.9 years/ S.D. = 6.7), who were recruited for participation in the ongoing Pittsburgh Vulnerability Study. Schizophrenic probands were identified primarily through the Highland Drive VA Medical Center in Pittsburgh. Informed consent was obtained from patients to contact their family members, and siblings were then selected randomly within each family and contacted for recruitment. Of the 58 siblings evaluated, 10 (17%) received a DSM-III diagnosis of SPD, probable (3 symptoms) or definite (4 or more symptoms). This rate is comparable to earlier findings (Baron et al., 1985; Kendler et al., 1981, 1984). Those siblings diagnosed with SPD are the focus of the present report (n = 10).

Schizotypal individuals without a family history of schizophrenia (n = 15) were recruited from the general population via media advertisement of two types. The first advertisement was designed specifically to target schizotypy, and expressed an interest in testing individuals with a history of mystical and unusual experiences (e.g., ESP, telepathy, clairvoyance). This advertisement appeared in local media, several times weekly over a two month period. This approach was successful in attracting an initial 64 respondents (males = 20; females = 44), who were screened for the general exclusion criteria and family history of schizophrenia described below (see Psychiatric Assessment). Of those respondents eligible for laboratory testing, 11 were assigned a DSM-III diagnosis of SPD, probable (3 symptoms) or definite (4 or more symptoms). None had a family history of schizophrenia. The remaining SPD subjects without schizophrenic relatives (n = 4) and the normal controls (n = 24) were identified among individuals who responded to an advertisement that stated an interest in testing the general information processing capacities of normal, healthy adult males.

Normal controls and siblings of schizophrenics were matched on age, education, and sex. There were no significant differences between the two SPD groups on age and education, but the SPD group without a family history of schizophrenia included females (n = 9). Because gender differences in clinical profile and social functioning have been reported for schizophrenic patients (Haas et al., 1990), and for SPD inpatients (McGlassan and Bardenstein, 1988), additional analyses were conducted to evaluate the potential effects of gender on the variables of interest in the present study. This was accomplished by collapsing the family history factor, and comparing the male (n = 16) and female (n = 9) SPD subjects on each of the measures examined in this study.

Informed consent following explanation of pro-
PROCEDURES

Psychiatric assessment

DSM-III Axis I and Axis II diagnoses (American Psychiatric Association, 1980) were determined in case conferences involving a psychiatrist, a psychologist, and research staff. Diagnoses were based on the results of semi-structured clinical interviews (Axis I: Schedule for Affective Disorders and Schizophrenia-Lifetime (SADS-L) (Spitzer and Endicott, 1979), and Alcohol and Drug sections, Diagnostic Interview Schedule (DIS) (Robins et al., 1981); and Axis II: Structured Interview for DSM-III Personality (SIDP) (Pfohl et al., 1983), and Schedule for Schizotypal Personalities (SSP) from the Schedule for Interviewing Borderlines (SIB) (Baron, 1980)). Family history of psychiatric disorder was determined using the Family History Research Diagnostic Criteria (Andreasen et al., 1977). Interrater reliability for the diagnosis of interest (SPD) was 0.81 (kappa statistic), as assessed by two independent raters on a subset of the clinical interviews (n = 11).

General exclusion criteria included history of major medical disease (e.g., diabetes, heart disease), and neurological injury or disorder (e.g., incidents of loss of consciousness, epilepsy). None of the subjects were taking neuroleptic medication.

Cognitive behavioral assessment

Attentional vigilance was assessed using a degraded visual stimulus version of the Continuous Performance Test. The task was to press a key when a target, the number 0, was recognized. Stimuli (numerals 0–9) were presented on video monitor and subtended a visual angle of 3.1° by 2.1°. The image was blurred by placing a rear-projection screen directly on the face of the monitor. The target appeared on 25% of the trials. A practice run of 162 stimuli was followed by 486 test stimuli. Stimuli were presented for a duration of 50 ms with an interstimulus interval of 1000 ms. Proportions of correct responses (hits) and errors of commission (false alarms) were used to calculate \( d' \) level, a parametric measure of visual sensitivity that reflects the ability to discriminate the target (0) from distractors (1–9).

Cognitive psychophysiological assessment

The P300 component of the event-related potential (ERP) was elicited using an ‘oddball’ paradigm during auditory counting and choice reaction tasks. ERPs were recorded from six scalp locations (FZ, CZ, PZ, P3, P4, O2; 10/20 system) and an eye artifact channel (EOG) using a bandpass of 0.01–30 Hz amplified \( \times 20,000 \). Data were digitized for 1200 ms at 125 Hz, beginning at 200 ms prestimulus. Conditional probability was manipulated with the rare event occurring on 25% of the trials in both tasks. Auditory stimuli were high (1500 Hz) and low (800 Hz) tones presented for 40 ms at 65 dB SL. The two tasks were to count the rare tone (counting), and to make a different motor response (button press) to the two tones (choice reaction).

Artifact-free trials were used to compute the ERPs. For the present study, analyses of peak amplitude and latency were restricted to the midline parietal electrode (Pz), which typically yields the largest P300 in this paradigm. P300 amplitude and latency were recorded automatically following the determination of the maximum positivity in the 250 to 500 ms time window, and the substraction of the median voltage recorded at the pre-stimulus baseline.

Neuropsychological assessment

Measures of general cognitive functioning included: two tests sensitive to anterior brain dysfunction: Trails-B and Wisconsin Card Sorting Test (WCST) (Levin et al., 1989); a test of visuocostrucational ability sensitive to posterior brain dysfunction: WAIS-R Block design (Lezak, 1983); a test of general knowledge: WAIS-R Information; and a test of comprehension of grammatical constructions: Relational Concepts Test (Luria-Nebraska Neuropsychological Battery).

RESULTS

Statistics

Due to the number of comparisons, the standard significance level of \( p = 0.05 \) was adjusted using the modified Bonferroni test (Keppel, 1982). For the clinical measures (12 comparisons), significance was set at \( p = 0.008 \), with a trend considered present if \( p = 0.02 \). For the neurocognitive measures (11
comparisons), significance was set at $p = 0.009$, and trend significance at $p = 0.02$.

Homogeneity of variance was tested for all measures (Levene’s test). Several of the measures showed significantly different group variances (viz., education, Trails-B, WCST-Categories, Relational Concepts, and P300 amplitude-counting task), and a nonparametric analysis of variance was employed in those cases (Kruskal-Wallis H-test). Because occupational status and the neuropsychological measures are likely associated with level of formal education, analyses of covariance were also conducted to adjust for the potential influence of this variable on these measures. One variable involved measurement at the ordered metric level (viz., the Hollingshead Occupational Status scale). Following others (Abelson and Tukey, 1959; Labovitz, 1970), this level of measurement was treated as interval data and analyzed accordingly.

As reflected in Table 3, complete data were not available for all subjects. For normal controls ($n = 24$), psychophysiological data were missing for two subjects due to the attrition of one subject and the unreliability of ERP data for the second subject. Neuropsychological data were not available for a third normal control. For the SPD sibling group ($n = 10$), laboratory data were missing for two SPD siblings, and laboratory and neuropsychological data for two additional SPD siblings, all due to subject attrition. In the SPD group without schizophrenic relatives ($n = 15$), laboratory and neuropsychological data were not available for one subject due to attrition, and cognitive behavioral data were missing for a second subject due to a procedural irregularity.

**Demographic characteristics**

Table 1 presents the demographic characteristics of the three groups. Age was not significantly different among the three groups ($F = 2.11, df = 2.46, p = n.s.$), nor were there significant differences for level of education ($H = 2.82, df = 2, p = n.s.$). Highest occupational status for the preceding year was significantly different between groups ($F = 7.95, df = 2.46, p = 0.001$), as measured by the 9-step scale of Hollingshead’s revised Four Factor Index of Social Status (1 = farm and service workers; 5 = clerical and sales workers, and small farm and business owners; 9 = professionals and executives, and large business owners) (unpublished paper, Yale University, 1975). However, this difference did not retain significance following covariance of education ($F = 3.67, df = 2.39, p = 0.03$).

**Psychiatric functioning**

The pattern of schizotypal symptoms exhibited by the two SPD groups did not differ significantly. No differences were observed for frequency of any of the DSM-III schizotypal personality disorder symptoms (for all 8 diagnostic criteria: Fisher’s exact test, $p = n.s.$). The two groups were also not significantly different on any of the indicators of clinical severity, which are summarized in Table 2 (for all measures, Fisher’s exact test, $p = n.s.$): number of SPD diagnostic criteria met (probable versus definite), presence of concurrent Axis I disorders, history of psychiatric treatment, and employment dysfunction associated with psychopathology. Only one subject reported previous psychiatric hospitalization (SPD sibling group).

In summary, the SPD sibling group was not more socially isolated and anxious, as evaluated during the psychiatric interview. And, although the SPD sibling group showed a lower absolute level of occupational achievement, the effect of educational background likely accounts for this difference. Therefore, no support was provided for our first hypothesis, which predicted more impaired social functioning for the SPD sibling group. In addition, no support was obtained for the second hypothesis, which predicted that more positive symptoms (e.g., magical thought and illusions) would be exhibited by the SPD without schizophrenic relatives.

**Psychiatric profile and gender differences**

Male and female SPD subjects did not differ significantly on any of the demographic, clinical, or occupational status measures evaluated in this study. Of interest, a trend significance was observed for one SPD symptom, ideas of reference (Fisher’s exact test, $p = 0.01$), with more males exhibiting this symptom (10/63%) than females (1/11%).

**Neurocognitive measures**

Table 3 presents the means and standard deviations for the measures of neurocognitive functioning for the two SPD groups and the normal controls.
TABLE 1
Demographic characteristics associated with schizotypal personality disorder (SPD) in individuals with and without schizophrenic relatives and for normal controls

<table>
<thead>
<tr>
<th></th>
<th>SPD family history of schizophrenia</th>
<th>SPD no family history of schizophrenia</th>
<th>Normal controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 10)</td>
<td>(n = 15)</td>
<td>(n = 24)</td>
</tr>
<tr>
<td>Age</td>
<td>36.4 (7.7)</td>
<td>37.1 (7.1)</td>
<td>32.8 (6.6)</td>
</tr>
<tr>
<td>Formal education (years)</td>
<td>12.5 (1.4)</td>
<td>14.7 (2.7)</td>
<td>14.9 (2.0)</td>
</tr>
<tr>
<td>Occupational status</td>
<td>2.7 (1.8)</td>
<td>5.8 (2.5)</td>
<td>5.7 (2.1)</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single, never married</td>
<td>5 (50%)</td>
<td>3 (20%)</td>
<td>10 (42%)</td>
</tr>
<tr>
<td>Married</td>
<td>5 (50%)</td>
<td>8 (53%)</td>
<td>12 (50%)</td>
</tr>
<tr>
<td>Separated or divorced</td>
<td>0 (--)</td>
<td>4 (27%)</td>
<td>2 (8%)</td>
</tr>
</tbody>
</table>

Note. The values for the first three items are means (SD), for marital status f(%).

TABLE 2
Clinical severity associated with schizotypal personality disorder (SPD) in individuals with and without schizophrenic relatives

<table>
<thead>
<tr>
<th>Clinical severity</th>
<th>SPD family history of schizophrenia</th>
<th>SPD no family history of schizophrenia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of SPD symptoms</td>
<td>f(%)</td>
<td>f(%)</td>
</tr>
<tr>
<td>Probable (3)</td>
<td>1(10)</td>
<td>7(47)</td>
</tr>
<tr>
<td>Definite (4 or more)</td>
<td>9(90)</td>
<td>8(53)</td>
</tr>
<tr>
<td>Concurrent Axis I disorder(s)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>2(20)</td>
<td>5(33)</td>
</tr>
<tr>
<td>Present</td>
<td>8(80)</td>
<td>10(67)</td>
</tr>
<tr>
<td>Outpatient treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>4(44)</td>
<td>5(36)</td>
</tr>
<tr>
<td>Brief consult to long-term</td>
<td>5(56)</td>
<td>9(64)</td>
</tr>
<tr>
<td>Employment dysfunction due to psychopathology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>5(63)</td>
<td>11(79)</td>
</tr>
<tr>
<td>Present</td>
<td>3(37)</td>
<td>3(21)</td>
</tr>
</tbody>
</table>

Cognitive behavioral functioning
Vigilance performance, as measured by \(d'\) level, was significantly different for the three groups (\(F = 9.68, df = 2.42, p = 0.0003\)). Planned comparisons determined that the normal controls were superior to the SPD sibling group (\(F = 14.88, p = 0.0004\)), and to the SPD without schizophrenic relatives (\(F = 10.01, p = 0.003\)). Attentional vigilance of the two SPD groups did not differ (\(F = 1.17, p = n.s.\)).

Cognitive Psychophysiological functioning
Fig. 1 presents the artifact-free ERPs elicited by the rare stimulus at Pz during the two tasks, with the three groups superimposed. The three groups did not differ significantly for amplitude or latency of the P300 component during either the counting or choice reaction tasks. The variations that are apparent in the earlier ERP components (i.e., P200, N250) will be addressed fully elsewhere.

Neuropsychological functioning
Significant group differences were observed for comprehension of grammatical constructions (Relational Concepts Test: \(H = 17.37, df = 2, p = 0.0002\)). Planned comparisons determined that the SPD sibling group exhibited poorer comprehension than both the normal controls (\(z = 3.94, p < 0.001\)), and the SPD without schizophrenic relatives (\(z = 3.80, p < 0.001\)). The SPD without schizophrenic relatives did not differ from normal controls (\(z < 1, p = n.s.\)). This group effect retained significance following covariance of education (\(F = 13.80, df = 2.39, p < 0.0001\)). No significant group differences were observed for Trails-B, the WAIS-
TABLE 3
Neurocognitive functioning associated with schizotypal personality disorder (SPD) in individuals with and without schizophrenic relatives and in normal controls

<table>
<thead>
<tr>
<th>SPD family history of schizophrenia</th>
<th>SPD no family history of schizophrenia</th>
<th>Normal controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive behavioral</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPT d'</td>
<td>n = 8</td>
<td>n = 13</td>
</tr>
<tr>
<td>Electrophysiological</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-P300 to rare event</td>
<td>n = 8</td>
<td>n = 14</td>
</tr>
<tr>
<td>Amplitude-counting task</td>
<td>13.59 (6.04)</td>
<td>13.81 (8.51)</td>
</tr>
<tr>
<td>Amplitude-choice reaction</td>
<td>11.46 (5.82)</td>
<td>14.54 (7.13)</td>
</tr>
<tr>
<td>Latency-counting task</td>
<td>357.00 (16.53)</td>
<td>362.29 (33.42)</td>
</tr>
<tr>
<td>Latency-choice reaction</td>
<td>369.00 (79.76)</td>
<td>378.86 (51.52)</td>
</tr>
<tr>
<td>Neuropsychological</td>
<td>n = 6</td>
<td>n = 14</td>
</tr>
<tr>
<td>WCST categories</td>
<td>5.83 (0.41)</td>
<td>5.21 (1.48)</td>
</tr>
<tr>
<td>WCST perseverative errors</td>
<td>11.67 (10.61)</td>
<td>12.29 (11.14)</td>
</tr>
<tr>
<td>Trails-B</td>
<td>99.17 (68.11)</td>
<td>66.00 (18.62)</td>
</tr>
<tr>
<td>WAIS-R block design</td>
<td>13.17 (3.06)</td>
<td>11.36 (2.31)</td>
</tr>
<tr>
<td>WAIS-R information</td>
<td>10.83 (4.12)</td>
<td>11.43 (2.62)</td>
</tr>
<tr>
<td>Luria-Nebraska relational concepts (Errors)</td>
<td>3.67 (2.16)</td>
<td>0.43 (1.16)</td>
</tr>
</tbody>
</table>

Adjusted alpha (p = 0.05): p = 0.009. Values are means (SD).
*p < 0.001.

Fig. 1. Grand mean event-related potentials from the midline parietal electrode (Pz) superimposed for the three subject groups, separated by task (counting and choice reaction): normal controls (solid line), schizotypal siblings of schizophrenic relatives (dotted line), and schizotypal subjects without schizophrenic relatives (dashed line). P300 is the late positivity occurring between 300 and 400 ms. Apparent group differences were not significant. (Note that the grand means do not reflect the unweighted mean amplitudes unbiased by latency variation; exact mean amplitudes are provided in Table 3.)

R Block design and Information subtests, or for the Wisconsin Card Sorting Test-Categories and Perseverative Errors.

In summary, vigilance performance (d' level) was similarly impaired in both SPD groups. Thus, as hypothesized, schizotypal subjects exhibited neurocognitive impairment regardless of their family history status. Furthermore, the SPD sibling group showed an additional cognitive deficit, impaired comprehension of grammatical constructions, that was not observed in the SPD without schizophrenic relatives.

Neurocognitive functioning and gender differences
There were no significant differences between male and female SPD subjects on any of the neurocognitive measures evaluated.

DISCUSSION

This study provides a direct comparison of the clinical profiles and neurocognitive functioning of individuals who were diagnosed with SPD, but who differed with respect to family history of schizophrenia. To our knowledge, there are no published studies that compare directly these two types of schizotypal individuals on the combination of measures evaluated in the present study. Although the small sample size makes it impossible to draw firm conclusions, these data provide initial information regarding the neurocognitive functioning associated with schizotypy as a function of family history of schizophrenia.

Results suggest that SPD is associated with a liability for neurocognitive deficits that increases
as a function of family history of schizophrenia. First, both SPD groups exhibited impaired vigilance performance ($d'$ level). This finding indicates that deficient vigilance performance is not restricted to an association with familial schizophrenia, and suggests the presence of a core neurocognitive deficit that is expressed along with the schizophrenia-spectrum. Thus, results of our direct comparison are consistent with separate reports of vigilance deficits in schizophrenic patients and their first degree relatives (Nuechterlein, 1991; Nuechterlein and Dawson, 1984), and in schizotypal subjects identified in a non-clinical college population (Lenzenweger et al., 1991). Second, the SPD sibling group was characterized by an additional neurocognitive deficit that was not present in the SPD group without schizophrenic relatives, namely, impaired comprehension of grammatical constructions (Relational Concepts Test).

The multi-dimensional data examined in the present study therefore suggest that a continuum of liability for neurocognitive deficits is associated with SPD. Specifically: (1) if SPD is present, then impaired vigilance performance is probable; and (2) if SPD and a family history of schizophrenia are both present, then impaired vigilance performance and impaired comprehension of grammatical constructions are both likely. A continuum of liability is generally consistent with the Vulnerability model (Zubin and Spring, 1977; Zubin and Steinhauer, 1981), which predicts that the indicants of the core deficit of schizophrenia should also be observed in the non-schizophrenic relatives of patients. Findings of the present study extend the implications of that hypothesis. The presence of an identical neurocognitive impairment in both groups of schizotypal individuals, who differed with respect to familial schizophrenia, strengthens the argument that the schizophrenia-spectrum disorders reflect the core trait (e.g., Mechl, 1962; 1989; Schulsinger et al., in press). Moreover, multiple levels of liability are suggested by the presence of an unique, additional neurocognitive impairment in the schizotypal individuals with schizophrenic relatives.

Of interest, the neurocognitive impairments observed are somewhat consistent with current hypotheses that implicate hemispheric imbalance (Gruzelier et al., 1988) and dysfunction of anterior brain systems (Levin, 1984; Scidman, 1983; Weinberger, 1987) in the neuropathology of schizophrenia. Recent evidence from studies using positron emission tomography (PET) indicates that vigilance performance in schizophrenics is associated with lower than normal activation in the prefrontal and right temporoparietal cortex (Buchsbaum et al., 1990; Cohen et al., 1987). In addition, difficulties in comprehension of grammatical constructions have been observed in patients with severe frontal lesions (Luria, 1980). It is important to note, however, that neither SPD group showed impairment on the Wisconsin Card Sorting Test or Trails-B, tests with previously established sensitivity to anterior dysfunction (Levin et al., 1989). Thus, impairment was seen on some, but not all, of the tests that are currently believed to be sensitive to anterior brain dysfunction. Furthermore, neither SPD group exhibited abnormal P300 amplitude, a measure that has reliably detected information processing deficits in schizophrenic patients (Friedman, 1991).

Clinical profiles did not differ for these two SPD groups, a finding that is contrary to what was expected based on the separate clinical reports concerning these two types of schizotypy (Gunderson et al., 1983; Jacobsberg et al., 1986; Kendler, 1985; Torgersen, 1985). One possible explanation for the absence of differences in symptomatology concerns the level of clinical severity observed in the SPD without schizophrenic relatives. As noted, the two SPD groups were similar across the indicants of clinical severity. The earlier clinical descriptions of the SPD without schizophrenic relatives were based on individuals who were identified in psychiatric treatment settings (Jacobsberg et al., 1986; Kendler, 1985). In contrast, in the present study, the SPD without schizophrenic relatives were recruited from the general population through media advertisement. This recruitment method may have resulted in a sample of SPD without schizophrenic relatives who were less clinically impaired, and, as a result, exhibited fewer florid symptoms than those patients described in the earlier reports (Jacobsberg et al., 1986; Kendler, 1985). An additional consideration is that more sensitive measures of social adjustment may determine the SPD with schizophrenic relatives to be more impaired in this domain. This is suggested by their lower absolute level of occupational achievement, a difference that was not significant.
when an adjustment was made for educational background.

In summary, SPD was associated with a liability for neurocognitive deficits that increased as a function of family history of schizophrenia. Schizotypal subjects displayed impaired vigilance performance regardless of their family history of schizophrenia. An additional cognitive deficit, impaired comprehension of grammatical constructions, was observed in the SPD sibling group, but not in the SPD without schizophrenic relatives. Thus, a continuum of neurocognitive vulnerability is suggested by these data. Also, the neurocognitive deficits observed are generally consistent with the neuropathic models of schizophrenia which implicate hemispheric imbalance and dysfunction of anterior brain systems. These findings have significance for our understanding of the neurocognitive deficits and clinical impairments associated with a vulnerability to schizophrenia.

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