Working memory deficits, antisaccades, and thought disorder in relation to perceptual aberration

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The larger than usual number of authors of this paper (listed, except for the first author, alphabetically) indicates that this research was a team effort in which three studies were conducted simultaneously by three teams of investigators. The effort began when Mark Lenzenweger conceived of the idea to study normally functioning college students who, on psychometric examination, showed significant elevations on traits that Loren and Jean Chapman have identified as schizotypal. Lenzenweger thought that it would be informative to determine how these students performed on tests that distinguish schizophrenic patients and their relatives from both the normal population and patients with nonpsychotic illnesses. The scientists in our laboratory agreed.

Three teams of investigators set out from the Harvard-McLean Hospital laboratory to work at Cornell University, where they completed the testing of 57 people in Lenzenweger’s population. Each of the teams completed a report of its own, which has been submitted for publication; these reports are summarized here. In addition, this chapter considers the performance of these college students on all of the tasks, which could not be done in the individual reports. The three tasks are (1) delayed response, from which we assess working memory; (2) antisaccade eye movements, and (3) thought disorder assessed from the Thought Disorder Index (TDI).
Rationale

The measures

For many years, the Psychology Research Laboratory at McLean Hospital has been studying psychological and psychophysiological characteristics that are associated with schizophrenia. Some of these performance measures show a familial aggregation among the relatives of schizophrenic individuals, which enhanced their significance beyond the mere demonstration that performance was deviant in a group of psychotic people. Examples of the behaviors that showed familiarity are eye tracking dysfunctions (ETD; e.g., Levy, Holzman, Mambyse, & Mendell, 1993), specific qualities of thought disorder (e.g., Shenton, Solovay, & Holzman, 1987; Solovay, Shenton, & Holzman, 1987), and delayed response performance (e.g., Park & Holzman, 1992).

Some of these performance deviations, like thought disorder, seem obviously phenomenologically related to schizophrenic pathology since most schizophrenic patients show characteristic thought slippage, others, like antisaccade eye movements and delayed response, do not. But the absence of an obvious relation between schizophrenia and a particular task deficit presents an opportunity for probing in greater depth the nature of schizophrenic pathology. In other diseases - phenylketonuria (PKU), for example - such associations are not unusual. In PKU, light skin pigmentation and subnormal intelligence are associated, but that relation became understandable only after it was learned that tyrosine hydroxylase is the key to that puzzle. Therefore, when, in the course of investigating vestibular integrity in schizophrenic patients, we discovered that eye tracking was disordered, we were not dissuaded from investigating the significance of this finding merely because the relation of eye tracking dysfunctions to schizophrenia was not readily apparent.

The population

Most recurrence risk studies record clinical schizophrenia in only about 4% to 10% of the first-degree relatives of schizophrenic patients. The prevalence of schizophrenia in families is thus rather low. But beginning with Kraepelin and Bleuler, there has been a general recognition that schizophrenic pathology is not limited to the psychotic form of the illness. Bleuler, for example, remarked that "[T]he symptoms exist in varying degrees and gradations on the entire scale from pathological to normal; also, the milder cases, latent schizophrenics with far less manifest symptoms, are many times more common than the overt manifest cases." Later in his monograph, he wrote:
Delayed response, antisaccade, thinking

[T]here is also a latent schizophrenic, and I am convinced that this is the most frequent form, although admittedly these people usually ever come to treat... In this form, we can see in near all the symptoms and all the combinations of symptoms which are present in the manifest types of disease. Irritable, odd, moody, withdrawn, or exaggeratedly garrulous people appear, among other things, the suggestion of being schizophrenic... (Every form of this disease may take a latent course).” (Bleuler, 1911/1950, pp. 13, 239)

The recognition that the nonpsychotic forms of schizophrenia are more prevalent than the psychotic forms lay behind Rado’s (1953), Grinker’s (1969), and Meisel’s (1962) emphasis on schizotypy. The many modifiers of the term “schizophrenia” that were used in diagnostic practice prior to the introduction of the DSM-III and DSM-III-R, such as “pre-,” “latent,” “incipient,” “remitted,” “borderline,” and “pseudoneurotic” schizophrenia, and also “schizophrenic character” attempted to tag nonpsychotic forms of schizophrenia.

If the basic pathology of the latent or schizotypal form of the disorder is in crucial aspects the same as that of its psychotic form, investigation of the nonpsychotic manifestations of schizophrenia will be less influenced by the nosological distractions of poor motivation, inattention, medication effects, generalized performance deficits, and social deterioration, which plague most studies of schizophrenic patients. This rationale underlies our laboratory’s emphasis on studying “unaffected” first-degree relatives of patients (Holzman & Manly, 1996). And it provides the motivation for embarking on this collaboration with the Cornell laboratory in the study of “normal” students identified as psychometrically deviant by one of the scales introduced by Chapman and Chapman (1958). In short, we decided to examine a variety of functions associated with schizophrenia—working memory deficits, antisaccades, thought disorder—among those in the general population who have been selected by a psychometric measure of schizotypy.

Method

The subjects for this study were drawn from a larger, randomly ascertained sample of first-year undergraduates at Cornell University who voluntarily completed a 250-item objective psychological inventory entitled “Attitudes, Feelings, and Experiences Questionnaire” (AFEQ). The AFEQ included the 35-item Perceptual Aberration Scale (PAS; Chapman, Chapman, & Roulin, 1978), the scores on which represented the principal independent variable. We selected a random sample in order to maximize diversity within our pool of potential subjects as well as to minimize the effects of self-selection and
group-related test-taking attitudes that are often associated with sampling from students enrolled in introductory psychology courses. Of the 2,000 students who were invited to complete the inventory, 1,664 (51.3% women, 48.7% men) did so. The response rate of 84.2% suggests that we achieved representative sampling of this population. Of the 1,684 subjects, 35 (2.1%) were excluded from our sample as invalid, and an additional 9 subjects were dropped because of extensive missing data. The resulting final sample consisted of 1,646 cases.

Two subject groups were composed for the experiments described below, using the complete pool of 1,646 individuals. Group means and standard deviations on the PAS, computed separately for males and females, served as the basis for group composition. Following Chapman and Chapman (1983), subjects classified as high PAS scorers were required to have scores at least 2.0 standard deviations above the PAS group mean, whereas subjects classified as normal scorers were required to have scores no higher than 0.5 standard deviations above the group mean. By this method we identified 76 (4.0%) students who had high PAS scores and 1,371 (83.3%) students with low PAS scores. From these two groups we randomly selected 31 students (16 female) from the high PAS group and 26 students (12 female) from the low PAS group for the normed control subjects. Table 15.1 presents the demographic characteristics of this final sample.

High and low PAS subjects did not differ with respect to sex ratio, age, ethnicity, or contact rate. All subjects were screened for psychosis, and none had a diagnosis of any psychiatric illness at the time of testing. The three measures used are described as follows.

The Perceptual Aberration Scale (PAS)

The PAS is a well-established 35-item true-false self-report measure of distortions and distortions in perceptions of body image as well as other objects (Chapman et al., 1978). It includes such items as "Occasionally I have felt as though my body did not exist" (keyed true), and "I have never felt that my arms or legs have momentarily grown in size" (keyed false). The scale is described more fully elsewhere (e.g., see Chapman, Chapman, & Kwast, Chapter 5, this volume).

We chose the PAS score as the independent variable for this study because of the emphasis given to body-image and perceptual distortions in schizotypy by both Rado and Mehl. Multiple converging lines of evidence show that the PAS is a valid, though fallible, psychometric indicator of traits associated with schizotypy (cf. Cramer & Mehl, 1959).
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2.2% suggests that we achieved
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additional 3 subjects were
the resulting final sample con-

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<table>
<thead>
<tr>
<th>Table 1. Demographic characteristics of study subjects</th>
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<tbody>
<tr>
<td><strong>Age</strong></td>
</tr>
<tr>
<td><strong>Total sample</strong></td>
</tr>
<tr>
<td><strong>57</strong></td>
</tr>
<tr>
<td><strong>27</strong></td>
</tr>
<tr>
<td><strong>13</strong></td>
</tr>
<tr>
<td><strong>7</strong></td>
</tr>
</tbody>
</table>

| **Variable** | **Mean** | **SD** | **Frequency** |
| **Total sample** | **57** | **5.87** | **52** | **51.29** | **197 (12.3%)** |
| **57** | **5.87** | **52** | **51.29** | **31 (2.1%)** |
| **13** | **6.35** | **14** | **14.69** | **11 (0.7%)** |
| **7** | **7.77** | **14** | **14.69** | **2 (0.1%)** |
Psychological state measures

The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), a 21-item self-report inventory, was used to measure depressive/somatoform symptoms. The State-Trait Anxiety Inventory (Form Y; Spielberger, 1983), a 40-item self-report inventory, was used to measure state and trait anxiety.

Intellectual functioning measures

An estimate of general intellectual functioning was obtained from the Digit Symbol sub-scale of the Wechsler Adult Intelligence Scale Revised (WAIS-R) (Wechsler, 1981). Furthermore, each subject provided written consent to release the official Scholastic Aptitude Test (SAT) scores, verbal and quantitative, from his or her Cornell record.

The subjects groups did not differ significantly on any of these measures of intellectual achievement, namely digit symbol (raw score), SAT Quantitative score, and SAT Verbal score, indicating that the high PAS group did not have a generally lower intellectual capacity. The high PAS subjects displayed significantly greater levels of depressive symptoms (p < .001, 2-tail), state anxiety (p < .05), and trait anxiety (p < .01), as one would expect of such subjects (cf. Mezzich, 1990).

Subjects were tested individually in quiet and conventionally lighted laboratory rooms at Cornell University. All procedures were administered by the investigators, who were assisted by trained research staff. Because a complex coding scheme was employed to disguise the group status of the subjects, both study staff and the investigators were blind to group membership throughout recruitment, testing, and scoring. All subjects were paid $50.00 for participating and all gave written informed consent.

Experiment 1: the delayed response test

One of the several cognitive deficits of schizophrenic conditions appears to be a dysfunction of working memory that leads to a breakdown of behaviors guided by internal representations (Pais & Holzman, 1992). Neuroanatomical and neurophysiological observations point out the important role of the prefrontal cortex in working memory deficits (Fournier, Bruce, & Goldman-Rakic, 1994). Goldman-Rakic, 1991). Surgical and chemical lesions in the dorsolateral prefrontal cortex—in particular, the region of the principal sulcus in the rhesus monkey (roughly equivalent to Brodmann's Area 46)—lead both...
to severe deficits in working memory as assessed by an oculomotor delayed response task, and to some symptoms that resemble those of schizophrenia, such as distractibility and perseveration.

Park and Holzman (1992) developed a human analogue of the oculomotor spatial delayed-response paradigm in order to test the hypothesis that schizophrenic patients show working memory deficits. They found that schizophrenic inpatients were significantly impaired on memory-guided delayed response tasks (DRT), whether the same modality was visual or haptic, but the same patients showed almost no impairments on a sensory-guided DRT.

Bipolar patients, in contrast, showed no impairments on the same oculomotor spatial DRT. Park and Holzman (1993) replicated the oculomotor DRT deficit in schizophrenic inpatients, and also found that 40% (6 of 15) of the first-degree relatives of these patients also showed the same oculomotor DRT deficits (Park, Holzman, & Levy, 1993).

Procedure

A total of 50 subjects were tested on the oculomotor DRT; 28 were in the high PAS group and 22 were in the low PAS group. The procedure was identical to that used by Park and Holzman (1992, 1993). Subjects sat with their heads stabilized on a chin and head rest and were asked to look at a stimulus display screen. A red fixation point (a small dot, 0.5° of visual angle) was located in the center of the screen. The target was a small black circle that measured 2° of visual angle. The location of the target varied from trial to trial. There were eight possible target locations, each separated by 45° on the circumference of an imaginary circle.

In the oculomotor memory task, which assesses working memory, a target (black circle) was flashed on the screen for 200 ms at one of the eight positions. During the brief period, the subject was asked to fixate at the center. Immediately afterward, there was a 10-second delay period, during which the subject performed a distractor task, which prevented rehearsal and kept the subject looking at the location of the fixation point during the 10-second delay period.

After the delay period, the fixation point and eight "reference" circles (empty, rather than black) appeared on the screen. Subjects were required to move their eyes to the position that the target circle had occupied prior to its disappearance. If the subject did not move their eyes to the correct location within a 10-second time limit, the reference circles disappeared.

To control for the working memory component of the delayed response task, the oculomotor sensory task was an oculomotor sensory task. This task was identical to the oculomotor
memory task in all respects except that the target remained on the screen at all times, and therefore no working memory was required to perform this task. There were 64 trials in each of the tasks.

**Apparatus**

An infrared reflective high-speed record eye movements via a video-camera that was connected to an ISCAN XG-26 pupil/ocular reflection tracking system. Eye position information was stored on a Macintosh II computer. The system is fully described in Park and Holzman (1992).

**Scoring**

The dependent variable was accuracy, defined as the percentage of trials that were correct. A response was scored as correct only if the eye moved directly to within 1.5° of the center of the target position. If the eye moved to an incorrect position first and then moved to the correct target position, the trial was counted as incorrect.

**Results**

Table 15.2 presents the means for the two subject groups with respect to the delayed response memory and memory tasks. The high PAS group made significantly more errors than the normal control group on the ocular motor memory task. (df = 49, \( p < .04 \, \text{tail} \), but the two groups did not differ on the sensory control task. The effect size estimate is .49 (Cohen’s d; Cohen, 1988), a “medium” effect size.

Figure 15.1 shows a scatter plot of the DRT scores. It is clear from this figure that not everyone with a low DRT score has a high PAS score. If one uses an arbitrary cutoff point of 50% accuracy, which is the lowest accuracy score in Park and Holzman’s (1992) normal sample, the 12 people with the lowest DRT score (mean of 9 high PAS scores and 3 low PAS scores). It is also noteworthy that 5 of these 9 high PAS subjects have first-degree relatives with an DSM-III-R Axis I disorder (Table 15.3).

Mental state variables (anxiety and depression) were not associated with performance on the DRT, indicating that DRT deficits were not solely the result of transient mental state factors. The SAT math and verbal scores were also not associated with DRT performance, whereas the Digit Symbol score was modestly correlated with DRT performance (\( r = .30, \, p < .03, \, 2-tail \)), which may reflect the shared motor component of both the DRT and Digit
Delayed response antisaccade, thinking

Table 15.2: Principal scores (Mean ± SD) on the delayed response task, antisaccade task, and Thought Disorder Index for high and low PAS students

<table>
<thead>
<tr>
<th></th>
<th>High PAS</th>
<th></th>
<th>Low PAS</th>
<th></th>
</tr>
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<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
<td>SD</td>
<td>N</td>
</tr>
<tr>
<td>Delayed response task</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent correct</td>
<td>20</td>
<td>89.05</td>
<td>7.92</td>
<td>20</td>
</tr>
<tr>
<td>Antisaccade task</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No errors</td>
<td>30</td>
<td>3.04</td>
<td>2.90</td>
<td>30</td>
</tr>
<tr>
<td>Thought Disorder Index</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. TD responses</td>
<td>20</td>
<td>7.27</td>
<td>10.52</td>
<td>20</td>
</tr>
<tr>
<td>Total TDI</td>
<td>30</td>
<td>8.83</td>
<td>15.30</td>
<td>20</td>
</tr>
<tr>
<td>Idiosyncratic verbs</td>
<td>30</td>
<td>4.87</td>
<td>8.39</td>
<td>20</td>
</tr>
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</table>

Symbol tasks. There was no effect of handedness or sex on performance of the oculomotor delayed response task. These data suggest that the significantly poorer DRT performance of the high PAS subject was not likely due to an intellectual deficit or to anxiety or depression in the subjects.

Discussion

Subjects with high scores on the PAS made significantly more errors on the oculomotor memory DRT than those with low PAS scores. 75% of the low accuracy DRT subjects were high PAS scores. This result adds psychometrically ascendant schizophrenia (those with high PAS scores) to schizophrenics and first-degree relatives of schizophrenics as showing deficits in working memory.

We note that only a subgroup of the high PAS scores shows the working memory impairments. Two issues must be considered with respect to these results. First, the PAS served as an endorsement of only a subset of symptoms associated with schizophrenia: perceptual aberrations and body image distortions. The PAS is not intended to identify all individuals with schizophrenic characteristics, that is, individuals who manifest schizophrenic characteristics other than perceptual aberrations and body image distortions. Nor does the PAS claim to exclude individuals whose perceptual aberrations and body image distortions affect conditions other than schizophrenia. Therefore, the optimal expectation for this psychometric instrument is that it identifies a group of individuals, some of whom have circumscribed symptoms associated with
schizotypic pathology. These issues will be further discussed in the final section of this paper.

Second, the DRT is targeted at a specific impairment—working memory deficits—and the DRT will therefore tag those individuals with working memory deficits. DRT deficits are, however, also associated with conditions other than schizophrenia-related pathology, such as clearly diagnosed frontal lobe lesions. Therefore, not everyone with DRT deficits will have a high PAS score, and not everyone with a high PAS score will have schizophrenia-related psychopathology.

These two factors—the imperfect specificity and sensitivity of the PAS measure, and the absence of an exclusive relation between DRT performance deficits and schizophrenia-related pathology—can account for the predicted modest but significant relation found between working memory and PAS scores.

### Table 15.3

<table>
<thead>
<tr>
<th>Delayed resp.</th>
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<tbody>
<tr>
<td>Scored 100</td>
<td>100</td>
</tr>
<tr>
<td>Scored 90</td>
<td>90</td>
</tr>
<tr>
<td>Scored 80</td>
<td>80</td>
</tr>
<tr>
<td>Scored 70</td>
<td>70</td>
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<td>Scored 60</td>
<td>60</td>
</tr>
<tr>
<td>Scored 50</td>
<td>50</td>
</tr>
<tr>
<td>Scored 40</td>
<td>40</td>
</tr>
</tbody>
</table>

**Group**

- **High PAS** (n = 28)
- **Low PAS** (n = 22)

Fig. 15.1. Scatter diagram of percent accuracy scores for high and low PAS students on the delayed response task. The shaded area represents the range of scores by normal subjects in the Park and Holzman (1992) study.

### Relevant diagnoses

- Major depression
- Schizophrenia
- Major depressive
- Bipolar disorder
- Major depression
- Bipolar disorder
- Schizophrenia
- Affective disorder
- Bipolar disorder

*Abnormal scores*
Delayed response, antisaccade, thinking

Table 15.3. List of subjects’ relatives with Axis I conditions and their delayed response accuracy score, antisaccade error score, and number of thought-disordered responses

<table>
<thead>
<tr>
<th>Relative’s diagnosis</th>
<th>Student PAS group</th>
<th>DRT (% correct)</th>
<th>Antisaccade (No. errors)</th>
<th>No. TD responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depressive disorder</td>
<td>high</td>
<td>93.8</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>high</td>
<td>78.3*</td>
<td>2</td>
<td>40*</td>
</tr>
<tr>
<td>Major depressive disorder</td>
<td>high</td>
<td>81.3*</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td>high</td>
<td>81.2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Major depressive disorder</td>
<td>high</td>
<td>96.8</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td>high</td>
<td>100.0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>high</td>
<td>97.9</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Affective disorder</td>
<td>high</td>
<td>80.3*</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td>high</td>
<td>78.1*</td>
<td>9*</td>
<td>2</td>
</tr>
</tbody>
</table>

*Abnormal scores

Poor accuracy on the DRT also occurs in about 40% of the otherwise unaffected first-degree relatives of schizophrenic patients, as shown by the results of Park, Holzman, and Levy (1993). In the present study, 9 subjects—all with high PAS scores—have first-degree relatives with major psychopathology, and 5 of these are in the group with low DRT accuracy scores. Four of the 9, however, have very good DRT scores. None of the low PAS scorers had a first-degree relative with major psychopathology (Table 15.3). In summary, there is a statistically significant association between high PAS scores and low accuracy on the DRT, and these same high PAS individuals tend to have relatives with an Axis I psychiatric disorder. The high scores on the PAS account for three times as many of the subjects with working memory deficits as do the normal students.

Experiment 2: antisaccade performance

Schizophrenic patients can shift their eyes in a target as rapidly and accurately as normal people can. When these rapid shifts of gaze, called “saccadic eye movements,” are under 10° amplitude, the latency, accuracy, and velocity of the saccade made by a schizophrenic patient are indistinguishable from those made by a normal person (دازور، توسن، & جونسون، 1961; ليفين، هولمز، روثباتربيرغ، & ليپتون، 1981). For saccadic amplitudes of greater than 10°, however, Levin and colleagues (ليفن، جونز، سكرك، ميرن، &
Holzman, 1982) reported that schizophrenic patients show longer latencies than do normal controls.

On an antisaccade task, in contrast, individuals are instructed not to look at the target when it moves, but instead to look as quickly as possible in the opposite direction. Thus, a saccade and an antisaccade task differ both in the location to which the person makes an eye movement in response to target movement and in whether the eyes move in response to a foreshadowed target. Studies of antisaccade performance in schizophrenia show that these patients have higher error rates and longer latencies than do normal individuals (Fukushima, Fukushima, Chiba, Tanaka, & Yamashita, 1988; Fukushima, Fukushima, Morita, & Yamashita, 1990; Fukushima, Morita, Fukushima, Chiba, Tanaka, & Yamashita, 1990; Thaker, Nguyen, & Tamminga, 1989). The nature of the performance deficit shown by schizophrenic patients differs from that seen in patients with frontal lesions, however. Frontal lobe patients are unable both to suppress reflexive glances to the target and to make an antisaccade even after making an incorrect saccade to the target; schizophrenic patients make more reflexive saccades to the target than do control subjects, but their initial errors are corrected by appropriate antisaccades (Levy, in press). We chose this task to discover whether high PAS scorers also show a performance deficit on this task.

Procedure

Data were collected from 56 students, 25 of whom had low PAS scores and 31 of whom had high PAS scores. Horizontal eye movements were recorded from both eyes, by an infrared reflection system mounted on eye glass frames. Eye position was calibrated using six target positions on the screen. Sampling rate was 5 ms. Data were recorded on a computer that generated position and velocity tracings and provided automated analysis of saccade direction, amplitude, peak velocity, and duration.

At the beginning of each trial, a small solid square, 1" x 1", appeared at the center of the screen for 800, 1,000, or 1,200 ms. The period was unpredictable, but each time interval was represented equally often in every testing block. Coinciding with the offset of the fixation point, a peripheral target of the same size flashed for 100 ms at 15° to the left or right of the fixation point. The direction of the target was pseudorandom, with the restriction that the target appear an equal number of times on the right and the left, and that it appear a maximum of three times consecutively on the same side. After the peripheral target flashed, the screen remained blank for 1 second to allow the subject to make a saccade to the mirror position on the screen (antisaccade). After making the

Delayed response antisaccade, subjects respond immediately to the target.

Data were collected on all movements.

Antisaccades following saccades follow the movement of the eye. Data from all antisaccades were analyzed to determine if all people under.

Results

The high PAS group had significantly more antisaccade errors than the low PAS group, $p < .01$. The effect size, calculated as the difference in error scores divided by the standard deviation of the mean error scores, was 26% for the high PAS group and 8% for the low PAS group. The high PAS group had almost 14% more antisaccade errors than the low PAS group.

The eight high PAS group subjects made more antisaccade errors than the other subjects. Although the DRT and DDT errors were calculated for each subject, the DRT error was not used in the analysis of the antisaccade errors.

There was an Axis 1 psychiatric diagnosis for each person with high PAS scores. None of the subjects had a diagnosis of antisaccade dysfunction. The high PAS group subjects were significantly more anxious.
antissaccade, subjects were not to wait at the periphery, but to return their eyes immediately to the center to await the reappearance of the fixation point.

Data were collected for three blocks of 15 trials each, for a total of 45 trials. Data were entered automatically into a computer program that notes eye movements.

Antissaccades were scored as right or wrong solely on the basis of the first saccade following offset of the fixation point. Saccades of greater than 1° in the direction opposite that of the target were considered correct responses. Data from all three blocks of trials were included in the data analysis. All antissaccade errors were immediately corrected by the subject, indicating that all people understood the task and were attempting to comply.

### Results

The high PAS students made significantly more errors (mean of 2.84 errors) than the low PAS students (mean of 1.16 errors) in the 45 trials ($t = 2.66, df = 54, p < .01, 1-tail). The effect size (Cohen's $d$; Cohen, 1988) was .60, a "large" effect size. Table 12.2 presents these data. Figure 12.2 shows a scattergram of the error scores for the two subject groups. Eight high PAS students (about 26% of the high PAS group) made 5 or more errors on the 45 trials — that is, more errors than were made by any of the low PAS students. A Pearson correlation coefficient indicates that this association between high antissaccade errors ($r$ or more) and high PAS scores is statistically significant and accounts for almost 14% of the variance in antissaccade error scores ($r = .367, p = .005$).

The eight high PAS students with high antissaccade error scores were not necessarily the same subjects who made high numbers of delayed response errors. Although only three high PAS students performed abnormally on both the DRT and antissaccade tasks, the Pearson correlation between antissaccade errors and DRT errors, however, is statistically significant ($r = .276; p < .05$, 2-tail), indicating that those with poor DRT accuracy tended to make more antissaccade errors.

There was virtually no relation between number of antissaccade errors and Axis I psychopathology in first-degree family members ($r = .024, n = 40$). Only one person with more than five antissaccade errors had a first-degree relative with an Axis I diagnosis (Table 15.3). This person also had a high number of DRT errors. All other subjects with a first-degree relative with an Axis I diagnosis performed normally on the antissaccade task. That of intellectual functioning and attitude, such as the WAIS and the SAT, showed no relation to antissaccade performance. Neither the Beck Depression Inventory nor the anxiety measures showed any significant relation to antissaccade performance.
Discussion

Interest in antisaccades first emerged from studies of patients with frontal lesions. In a series of papers, Guittton, Buchtel, and Douglas (1982, 1985) reported that patients with unilateral lesions of the dorsolateral and mesial regions of the frontal lobes had difficulty suppressing reflexive saccades to a target and initiating voluntary saccades to the side opposite that of the target. The poor performance of schizophrenic patients on antisaccade tasks has usually been discussed in terms of frontal dysfunctions—particularly of the dorsolateral prefrontal area.

The antisaccade task, however, is not specific for damage to any single brain area, including specific areas of the frontal lobes, or for any specific central nervous system disease. Not only do the anterior prefrontal cortex but also the mesial frontal cortex have been implicated in impaired antisaccade performance (Guittton et al., 1982, 1985). Patients with Huntington's disease, pro-

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**Experiment**

Schizophrenia: A descriptive study

The deviant's deviant

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Observers have found that very mild forms of schizophrenia are attenuated for auditory input and require valid auditory input.

Although this is a psychopathological issue, the TDI has been measured without a psychopathological degree of audio and visual input and without a psychopathological degree of audio and visual input. The TDI has been measured without a psychopathological degree of audio and visual input.

**Fig. 13.2. Scatter diagram of the number of errors made by high and low PAS students on the antisaccade task.**

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Delayed response
Exhibit 3: Thought disorder

Schizophrenic patients think in peculiar and sometimes bizarre ways. This observation, systematically presented as early as Kraepelin's (1896/1919) descriptive efforts and Bleuler's (1911/1950) attempts to characterize the nature of the thought disorder, remains one of the most conspicuous and salient diagnostic symptoms of schizophrenia. Since Kraepelin's and Bleuler's descriptions of schizophrenic thought disorder, there have been many efforts to define the essence of the thought disorder. The nature of schizophrenic thinking eludes a simple categorization or dimensionality. The deviances in schizophrenic thinking are multifaceted and multidimensional, in that autistic logic, neologistic word formations, confabulatory tendencies, confusion, among many others, all occur, and in varying degrees. Schizophrenic thinking also differs from the thinking disorders of other pathological conditions such as mania, delusional states, and dementias associated with various brain disorders.

Observers such as Kline (1953) and Meled (1973) have noted that people with very mild forms of schizophrenia manifest a thought disorder that resembles, in intensified form, that observed in schizophrenic psychoses. These observations require validation, and validation requires a reliable measuring instrument.

Although there are several instruments for assessing thought disorder, we chose the Thought Disorder Index (TDI; Johnson & Holzman, 1979), which is a psychometric tool for identifying and measuring disturbances in thinking. The TDI has been validated on a variety of groups including those with and without psychiatric illness, and relatives of psychiatric patients. It has a high degree of interrater reliability (Coleman et al., 1993). The TDI yields qualitative and quantitative indexes of the ways in which ideational and perceptual organization and verbalizations can be distorted in psychopathological and neuropathological conditions.
Johnston and Heiman, the developers of the TDI, attempted to refine and extend Krasnoff's (1956/1919) clinical observations, which were, to a large extent, incorporated and detailed by Rapaport, Gill, and Schaffer (1946/1968) in their work on diagnostic psychological testing. Rapaport, Gill, and Schaffer's (1946/1968) qualitative descriptions of thought slippage were subsequently quantified by Wadbeh and Starfish (1952) in the Delta Index, a predecessor of the TDI (Johnson & Holzman, 1969).

Previous studies have shown that elevated levels of thought disorder are found not only in psychotic patients, both schizophrenic and bipolar (Shannon et al., 1987; Soloway et al., 1987), but also in a significant number of their otherwise unaffected first-degree relatives (Johnson & Holzman, 1979; Shannon, Holzman, & Soloway, 1989), including children (Arbola & Holzman, 1985), and in borderline and schizotypic patients (Edei, 1987). Although the amount and severity of thought disorder, as measured by the TDI, are significantly reduced by neuroleptic treatment (Hari, Holzman, & Davis, 1983), Patterson, Stehle, Bagule, & Hayes, 1986), the thought disorder detected at any particular time reflects both a propensity for thought slippage (a unit characteristic) and adventitious events (state variables, such as clinical state and medication variables). The hypothesis in the present study is that there will be higher than normal levels of thought disorder in subjects with high PAS scores, and that this propensity for manifesting thought disorder represents a feature of the thinking of these high PAS students.

Method
Thirty high PAS and 26 low PAS subjects were tested by two examiners. All subjects were administered a 10-card Rorschach test (Rorschach, 1921), from which the TDI is assessed. Administration of each Rorschach protocol followed the procedure described by Rapaport et al. (1946/1968). All sessions were audiotaped and subsequently transcribed verbatim. The protocols were scored for thought disorder by consensus decision by three trained scorers, according to the TDI manual (Soloway, Shenton, & Holzman, 1986). Both Rorschach administration and TDI scoring were performed without knowledge of the PAS status of the subjects.

Instrument description and scoring
The 23 TDI categories represent most of the types of deviant verbalizations encountered as disordered thought of a formal nature, such as ideopathic
the TDI, attempted to refine observations, which were to a Rapport, Gill, and Schuler Rapport, Gill, clinical levels of thought disorder were autistic (1952) in the Delta autistic, including children (Anteolida Schuster autistic patients (Essex, 1967). A disorder, as measured by the treatment (Hart, Holzman, & A. 1986), be thought disorder a propensity for thought slips where several state variables, such as there will be higher than nor almost high PAS scores, and that represents a feature of the

 Delayed response, antisaccade, thinking...

speech, combinatorial thinking, associative thinking, and disorganized thinking. The TDI matrix contains complete descriptions of the categories as well as the psychometric characteristics of the instrument (Johnson & Holzman, 1979; Sokolov et al., 1986).

The total TDI score is computed as the number of instances of thought disorder tagged by the TDI, multiplied by its category weight (e.g., .25, .50, .75, or 1.0), divided by the number of Rorschach responses to control for verbal productivity, and multiplied by 100 to represent the value as a percent.

Results

High PAS subjects had a significantly higher mean total TDI score (8.83) than low PAS subjects (3.65) (t = 1.75, df = 54, p < .05, 1-tail). High PAS students also had a significantly higher mean number of responses that were scored as disordered (7.27) than low PAS students (3.00) (t = 2.10, df = 54, p = .04, 1-tail). In addition, the high PAS group showed a significantly higher mean number of idiosyncratic verbalizations (i.e., peculiar and queer verbalizations and absurd responses) (4.87 vs. 1.69; t = 1.98, df = 54, p < .02, 1-tail; see Table 15.2). The effect sizes for all three TDI variables range between .46 and .55 (Cohen’s d, Cohen, 1988), considered to be “medium” effect sizes. Four individuals showed very high levels of idiosyncratic verbalizations, and all four were in the high PAS group. None of the other TDI categories distinguished the two groups of students. Figure 15.3 shows the distribution of the number of thought-disordered responses for the two groups of students.

Only one of the high PAS students with high TDI scores had a first-degree relative with an Axis I psychiatric disorder (Table 15.3). One of the four high PAS students who showed a high level of idiosyncratic verbalizations also had the highest DET score. There were no significant relations between the total number of thought-disordered responses, the TDI score, or number of idiosyncratic verbalizations and the anxiety measures, SAT scores, or the Digit Symbol Tests. The total number of thought-disordered responses showed a marginally significant tendency to be related to scores on the Beck Depression Inventory, although the total TDI score and idiosyncratic verbalizations were not at all related to the Beck scores.

All three TDI scores were significantly associated with antisaccade performance at moderate levels, such that those with higher amounts of thought disorder, however measured by the TDI, made more antisaccade errors. The correlations between antisaccade errors and the three TDI scores ranged from .35 to .47, all of which are statistically significant at ≤.005.
Fig. 15.3. Scatter diagram of the number of thought-disordered responses made by high and low PAS students on the Rorschach test, as scored according to the Thought Disorder Index.

Discussion
The high and low PAS groups differ in the amount of thought slippage detected by the TDI, with the high PAS group producing a significantly greater number of thought-disordered responses. The principal type of thought disorder that characterized the high PAS group was ideosyncratic verbalizations—the mild peculiarities of speech that indicate an elliptic, a failed effort to self-edit, or even an autistic idea. Nine of 30 high PAS students (30%) showed significant elevations on the TDI, compared with 4 of 27 students with low PAS (18%) scores. The high PAS group showed not only peculiar and queer verbalizations, but in several instances incoherence, autistics psychology, and ideational looseness occurred as well. These features of thought disorder in the high PAS group are also characteristic of clinical populations, particularly schizophrenics. These results are compatible with those reported by Ekel and activity present

Reflection
The present sample of patients was drawn from a group of 28 (329) students, as was also the Holzman PAS scores. The discussion was also tentative in such a way. Although the deviant DRT was the task between the high and low student means.

The low DRT scores suggest that people may have schizophrenia. The low nae for the method.

1. How many patients from this group were diagnosed as schizoa...
by Edell and Chapman (1979) using the Detox Index. Combinatory ideational activity accounted for the slippage shown by the low PAS group and was also present in the high PAS group.

Reflections and speculations

The principal findings of this investigation show that some college students who were selected for proneness to perceptual abstractions and body image disturbances show performance patterns like those seen in schizophrenic patients and in some of their first-degree biological family members. Nine of 28 (32%) of the students with high PAS scores, compared with 3 of 22 (14%) of students with low PAS scores, had DRT accuracy of less than 86%, which was also the lowest score obtained by the normal group tested by Park and Holtzman (1992). On the antisaccade task, eight students (26%) made more than five antisaccade errors, and all were identified as having high PAS scores. Higher levels of thought disorder were also detected among the students with high PAS scores. Our sample of psychoticomimic "schizotypal" was identified on the basis of perceptual abstractions and body image distortions. Thought disorder may also accompany other characteristics of schizotypy, such as magical thinking, odd speech, and odd, absurd affect.

Although a substantial proportion of the high PAS students performed deviantly on each of the three tasks, the students who performed poorly on the DRT were not necessarily the same ones who performed poorly on the antisaccade task or who showed significant amounts of thought disorder. Each task identifies approximately the same proportion of high PAS students - between 20% and 30% - as having schizophrenia-related deficits. Of students with high PAS scores, 60% had deviant scores on at least one of the dependent measures, compared with 22% of the students with low PAS scores.

The PAS identifies many more people as deviant than do the DRT, the antisaccade task, or the TDI. Further, the PAS probably fails to identify some people who have schizotypic traits other than perceptual abstractions or body image distortions, such as those that accompany impaired interpersonal relatedness, social isolation, or odd appearance. Therefore, only a subgroup of schizophrenic individuals will be identified by the PAS, yielding an indeterministic false negative and false positive rate of detection. This psychometric method, then, poses several questions, which we attempt to address here.

1. How diagnostic is schizotypy in the PAS? Our data suggest that there are many students with high PAS scores who probably do not have schizophrenia-related performance deficits. This yield is respectable both epidemiologically and clinically, since many of the traits tapped by the PAS are associated
with many other disorders. Consider the symptom of body image distortion, which is an aspect of depersonalization. Depersonalization refers to a feeling of unreality about oneself, a sense of disconnection with one’s own body and thoughts, emotional numbness, a sense that one’s voice, actions, or feelings are not under one’s control. All these experiences can occur among people in the general population. When not violent or dangerous, they can persist for years. However, if violent or dangerous, they can cause profound distress and disability. Depersonalization experiences may also occur in anxiety, panic, depression, schizophrenia, and personality disorders, and, of course, in the identifying characteristic of depersonalization disorder. Mayer-Gross regarded depersonalization as a nonspecific brain response that is built into the CNS, much like a fever response to an organic insult (Mayer-Gross, 1935). It is therefore expected that the PAS will test a very wide net and detect people who share certain symptoms that reflect depressive psychiatric conditions and thus produce a number of false positive detections.

As we have already remarked, the PAS constitutes an experiences of perceptual and body distortions, and thus does not focus at all on the other defining dimensions of schizophrenia, such as disturbed social interactions, magical or referential thinking, behavioral eccentricities, or odd speech. As a consequence, the PAS will fail to identify people who possess other identifying characteristics of schizophrenia, but who do not experience perceptual and body distortions. A number of false negative identifications will thus occur in the low PAS or normal group, thereby misclassifying some valid schizophrenic individuals. In such connection, it is noteworthy that six students in the low PAS group had either very low accuracy (IRT < 2.0), high levels of thought disorder (n = 3), or both (n = 1). A more intensive examination of these students would shed light on whether they also have schizophrenic symptoms not captured by the PAS.

Similar heterogeneity of schizophrenic symptoms and traits was shown by Kendler et al. (Kendler, Oden, Gurian, South, & Mark, 1981), who studied 29 pairs of twins, monzygotic (MZ) and dizygotic (DZ), selected from a population registry. Seven measures of schizophrenia were obtained, including schizophrenic symptoms (from the Structured Interview for Schizophrenia), schizophrenic personality traits (from a self-report questionnaire which included the Chart 3 PAS scale), and a neuropsychological attention battery. A factor analysis of the schizophrenia measures showed two inde-

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pendsent factors. The first factor had high loadings on "positive symptoms" such as speech oddities, avoidant traits and social anxiety. The second factor had high loadings on poor modulation of affect, and oddness in social behavior, including social isolation. The attentional battery screening score correlated significantly with this second schizophrenia factor, a result that is similar to that of Asarnow, Nuechterlein, and Mednick (1983), who reported a significant association between scores on the Span of Apprehension test and only a subset of symptoms of schizophrenia.

Contemporary research studies of normalcy at risk for schizophrenic conditions have selected their subjects in three different ways. The first is to focus on the biological first-degree relatives of schizophrenic patients; the second is to select patients with a clinical diagnosis of a schizophrenia-spectrum disorder, such as schizotypal, paranoid, or schizoid personality disorder; the third, the method used in this study, is to select members of the general population who perform in a deviant way on certain psychomotor or physiological indices associated with schizophrenia.

The first two methods have been generally successful in finding deviance on several measures that parallel the anomalies found in schizophrenic patients. For example, eye tracking dysfunction (ETD) is prevalent in patients with schizophrenia-spectrum disorders as it is in first-degree relatives of schizophrenic patients and in patients with schizophrenia (Holzman, Proctor, Levy, Yoel, Meltzer, & Hart, 1976; Everet al., 1990; Smiraglia et al., 1987).

The psychometric method employed in this study identified all nine students who had a first-degree relative with an Axis I psychiatric disorder (Table 15.3). Five of these nine students performed in a deviant way on at least one of the 3 dependent measures; thereof, the method of the psychometric test used in this study may indeed validly identify those who may be gene carriers, although there is a price to be paid in an unknown risk of false positive and false negative identifications.

1. The presence of perceptual abnormalities, body image distortions, or other "schizotypic" characteristics identify people who have a greater than base rate probability for developing schizophrenia?

This question sheds two different issues. The first would ask whether the PAS identifies "gene carriers," who, by virtue of a genetic endowment, inherit the schizophrenic characteristics that can be considered phenotypic expressions of the schizophrenia genotype. Only none of these schizotypic people would, sooner or later, become clinically schizophrenic. Others may be able to main-
tain a state of compensated adaptation, but this adaptation probably exists to the functional efficiency of various psychological processes. The term “risk” in these instances seems better replaced by the term “liability” or “vulnerability,” since the issue in one of a diagnosis that represents a necessary but not sufficient condition for the two clinical outcomes described above. Still, other people, some with and some without the schizophrenia genotype, may show schizophrenic characteristics but do not succumb to psychosis or show deficits in performance on relevant psychological tasks. In this instance, the presence of schizophrenic characteristics determined psychometrically, may or may not be an expression of vulnerability. Given only the psychometric information concerning the presence of schizophrenic characteristics, one cannot utilize a specific individual whether these schizophrenic traits represent a necessary condition for a schizophrenic condition.

The second issue implicit in the question involves a different model, one in which perceptual aberrations and body image distortions are not an outcome of a schizophrenia disease, but are independent of it. These characteristics may function as a potential of schizophrenia-like pathology in those who inherit the hypothesized schizophrenia disease, but they are not exclusively coupled with the disease. In this sense, behaviors like those identified by the PFA could, if present, predispose a person to develop schizophrenia, just as high dietary cholesterol, smoking, and obesity can place a person at risk for developing cardiovascular disease. “Risk” in this model is a statistical term that refers to an increase in the probability for developing the disease. (See Holzman, 1982, for a discussion of the issues of vulnerability and risk.) For clarity, we have diagrammed the three causal models discussed in this section.

Although we require data for our preference, we regard the PFA as an identifier of vulnerability for schizophrenia. If one keeps in mind the possibility of the instrument, the true that characterizes the high PFA scores can be viewed as some of the phenotypic expressions of a schizophrenia disease, which has many manifestations. These symptoms can be considered as another expression of a latent trait, along with EFD and schizophrenia (Matthysse, Holzman, & Lang, 1960). Figure 15.5 presents the model.

The psychometric method, as we have noted, identifies a number of false positives and false negatives, and does not include a principal vulnerability factor: whether a person is a gene carrier. It therefore approaches the issue of risk from a side not yet addressed by the “genetic high-risk method.” It asks whether the possession of certain personality traits is significantly associated with schizophrenic (or perhaps other psychosis) condition, as well as the compensations in psychological functioning associated with these conditions.
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Fig. 15.4 Diagram of the hypothetical causal model in which a gene (or genes) for schizophrenia gives rise to a predisposition, which is not directly observable (the shaded rectangle denotes the hypothetical entity). The predisposition can give rise to schizophrenia and to deviant behaviors or measures such as those investigated in this study. These two outcomes are independent of each other and conditioned on the presence of the hypothetical predisposition. Schizophreny, an observable constellation of cognitive, perceptual, affective, and interpersonal deviations, is not caused by the gene or genes that give rise to the schizophrenia disposition, but independently acts to potentiate the predispositions to lead to a schizophrenia outcome.

The method provides the opportunity to examine conditions that protect a person from developing the disease. These conditions may be interpersonal, interpersonal, biological, and social. The genetic risk method also affords such opportunities, but they have rarely been exploited for this purpose.

4. Why are the differences between the groups not greater? We anticipated relatively subtle, but statistically reliable, differences between the groups for reasons related to our subject selection strategy. We selected our experimental group using the PAS, a fallible psychometric marker, that most likely generated an admixture of people, some of whom may be compensated schizophrenies (only a subset of whom will ever decompensate), an unknown proportion of false positives who represent a variety of personality types, and an unknown number of false negatives whose schizophrenic symptoms differ from those identified by the PAS. Thus, we most likely identified individuals representing a diversity of liabilities.

Finding subtle differences between groups on such tasks is consistent with the usual results from "high-risk" studies (Cornblum & Erlenmeyer-Kimling, 1985; cf. Hesron, Gotteeman, & Meinl, 1977). Clearly, the goal of the high-risk approach in psychopathology research is the isolation of reliable objective behaviors that might aid in more efficient identification of schizophrenia (or psychosis) liability. Even if individual objective markers reflect relatively
Fig. 105. The latent trait model is essentially equivalent to the causal chain depicted in this figure. The gene or genes give rise to a schizophrenia predisposition, which McHale et al. (1966) called a latent trait (plotted to indicate its inherent variability). This predisposition can independently give rise to schizophrenia (measured psychometrically or clinically observed), clinical schizophrenic and delusional laboratory measures. These outcomes are independent of one another, but conditioned on the presence of the schizophrenia predisposition (latent trait).

5. How can we interpret the finding that different tests identify different people in the schizophrenia group? One model that seems reasonable is that of some pleiotropic disorders, such as neural tube defects or osteogenesis imperfecta, in which there is one genotype but several phenotypes. In these instances, a parent may have particular manifestations of the disorder, but the offspring may have different ones. Thus, in a case of osteogenesis imperfecta described by Pignani and Tunno (1992), the mother's symptoms included blue sclerae, deafness, and osteoporosis; the child's symptoms included multiple bone fractures that necessitated the placing of steel rods in the bones. In the case of schizophrenia, one might postulate that the genotype, as yet undefined, unfolds as several phenotypes, which we measure only imperfectly with our tests and with clinical assessment.

6. How should this set of findings be integrated with the family studies of...
schizophrenia? Since the psychometric method has identified a significant number of persons with schizophrenia-like traits, and since a significant proportion of these people show deficit performance on some cognitive tasks associated with schizophrenia, these people should be followed longitudinally, as the Chalmers (Chapman & Chapman, in press) and as Lenznewenger are doing.

This method alone cannot address one important issue, however: Is schizophrenia the inevitable route to schizophrenia? This psychometric method must be used in conjunction with longitudinal and familial studies of schizophrenia. The study of MZ twins would be especially useful for answering this question. We would ask whether, in MZ twins discordant for schizophrenia, schizophrenia always occurs in the well twin and most always precede the development of schizophrenia in the affected twin. Do schizotypic traits in the well twin increase the risk for eye movement dysfunctions, working memory deficits, antisaccade errors, and thought disorder? If they do, one must then entertain a model of the transmission of schizophrenia that postulates that schizotypy is the channel through which these traits may pass (see Figure 15.6, which depicts this model). If, however, these performance deficits are only modestly associated with schizotypic characteristics, it appears to be the case in this study's population, then support is given for another kind of model that is more consistent with the Mendelian latent structure model proposed by Matthyse et al. (1986; see Figure 15.5).

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Fig 15.6. This model is closely related to the advanced by Mehl (1990). A specific gene given rise to a hypothetical entity called "schizotypal" (shaded in the model), which, in the presence of certain as yet unknown environmental influences, causes schizotypy, recognizable by specified signs and symptoms as well as through laboratory measures. Although schizotypy is a necessary precondition for clinical schizophrenia, it is not a sufficient cause, since the outbreak of schizophrenia requires both schizotypy and certain environmental stressors as precipitants. This model differs from the latent trait model (Figure 15.5). There, schizophrenia and schizotypy are independent of each other, the latent trait being a necessary condition for both.

References


Delayed response, antiscadacue thinking


