Psychometric Detection of Schizotypy

William M. Grove
University of Minnesota

Genetic studies of typical schizophrenia require accurate means for the detection of carriers of the heritable diathesis. Psychometric methods have been developed, often for studying psychosocial transmission of schizophrenia, that might be useful for genetic studies. Studies of schizotypy detection using the Goldstein-Scheerer Object Sorting Test, the Rorschach and Thematic Apperception Tests, and the Minnesota Multiphasic Personality Inventory are reviewed. Almost all studies suffer from serious conceptual and methodological flaws that have made replication a rarity. Recent approaches emphasizing clinical signs and symptoms that bear a close content relationship to the typical schizophrenic syndrome are asserted to be more likely to bear fruit.

Family, twin, adoption, and high-risk studies have all contributed to establishing that there is a genetically transmitted diathesis for the manifestation of typical (Kraepelinian) schizophrenia (Kessler, 1980). The details of the causal chain leading from genes to psychosis—with complex interactions between genes, early environment, and later stress—remain less well established. All genetic theories postulate a central state, partially or largely under genetic control, that acts as a disposition. This disposition, which Meehl (1962) called schizotaxia, presumably manifests itself at a biological level. Because the ultimate outcome in question, schizophrenia, is defined in terms of molar behavioral referents, it seems reasonable to assume that schizotaxia has correlates at the molar behavioral level, too. Meehl and Rado (1962) called this behavioral organization schizotypal.

The concept of a personality organization reflecting an underlying schizotypic biotype has a long history. Kraepelin (1913/1919) referred to it, and Hoch and Polatin (1949) described clinical features of a group of outpatients seeking treatment for anxiety disorders whom they believed to be predisposed to develop schizophrenia. Hoch and Cattell (1959) presented more refined criteria for making this diagnosis, which they called pseudoneurotic schizophrenia. Unfortunately, the signs and symptoms that count toward the diagnosis are highly inferential and would be difficult to measure reliably. It is probably for this reason that no one has replicated their finding that a large fraction of pseudoneurotic schizophrenics have to be certified as typically schizophrenic at follow-up (Hoch, Cattell, Strahl, & Pennes, 1962).

Removing such attributions as schizotypy from the realm of unaided clinical judgment offers advantages. First, it would ease the burden on the inferential powers of the clinician by organizing the decision process around systematically gathered observations according to specified rules. Second, greater validity should be obtainable by psychometrically assessing schizotypy, leading to more sensitive research on the origins of schizophrenia. Third, if great validity were obtainable, primary prevention efforts might be feasible.

The success of the psychometric endeavor rests on the verisimilitude of two hypotheses: first, that schizophrenia is etiologically one illness. If it is not, studies using “schizophrenics” of varying sorts will presumably vary in the nature of the predisposition measured, and replication will be difficult. Unfortunately, the quality of description of schizophrenic subjects in research is usually rather poor (Garfield, 1978), which makes comparison of studies difficult. Second, the predisposition must appear at the molar be-

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Requests for reprints should be sent to William M. Grove, Department of Psychology, Elliott Hall, University of Minnesota, 75 East River Road, Minneapolis, Minnesota 55455.


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havioral level. It is not clear that it invariably does. There are cases of outgoing, well-liked, academically successful young people suddenly developing schizophrenia. Perhaps a more intimate knowledge of the person would have disclosed an underlying schizophrenia proneness; but perhaps not.

It is further necessary that schizotypy be measurable with a modicum of interobserver agreement so that workers in different laboratories can replicate one another’s results. The measurement must be sensitive (to borrow a term from epidemiology): Given schizotypy, a positive test result must be likely. It must also be (using another epidemiological term) highly specific: The false positive rate must be very low. The importance of these two parameters varies with one’s genetic model. Partially dominant gene models predict that every schizophrenic has at least one schizotypal parent and that half of his or her siblings are schizotypes. Recessive models predict that every parent of a schizophrenic is a carrier (who may or may not be a schizotype, depending on penetrance in the heterozygote) and that a quarter of the siblings are schizotypes. Polygenic theories posit that it is to some degree arbitrary who one calls schizotypal because the probability of becoming schizophrenic grades insensibly from zero to one. For this reason, polygenic theories are not featured here, and the dominant gene model is used to make predictions about how a schizotypy measure ought to behave.

Insofar as all three models predict that many relatives of schizophrenics should be schizotypes, this choice of reference model is simply one of illustrative convenience. Insofar as sensitivity and specificity frequently differ, rates of schizotypy in relatives will be misestimated. The further the base rates are from 50% and the more unequal sensitivity and specificity are, the worse the estimations will be (Kraemer, 1979). For base rates less than 50%, higher specificity than sensitivity is needed; this would occur in a sibling study under recessivity. Under a dominant gene model, sensitivity and specificity should be equal (and, of course, as high as possible). One implication of these considerations is that measures built for one model may be unsuited for use in studies based on another model.

No instruments meet these requirements of accuracy. When investigators have tried to meet these criteria, results have been negative. Insofar as this area has seen several sustained research efforts in multiple laboratories, this is a disappointing conclusion. I review research relating to the four best-validated approaches to schizotypy detection: the Object Sorting Test (OST), the Rorschach and Thematic Apperception Tests (TAT), and the Minnesota Multiphasic Personality Inventory (MMPI). I then argue that this research has not been more fruitful because subject selection was not homogeneous, syndrome measurement was not sophisticated, and there has been no close content relationship between predictor (psychometric instrument) and predictand (future schizophrenia). Finally, I review recent efforts that may establish some molar behavioral referents for the concept of schizotypy.

The Object Sorting Test

Rapaport (1945) described an object sorting task using 33 common objects. The subject is first asked to group the objects so that all belong with an object provided by the examiner and is then asked why they belong together. The answer is recorded on each of seven trials of this type. In the second part of the task, the subject is shown 12 standard sorts and is asked why these objects belong together. The answers are again recorded. Lovibond (1954) introduced an irrelevant associations score, which quantifies the irrelevance of the reasons given by the subject for a given sort. If the reason would apply equally well to half of the remaining objects, the reason scores one point. If the reason would apply to all of the remaining objects, it scores two points. Otherwise, it scores no points. Scores for all 19 trials are summed to give an overall score. A number of studies have reported that relatives of schizophrenics often score high on the Lovibond measure. On the whole, however, the OST deficit is weak, difficult to replicate, and open to
criticism of lack of diagnostic specificity. No one seems willing to retire the test, but re-
search on it has tapered off sharply.
McConaghy (1959) tested parents of 10
schizophrenic patients with the Object Sort-
ing Test, computing a Lovibond score for
each parent. He found that every patient had
at least one parent scoring 7 or higher. It is
a very striking result because this cutting
point was not determined from the data but
was derived from Lovibond’s work. Under
a dominant gene model, one would predict
exactly the result that was obtained, and so
McConaghy interpreted his data as favoring
a simple dominant gene predisposing to
schizophrenia. It is essential to note that not
one of the parents was apparently mentally
ill according to clinical criteria. This test
makes theoretical sense, and it was highly
sensitive (100% on a dominant gene model)
and specific (only 6 of 65 controls scored
high). This latter figure, about 10%, is in-
teresting because Kidd and Cavalli-Sforza
(1973) found that a gene frequency of about
.1 gave best fit to the Kallmann data under
a single partially dominant gene model.
Therefore, the 10% of “false positives” may
have been true positives who were compens-
ated schizotypes.
Lidz, Wild, Schafer, Rosman, and Fleck
(1962), however, found that only 25% of the
patients’ parents (N = 10 patients) scored
pathologically, whereas 12% of the controls’
parents (N = 21 controls) did so. They did
not report their data in terms of pairs of
parents. After consultation with Lovibond,
they rescored the protocols, raising sensitiv-
ity to 70% but lowering specificity to 81%.
They explained this discrepancy with earlier
work by pointing out that their subjects had
much more education than did the Lovibond
and McConaghy subjects.
The Lidz group then enlarged their ma-
terial to include parents of 34 schizophrenics
and 115 parents of controls. The result, re-
ported by Rosman, Wild, Ricci, Fleck, and
Lidz (1964), was therefore not numerically
independent of their previous study because
all of the patients in that earlier study were
included in the latter. They reported overall
sensitivity of 76% and a specificity of 43%.
Nevertheless, now 57% of the controls had
one or both parents with a score that was
greater than 6.
The Lidz group collaborated with Mar-
garet Singer, who developed a manual for
coding disturbances of attention, meaning,
and interpreting objects. Two trained raters
of the Lidz group correlated .66 with one
another on a summed Singer score of all
pathological verbalizations on the OST. The
summary score was related to education and
age; therefore, parents of 25 patients were
matched to 49 control parents on these vari-
ables. Wild, Singer, Rosman, Ricci, and
Lidz (1965) split the pooled group of parents
at the median, yielding a sensitivity of 84%
and a specificity of 69% on a dominant gene
model. This implies that the gene frequency
is about 30% and that 16% of schizophrenics
are phenocopies if the test is perfectly valid
at this a posteriori cutting point. It does not
seem that the new scoring system led to a
constructive replication of McConaghy’s
original finding. All of the Lidz group re-
ports show an excess of false positive scores,
which with a low base rate condition leads
to more erroneous than correct assessments.
Stabenau, Tupin, Werner, and Pollin
(1965) studied whether pathological Lovi-
bond OST scores may be found in parents
of delinquent children. They counted the
number of trials on which parents obtained
a “zero” score, which indicates clear con-
ceptualization. Parents of delinquents did as
poorly as parents of schizophrenics; more-
ever, delinquents themselves did as poorly
as schizophrenics. Romney (1969) found no
significant difference in mean Lovibond score
between parents or siblings of schizophren-
ic, neurotics, and controls. Finally, Wright
(1973) studied parents of poor premorbid
male schizophrenics and of normal young
men. Modifying the Lovibond scoring sys-
tem, he collapsed all non-“zero” scores into
what he called an overinclusive score. Analy-
osis of variance revealed no significant dif-
ference between patients’ and controls’ par-
teins. Quinlan, Schultz, Davies, and Harrow
(1978) used Singer’s concept of “transac-
tional thinking” and significantly discrimi-
nated between schizophrenics and non-
 schizophrenics and between their respective
parents on the OST. The difference between
cate Singer and Wynne’s (1966a) earlier results. Although they had consulted with Singer and Wynne and had found high interjudge reliability of scoring (r = .87), they were unable to separate parents of variously diagnosed patients by practically significant amounts. Their subjects were parents of consecutively admitted acute schizophrenics, acute depressives, phobic or obsessive patients. Diagnoses were made following administration of the Present State Examination (PSE), and only clear-cut cases were included. Their refusal rate was only one in 81 consecutive cases, yielding 40 schizophrenics and 40 neurotics.

Although the mean of deviance scores for parents of schizophrenics reliably exceeded that for parents of neurotics, the overlap in distributions was essentially complete; only 15% of schizophrenics’ parents exceeded 90% of neurotics’ parents. The medians of the two distributions were identical. Forty percent of schizophrenics’ parents had scores below the pooled median, and Hirsch and Leff (1971) concluded that the parental communication deviance is not necessary for the transmission of schizophrenia. What is most damaging to the Singer-Wynne measure is that the number of words spoken and the deviance score correlated .57 among schizophrenics’ parents and .76 among neurotics’ parents, calling into serious question the interpretation of parental deviance scores as schizophrenogenic or as schizotypal. Hirsch and Leff then used analysis of covariance to adjust observed scores of schizophrenics’ and neurotics’ parents for verbosity. Wynne, Singer, Bartko, and Toohey (1975) showed that verbosity could account for deviance score differences in the Hirsch and Leff sample but not in the Singer and Wynne NIMH data.

Wynne et al. (1975) objected to the way Hirsch and Leff (1971) administered the Rorschach, asserting that it differentially prevented schizophrenics’ parents from scoring deviately. A standard Rorschach administration procedure was used until Wynne criticized it in midstream of the Hirsch-Leff study. Hirsch and Leff then changed their procedure with the result that more deviance was elicited from both schizophrenics’ and neurotics’ parents; they found no evidence that their first procedure differentially affected groups (Hirsch & Leff, 1975).

In sum, Hirsch and Leff’s (1971) failure to replicate cannot be laid to poor experimental technique. Hirsch and Leff (1975) pointed out that their own criteria for diagnosing schizophrenia are so much narrower than Singer and Wynne’s (1965, 1966a) that many of Singer and Wynne’s schizophrenics, especially “borderlines,” would be called personality disorders in Britain (or under DSM-III, I would add). Moreover, they pointed out that Singer and Wynne may have studied cases referred to NIMH because of interesting familial communication patterns. In terms of subject diagnosis and sample representativeness, the British study, based as it was on consecutively admitted patients who were administered the PSE, is much the stronger of the two. In another failure to replicate, Wender, Rosenthal, Rainer, Greenhill, and Sarlin (1977) used Singer’s scoring method on Rorschach and TAT protocols of biological parents of schizophrenic and mentally retarded adoptees, finding no significant differences between groups. Evidently, the Singer scoring manual fails to capture the discriminant information in parental protocols.

Jones (1977) and Doane, West, Goldstein, Rodnick, and Jones (1981) reported on use of parental Singer-Wynne TAT scores in prospectively predicting adult psychopathology in disturbed adolescents. Jones factor analyzed the TAT scoring categories and computed six factor scores for each parent. He found that every future borderline, remitting, or chronic schizophrenic had at least one parent with a factor T score over 60, but two-thirds of the neurotic and personality-disordered offspring also had such a parent. Clearly, the pathology was not specific to schizophrenia. Doane et al. predicted “schizophrenia spectrum disorder” among offspring; they included, however, cyclothymia and schizoaffective disorders in the spectrum. The former is generally thought to lie in the spectrum of bipolar affective disorder, and the latter is probably sometimes genetically a variant of affective disorder (Pope & Lipinska, 1978). Therefore, their finding that parental deviance predicts offspring spectrum disorders is difficult to interpret.

To summarize, the only study that tried
& Novick, 1968). This means that within schizotypal and nonschizotypal classes, the items are statistically independent (i.e., that the dichotomy schizotypal/nonschizotypal accounts for the observed covariation between items). Using an estimation procedure that is too complicated to explain here, they selected seven items based on responses of 211 psychiatric outpatients, none of whom were diagnosed as schizophrenic. Estimating the base rate of schizotypy at about .4, they classified cases by Bayes formula into schizotypal and nonschizotypal classes. The mean MMPI profile of schizotypes corresponded closely to Peterson’s (1954) preschizophrenic sample profile. The convergence of the two studies suggests that this seven-item scale might have potential for detecting schizotypy.

What conclusions can be drawn from research using the OST, Rorschach, TAT, and MMPI? First, differences between high-risk groups and controls are evanescent. Second, construction of single scales or batteries of scales to assess dimensions of difference between schizotypes and nonschizotypes has not delivered on early promises. I believe that the use of dimension-detecting procedures in syndrome delineation has hampered research in this area. The Golden and Meehl (1979) approach, which is a true syndrome delineation method, represents a significant step toward a more fruitful conception of schizotypy. Another possibility, however, is that the dimensional approach failed because schizotypy is not highly transmissible. This is unlikely, though, because twin data (Gottesman & Shields, 1972) indicate that schizophrenia is moderately heritable. The diathesis should be as heritable as the disorder.

In my opinion, the most important strategic error in schizotypy research was made in choosing schizotypy indicators. Workers, influenced by Bleuler (1911/1950), chose manifestations of schizophrenia that they thought were closest to the central defects in schizophrenia. They theorized that these highly inferential central states would be closer to the core condition and thus more meaningful. They shunned symptoms like delusional ideas and brief hallucinations, assuming that such symptoms were restitutive or secondary. Nevertheless, evidence is accumulating that using clinical signs and symptoms as schizotypy indicators works better because the content of the predictor and of the predictand overlap.

Recent Efforts Using Signs and Symptoms

Two groups have worked with signs and symptoms of schizotypy: the Biometrics Research Unit at New York State Psychiatric Institute and the Chapmans at the University of Wisconsin. In New York, Spitzer, Endicott, and Gibbon (1979) consulted Rosenthal, Wender, and Kety in developing clinical signs of what the latter called the schizophrenia spectrum. These disorders range from inadequate personality (DSM-11) to chronic schizophrenia. The spectrum is similar to the schizotypy concept, insofar as relatives of schizophrenic adoptees show concentrations of spectrum disorders. Spitzer et al. derived eight sign clusters: odd communication (short of gross formal thought disorder), ideas of reference, suspiciousness, recurrent illusions (or depersonalization or derealization), magical thinking, inadequate rapport (including constricted affect), undue social anxiety, and social isolation. Internal consistency reliability (K-R 20) for the eight-item “scale” was only .54, which Spitzer et al. found disappointing. Nevertheless, if these items are signs and not dimension-measuring items, the low K-R 20 may be due to a substantial lack of correlation among signs within the schizotypal and nonschizotypal groups. Sensitivity of the eight-item scale (with a cutting score of three) in the training set of 36 spectrum cases was 86%, and specificity among 43 noncases was 95%. On cross-validation, sensitivity among 30 cases fell to 63%, but specificity with 31 noncases was 100%. This indicates that behavioral signs may be used as moderately strong schizotypy indicators. These signs are the basis of the DSM-III (American Psychiatric Association, 1980) diagnosis of schizotypal personality.

The Chapmans have studied clinical signs, using questionnaire as well as structured interview formats to obtain data. Each of the several scales and schedules they have developed corresponds to just one sign of schi-
to duplicate the results of Singer and Wynne, (1965, 1966a), incorporating psychiatric controls, reliable diagnoses, and representative sampling, failed. Other studies, using related but not identical TAT scoring schemes, have reported significant relationships between prospectively assessed parental deviance and offspring psychopathology, but the claim that this pathology is usually schizophrenic must be questioned.

Minnesota Multiphasic Personality Inventory

In the detection of schizotypy, structured personality inventories offer in principle certain advantages and suffer certain disadvantages in comparison with projective tests. They offer ease of administration and scoring, and they lend themselves to mass storage so that archival studies can yield new scales with evidence of validity on creation. On the other hand, they do not offer the sensitive assessment of thought disorder that projectives have promised and that has kept the Rorschach in the portfolio of schizophrenia researchers. Moreover, the prototype of these inventories, the MMPI, has not proved very sensitive in most studies.

Peterson (1954) was apparently the first to use the MMPI in schizotypy detection. He compared 33 white male outpatients initially called nonschizophrenic who were later diagnosed schizophrenic (false negatives) with 33 other outpatients who were never called schizophrenic (true negatives). Finally, he found 27 cases called incipient, latent, subclinical, or remitted schizophrenia who were later hospitalized as schizophrenic (true positives). All cases had taken the MMPI at initial evaluation. The false negatives had profiles of the same shape as, but with greater elevation than, the true positives, earning a mean 8-7-2 three-point code. Insofar as the mean Sc scale T score for false negatives was about 85, it seems that a diathesis for later schizophrenia was probably being detected.

Toms (1955) gave the MMPI to mothers of 25 male paranoid schizophrenic veterans. Comparing them with 30 control mothers, she found significant mean differences on L, K, and Hs, but the differences were only five or six T scores in each case. The Peterson (1954) findings might lead one to expect that schizophrenics’ mothers would have a 7-8-2 profile; clearly, they do not.

Mosher, Pollin, and Stabenau (1971) examined what is the most certainly schizotypal cohort available: discordant identical twins of a schizophrenic. They administered the Mf and Baron Es scales of the MMPI to such twins; only the Es scale separated the discordant twins from the matched controls. Score distribution overlap was not computed, but the reported significance level indicates an approximately 85% hit rate for a base rate of .5. Nevertheless, one can expect Es to lack convincing discriminant validity as a schizotypy scale; Es will probably detect “weak egos” due to many causes.

Gottesman and Shields (1972) used the OST and MMPI in their study of consecutively admitted schizophrenic twins at the Maudsley Institute. They were unable to use this information to improve their MZ/DZ concordance ratio over that obtainable from psychiatric interview or to detect oddities in the discordant twins. Their results are in line with those of Toms (1955).

As a part of the Great Copenhagen Adoption Study, Haier, Rosenthal, and Wender (1978) administered the MMPI to the adopted-away offspring of schizophrenic parents. About 80% of such adoptees participated in the study, resulting in 64 adoptees and 64 matched control adoptees. The mean profiles of male and female high-risk adoptees were very similar; the mean profile was a 3-2-1 code. This profile shape was the same as that of the matched controls, and multivariate analysis of variance revealed that the profile elevation difference (3-5 T scores) was not significant. Insofar as their result should have replicated Peterson (1954), clearly, Peterson’s finding is not general.

All of these studies used existing MMPI scales as signs of schizotypy. Golden and Meehl (1979) took a radically different tack in scale creation, treating MMPI items as signs of a syndrome. Rather than picking items that correlated highly with each other or with some criterion, they chose items from a face-valid candidate list that fit a latent class model with local independence (Lord
zototypy so that the instruments may be used together. They have developed scales for physical and for social anhedonia (Chapman, Chapman, & Raulin, 1976), for body image aberration (Chapman, Chapman, & Raulin, 1978), and for rating psychotic-like experiences (Chapman & Chapman, 1980). These efforts were inspired by the Meehl (1962) and Rado (1962) concepts of schizotypy.

Scale development proceeded by choosing face-valid items for the domains of anhedonia, body image aberration, and micropsychotic episodes. A typical social anhedonia item is “Getting together with old friends has been one of my greatest pleasures.” A physical anhedonia item is “The beauty of sunsets is greatly overrated.” A body image item is “My hands and feet have never seemed so far away.” A psychotic-like experience rating scale for “transmission of own thoughts” is anchored at the pathological end (rating of 10) by “S[subject] has actively experienced thoughts leaving his head so that anyone in the area could hear the thoughts through his ears,” which is the Schneiderian first rank symptom of thought broadcasting. The same scale is anchored at 5 by “S has suspected on the basis of direct experience that... single individuals have heard his thoughts through their ears without S’s actively trying to achieve thought transmission, or has had the experience, with belief, that single individuals have read his mind against his will (or felt it was happening even though he knew better).” Anchors are provided for all scale points (Chapman & Chapman, 1980, pp. 482-483).

Preliminary reliability and validity studies have been encouraging. Reliabilities (K-R 20) of the social anhedonia scale among 241 normal noncollege men and 263 normal noncollege women were .85 and .82, respectively (Chapman et al., 1976). Reliabilities of the body image aberration scale among 631 normal college men and 718 normal college women were .88 and .90. Physical anhedonia reliabilities in the same samples were .79 and .78 in men and women, respectively (Chapman et al., 1978). The scales discriminate between normal persons and schizophrenics. Normal and male chronic schizophrenic samples (N = 121) had the same modal score on physical anhedonia, but the schizophrenic score distribution was bimodal, yielding a sensitivity of 33% and a specificity of 93% (Chapman et al., 1976). Social anhedonia scores were higher than those of normal persons: A quarter of the schizophrenics lay above the 90th percentile of the normal sample’s distribution. On body aberration, 74 male schizophrenics (diagnostic criteria unspecified) scored .4 standard deviations higher than 100 normal men (Chapman et al., 1978).

Both anhedonia and body image aberration scales have demonstrated discriminant validity. Small samples of nonpsychotic outpatient psychiatric clinic patients have means lying between those of normal persons and schizophrenics (Chapman et al., 1976, 1978). Evidence that these scales tap schizophrenia proneness and not simply psychosis proneness may come from examining high scorers for correlates of schizophrenia. Haberman, Chapman, Numbers, and McFall (1979) studied social competence in 16 physically anhedonic and 20 perceptually aberrant college men (scores with more than 2 standard deviations on respective schizotypy scales) and 19 controls (scores with no more than .5 standard deviation on either scale). Each subject role played responses to 24 problematic social situations as a test of interpersonal competence. Responses were rated on a 3-point scale. Interrater reliability (Pearson r) for the total score was .80. The physically anhedonic group did worse than the controls, but the perceptually aberrant group did not. The difference between anhedonic and control means was about .8 standard deviation, implying about 65% sensitivity and specificity for a .5 base rate.

Edell and Chapman (1979) examined delta and alpha Rorschach signs among 26 physically anhedonic, 27 perceptually aberrant, and 26 normal college students. Previous research (Kataguchi, 1959; Piotrowski & Berg, 1955) found schizophrenics elevated on both indices. Edell and Chapman found twice as many high scorers on both Rorschach indices among the anhedonic and perceptually aberrant as among controls. Penk, Carpenter, and Rylee (1979) examined the relationship between MMPI scales, MMPI profile scales (Gilberstadt, 1970),
and the anhedonia scales. Their subjects were 245 consecutively admitted male veterans on a drug abuse unit. They reported correlations between both anhedonia scales and the MMPI Sc scale in the mid-.30s. Social anhedonia correlated .42 with the 2-7-8 profile scale and .35 with the 8-6 profile scale. It also, however, correlated .22 with the 2-7 profile scale. This suggests a lack of phenomenological specificity because the 2-7 profile is usually considered a neurotic and anxious one. Nevertheless, the choice of subject population was not ideal for the study of schizotypy, and we have seen that clinical MMPI scales are not very sensitive to schizotypy.

Chapman, Edell, and Chapman (1980) recently showed that the perceptual aberration scale measures proneness to schizophrenia and not merely to psychiatric illness. They studied 50 physically anhedonic, 65 perceptually aberrant, and 66 control white college students. The two scales were essentially independent in a screening population of 2,576 students, in line with Meehl's (1962) taxonomic theory that independence must hold in a purely schizotypal or purely nonschizotypal population, even though both measure schizotypy. If the gene frequency is about .1 (on a partially dominant gene model), the base rate of schizotypy among college students will be substantially less than 10%. This is because college students are selected for academic and social success; preschizophrenics are often neither (Barthell & Holmes, 1968). Thus, the Meehl theory predicts negligible correlations among schizotypy indicators in this group.

Chapman et al. (1980) examined subjects’ history of Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) disorders (based on a Schedule for Affective Disorders and Schizophrenia—Lifetime version, SADS-L, interview) and of psychotic-like experiences using their structured interview scales. They found that 17% of the perceptually aberrant subjects had had psychotic symptoms (versus 2% of the controls), but only 2% of the physically anhedonic subjects had had them. Every type of psychotic-like symptom studied was at least three times as common among perceptually aberrant subjects as among controls. There was a tendency for anhedonic subjects to have more psychotic-like experiences than did the controls, but the experiences did not seem to achieve delusional conviction or hallucinatory intensity. Every one of the Spitzer et al. (1979) schizotypal features, included in the SADS-L, were also much more common among perceptually aberrant and anhedonic subjects than among controls.

No subject had ever had mania (although most of the risk period had not been surpassed). Depression was much more common in perceptually aberrant than in anhedonic or control subjects: It occurred in 17% of perceptually aberrant, 8% of anhedonic, and 3% of control subjects. Given that the psychotic-like experiences reported were usually brief, most subjects would not qualify for an RDC diagnosis of schizophrenia, let alone a DSM-III diagnosis (RDC requires 2 weeks’ duration; DSM, 6 months). Nevertheless, because DSM-III includes prodromal and residual phases of illness in the determination of duration, some subjects might have qualified. Two (3%) perceptually aberrant subjects had been psychiatrically hospitalized, neither for mania but one for what sounds like a typical schizophrenia. Fourteen percent of the perceptually aberrant, 2% of the anhedonic, and 3% of the control subjects had seen a psychiatrist or psychologist. These figures suggest that the risk of schizophrenia and of depression, but not of mania, is raised in perceptually aberrant subjects. The physical anhedonia scale is not powerful as a schizotypy indicator.

In summarizing the Chapmans' work, it must be said that each one of their indicators, except the psychotic-like experiences scales, only modestly distinguishes schizophrenic and nonpsychotic patient samples. The perceptual aberration scale, however, seems quite capable of isolating a group of subjects at high risk for schizophrenia, and the psychotic-like experiences scales, almost (but not quite) by definition, indicate a very high risk of clinical schizophrenia. The problem with all of these scales is that high scores could be obtained by ambulatory schizophrenics, making the detection of schizotypy merely a diagnosis of schizophrenia. No data have been published that show that elevated scale scores on the Chapman et al. (1976,
1978, 1980; or Spitzer et al., 1979) scales precede the onset of outright schizophrenia. Gilles (1958), however, reported such precedence of signs before disorder. Moreover, because it is impossible that the base rate of clinical schizophrenia in college students is 17%, and because only one of 65 perceptually aberrant subjects (1.5%) has thus far been hospitalized for typical schizophrenia, it seems probable that schizotypy, not schizophrenia, is being detected. Only further research, distinguishing schizophrenics from manics and relatives of schizophrenics from controls with the scales, along with the continued follow-up of the Chalmers' cohort, can answer such questions definitively.

Summary

Clinicians have long been aware that certain personalities are predisposed to development of typical schizophrenia. The long history of efforts to objectify judgments about cognitive disorder in relatives of schizophrenics by means of psychometric devices has mostly been a series of failures to replicate. The reasons for these failures are several. First, it may be that schizotypy is not so transmissible as has often been thought, although the evidence does not favor this possibility. Second, the index subjects whose relatives were studied may not all have suffered from typical schizophrenia. Because the risk of schizophrenia in relatives of atypical schizophrenics is lower than that in relatives of typical schizophrenics, investigators with mixed samples operated at a disadvantage. Third, a conception of measures as assessing dimensions that separate schizophrenics from nonschizophrenics has customarily been invoked. This concept entails the construction of single measures with good reliability properties. Recently, evidence of superiority for a multi-sign syndrome approach requiring low intercorrelation of measures has begun to emerge. Fourth, the choice of constructs to measure was originally too subtle. Highly inferential constructs like "transactional communication deviance" and "overinclusive concepts" were seen as closest to the endophenotype and were therefore preferred to signs and symptoms of obvious mental disorder. Again, recent evidence suggests that rather obvious, overt psychopathology may offer advantage as an indicator of the genetically transmitted diathesis for schizophrenia.

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