Abstract

This study investigated the factor structure and etiology of four self-report schizotypy questionnaires during young adulthood (age 18–27) in 98 monozygotic and 59 same-sex dizygotic twin pairs from the community. A single phenotypic factor was identified that was primarily associated with Perceptual Aberration, Magical Ideation, and the Rust Inventory of Schizotypal Cognitions scales, and less so with Social Anhedonia. Univariate etiologic models suggested that in addition to nonshared environmental influences, Perceptual Aberration and Social Anhedonia were significantly influenced by either genes or shared family environment, whereas Magical Ideation and the Rust Inventory were influenced by shared family environment, but not genes. Multivariate twin analyses detected a common schizotypy factor, primarily defined by Perceptual Aberration, Magical Ideation, and the Rust Inventory scales, that was influenced by genes or shared environment as well as nonshared environment. Contrary to expectations, these results suggest that, at least in community-based samples, these “positive” schizotypy questionnaires are not strongly genetically influenced.

Keywords: Schizotypy, twin study, genetics, perceptual aberration, magical ideation, social anhedonia


Rationale

Any theory of the etiology of schizophrenia must account for data that indicate liability to the diagnosis is genetically influenced: probandwise concordance rates for schizophrenia of approximately 48 percent are reported between monozygotic (MZ) twins compared with 17 percent for dizygotic (DZ) twins and a 1 percent morbid risk in the general population (for review, see Gottesman 1994). At the same time, because MZ concordance is less than 100 percent, genetic liability does not appear to be a sufficient cause. Some nongenetic environmental component must contribute. It follows that there may be a portion of the population (as high as 10% by some estimates [Meehl 1990]) with a genetic liability to schizophrenia that does not yet, or may never, manifest clinical schizophrenia (e.g., Gottesman and Bertelsen 1989).

In an attempt to identify this “unexpressed” genetic liability, a constellation of psychological traits have been investigated among the relatives of schizophrenia patients. Eccentricity, irritability, social isolation, suspiciousness, and superstitiousness have been identified and labeled “schizotypy” (for review, see Kendler 1985). Etiologic models as diverse as Meehl’s single major locus model (1962, 1990) and Gottesman and Shield’s multifactorial threshold model (1982; Gottesman 1994) agree that such schizotypal traits may reflect this genetic liability, which, when combined with environmental factors, results in symptoms of schizophrenia.

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One of the most widely used methods for assessing schizotypy, or more generally "psychosis proneness," has been self-report questionnaires, in particular three included in the present study: Perceptual Aberration (distortion of the perception of one's body and other objects; Chapman et al. 1978), Magical Ideation (the tendency to accept forms of causality that are not viewed as valid in Western culture; Eckblad and Chapman 1983), and revised Social Anhedonia (the tendency to avoid interpersonal interactions, hereafter Social Anhedonia; Chapman et al. 1976; Eckblad et al. 1982). Another measure included in the present study is the Rust Inventory of Schizotypal Cognitions (Rust 1989), which has been less widely used. Items for these scales have been selected to tap experiences and beliefs that are not biased toward social desirability and are reliably reported (see Rust 1989; Vollema and van den Bosch 1995).

The hypothesis that such measures index genetic liability to schizophrenia has been investigated using several methods. First, the hypothesis predicts that scores on schizotypal personality measures should be elevated in schizophrenia patients. Evidence for this is mixed, however (Chapman et al. 1976, 1978; Rust 1989; for negative results, see George and Neufeld 1987; Edell 1995). Second, individuals scoring high on schizotypy measures should also be at an increased risk of later being diagnosed with a psychotic disorder. One combination of scales, Magical Ideation and Social Anhedonia, showed a weak but significant relationship in an initial report (Chapman et al. 1994; for a failure to replicate, see Kwapił et al. 1997), whereas a followup report on the same sample found a stronger relationship between psychotic disorders and Social Anhedonia alone (Kwapil 1998). Third, schizotypy scores should be elevated in the relatives of schizophrenia patients, for which evidence is also mixed (Katsanis et al. 1990; Huxley 1993; Kendler et al. 1996; Craver and Pogue-Geile 1999; for negative results, see Rust 1989; Clementz et al. 1991). Fourth, individuals who score higher on schizotypy measures should have more relatives with schizophrenia, which has been reported in some studies but not in others (Lenzenweger and Loranger 1989; Chapman et al. 1994; for negative results, see Kwapił et al. 1997). Finally, a number of studies suggest a relationship between individuals scoring high on these measures and schizophrenia-like neuropsychological and psychophysiological profiles (for reviews, see Fernandes and Miller 1995; Lenzenweger 1998). Thus, despite negative findings in this area, an emerging literature indicates some suggestive associations between schizophrenia and questionnaire measures of a variety of strange experiences, beliefs, and behaviors.

In addition to cross-sectional, predictive, and familial correlations with diagnosed schizophrenia, a number of questions remain about schizotypy as a dimension of personality in the general population. In this case, the schizotypy hypothesis makes a number of testable predictions. An initial question concerns the interrelationship among the questionnaires, or rather, their convergent validity. Do the multiple measures of schizotypy all reflect a single latent personality factor, as might be predicted from a simple, unidimensional model of genetic liability to schizophrenia, or are there multiple latent factors? Several exploratory factor analyses have assessed the relationships among the Chapman and Rust scales and other measures of schizotypy. This research indicates that not one but between two and four factors are involved in schizotypy, depending on which measures are included (e.g., Raine et al. 1994; for review, see Vollema and van den Bosch 1995). Perceptual Aberration and Magical Ideation consistently load on a primary "positive" schizotypy factor characterized by odd beliefs or unusual experiences. Social Anhedonia loads on a second "negative" schizotypy factor characterized by social avoidance, which appears regularly in these analyses. In general, this second factor has been found to be uncorrelated with the first. The apparent existence of at least two latent factors among schizotypy questionnaires raises the question of whether one, both, or neither best reflects genetic liability to schizophrenia. The present study will address this issue further by investigating the etiologic factor structure of these schizotypy questionnaires using a twin study design.

The central question to be examined in the current study, initially suggested by Meehl's (1962) schizotypy hypothesis, is the extent of genetic influences on these traits in the general population. Evidence for heritability is mixed. Sibling studies have shown familial correlations for Magical Ideation and Social Anhedonia but not Perceptual Aberration (Berenbaum and McGrew 1993; Huxley 1993; Craver and Pogue-Geile 1999). In one twin study, Magical Ideation and Social Anhedonia were significantly heritable, but Perceptual Aberration was not, showing significant shared environmental effects (Kendler and Hewitt 1992), while in another study, Perceptual Aberration and Magical Ideation were heritable, but Social Anhedonia was "familial," that is, genetic or shared environmental influences were implicated (Miller 1993). The current study will attempt to adjudicate among these conflicting results and extend this previous work by analyzing the heritability of individual scales as well as the heritability of the structural components of schizotypy in a young-adult sample of twins from the community. By using twin study methods and multivariate analyses, we can examine several key aspects of the validity of schizotypy questionnaires in community samples. Specifically, we address the following set of questions:
1. What is the phenotypic factor structure of questionnaire-defined schizotypy?
2. Are the individual scales of schizotypy genetically influenced?
3. Are latent dimensions of schizotypy genetically influenced?

Method

Subjects and Measures. Twins were recruited from Western Pennsylvania as part of the ongoing University of Pittsburgh Twin Study through the Allegheny County, PA, voter registration and driver's license lists and the University of Pittsburgh student registry (Pogue-Geile et al. 1998). Individuals from these lists with the same last name, sex, and date of birth were mailed information about the study. Additional subjects were recruited through newspaper advertisements. Respondents who confirmed their twin status and expressed interest were admitted to the study if they were (1) 18–27 years old, and (2) without any currently treated physical or mental illness. Twins were generally tested on different days within 1 week of each other. Zygosity was determined by DNA fingerprint analysis. As part of a computer-administered questionnaire battery, subjects completed Perceptual Aberration, Magical Ideation, Social Anhedonia, and the Rust Inventory.

Model-Fitting Analyses. The Mx computer program (Neale 1995) was used to fit phenotypic and etiologic models to the twin variance-covariance data from schizotypy measures. Phenotypic confirmatory factor analyses were used to assess the fit of the measurement model. Etiologic model-fitting analyses were used to partition the variance of the measures into influences from latent variables, including additive genetic (A), common or shared environment (C, such as rearing environment), and nonshared environment (E, such as individual experiences and measurement error). The squared path coefficients between these latent variables and the observed measures represent partitions of variance, that is, parameters $a^2$, $c^2$, and $e^2$. The full ACE model included all three parameters, and the significance of each parameter was tested by comparing the reduced models, AE, CE, and E, to determine whether the removal of a parameter significantly degraded the chi-square goodness-of-fit statistic. Nonadditive genetic, or dominance, effects (D) were tested by comparing the AE to an ADE model. When two nonnested models, such as AE and CE, are compared, lower values of Akaike's Information Criterion (AIC, Akaike 1987) suggest which model is the more parsimonious. Models were not reduced using AIC, however, because resulting partitions of variance might capitalize on chance to bias parameter estimates. This twin model-fitting approach assumes that assortative mating, epistasis, and gene-environment interactions or correlations are minimal. Further, it assumes common environments do not differ systematically in ways relevant to schizotypy between MZ and DZ twin pairs.

Results

Complete schizotypy data were available for this report on 98 MZ twin pairs (MZ; 53 female, 45 male) and 59 same-sex DZ twin pairs (DZ; 33 female, 26 male), for a total sample of 314 individuals. The mean age of the sample was 21.4 (standard deviation [SD] = 2.9) years, with an average of 14.2 (SD = 1.8) years of education. The sample was largely European-American, with the exception of seven minority twin pairs. Zygosity status was not significantly associated with sex, age, education, or ethnicity. Means and standard deviations of the questionnaires are reported in table 1.

Table 1. Means, standard deviations, and correlations among schizotypy measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Perceptual Aberration</th>
<th>Magical Ideation</th>
<th>Social Anhedonia</th>
<th>Rust Inventory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceptual aberration</td>
<td>3.67 (4.57)</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magical ideation</td>
<td>6.75 (5.03)</td>
<td>0.64***</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Social anhedonia</td>
<td>7.57 (5.40)</td>
<td>0.30***</td>
<td>0.25***</td>
<td>1.00</td>
</tr>
<tr>
<td>Rust inventory</td>
<td>29.75 (9.37)</td>
<td>0.57***</td>
<td>0.62***</td>
<td>0.19**</td>
</tr>
</tbody>
</table>

Note.— M = mean; SD = standard deviation; K-S = Kolmogorov-Smirnov statistic; ns = nonsignificant.

1 Correlations based on transformed (as appropriate) linearly sex- and age-corrected scores, as follows: Perceptual Aberration (logarithmic, K-S = 0.14, $p < 0.0001$), Magical Ideation (logarithmic, K-S = 0.07, $p < 0.005$), Social Anhedonia (logarithmic, K-S = 0.03, ns), Rust Inventory (not transformed, K-S = 0.03, ns). Sex and age were regressed out of scores, but only two correlations were significant: Social Anhedonia with sex ($r = 0.20, p < 0.001$, males higher), and Rust Inventory with age ($r = -0.12, p < 0.05$).

** $p < 0.01$; *** $p < 0.001$ (2-tailed)
Schizotypy measures were transformed as appropriate (see table 1) and adjusted for sex and age, and standardized residual scores based on regression equations were used in subsequent analyses, unless otherwise noted. Sex correlated significantly only with Social Anhedonia (men scoring higher), and age correlated significantly only with the Rust Inventory (with younger subjects scoring higher, see table 1). Education significantly correlated only with Magical Ideation, and this was marginal ($r = -0.11, p = 0.05$). There were no ethnic differences on scores, although our power to detect such differences was quite low. There were no significant differences between MZ and DZ twins on the schizotypy scores. Because of the parametric assumptions of the Pearson $r$, Spearman's $\rho$ was also calculated and similar results were observed.

**Phenotypic Convergent Validity.** Correlations among subjects' scores are presented in table 1. With respect to convergent validity, correlations among the four measures of schizotypy were strong, except for Social Anhedonia, which was weakly correlated with the other three.

To examine convergent validity, a series of models tested the hypothesis that a common schizotypy factor adequately fit the data for the four schizotypy measures and whether sex differences existed. Details of this model reduction are reported in Appendix table A1. Figure 1 illustrates the paths of the best fitting model. The initial single common factor model did not reduce fit compared to a Cholesky decomposition (i.e., a saturated model). Subsequent reductions suggested sex differences were not significant. Overall, Perceptual Aberration, Magical Ideation, and the Rust Inventory were significantly and equally associated with the common factor, as was Social Anhedonia, although to a lesser degree. Parameter estimates from the best fitting model indicated that the common schizotypy factor accounted for 61 percent of the variance in Perceptual Aberration, Magical Ideation, and the Rust Inventory, and approximately 11 percent of the variance in Social Anhedonia. In effect, 89 percent of the variance in Social Anhedonia was specific, residual variance, unaccounted for by the schizotypy factor. Thus, the schizotypy factor appeared to be primarily a positive dimension associated with unusual experiences and

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**Figure 1.** Final model from convergent validity confirmatory factor analyses

<table>
<thead>
<tr>
<th>Perceptual Aberration</th>
<th>Magical Ideation</th>
<th>Rust Inventory</th>
<th>Social Anhedonia</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.39</td>
<td>0.39</td>
<td>0.39</td>
<td>0.89</td>
</tr>
</tbody>
</table>

**Note.**—S = specific variance. Arrows represent significant paths with corresponding proportions of variance from the confirmatory analyses. The illustrated model fit $\chi^2 = 18.68$ (df = 14), $p = 0.18$ (for details of model reduction sequence, see Appendix table A1).
beliefs. As a next step, etiologic analyses were conducted to determine what these observed correlations indicate about the origin of schizotypy.

Etiologic Models of Schizotypy

Univariate models. Table 2 presents the correlations between twins on the four measures. Initial models suggested no sex differences for any of the scales. Data from both sexes were combined to test five successive etiologic models, including ACE, AE, CE, E, and ADE, also reported on table 2.

Two of the schizotypy scales, Magical Ideation and the Rust Inventory, were significantly influenced by the shared and nonshared environment, but not genetic factors. Perceptual Aberration and Social Anhedonia both showed significant familiality, that is, either the genetic or shared environment parameters could be dropped from the model, but not both. Nevertheless, in the full ACE model, genetic parameters, A, for both these scales (a² = 0.33 and 0.27, respectively) were greater than C estimates (c² = 0.09 and 0.00), and AE models fit marginally better than CE models. In all cases, the amount of variance explained by nonshared environment was substantial (e² = 0.58–0.73).

Multivariate models. A multivariate etiologic model was implemented to evaluate the origins of the schizotypy factor found in the phenotypic factor analysis. To do this, we combined the twin data for the schizotypy measures into a single model. The model reduction strategy was based on the phenotypic factor structure of the confirmatory model derived above and illustrated in figure 1. Thus, in the context of a "common pathway" model as described by Neale and Cardon (1992), all etiologic paths, including additive genetic and shared and nonshared environmental influences, were tested for significance (for details of this model reduction, see Appendix table A2).

The resulting model along with final parameter estimates is presented in figure 2. These findings show the common schizotypy factor was significantly familial. Although the genetic (AE) and shared environmental (CE) models for the schizotypy factor could not be significantly distinguished, the shared environment accounted for more variance than genetic influences in the full ACE model (45% and 15%, respectively). Factor loadings of the common factor on the observed variables were similar to those found in the phenotypic model, with Perceptual Aberration, Magical Ideation, and the Rust Inventory loading strongly on the schizotypy factor (61% of variance), with a significant but lower loading for Social Anhedonia (11%).

The etiologic influences on the specific variance of each variable were also tested and are reported in figure 2. Perceptual Aberration, Magical Ideation, and Rust Inventory specific variance could be accounted for by nonshared environmental factors (E models). For Social Anhedonia, familial models (AE and CE) fit the data significantly better than models with only nonshared (E) influences.

This etiologic examination of the factor structure of schizotypy suggests that this common schizotypy factor, largely defined by the pattern of unusual experiences and beliefs tapped by Perceptual Aberration, Magical Ideation, and the Rust Inventory, is familial with shared environmental or perhaps genetic influences. The measure-specific variance of these scales, however, resulted from nonshared environmental influences. Further, while this factor is significantly related to Social Anhedonia, this measure largely taps a familial aspect of personality not related to the common factor.

Table 2. Twin correlations and univariate models of the etiology of schizotypy

<table>
<thead>
<tr>
<th>Measure</th>
<th>Twin Correlations</th>
<th>Partition of Variance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MZ</td>
<td>DZ</td>
</tr>
<tr>
<td>Perceptual aberration</td>
<td>0.43*</td>
<td>0.24</td>
</tr>
<tr>
<td>Magical ideation</td>
<td>0.38*</td>
<td>0.46*</td>
</tr>
<tr>
<td>Social anhedonia</td>
<td>0.29*</td>
<td>0.04</td>
</tr>
<tr>
<td>Rust inventory</td>
<td>0.35*</td>
<td>0.46*</td>
</tr>
</tbody>
</table>

Note. — MZ = monozygotic; DZ = dizygotic; a² = heritability; CI = confidence interval; c² = shared environment; e² = nonshared environment; D = nonadditive genetic, or dominance, effects.

1 A comparison of two initial ACE models, with and without sex differences, indicated no significant effect for sex in the etiology of any variables. a², c², and e² and the 95% CI from the sex-combined ACE model are presented. The changes in c² for AE, CE, and E models were compared to ACE models to determine significant parameters. In no case were ADE effects significant when tested against AE.

2 Perceptual Aberration and Social Anhedonia showed significant familiality, that is, either a² or c² must be present. Analyses did not distinguish between these models at the 0.05 level, but in each case the AE model fit better than the CE model according to Akaike's Information Criterion.

* p < 0.05; p < 0.01; p < 0.001
Discussion

In evaluating the factor structure and etiology of questionnaire-defined schizotypy in a community-based sample, we found the following:

1. Phenotypic correlations and confirmatory factor analysis indicated that Perceptual Aberration, Magical Ideation, and the Rust Inventory were strongly associated with a single, latent common schizotypy factor. Social Anhedonia was weakly but significantly associated with this common factor (table 1, figure 1, Appendix table A1).

2. Etiologic twin analyses indicated that in addition to nonshared environmental influences, Perceptual Aberration and Social Anhedonia resulted from familial (either genetic or shared environmental) influences, while Magical Ideation and the Rust Inventory resulted from shared environmental influences (table 2).

3. Multivariate twin analyses also detected a common schizotypy factor, largely defined by Perceptual Aberration, Magical Ideation, and the Rust Inventory. This common factor was influenced by shared environmental, nonshared environmental, and perhaps genetic influences. Specific variance of these scales was nonfamilial. Social Anhedonia was only slightly (although significantly) related to the common factor, while its residual variance was familial (figure 2, Appendix table A2).

Integration With Previous Literature. As in any assessment of factor structure, our first result is strongly influenced by the specific measures chosen to tap the schizotypy construct. The empirically derived common schizotypy factor is very similar to factors found in previous studies characterized by unusual beliefs and experiences frequently referred to as positive schizotypy (for review, see Vollema and van den Bosch 1995). The large residual variance of Social Anhedonia is consistent with many of these same studies in finding that schizotypy is not a unitary construct and involves a social withdrawal dimension negatively correlated with extraversion and only loosely related to unusual experiences. This second dimension, often called negative schizotypy, would emerge only when several relevant measures were included in the assessment battery. Thus, while we found one common schizotypal dimension, our findings are consistent with models of schizotypy as a multidimensional construct. Of course, our results do not apply to other aspects of schizotypy, such as disorganization, that were not measured (e.g., Raine et al. 1994).

The second result was initially surprising for us. In a sample of 70 MZ and 63 DZ twins, Kendler and

Figure 2. Final model (for each twin) from the multivariate twin analyses

![Diagram](image)

Arrows represent significant paths, unless otherwise noted, and their corresponding proportions of variance. The illustrated model fits \( \chi^2 = 76.13 (df = 61), p = 0.92 \) (for details of model reduction sequence, see Appendix table A2).

1 Influences are significantly familial, but AE and CE models could not be distinguished.
Hewitt (1992) reported no significant heritability for Perceptual Aberration but heritabilities of over 0.50 for Magical Ideation and Social Anhedonia without any influence of shared environment. A large proportion of the apparent discrepancy arises because our estimates are derived from an ACE model tested for significant effects rather than a reduced model based on AIC without testing the significance of each parameter, as was done by Kendler and Hewitt. A reanalysis of Kendler and Hewitt's data using univariate ACE models indicates familiarity for Perceptual Aberration and Magical Ideation, significant heritability for Social Anhedonia, and a large overlap with the confidence intervals of the current study (table 2). 1 Apparently, choices made in reporting the data and the wide confidence intervals for these sample sizes account for much of this discrepancy. Still, noteworthy differences remain: Magical Ideation is more due to genetic influences in their sample and more due to shared environment in ours; and though Social Anhedonia appears genetically influenced in both samples, their heritability estimate is more than twice that in our sample. Methodological decisions may also account for some of these differences. Kendler and Hewitt used an older sample with a wider age range (mean age = 37.7 years, SD = 13) which could affect heritability estimates if true heritability changes with age. Further, because the authors included opposite-sex twins in the DZ sample, sex effects that reduce the DZ correlations may have inflated heritability estimates. Finally, Kendler and Hewitt used shortened versions of the Chapman scales and included in the analysis any scale that was at least 60 percent complete, which substantially reduced the reliability of the scales (see Kendler et al. 1996).

Among 103 MZ and 73 DZ young adult male twins, Miller (1993) reported significant heritability for Perceptual Aberration and Magical Ideation, and familiality for Social Anhedonia. 2 Again, in part because of wide confidence estimates, these results are similar to the current findings except for the heritability of Magical Ideation in Miller's sample. This does not appear to be due merely to combining sexes in the current sample, as we found that the correlations of male and female twin pairs were quite close. It is possible that different methods of ascertainment, mailed as opposed to computer-administered questionnaires, regional differences, and sampling variation may account for this discrepancy. Nevertheless, given these methodological differences, these studies are consistent in that they show familial influences accounting for a significant proportion of variance in several measures of the schizotypal personality construct.

There is much theoretical overlap between the constructs that underlie these scales, and there is a consistent pattern of intercorrelation between questionnaire-defined schizotypy. Thus, an important step in validating these scales as related to schizophrenia proneness is to evaluate the etiologic influences on this multivariate factor structure. Previous analyses of the etiology of schizotypy factors (Kendler and Hewitt 1992; Miller 1993) indicated significant heritability for the factor characterized by unusual beliefs and experiences. As noted in the third result, this factor was significantly familial in the current sample, with some evidence of shared environmental influences. However, again, choices made in presenting the results may overstate the differences between Kendler and Hewitt's and our own data. For example, the heritability of Kendler and Hewitt's factor that included Perceptual Aberration and Magical Ideation was approximately 0.62 in the AE model. The authors report the AE because it was the most parsimonious model based solely on AIC. The heritability of our common schizotypy factor from our best fitting multivariate AE model (Model 2, Appendix table A2) is 0.56 [95% confidence interval 0.40, 0.76]. Miller (1993) reports a high correlation (0.79, p < 0.001) between the genetic components of the AE models that determine Perceptual Aberration and Magical Ideation, but because of the difference between our analyses, it is difficult to make a more direct comparison. Despite these differences, the combined evidence suggests a common schizotypy factor, characterized by unusual experiences and reliably indexed by a number of instruments, that is familiality influenced, although not necessarily heritable.

The multivariate analyses also suggested that Social Anhedonia was significantly but not strongly associated with the first factor. This finding is consistent with previous research on the dimensionality of schizotypy, some of which suggests that anhedonia and social anxiety, rather than unusual experiences, are more closely related to a vulnerability to psychosis (Thaker et al. 1993; Kwapil 1998) and the likelihood of having relatives with schizophrenia (Kendler et al. 1996). Previous twin analyses report conflicting results for this factor. Kendler and Hewitt report that the factor

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1 The reanalysis of Kendler and Hewitt's (1992) correlations yielded the following estimates (and 95% confidence intervals) for Perceptual Aberration (a² = 0.07 [0.00, 0.46], c² = 0.20 [0.00, 0.41], e² = 0.73 [0.54, 0.92]), Magical Ideation (c² = 0.46 [0.00, 0.69], c² = 0.11 [0.00, 0.52]), Social Anhedonia (c² = 0.70 [0.42, 0.79], c² = 0.00 [0.00, 0.23], e² = 0.30 [0.21, 0.44]). Note that this reanalysis is based on published correlations, not covariances.

2 Miller's (1993) study reported the following estimates (and 95% confidence intervals) for Perceptual Aberration (a² = 0.34 [0.16, 0.48], e² = 0.66 [0.52, 0.84]), Magical Ideation (c² = 0.44 [0.27, 0.56], e² = 0.57 [0.44, 0.73]), and Social Anhedonia (c² = 0.13 [0.00, 0.48], c² = 0.22 [0.00, 0.44], e² = 0.65 [0.54, 0.77]).
One of the strengths of the current study is that it employs a sample from the community. In contrast, the majority of research on the psychometrics of schizotypy questionnaires is based on undergraduate student populations, which has been a source of controversy (e.g., Balogh and Merritt 1990). Our sample scores were consistent with other large studies of schizotypy in community samples (e.g., Katsanis et al. 1990), effect sizes = 0.13 on Perceptual Aberration and 0.09 on Social Anhedonia comparing their sample means to the current sample). Undergraduate samples, on the other hand, tend to show higher schizotypy scores than the current and other community samples (e.g., Chmielewski et al. 1995; effect sizes were: Perceptual Aberration 0.48, Magical Ideation 0.42, Social Anhedonia 0.08, and Rust Inventory 0.79; Rust 1989). These differences may be the result of selection for college admission, age, or both. At the same time, the current sample was restricted to a narrow band around the peak age of onset of psychotic disorders and is therefore representative of a population of interest to schizophrenia researchers because the analyses are not confounded by potentially artifactual age effects. Also unlike previous twin studies, the current sample was sex balanced, only same-sex twins were included, and sex effects were tested for significance in the analyses.

Conclusions. A central hypothesis tested in the current study—that dimensions of questionnaire-defined schizotypy, or psychosis proneness, are strongly influenced by genes—was not supported. Although there were trends for Perceptual Aberration and Social Anhedonia, and a small genetic effect on the schizotypy dimension, the general picture was not consistent with large genetic effects on these scales. These results imply that an association between schizotypy assessed in this...
manner and a genetic liability to schizophrenia in the general population will be difficult to demonstrate in the long run and, if it does exist, will require much larger samples than have been used to date. This difficulty is certainly because strange experiences and beliefs and an introverted lifestyle can result from many different causes. “Schizotypal genes” may contribute to only a small proportion of the total variation of such behaviors. Highlighting this view, Berenbaum (1995) has rechristened this phenotype broadly as “peculiarity,” suggesting that, besides a genetic liability to schizophrenia, many traditional schizotypal traits may be associated with childhood experiences such as trauma. Our findings of shared environmental effects also suggest cultural effects on superstitiousness. Overall, the present results support this more agnostic approach, labeling the measures descriptively and letting subsequent research determine if they are genetically associated with schizophrenia.

As it stands, the current study challenges only the questionnaire method for assessing the positive and negative dimensions of schizotypy. It leaves open the possibility that, as part of a larger assessment procedure, questionnaire measures of schizotypy may still have an important role in the future of schizophrenia-liability research. For example, in developing a recent questionnaire, Raine (1991) took advantage of advances through the 1970s and 1980s in schizophrenia-spectrum nosology and found that 55 percent of individuals scoring in the top decile on his questionnaire were subsequently diagnosed with DSM-III-R schizotypal personality disorder after interviews. Thus, one way to improve power and further hone the phenotype of schizotypy may be to follow questionnaires with interviews that allow for further probing, interpretation, and behavioral ratings (e.g., Kendler et al. 1995). In any case, the difference between individuals who are peculiar for other reasons and those who may be liable to schizophrenia has important implications for understanding the genetics and cognitive precursors of schizophrenia. A growing body of work suggests that detecting this important distinction will be the challenge for future measures of genetic liability to schizophrenia.

Appendix

Table A1. Convergent validity confirmatory factor analysis

<table>
<thead>
<tr>
<th>Model Description</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$p$</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Common factor paths to PA, MI, RI, and SA; paths for specific variance; separate models for men and women.</td>
<td>4.90</td>
<td>4</td>
<td>0.30</td>
</tr>
<tr>
<td>2</td>
<td>Model 1, models for men and women equated.</td>
<td>16.70</td>
<td>12</td>
<td>0.16</td>
</tr>
<tr>
<td>3</td>
<td>Model 2, with common factor path to SA omitted.</td>
<td>46.33</td>
<td>13</td>
<td>0.00</td>
</tr>
<tr>
<td>4</td>
<td>Model 2, with common factor path to PA, MI, and RI set equal.*</td>
<td>18.68</td>
<td>14</td>
<td>0.18</td>
</tr>
<tr>
<td>5</td>
<td>Model 4, with common factor path to PA, MI, RI &amp; SA set equal.</td>
<td>67.93</td>
<td>15</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Note. PA = perceptual aberration; MI = magical ideation; RI = Rust Inventory of Schizotypal Cognitions; SA = social anhedonia; AIC = Akaike’s information criterion.

* indicates the most parsimonious model.

1 The first model did not decrease fit significantly compared to an optimal Cholesky decomposition. Comparisons among successive models were as follows: Model 2 vs 1: $Dc^2(df=8) = 11.80$, $ns$ = indicating no sex differences; Model 3 vs 2: $Dc^2(1) = 29.63$, $p < .005$, indicating SA significantly contributed to the common factor, as did the other three measures; Model 4 vs 2: $Dc^2(2) = 1.98$, $ns$ = indicating PA, MI, and RI contributed equally to the common factor; 5 vs 4: $Dc^2(1) = 49.25$, $p < .005$, indicating SA could not equally contribute to the common factor. Final paths and proportions of variance derived from Model 4 are illustrated in Fig 1.
Table A2. Etiological factor structure of schizotypy

<table>
<thead>
<tr>
<th>Model Description</th>
<th>$\chi^2$</th>
<th>$df$</th>
<th>$p$</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 ACE paths to common schizotypy factor, similar to phenotypic Model 4; ACE paths for specific variance to all observed variables</td>
<td>69.76</td>
<td>55</td>
<td>.09</td>
<td>-40.24</td>
</tr>
<tr>
<td>2 Model 1, with C path to common factor omitted</td>
<td>72.07</td>
<td>55</td>
<td>.07</td>
<td>-39.93</td>
</tr>
<tr>
<td>3 Model 1, with A path to common factor omitted</td>
<td>69.98</td>
<td>56</td>
<td>.10</td>
<td>-42.02</td>
</tr>
<tr>
<td>4 Model 1, with C and A paths to common factor omitted</td>
<td>104.9</td>
<td>57</td>
<td>.001</td>
<td>-9.10</td>
</tr>
</tbody>
</table>

Note: — ACE = additive genetic (A), shared (C), and unshared (E) environmental etiological influences; AIC = Akaike’s Information Criterion.

1 Model 1 did not significantly reduce fit compared to an ACE Cholesky model, $\chi^2 (13) = 14.76$, ns = indicating no sex differences. Comparisons between successive models were as follows: Models 2 vs 1: $\chi^2 (1) = 2.31$, ns = indicating no sex differences, and model 3 vs 1: $\chi^2 (1) = 0.22$, ns = indicating no sex differences, indicating neither A nor C individually significantly contributed to the common schizotypy factor; models 4 vs 1: $\chi^2 (2) = 35.14$, $p < .005$, indicating significant familiality for the common factor. Given ACE influences on the schizotypy common factor, followup analyses on the etiological parameters of specific variance indicated that nonshared environmental factors significantly accounted for perceptual aberration, magical ideation, and Rust Inventory. Familial models (AE or CE) accounted for social anhedonia and could not be significantly distinguished. Final parameter estimates are reported in Fig 2.

References


**Acknowledgments**

Preparation of this article was supported in part by grant NHLBI 40962. Portions of this report were presented at the annual meetings of the Behavior Genetics Association (1996) and the Society for Research on Psychopathology (1996). We wish to thank the twin study participants, Thomas Kamarck, Ph.D., and the twin study staff and associates: Elizabeth Bachen, Ph.D.; Eric Delliquadri; Tara Fazzari, M.A.; John Hall, Ph.D.; Gary Katz, Ph.D.; Uzma Kazi; Jennifer Koehler, M.A.; Susan Mallkoff-Schwart, Ph.D.; Anna Marsland, Ph.D.; Sheila Metzler; Joanna Polefrone, Ph.D.; Melissa Smith; Christopher Stewart; and Shari Waldstein; as well as Robert Ferrell, Ph.D., Department of Human Genetics, University of Pittsburgh, for zygosity diagnoses.
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